

**ADVANCING UNDERSTANDING OF THE ROLE OF NON-PHYSICAL
CHILDHOOD ABUSE IN ADULT EATING DISORDER PATIENTS: A MIXED
METHODS APPROACH**

Submitted by

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STUDENT DECLARATION FORM

Type of Award **Doctor of Philosophy**

School **Psychology**

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General Abstract

Eating disorders are complex regarding aetiology and symptom maintenance but a key focus of theory and research is the physical rather than non-physical forms of childhood abuse. This thesis displayed how the non-physical forms of childhood abuse are related to eating pathology via core mediating psychological factors conceptualising family dysfunction and invalidating environment as childhood non-physical abuse.

Study 1 assessed the mediating role of self-esteem between self-reported family dysfunction and both eating pathology and psychosocial QoL in ED patients ($n = 154$) and healthy controls ($n = 153$) using Structural Equation Modeling. Self-esteem mediated the relationship between family dysfunction and both ED and psychosocial QoL in both groups.

Study 2a examined the relationship between self-reported childhood non-physical abuse (predictor) with eating pathology and psychosocial QoL (outcome variables) in ED patients ($n = 80$) and healthy controls ($n = 188$) using self-esteem, attachment and emotion regulation as mediators in SEM models. Only self-esteem mediated the relationship between childhood non-physical abuse and eating pathology in patients. The path between childhood non-physical abuse and psychosocial QoL was mediated both by self-esteem and emotion regulation with self-esteem being the strongest mediator. Attachment did not mediate any relationship in the clinical sample. In controls, only emotion regulation mediated childhood non-physical abuse and ED and all mediators were significant for psychosocial QoL with self-esteem being the strongest one. Study 2b examined an adaptation of biosocial theory whereby the relationship between non-physical abuse and four outcomes (self-esteem, emotion regulation, eating pathology and psychosocial QoL) is mediated by attachment and temperament in ED patients and controls. Attachment mediated all relationships except for

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ED in patients, while temperament mediated all the proposed relationships in the same group. In controls, both attachment and temperament mediated all the proposed relationships.

Study 3 used Interpretative Phenomenological Analysis to explore the lived experiences of ED patients ($n = 6$), who have experienced non-physical childhood abuse and are still victims in adult life: also explored the potential psychological factors behind this continuing phenomenon along with its consequences on psychosocial QoL and ED maintenance. The three master themes drawn from the analysis are: continuation of non-physical abuse across the lifespan, developmental factors and non-physical abuse, non-physical abuse and eating pathology.

The findings of the studies combined have implications for prevention and treatment. Self-esteem is the most important factor for ED patients and emotion regulation for the controls. Non-physical childhood abuse was identified as a psychological mechanism that has an effect on eating pathology and psychosocial QoL in adult life passing through self-esteem, emotion regulation, attachment and temperament. These psychological factors are linked to other psychopathology types indicating that this proposed theoretical mechanism for EDs could be tested for other mental disorders. It is also evident that the psychological consequences of childhood non-physical abuse are continuous and should be targeted in treatment in order to be prevented.

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Dedication

This thesis is dedicated to my father and brother and to the memory of my beloved mother.

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A thing which has not been understood inevitably reappears; like an unlaidd ghost, it cannot rest until the mystery has been resolved and the spell broken.

(Freud, 1909)

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List of Abbreviations

ED	Eating Disorders
QoL	Quality of Life
CEA	Childhood Emotional Abuse
CEN	Childhood Emotional Neglect
CPN	Childhood Physical Neglect
CSA	Childhood Sexual Abuse
CPA	Childhood Physical Abuse
BPD	Borderline Personality Disorder
EDE-Q	Eating Disorders Examination Questionnaire
RSES	Rosenberg's Self-Esteem Scale
FAD	McMaster Family Assessment Device
WHO	World Health Organisation
ECE-R	Experiences in Close Relationships Revised
DERS	Difficulties in Emotion Regulation Scale
ATQ	Adult Temperament Questionnaire
CTQ	Childhood Trauma Questionnaire
IPA	Interpretative Phenomenological Analysis
SEM	Structural Equation Modeling
ACE	Adverse Childhood Experiences
SIB	Self-Injury Behaviour
MNBS	Multidimensional Neglectful Behaviour Scale
DBT	Dialectical Behaviour Therapy

Chapter 1: Introduction to the Rationale and Aims of the Studies

1.1. Overview and Thesis Organisation

Research on eating disorders (EDs) has overlooked the role of childhood non-physical abuse and the psychological factors that mediate its relationship with the onset and maintenance of eating pathology. This chapter will explore the term ‘childhood non-physical abuse’, highlighting gaps in the literature regarding these abuse forms in relation to EDs. Following this, the background and rationale for the PhD studies will be explained, along with their significance and original contribution to knowledge. Elaboration on the factors and their associations is beyond the scope of this chapter. It briefly presents the origins of these studies to enable readers’ understanding of the proposed empirical models. Chapters 2 and 3 present the literature review, providing definitions and associations of all the factors mentioned in the PhD studies. Chapter 4 addresses the studies’ methodologies. Chapters 5, 6 and 7 present the empirical studies, and Chapter 8 closes the thesis with a general conclusion.

1.2. Statement of the Problem Regarding Non-physical Abuse and EDs in the Literature

Adverse childhood experiences (ACEs) are the most fundamental cause of many health-risk behaviours in adolescence and adulthood (Garrido et al., 2018; Norman et al., 2012). It is also empirically evidenced that experiencing psychological trauma at an early age increases the risk of addictive behaviour in adolescence and adulthood (Felitti, 2004; Norman et al., 2012). EDs could be considered a form of addiction (Curtis & Davis, 2014) as food is used to alleviate stress in the same way as alcohol (Kinzl & Biebl, 2010). Various authors have discussed the similarities between the psychological mechanisms involved in alcoholism and EDs (Vandereycken, 1990) with respect to the uncontrollable drive to engage in maladaptive behaviour (Barbarich-Marsteller et al., 2011), emphasising the role of ACEs as an important precursor (Theodoropoulou et al., 2009).

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Evidence has demonstrated that a significant proportion of people suffering from an ED or engaging in disordered eating report a history of childhood abuse compared to individuals without EDs (Favaro & Santonastaso, 1995; Kong & Bernstein, 2009; Sansone & Sansone, 2007; Schmidt et al., 1997; Wiederman et al., 1998). Research also indicates that childhood abuse is associated with body image disturbances in adulthood (Sansone & Sansone, 2007). Childhood emotional abuse (CEA) in particular is responsible for damaged trait self-esteem and immature defence mechanisms that can trigger adult psychopathology (Finzi-Dottan & Karu, 2006). Moreover, there is a theoretical and empirical suggestion that ACEs play an aetiological role in the development of EDs (Holmes, 2018; O'Shaughnessy & Dallos, 2009).

There has been substantial research into the relationship between physical forms of childhood abuse (sexual, physical) and EDs, with a great emphasis on sexual abuse (Hund & Espelage, 2005; Messman-Moore & Carrigus, 2007; Schmidt et al., 1997; Smyth et al., 2008). Despite the emphasis on childhood sexual abuse (CSA), research into ACEs in ED development dispels the myth of CSA being the unitary cause of this disorder (Guillaume et al., 2016; Schmidt et al., 1997) or an absolute risk factor, as clinicians used to believe. This highlights the importance of researching non-physical forms of abuse (Burns et al., 2012; Welch & Fairburn, 1994).

Nevertheless, non-physical forms of childhood abuse (emotional abuse, emotional neglect, physical neglect) remain under-researched in relation to EDs and, consequently, less well understood, even though there is evidence of a phenomenological link between them (Claes & Vandereycken, 2007; Hund & Espelage, 2006; Kimber et al., 2017; Pignatelli et al., 2017; Waller et al., 2007; Vajda & Láng, 2014). Waller and colleagues (2007) attribute the research gap regarding non-physical abuse forms and EDs to the difficulty in defining the

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experience of non-physical abuse in a clinically meaningful way. Emotional abuse is not clearly characterised by specific actions of the abuser, so the definition depends on the victim's perceptions and interpretations of the abuse. Another explanation for this gap could be that non-physical childhood abuse is perceived as less severe and thus less important in empirical studies (Melton & Davidson, 1987). However, researchers who have examined non-physical abuse with respect to eating pathology development concluded that CEA may be the most damaging form of childhood abuse (Kent & Waller, 2000; Kent et al., 1999; Rorty et al., 1994). This conclusion could possess merit considering the fact that CEA is responsible for the development of negative cognitions in children (Courtney et al., 2008).

There are limited studies on CEA, which is sometimes termed psychological abuse, emotional maltreatment or psychological maltreatment (Baker & Maoiorino, 2010; Briere et al., 2012; Burns et al., 2012; van Harmelen et al., 2010; Kent & Waller, 1998; Mills, 2011; Witkiewitz & Dodge-Reyome, 2000). These studies offer little to no understanding of the psychological factors that could sufficiently explain the relationship between ED onset and maintenance (Kent & Waller, 2000; Levitt & Sansone, 2007; Waller et al., 2007). This lack of agreement about the term's definition (Morgan & Wilson, 2005) generates confusion regarding what these studies measured, making comparability and aggregation of the results a difficult task. These studies typically examine a non-clinical population and report contradictory findings due to variations in definition, terminology, measures and methodology, as well as due to small sample size (Hawkins, 2003; Kent & Waller, 2000; Witkiewitz & Dodge-Reyome, 2000). Moreover, family dysfunction has also been used in the literature to imply emotional abuse (Kent & Waller, 2000) and linked to the concept of the invalidating environment (Linehan, 1993) in EDs (Crowell et al., 2009; Haslam et al., 2012). However, the pathway that leads to eating pathology, no matter how the term is defined, has

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not been explained. In sum, the use of different terms in the literature further complicates an already complex phenomenon (Clarke, 2015).

Literature reviews (e.g. Kent & Waller, 2000) suggest a link between the CEA found in adverse familial contexts and eating pathology development, proposing that the link could be indirect and passes through low self-esteem. Trait self-esteem's significant role in the development of eating pathology has been overlooked in the leading models of EDs, even though it has been empirically associated with EDs (see Chapter 2, Section 2.3.1). Most researchers agree on the scientific need to elucidate the psychological mechanisms linking traumatic childhood experiences to EDs through mediation analysis, as this will clarify the pathway through which these traumatic experiences affect ED development and maintenance (Hawkins, 2003; Kennedy et al., 2007; Kent & Waller, 2000; Kong & Bernstein, 2009; Mazzeo & Espelage, 2002; Mitchell & Mazzeo, 2005). However, reviews indicate that mediation in EDs is in the early stages (Cortés-García et al., 2019).

Mediation is a context in which the independent variable causes a mediating variable, which in turn causes a dependent variable (McKinnon & Luecken, 2008). The most common mediators that have been empirically supported with respect to all forms of childhood trauma and eating pathology are attachment, depression, self-esteem, alexithymia, shame, impulsivity, ineffectiveness, affective instability, substance misuse, emotional distress, borderline personality disorder (BPD), emotion regulation, anxiety, dissociation, obsessive-compulsive symptoms, self-criticism and core beliefs (Andrews, 1997; Casper & Lyubomirsky, 1997; Dunkley et al., 2010; Groleau et al., 2012; Hawkins, 2003; Kennedy et al., 2007; Kent et al., 1999; Kong & Bernstein, 2009; Murray & Waller, 2002; Rabito-Alcón et al., 2021; Tasca et al., 2013; Vajda & Láng, 2014; Waller, 1993; Waller et al., 2001; Witkiewitz & Dodge-Reyome, 2000; Wonderlich et al., 2001). A closer look indicates that

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many of these mediators represent aspects of a major developmental factor, such as emotion regulation and trait self-esteem. Others are distinct mental disorders themselves, and their placement as mediators can scarcely explain the origins of EDs, as eating pathology is a distinct mental disorder and comorbidity cannot explain or imply causality (Valderas et al., 2009). Some of them are specific emotions or mood states. However, specific emotions and mood states result from many conditions (e.g. having a chronic mental disorder, being abused) or disrupted psychological factors (trait self-esteem, emotion regulation) that can cause psychological distress. Moreover, participants are measured at a specific point in time during which any given life event can affect their mood and, thus, their coping behaviours. Some of these studies' findings also suggest that childhood non-physical abuse could be the primary form of childhood trauma that results in low self-esteem and, consequently, in body dissatisfaction, which are both major risk factors for ED development, maintaining that further exploration of the association between non-physical abuse and EDs is needed (Kennedy et al., 2007). Additionally, meta-analytic reviews on risk and maintaining factors for EDs suggest that they have not been adequately identified (Stice, 2002).

In sum, the literature highlights the importance of shifting attention from physical to non-physical childhood abuse (Kennedy et al., 2007) since physical forms of abuse have failed to provide a lucid explanation for the pathway to ED development. In addition, the major developmental factors affected by childhood non-physical abuse (attachment, trait self-esteem, emotion regulation) should be explored as mediators because in order to understand the origins of any condition, it is necessary to examine the starting point and how it developed. One of the few consistently undisputed tenets in psychology is that the development of the self and all its associated concepts and major functions takes place in the early years of life (Bee, 1999; Rochat, 2003). The development of the self, for example, is an important factor in anorexia nervosa (AN) linked with attachment (Amianto et al., 2016). The

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authors proposed that functions of the self are not integrated due to disrupted attachment and as a result, body experiences and emotional expressions are detached from the self (Amianto et al., 2016). This aligns with the creation of a false self to preserve attachment as the true self is not accepted by significant others (Winnicott, 1965). This concept has been empirically supported by AN patients' accounts indicating that the sense of self is important in AN maintenance and that it is enmeshed with the disorder as the disorder takes control of the real, 'fragile' self (Williams et al., 2015). Even though it is not possible to study the developmental process in adults, the outcomes of an already established self (e.g. trait self-esteem, emotion regulation skills) can be measured, offering insight when linked to childhood non-physical abuse and eating pathology. In addition to the importance of exploring non-physical abuse regarding ED development, the difficulty in treating EDs must be taken into consideration as well, stressing the necessity of clarifying the specific maintaining psychological factors that need to be targeted in therapy, as they could be the same ones responsible for the onset. Considering that all humans undergo the same developmental process (Bee, 1999), along with the empirical suggestion that the factors involved in the development of EDs are presumably transdiagnostic (Milos et al., 2005), the proposed developmental pathways in this thesis adopt a transdiagnostic perspective, and it is hypothesised that the factors under study are involved in both the origins and maintenance of EDs.

In this sense, it could be of explanatory significance to first study family dysfunction and eating pathology with trait self-esteem as a mediator to initially establish an empirical developmental link (Study 1), before proceeding to a developmental model that combines all aspects of childhood non-physical abuse with respect to EDs, plus other mediators that have been empirically supported as important for EDs, such as attachment and emotion regulation

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(Study 2). The inclusion of a healthy control group could enhance the role of the proposed factors by further testing them beyond the presence of psychopathology.

1.3. Background and Rationale for the PhD Studies

To the best of my knowledge, my MSc study (Theodoropoulou, 2014) employing regression analyses was the first to explore family functioning and trait self-esteem together as predictive factors of psychosocial (psychological health and social relationships) quality of life (QoL) in officially diagnosed ED patients (AN, bulimia nervosa, binge ED), comparing the results to a healthy group. The healthy group was defined as a group with no self-reported mental or physical illnesses and no ED or subclinical ED as measured by the Eating Disorder Examination Questionnaire (EDEQ).

Earlier studies have examined the impact of family function on trait self-esteem (Ha et al., 2006; Lau & Kwok, 2000; Smets & Hartup, 1988), of family function on EDs (McDermott et al., 2002; Erol et al., 2007; Wisotsky et al., 2003), of self-esteem on QoL (de la Rie et al., 2005) and of self-esteem on EDs (Cervera et al., 2003; Gual et al., 2002; Katsourani, 2009; Mendelson et al., 2002; Newns et al., 2003; Sassaroli & Ruggiero, 2005). However, there were no studies examining both factors together regarding psychosocial QoL in a clinical ED population. Moreover, many studies have examined QoL in ED patients (Abraham et al., 2006; Bamford & Sly, 2010; Mond et al., 2005; de la Rie et al., 2007; de la Rie et al., 2005; Watson et al., 2012), mostly focusing on physical QoL instead of psychosocial QoL (Engel et al., 2009). The MSc study filled this gap, contributing to the exploration and understanding of the factors that could affect ED patients' psychosocial QoL.

The results of the MSc study indicated that some aspects of family functioning (roles, communication, problem-solving, general functioning) and trait self-esteem predicted overall QoL and specific aspects of QoL (psychological health, social relationships). Trait self-

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esteem emerged as the strongest predictor of psychosocial QoL, especially in BN patients, and it was a significant predictor for all sample groups (AN, BN, BED, non-clinical group) in the psychological domain of QoL. It was the only predictor of psychological health for both AN and BN patients and the only predictor of social relationships in the BN sample. In summary, high trait self-esteem was associated with higher QoL in controls and low trait self-esteem was associated with low QoL in patients. I discussed the results with respect to psychological QoL and how they supported past literature arguing that self-esteem in individuals with EDs is affected by family dysfunction (Ha et al., 2006; Lau & Kwok, 2000) and that self-esteem can significantly affect psychological well-being (Baumeister et al., 2003). The study concluded that self-esteem might also be severely damaged in this clinical population because it is the most crucial predictor regarding their psychological health. By this, I theoretically implied that family dysfunction in ED patients consequently creates low self-esteem over time – or at least its consequences are manifested as such. Low self-esteem has been found to affect the social lives of people with chronic illnesses (Juth et al., 2008), such as EDs, since it affects individuals' physical and psychosocial health. The ED groups did not display statistically significant differences amongst them regarding psychosocial QoL, but they all had statistically significant lower QoL scores than healthy controls. Figure 1 shows the predictors and outcome variable of the MSc study.

Figure 1. Family functioning and self-esteem as predictors of psychosocial QoL in the MSc study.



Figure 1. Family functioning and self-esteem as predictors of psychosocial QoL in ED patients (n = 88) and controls (n = 44) in the MSc study. In the analysis, the ED subgroups were used (AN = 27, BN = 31, BED = 30).

1.3.1. Extension of the MSc Findings and Proposed Links Leading to the PhD Studies

As the MSc findings indicated, family dysfunction and self-esteem could be related to ED development, and, therefore, these results could be expanded by testing self-esteem as a mediator between family functioning and ED/psychosocial QoL in ED patients and controls. Obtaining a comprehensive picture of the role of family functioning and self-esteem by testing them through mediation would subsequently allow the design of more complex models with respect to eating pathology. Considering the population employed for the MSc study along with its results, family dysfunction and self-esteem could be regarded as transdiagnostic factors in EDs.

Based on the results of the MSc study, as well as existing theoretical and empirical literature, seven conclusions were drawn that led to the design of three studies for this PhD: first, women with an ED report significant levels of family dysfunction (Wisotsky et al., 2003) and less secure attachment to their parents (Cunha et al., 2009). Family dysfunction has also been associated with childhood abuse (Mullen et al., 1996) and low self-esteem (Ha et al., 2006). It is argued that family dysfunction does not directly lead to disordered eating, rather it affects emotion regulation through attachment and core beliefs about the self (self-

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esteem), which in turn influence ED development (Ross & Green, 2011; Vajda & Láng, 2014). It has further been suggested that the intense focus on food replaces the inadequate regulatory functions of early attachment (Pearlman, 2005) linking attachment with EDs. These factors are discussed in more detail in Chapter 3, as they are relevant to the PhD studies.

Second, attachment is considered crucial in shaping self-representations and the emotional consequences of self-confidence and self-doubt; the formation of a psychologically proper and strong ego identity and maturity; and the shaping of working models (representations) of self, morals, emotions and relations (Ross & Green, 2011; Thompson, 1998). An insecure attachment style could have a negative effect on the individual's psychological health and later social relationships (Mikulincer & Shaver, 2012; Pearlman & Courtois, 2005), especially in enmeshed families that do not allow personal growth (Winston, 2009). This indicates a link with attachment and psychosocial QoL. Attachment has also been associated with EDs and childhood maltreatment, and it is considered responsible for the development of emotion regulation (Barth, 2008; Cicchetti & Doyle, 2016; Dakanalis et al., 2014; Gander et al., 2015; Morris et al., 2013; Shaver & Mikulincer, 2007; Tasca & Balfour, 2014; Tasca et al., 2013).

Third, failure to regulate emotions can result in psychopathology including EDs (e.g. Gross & Munoz, 1995). Emotion regulation is a developmentally significant accomplishment and is essential for positive social adjustment (Stifter et al., 2011), indicating that it could affect individuals' social QoL. Crucially, emotion regulation has been found to be affected by non-physical childhood abuse (Vajda & Láng, 2014) and has been associated with disordered eating (Svaldi et al., 2012). It is argued that people's ability to regulate their emotions may vary according to their temperament because differences in the temperamental approach may

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produce different emotions that could need regulation (Burt et al., 2015; Stifter et al., 2011). In addition, certain temperamental traits, such as negative affect and effortful control, are considered individual risk factors for ED development (Burt et al., 2015).

Fourth, although temperament is believed to be biologically based (Tortella-Feliu et al., 2012), suggesting an innate predisposition for emotional skill, some authors argue that learning to regulate emotional expressions also depends on attachment and family dynamics (Fox, 1998; Wilson et al., 2000), rather than being a genetic trait (Martins et al., 2012). This means that temperament could be affected by the environment as well.

Fifth, low trait self-esteem, a well-known accompanying trait of disordered eating (Sassaroli & Ruggiero, 2005), is a major risk factor for ED development and maintenance (Adamson et al., 2019; Biney et al., 2019; Giovazolias et al., 2013) and severely affected by CEA (Kent & Waller, 2000). Studies indicate that it may play a more substantial role than emotion regulation in EDs (Mollen et al., 2015; Monell et al., 2020). Surprisingly, even though scholars have either implied or empirically supported its important role both in ED development and in non-physical childhood abuse, self-esteem has not been used as a mediator between non-physical abuse forms and ED in clinical populations to date.

Sixth, non-physical forms of childhood abuse remain under-researched (Vajda & Láng, 2014; Waller et al., 2007), even though there is evidence of significant association with eating pathology (Moulton, 2013; Vajda & Láng, 2014). Reviews indicate that family dysfunction has been implied in ED literature as a form of non-physical abuse (Kent & Waller, 2000) and has been linked with the invalidating environment (Crowell et al., 2009; Gonçalves et al., 2019). Moreover, non-physical childhood abuse can be also found in the definition of invalidating environment (Linehan, 1993) with the latter having been extended to ED research (Fox, 2009; Gonçalves et al., 2019; Haslam et al., 2008; Haslam et al., 2012).

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Non-physical childhood abuse has been found to affect attachment (Tasca et al., 2013), self-esteem (Waller et al., 2007) and emotion regulation (Vajda & Láng, 2014) in EDs. These psychological factors are also impaired in BPD patients (Linehan, 1993), in whom an invalidating environment is also present.

Seventh, the invalidating environment, family dysfunction, low self-esteem, emotion dysregulation, poor attachment, temperament and low QoL are present in all EDs, regardless of the subtype (Adamson et al., 2019; Biney et al., 2019; Cerniglia et al., 2017; Cortés-García et al., 2019; Fassino et al., 2002; Ford et al., 2011; Gander et al., 2015; Gonçalves et al., 2019; González et al., 2001; Haslam et al., 2008; Mazzeo & Bulik, 2009; Monell et al., 2018; Newns et al., 2003, de la Rie et al., 2005; Tasca et al., 2019).

To summarise, non-physical childhood abuse disturbs the attachment process, which impacts emotion regulation and trait self-esteem. Both emotion regulation and self-esteem are associated with EDs and are regarded as major risk factors for the development and maintenance of eating pathology. Furthermore, an invalidating environment, poor attachment, low self-esteem and emotion dysregulation are factors found in all EDs and are thus transdiagnostic in nature. Elaborating on the combination of the previously mentioned factors, the MSc study triggered the need to test theoretical models that capture these relationships and further explore factors that correlate with and may, therefore, contribute to ED development. Consistent with the MSc, psychosocial QoL remained the outcome variable with the hypothesis that it is affected by the proposed factors, and this is the reason it remains low even after ED symptoms improve.

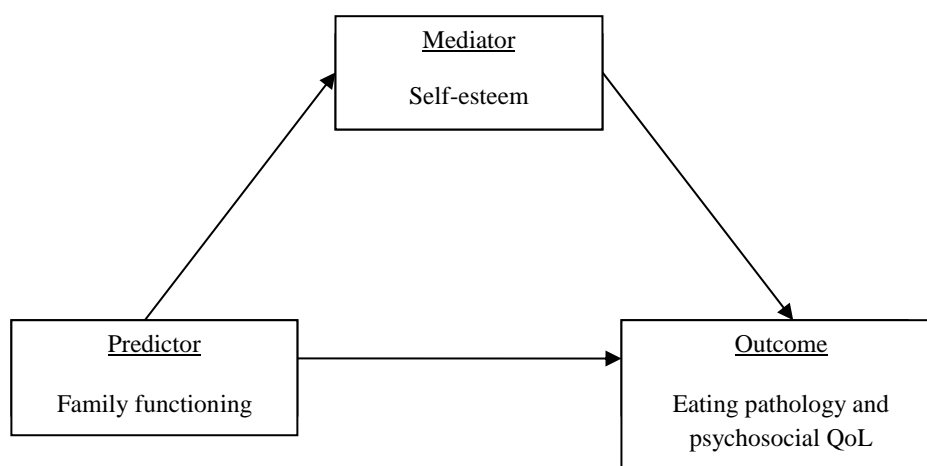
1.3.2. Design and Aims of the PhD Studies

Study 1. The first PhD study extends the original MSc study by examining the association of family functioning and self-esteem with psychosocial QoL and clinical

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characteristics of ED patients using mediational models (see Figure 2). It does this in comparison to normal controls to better understand the similarity of the models in both groups. The sample size for this first study was larger than that of the MSc study, and the variable of eating pathology was also added to permit more complex analyses, such as structural equation modelling (SEM).

Figure 2. Study 1: Effects of family function on eating pathology and psychosocial QoL with self-esteem as a mediator.



As shown in Figure 2, self-esteem was expected to mediate the relationship between family dysfunction and ED/psychosocial QoL. The findings from Study 1 were further extended by framing and interpreting them through different aetiological perspectives based on Kent and Waller's (2000) ED theory and Linehan's biosocial theory (1993) of BPD (Studies 2a and 2b, respectively).

Family dysfunction is linked with the invalidating environment (e.g. Crowell et al., 2009) and with Kent and Waller's (2000) theory of emotional abuse formulating the concept of childhood non-physical abuse. The potential emerging link from Study 1 and the theory behind this link is that family dysfunction can lead to insecure attachment and negative self-

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esteem, which in turn can lead to emotion dysregulation and, consequently, to maladaptive coping strategies (e.g. EDs) to handle the intolerable effects of the lifelong psychosocial consequences. The formation of these psychological processes (attachment, self-esteem, emotion regulation), which are regarded as sensitive to family dysfunction, takes place in the early years of life (Morris et al., 2013). Thus, Study 2 reconceptualised and further explored family dysfunction and its association with psychological factors that affect major developmental processes in relation to their impact on mental health and psychosocial QoL in adulthood. The adaptation and testing of two different models is important because all aspects of childhood non-physical abuse found in the literature are considered, making the independent variable more theoretically robust. This advances our understanding of how childhood non-physical abuse works since the conceptual combination of these theories (family dysfunction, emotional abuse, the invalidating environment) resolves the issue of different terminology across theories and studies, allowing the exploration of the possible pathways through which they manifest their psychological consequences.

Study 2. Part a. Family dysfunction was conceptualised as CEA, childhood emotional neglect and childhood physical neglect (which from this point will constitute the term *non-physical childhood abuse*) to understand its consequences regarding the relationship with ED and ED patients' psychosocial QoL. It was hypothesised that the reason family dysfunction affects ED patients' psychosocial QoL and contributes to ED development relies on the impact of the non-physical forms of childhood abuse on attachment, emotion regulation and self-esteem. This study simultaneously expanded Kent and Waller's model (2000), which proposed that emotional abuse plays a direct, causal role in ED development, by arguing that this relationship could also be mediated by low self-esteem and/or anxiety. It achieved this by adding 'neglect' to emotional abuse, by using different mediators and by including a clinical population instead of the non-clinical one used in their model (Kent et al.,

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1999). The study additionally included a non-clinical control group for comparison. Moreover, Study 2a conceptualises the factors discussed in the SPAARS-ED model (Fox & Power, 2009) in a concise developmental model. The SPAARS-ED model thoroughly explains the development of emotion dysregulation in EDs from a transdiagnostic perspective (see Chapter 3, Section 3.5.3). The three mediators were used with no specific sequence to explore which affects or has a higher effect on the dependent variables, as all three are affected by childhood non-physical abuse (see Chapter 3). The attachment literature indicates that self-esteem and emotion regulation are formed through attachment, meaning that attachment could chronically predate self-esteem and emotion regulation.

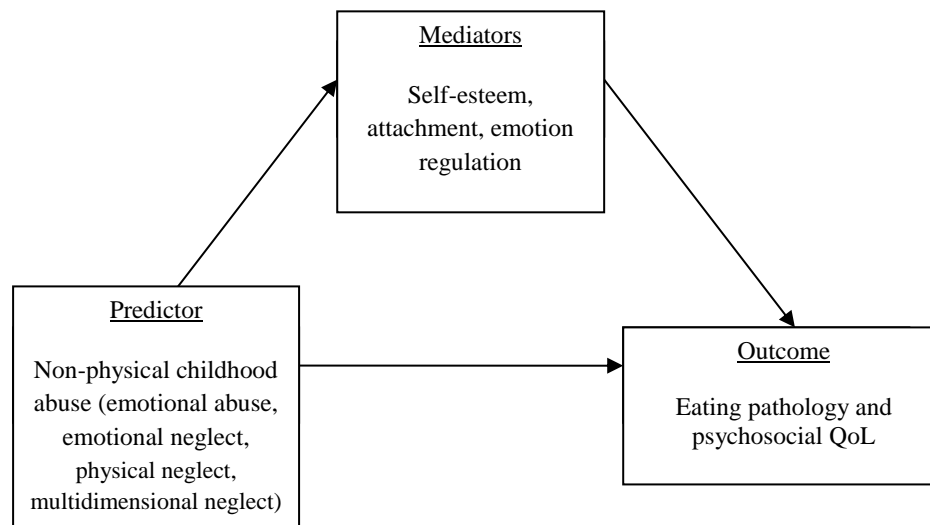
The purpose of the study was, therefore, to examine a developmental model to explain the origins of EDs. Put differently, this thesis's focus involved exploring the effects of childhood non-physical abuse on the developmental factors (self-esteem, attachment, emotion regulation) associated with the onset of EDs. The emotions generated by the presence of and impairment caused by the study's proposed factors have already been explained (Fox & Power, 2009). Each one of these factors (non-physical childhood abuse, insecure attachment, low self-esteem, emotion dysregulation, poor psychosocial QoL) can lead to psychological distress, including EDs themselves. For instance, difficulty in managing strong emotions can cause distress (Hund & Espelage, 2006; Krause et al., 2003), and this could explain depression and anxiety observed in people with emotion regulation difficulties, such as ED patients. Aspects of general psychological distress (e.g. sadness, anxiety) and affective symptomatology (e.g. depression) are comorbid conditions for EDs rather than causal factors (Hay & Williams, 2013) and can be found in almost all chronic physical and mental conditions. Moreover, general distress is not always present in eating pathology (García-Alba, 2004). Thus, in the context of this thesis, psychological distress is viewed as a consequence of EDs rather than as a mediating factor. Additionally, studies have

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demonstrated that trait self-esteem is a strong predictor of psychological well-being (depression, anomie, general anxiety resentment, anxiety, irritability, life satisfaction, guilt, happiness, negative affective states) (Rosenberg, 1995). Further to this, low trait self-esteem has been associated with high levels of general distress (den Heijer et al., 2011). This means that the low self-esteem observed in EDs could generate distress and low mood states, indicating that the reason behind negative emotions could be the impairment of a developmental factor. Moreover, psychological distress is marked by definition issues (Luckett et al., 2010), and its consideration in the models would only add to the mediation complexity observed in the ED literature. For example, psychological distress is either composed of anxiety and depression (den Heijer et al., 2011; Kessler et al., 2002) or perceived as a separate entity (Gulliver et al., 2012; Hay & Williams, 2013; Luckett et al., 2010). The thesis studies aimed to narrow down the main proposed causal factors and explore them in a developmental way in order to eliminate the complexity observed in the ED literature. There is no question that child abuse may lead to depression (Bradley et al., 2008), and reviews indicate a link between childhood neglect and anxiety and depression (Pignatelli et al., 2017). However, this thesis posits that general psychological distress, observed as a mediator between childhood abuse and EDs (e.g. Mazzeo et al., 2008), is caused by the impairment of developmental factors (e.g. attachment, self-esteem, emotion regulation) due to childhood non-physical abuse. The improvement of these factors in therapy will improve general distress. Thus, specific mood conditions and emotions related to the disorder and childhood abuse were not used as mediators.

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Figure 3. Study 2a: Effects of non-physical childhood abuse on eating pathology and psychosocial QoL with self-esteem, attachment and emotion regulation as mediators.



Part b. Study 2b tested an alternative theoretical model that explains the complexity of ED and its aetiological correlates. BPD patients and ED patients share common psychological features (e.g. the invalidating environment, emotion dysregulation, low self-esteem, poor attachment), suggesting that there could be a shared aetiological basis or at least common influential factors (Sansone & Sansone, 2007). The concept of the invalidating environment has been used to explain EDs (Gonçalves et al., 2019; Haslam et al., 2008; Haslam et al., 2012), and studies regarding EDs and emotion regulation already support the basic concept of biosocial theory (Fox, 2009). For the purpose of these studies, the invalidating environment is conceptualised as non-physical childhood abuse (a more detailed explanation can be found in Chapter 3). The new model adapted Linehan’s (1993) biosocial theory (Figure 4), retaining its basic tenets (key variables and pathways to emotion dysregulation) but incorporating additional predictors and potential mediators as pathways to EDs (see Figure 5 for the revised model). In response to this and continuing from Study 2a, which further expands Kent and Waller’s (2000) theory and the SPAARS-ED model (Fox & Power, 2009), Study 2b adapted Linehan’s theory by exploring the effects of non-physical childhood abuse and temperament on emotion regulation, self-esteem and psychosocial QoL

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in ED patients using attachment as an additional mediator and comparing the results to a healthy control group. Study 2b was designed as a separate study from Study 2a in order to test this adapted model, first with temperament as a mediator and then modified for EDs (using attachment as a mediator).

Figure 4. Linehan's (1993) biosocial theory model.

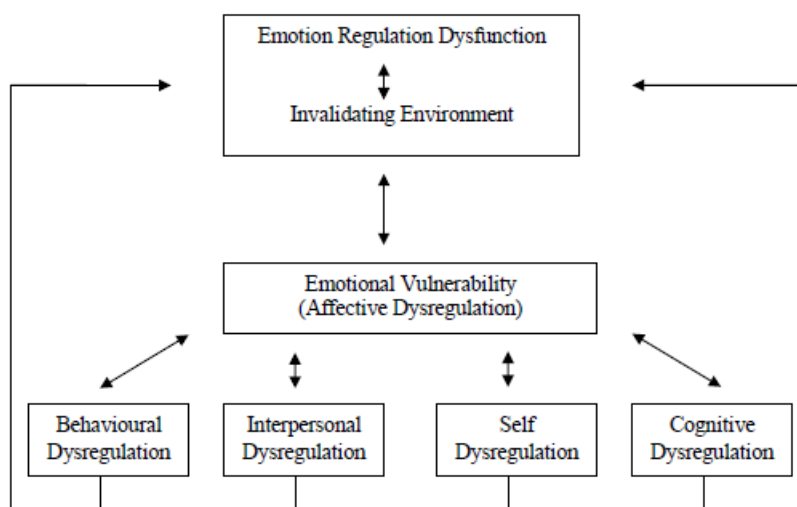


Figure 4. Linehan, M. (1993a). Cognitive-behavioural treatment of BPD. The Guildford Press. Reprinted with permission from the Guilford Press.

Figure 5. Study 2b revised model of biosocial theory.

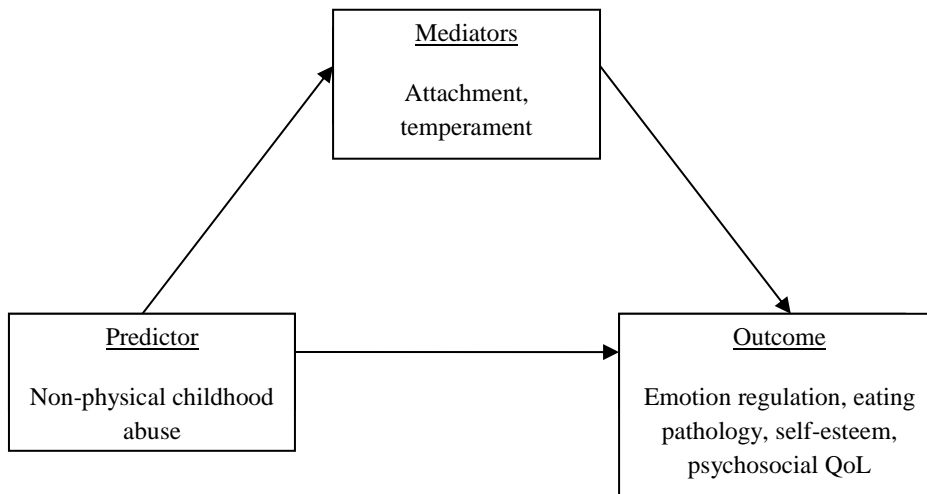


Figure 5. Effects of non-physical childhood abuse on emotion regulation, self-esteem and psychosocial QoL with attachment and temperament as mediators. Adapted with permission from the Guilford Press.

Study 3. The theoretical models of Studies 1 and 2 are extended in Study 3 where the non-physical forms of abuse experienced by women with an ED are explored in depth using a qualitative paradigm. Waller and colleagues (2007) extended O’Hagan’s (1995) emotional abuse definition to include adult experiences, as ED patients often report that this form of abuse continues into their adult years, either as a primary experience or as a revival of their childhood trauma. The latter is in agreement with the psychoanalytic view offered by Freud within the term *repetition compulsion* to explain that people seek comfort in the familiar (Freud, 1920). Repetition compulsion posits that the individual, who has experienced trauma, seeks symbolic repetition in order to master its unbearable psychological consequences and heal it (Winnikott, 1965). Waller et al.’s argument (2007) fits perfectly with my clinical experience, but what is missing from the literature is evidence indicating whether ED patients, who have suffered non-physical childhood abuse, endure this kind of abuse as adults due to their eating pathology and/or due to their past non-physical abuse and its enduring

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consequences in adulthood. Since this has not been researched before, Study 3 is a pioneer study.

1.3.3. Significance of the Studies

This series of studies offer a significant endeavour in the understanding of known factors (family functioning, childhood abuse, self-esteem, attachment, emotion regulation, temperament) related to EDs and psychosocial QoL by examining them through alternate perspectives that could clarify the way they relate to each other. The proposed models (Studies 1 and 2) and the explanation that ED patients provide regarding the continuity of their non-physical abuse in adult life and its link to EDs (Study 3) will expand existing psychological models and inform future theoretical and empirical work, as well as the design of more focused interventions for prevention and treatment.

1.3.4. Summary of Original Contribution to Knowledge

The mediational model proposed in Study 1 has not been researched before and extends an original MSc study (Theodoropoulou, 2014). The study is important for advancing understanding of the aetiological correlates between family dysfunction and EDs. The theoretical perspective used to interpret the consequences of family dysfunction in Study 2a is original, as well as the proposed reconceptualisation of family dysfunction and the invalidating environment (non-physical childhood abuse) along with the exploration of psychosocial QoL. Study 2a expands Kent and Waller's model (2000) by adding neglect to emotional abuse, testing only the non-physical forms of childhood abuse, using different mediators (self-esteem, attachment, emotion regulation) and different outcome variables (psychosocial QoL) and by using a clinical group as well as a non-clinical one to compare the results. It additionally offers a developmental design of the core concepts considered in the SPAARS-ED model (Fox & Power, 2009).

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Linehan's biosocial model (1993) was adapted for EDs in Study 2b using a specific operationalisation of the invalidating environment (non-physical childhood abuse), additional mediators (attachment) and different outcome variables (self-esteem, psychosocial QoL, EDs). The study addressed the question of how the invalidating environment is associated with EDs (Haslam et al., 2012), and Linehan's theoretical model was tested for the first time. Study 2 expanded and tested two different models using all of the important psychological factors for EDs together for the first time simultaneously linking two theories (Kent & Waller, 2000; Linehan, 1993) under the term 'childhood non-physical abuse'. Study 2 addresses the literature concern (Kent & Waller) regarding the link between family dysfunction and CEA, which was implied rather than expanded upon and researched. Additionally, Study 2 validates the theory behind the SPAARS-ED model (Fox & Power, 2009) through a core developmental presentation that originates in psychodynamic theories and links classic and contemporary psychology.

Study 3 explored the continuation of non-physical abuse from childhood to adulthood, along with its consequences in adult ED patients, which has never been researched before. This study also demonstrated that the factors associated with the development of EDs are equally responsible for their maintenance.

1.4. Summary

There is a gap in the literature regarding childhood non-physical abuse and its link to the onset of EDs. There seems to be a psychological link between non-physical abuse, self-esteem, attachment, emotion regulation and temperament that relates to the onset and maintenance of eating pathology also affecting psychosocial QoL. These factors, as well as their relationships with each other, are presented in Chapters 2 and 3 of the literature review. Chapter 4 describes the thesis studies' methodologies, while Chapters 5, 6 and 7 display the empirical studies. Chapter 8 closes this thesis with a general conclusion.

Chapter 2: Eating Disorders: Theoretical Causes and Impact on Psychosocial Quality of Life

2.1. Overview

This chapter will focus on the description of eating disorders (EDs) along with their theoretical causes and their impact on psychosocial quality of life (QoL). Eating pathology and psychosocial QoL are the main outcome variables of Studies 1 and 2. Initially, the diagnostic subtypes along with their criteria are presented, followed by the most prominent theories and models surrounding eating pathology. A rationale is presented for adopting a transdiagnostic approach. Then, the impact of EDs on psychosocial QoL is explained. Continuing the literature review, Chapter 3 presents the risk factors associated with EDs and subsequent poor QoL, focusing on their mediating role between family dysfunction, non-physical abuse, the invalidating environment and disordered eating development.

2.2. Eating Disorders

EDs have been characterised as disorders that are interesting to the public, complex for researchers and challenging for clinicians (Fairburn & Harrison, 2003). They are chronic in nature and characterised by relapse, with high rates of mortality and other comorbid mental disorders, causing serious implications to overall health and reproduction (O'Brien et al., 2017; Stice et al., 2013). Mortality rates among women with EDs, especially for those with anorexic nervosa (AN), are higher than for any other mental disorder and the suicide rates of AN patients are recorded as 12 times the corresponding number of women of matched age in the general population (APA, 2006). Moreover, the prevalent estimates of suicide attempts by adult ED patients in the US are reported as 15.7% for AN- restricting (AN-R) type, 44.1% for AN binge/purge (AN-B/P) type, 31.4% for bulimia nervosa (BN) and 22.9% for binge ED (BED) (Udo et al., 2019). Additionally, suicide attempts have a high incidence rate among ED patients in the UK, and AN patients are at higher risk (Cliffe et al., 2020).

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Over the last three decades, the prevalence of EDs has increased both in Europe and in the US, and their aetiology is still considered unclear (Bilali et al., 2010; Claudat & Lavender, 2018; Kaplan & Sadock, 2007; Keski-Rahkonen & Mustelin, 2016; Kyrgios et al., 2015; Micali et al., 2013; Rikani et al., 2013; Ringer et al., 2007). EDs' prevalence in the US general population is higher in women (2.7%) than in men (0.8%) (Afifi et al., 2017; American Psychiatric Association, 2013; Lafrance-Robinson et al., 2013), and this gender difference is present in Europe as well (Keski-Rahkonen & Mustelin, 2016). This gender difference has been largely attributed to the fact that societal messages about thinness are aimed at females rather than males, with the ideal shape for women becoming thinner in social media (Tiggemann et al., 2018).

The American Psychiatric Association (APA) (2013) relates the onset of EDs with a stressful life event, such as leaving home for college/university, separation or the death of a close family member. Dieting and body dissatisfaction among college women have been characterised as epidemic, raising concerns regarding unhealthy nutrition and eating disturbances (Germov & Williams, 1996; Graff-Low et al., 2003). In addition, ED symptoms and subclinical eating disturbances are highly prevalent and persistent in college populations, especially in college women (Chang et al., 2015; Eisenberg et al., 2011). According to studies, female college students diet, regarding themselves as overweight even though they have a non-overweight body mass index (BMI) (Fayet et al., 2012), suggesting weight and shape concerns are highly prevalent in college populations. The conclusion that college campuses have become breeding grounds for EDs is quite alarming, prompting researchers' efforts to sufficiently identify the aetiology and design effective prevention and intervention programmes (Berg et al., 2009; Compas et al., 1986; Kitsantas et al., 2003). A number of psychological risk factors have been consistently associated with eating pathology development, such as parental overemphasis on physical appearance and food, adverse life

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events, mood intolerance, exposure to criticism regarding physical appearance and the promotion of thin stereotypes (Mazzeo & Bulik, 2009). However, the origins of these common factors observed in EDs have not been explored and explained in a combined model.

There is disagreement in the literature regarding whether there exists successful evidence-based treatment for EDs (Costa & Melnik, 2016; Waller, 2016), and many treatment designs have been developed (Davis & Attia, 2019). Additionally, there is no effective treatment for severe cases, like AN and atypical ED (Abbate-Daga et al., 2013; Fairburn & Harrison, 2003; Touyz et al., 2013). Cognitive-behavioural therapy (CBT) is the standard psychotherapy used in EDs, with a variety of other psychotherapies being employed/combined for difficult and persistent cases (e.g. see Kass et al., 2013 for a review). Different models of psychotherapy seem to work for different subtypes, but the need for a treatment with long-term effects and low remission rates is still evident. Moreover, randomised control trials (RCTs) are lacking in the field of psychotherapy in EDs (Linardon et al., 2017), and the identification and targeting of maintaining factors in AN is still an issue (Zeeck et al., 2018). The development of several treatments for EDs could be attributed to the variation of treatment effectiveness among the ED subtypes, their overall complexity and unclear aetiology. Over the last several years, efforts have been made to incorporate dialectical behaviour therapy (DBT) (Linehan, 1993) into ED treatment with promising results (Ben-Porath et al., 2020), including improvements in patients' QoL (Amstadter & Squeglia, 2007; Rahmani & Omid, 2019; Rahmani et al., 2018; Shumlich, 2017). This indicates that the biosocial theory, which underpins DBT (Linehan, 1993), could be used to further understand eating pathology. However, more research is needed to garner empirical support and extend our understanding. Moreover, systematic literature reviews have identified that there is still an absence of effective prevention interventions regarding the ED

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risk factors (Le et al., 2017). This means that continued research is needed in this field to better identify the risk and maintaining factors of EDs so that they can be targeted in psychotherapy and prevention.

2.2.1. Anorexia Nervosa

According to the latest edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-V), published by the American Psychiatric Association (2013), a diagnosis of AN, a term introduced by William Gull in 1874 (Nolen-Hoeksema, 2003), requires a person's refusal to eat and maintain a body weight that is normal for their age and height, displaying a significantly low body weight (Criterion A). Significant low body weight is a weight less than minimally normal. Other criteria include an intense fear of gaining weight or becoming fat and/or persistent behaviour that interferes with weight gain (Criterion B) and disturbances in the way body shape is evaluated, the influence of body weight on self-evaluation and/or failure to recognise the seriousness of the current low body weight (Criterion C). AN is also specified by severity in the DSM-V, ranging from mild to extreme based on BMI. There are two types of AN: the restricting type, in which the person refuses to eat and can go for days without eating or only eating the minimum necessary for survival, and the binge eating/purging type, in which people engage in bingeing behaviour or purging, such as the misuse of laxatives and diuretics and self-induced vomiting. Both types may involve excessive exercise in order to burn calories, and the binge eating/purging type is more prone to substance abuse (APA, 2013). The onset of the disorder has been associated with a stressful life event, and there is rarely onset after the age of 40 as it commonly begins during adolescence. Some individuals may display atypical characteristics, such as denying the fear of becoming fat (APA, 2013). The mortality rate is 5% per decade due to suicide or medical complications resulting from the disorder. In fact, AN has the highest death rate of all psychiatric disorders due to its grave health implications (Chidiac, 2019), and the highest

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rate of suicide attempts among EDs (Kostro et al., 2014). Literature reviews indicate that there is an approximately 10:1 female-to-male ratio (Kostro et al., 2014).

People suffering from AN experience a distorted body image and they believe they are worthwhile only when they can control their eating and lose weight, with the latter serving as a reward (Walsh, 2013). Moreover, low self-esteem is considered a risk factor for relapse in AN (Carter et al., 2012). AN has proven to be highly resistant to treatment due to the ego-syntonic nature of restraint eating that leads to feelings of pride (Faija et al., 2017; Gregertsen et al., 2017; Mond et al., 2006; Peroutsi & Gonidakis, 2011), prompting ED behaviours, such as strict dieting, to become habitual (Abbate-Daga et al., 2013; Eshkevari et al., 2014; Halmi, 2013; Hay et al., 2012; Le Grange et al., 2014; Touyz et al., 2013; Walsh, 2013). Due to treatment resistance, AN patients are often detained under the Mental Health Act (Seed et al., 2016), which is a stressful procedure for patients, caregivers and therapists. The significant difficulty in treatment, combined with patients' resistance, particularly for the restrictive type, could possibly be explained by characteristics of autism found in AN patients (Baron-Cohen et al., 2013). Autism and AN behaviour share similarities, such as self-preoccupation, alexithymia and rigid attitudes (Baron-Cohen et al., 2013; Kelly & Davies, 2019). Moreover, women with an autism diagnosis display restrictive eating patterns, indicating possible overlap between autism and AN (Brede et al., 2020). This is also supported by similarities in the theory of mind (ToM) profile of AN and autism patients (Leppanen et al., 2018). Impairments in ToM are associated with social anxiety (Hezel & McNally, 2014), and this could support the suggestion that the autism traits observed in AN could be partially responsible for social anxiety in AN (Treasure et al., 2020). However, systematic reviews conclude that longitudinal studies are required to clarify the symptomatic overlap between AN and autism (Nickel et al., 2019).

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Common clinical findings in people suffering from AN include bradycardia, emaciation, dehydration, amenorrhea, hypotension, lanugo (a fine, downy body hair), electrolyte disturbances, low bone mineral density and thyroid gland disturbances (APA, 2013; Fairburn, 2008; Westmoreland et al., 2016), indicating that this disorder may severely affect physical health, thus making it quite dangerous compared to other mental disorders. Thinking processes and sleeping patterns are also affected by severe weight loss (Fairburn, 2008). Suicide risk rates are reported to be 12 per 100,000 per year according to meta-analysis studies (Preti et al., 2011), and the AN 12-month prevalence in young women is 0.4% (APA, 2013). Additionally, AN is reported to appear in high-income countries but its incidence in middle- and low-income countries is uncertain (APA, 2013). There is a cultural difference observed in Asian populations, where ED patients attribute their AN behaviour to gastrointestinal discomfort rather than the fear of gaining weight (Nolen-Hoeksema, 2003).

2.2.2. Bulimia Nervosa

The American Psychiatric Association's DSM-V criteria (2013) for a BN diagnosis are as follows: BN is defined by uncontrolled eating or bingeing (Criterion A), followed by compensating behaviours to prevent weight gain (Criterion B), and self-evaluation is overwhelmingly influenced by body shape and weight affecting self-esteem (Criterion D). Compensatory behaviours include self-induced vomiting, fasting, excessive exercise and abuse of laxatives and diuretics. Binge eating and compensatory behaviours should occur on average at least once a week for three months (Criterion C), and the disturbance should not occur exclusively during episodes of AN (Criterion E). Binge eating is conceptualised as eating an amount of food that is larger than most people would eat during a similar period of time in similar circumstances, combined with a sense of lack of control over feeding behaviour, the latter of which is essential for the definition of a binge eating episode. The average binge is about 1,500 to 3,500 calories, but there are variations among people with BN

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regarding the sizes of their binges (Cooper, 2009). Severity ranges from mild to extreme in terms of the frequency of compensatory behaviours (APA, 2013). A diagnostic crossover has been observed (10%–15%) from BN to AN and vice versa (APA, 2013). The onset of BN occurs between 15–29 years of age, and it is a chronic condition that occurs more frequently than AN. As with AN, onset is rare after the age of 40, and it has been reported to occur in industrialised countries with comparable prevalence between white samples and other ethnic groups (APA, 2013).

Health complications include imbalances in the body's electrolytes, which can lead to heart failure, rotten teeth from frequent exposure to stomach acid through vomiting, amenorrhea (Westmoreland et al., 2016) and, in severe cases, rectal bowel collapse from compulsive bowel emptying (Gonidakis et al., 2015). In a total of 79 outcome studies on BN involving 5,653 patients, it was found that 45% fully recover, 27% partially recover, 23% develop a chronic disorder and 0.32% die (Steinhausen & Weber, 2009). The 12-month prevalence of BN among young women is 1%–1.5%, and the mortality rate is 2% per decade (APA, 2013). There is an approximately 10:1 female-to-male ratio, as with AN.

2.2.3. Binge Eating Disorder

BED was first described by psychiatrist Albert Stunkard as the 'night eating syndrome' (Stunkard, 1959). The term 'binge eating disorder' was used to describe the same bingeing-type eating behaviour without the exclusivity of the bingeing occurring at night. Lifetime prevalence in the US is 2.6% (Guerdjikova et al., 2019), and the diagnostic criteria of BED (APA, 2013) include recurrent episodes of binge eating (Criterion A), and binge eating episodes are associated with at least three or more of the following: eating more rapidly than normal, eating until uncomfortably full, eating large amounts of food when not feeling hungry, eating alone due to embarrassment over the amount of food, or feeling

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disgusted with oneself and guilty afterwards (Criterion B); eating while markedly distressed about doing so (Criterion C) occurring at least once a week for three months (Criterion D), but there is an absence of compensatory behaviour (Criterion E). BN is categorised from mild to extreme severity in terms of the frequency of the binge eating episodes. The DSM-V evaluates prevalence as 1.6% for females and 0.8% for males in a 12-month period (APA, 2013).

The onset of BED usually occurs in adolescence or early adulthood, but it can also begin in later adulthood. It is considered the most prevalent ED among adults displaying similarity across racial and ethnic groups (Udo & Grilo, 2018), and the gender ratio is more balanced than AN or BN (Guerdjikova et al., 2019). BED sufferers seeking treatment are usually older than AN and BN individuals seeking treatment (APA, 2013). Medical consequences occur mostly due to obesity, which is prevalent in this disorder (Wassenaar et al., 2019). BED and obesity are two distinct conditions (van Hoeken et al., 2009), and levels of body overvaluation and psychiatric comorbidity are higher in obese people with BED than in obese people without BED, and, unlike obesity, there are evidence-based psychological treatments for BED (APA, 2013).

2.2.4. Other Specified Feeding or Eating Disorders

This ED category includes 1) atypical AN: all the criteria for AN are met, but despite significant weight loss, the patient's weight remains within or above the normal range; 2) atypical BN (of low frequency and/or limited duration): all of the criteria for BN are met, but binge eating and compensatory behaviours occur less than once a week and/or for less than three months; 3) atypical BED (of low frequency and/or limited duration): all of the criteria for BED are met, but binge eating occurs less than once a week and/or for less than three months; 4) purging disorder: recurrent purging behaviour to influence weight or shape

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(vomiting, laxative misuse, diuretics, other medications), but there is no binge eating behaviour; and 5) night eating syndrome: recurrent episodes of night eating manifested by eating on awakening from sleep or by excessive food consumption after the evening meal. In night eating syndrome, there is recall and awareness of the night eating, and it cannot be better explained by BED, any other mental/medical disorder or drug side effects (APA, 2013).

2.2.5. Unspecified Feeding or Eating Disorder

The unspecified ED category applies to conditions in which the symptoms of an eating disorder are not fully met but cause significant distress and impairing the individual's life. The unspecified ED category can be used in situations where the clinician chooses not to specify the reason the criteria for a specific ED have not been met, usually due to insufficient information (APA, 2013).

2.2.6. Differences Among Eating Disorder Subtypes

There are several differences between the subdiagnostic entities of EDs (APA, 1994, 2013; Barry et al., 2003; Nolen-Hoeksema, 2003), many of them by virtue of their diagnostic criteria. For example, there are noticeable differences between BN and AN-B/P. The criteria for the AN-B/P type require that a person has a significantly low weight, whereas BN does not have weight criteria, with BN patients usually being a normal weight or overweight. Another significant difference can be found in the size of binges. While people with BN consume large amounts of food in their binge episodes, AN-B/P usually eat a small amount of food, which is perceived by them as bingeing, and they purge it. Moreover, self-evaluation in BN is influenced by body shape, like in AN, but BN patients do not have a distorted body image like people suffering from AN do. Unlike BN and BED, some AN patients, mostly of the restrictive type, display a delusional fear of fat (Behar et al., 2018; Steinglass et al., 2007),

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but other studies have concluded that the delusional nature in AN is not like the one observed in psychotic disorders (McKenna et al., 2014). Also, the death rate in BN is not as high as in AN. There are also differences between BED and BN, as BED patients do not engage in any of this BN type's compensating behaviours to prevent weight gain from their binges, which are larger in amount compared to those in BN. Furthermore, people with BN are of a normal weight or are overweight, while people with BED are usually obese (Striegel-Moore et al., 2001). BN and BED do not differ dramatically in many clinical and psychological characteristics, including impulsive traits, psychiatric comorbidity and frequency of bingeing (Santonastaso et al., 1999), but they differ in treatment outcomes, as people with BED report greater improvement (APA, 2013).

Another difference found among the ED subtypes is the presence of traits of autism in AN, such as rigidity (Baron-Cohen, 2013; Odent, 2010), especially in the restrictive type. This could explain the difference observed in eating patterns between the restrictive and binge/purge types of AN: the AN-R eating pattern is stricter and more regular (Elran-Barak et al., 2014). Moreover, obsessive-compulsive personality disorder (OCD) is more prevalent in AN-R, and borderline personality disorder (BPD) is more prevalent in AN-B/P (Sansone & Sansone, 2011), further explaining the eating patterns of the two AN subtypes. Another difference between the AN types is that neurocognitive impairments are more severe in the AN-B/P than in AN-R (Tamiya, 2018). Reviews also indicate that there are gustatory differences among AN, BN and BED (Chao et al., 2020). Despite the differences in terms of symptoms' manifestation and severity, there exist certain psychological characteristics that are present in ED patients regardless of the diagnostic subtype.

2.2.7. Different Perspectives on Eating Disorders' Classification

Even though the official classification of mental disorders used in research, including EDs, is the categorical one offered by classification manuals, such as the DSM-V (APA, 2013), there has been much empirical debate and discussion of this categorical approach with some proposing a shift from the current categorical descriptive approach towards a more aetiological dimensional approach (e.g. First, 2017; Kendler & Solomon, 2016), something adopted by the current thesis studies. Clinicians need cut-off points (categories) to decide who warrants treatment, the type of therapy and whether the treatment is given as an inpatient or outpatient, especially in primary care settings (Klein et al., 2021). On the other hand, advocates of the dimensional models argue that these models are valuable for understanding core psychological factors and the underlying mechanism of a disorder (Goldberg, 2000; Stein, 2012), particularly in terms of the degree of severity rather than the presence or absence of dimensions (Luo et al., 2016). In reality, both approaches have been employed in quantitative research as the vast majority of the instruments used to measure psychopathology can be administered to non-clinical populations to assess the severity of traits' specific dimensions, such as ED traits, alongside cut-off points to determine the presence or absence of a diagnosis. This thesis posits that once an ED develops, there are noticeable differences in the subtypes (see Section 2.2.6) with specific symptoms and associated behaviours that require different handling. Nonetheless, the psychological mechanism behind the development and maintenance is the same for all EDs. In other words, the causal and perpetuating factors can be explained and therefore empirically explored dimensionally, and categories (i.e. ED subtypes) are required to understand and address the manifested symptoms in clinical practice. Therefore, the use of both the dimensional and categorical systems for different reasons (e.g. in research and practice, respectively) is more appropriate than a debate. This position could be extended to other psychiatric disorders, whereby a

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dimensional approach enables the understanding of the development of child to adult psychopathology, but once the psychiatric disorder is formed, we need presenting symptoms associated with that disorder to address specific symptoms and behaviours and to modify treatment. For some disorders, there is more support for a dimensional approach than others, such as personality disorders, for which the DSM-V (APA, 2013) includes a dimensional approach in terms of further consideration and future research (Regier et al., 2013). With respect to eating pathology, the dimensional versus categorical debate has not led to sufficient conclusions to disregard categories, and the core argument of the dimensional approach has been the observed diagnostic migration of ED subtypes, which, however, has not been the case for all ED patients (e.g. Eddy et al., 2008). The next paragraph presents the different proposed classifications of EDs, their limitations and inconsistent findings that result in the suggestion of a transdiagnostic perspective regarding common causal and maintaining risk factors rather than the absolute rejection of the subtypes.

It has been found that EDs that include binge/purge behaviour are discontinuous with normality, while those with restrictive behaviour are continuous with normality and thus dimensional in nature (Williamson et al., 2005). A possible explanation for this result could be that dieting is a behaviour found in the general population more so than bingeing/purging. Studies dealing with the dimensional aspects of EDs have also concluded that binge eating does not occur on a continuum with AN-R (Gleaves et al., 2000a; Williamson et al., 2005), suggesting a difference between restrictive and binge/purge presentations within the same ED subtype. However, this result favours differentiating between the AN-R and AN-B/P types in the categorical system. In addition, studies using both BN patients and non-clinical ED populations challenged the dimensional approach for BN (Gleaves et al., 2000b). Other studies employing general populations concluded that continuous measures produced dimensional results in the full sample but favoured more categorical models in extreme

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subgroup comparisons (Luo et al., 2016). Taken together, these results indicate that EDs can be conceptualised categorically or dimensionally, depending on the study's aims (Williamson et al., 2005).

Other authors have proposed classification based on three dimensional approaches, such as one approach to model the key dimensions of eating pathology, one approach to model the dimensions of comorbid psychopathology and one approach to model the neurological dimensions (e.g. the impulsivity and compulsivity dimensions) (Wildes & Marcus, 2013a, b). It has been proposed that the difference between the restrictive and binge/purge AN types, as discussed above (Williamson et al., 2005), could be attributed to comorbidity with a personality disorder. Authors suggest that OCD might partially account for restrictive eating pathology and BPD might partially account for binge/purge (impulsive) eating pathology (Sansone & Sansone, 2011). These authors indicate that OCD is the most common personality disorder found in AN-R, and BPD is the most common personality disorder observed in BN and AN-B/P and that their prevalence rates are higher in EDs compared to the general population (Sansone & Sansone, 2011). However, BED was not considered in this literature review, and these personality disorders have not been found in all ED patients. Furthermore, a psychiatric comorbidity comparison between Greek and French AN patients found that the most common personality disorder was BPD followed by OCD and avoidant personality disorder, with no significant differences uncovered between the two ethnic groups (Kountza et al., 2018). This study adds another personality disorder (avoidant) to AN, and diagnostic comorbidity can be found in a number of psychiatric conditions, not just in EDs (APA, 2013). Comorbidity is helpful for clinicians in terms of making an accurate differential diagnosis to eliminate mistakes and design appropriate individual treatments, rather than as a tool to define categories.

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Furthermore, findings from therapeutic studies do not support the over-controlled (OCD characteristics) and under-controlled (BPD characteristics) distinction in EDs. For instance, based on the under-controlled and over-controlled perspective of EDs, radically open DBT (RO-DBT) was developed for adults with AN (Lynch et al., 2013) offering promising therapeutic outcomes (Ben-Porath et al., 2020; Lynch et al., 2013). Lynch and colleagues (2013) based the RO-DBT design on the fact that even though DBT (Linehan, 1993) was found to be effective for BN and BED patients (Safer et al., 2001; Telch et al., 2001), thus offering support in the under-controlled (binge/purge) EDs perspective, there were no relevant studies regarding AN. Considering the theory behind DBT (Linehan, 1993), patients with under-controlled EDs have often experienced non-physical abuse and are characterised by emotion dysregulation. Although a DBT-based approach improves ED symptomatology in AN-R (Lynch et al., 2013), which is an over-controlled mental entity (Lynch et al., 2015), this does not support the restrictive (over-controlled) and binge/purge (under-controlled) dimensional classification, as one would expect that only AN binge/purge type would benefit from this intervention. What these studies do support, however, is a transdiagnostic approach regarding ED risk factors. That RO-DBT, which is transdiagnostic in nature (Lynch et al., 2013), links early non-physical abuse, emotion regulation skills and, most notably, emotional expression with the formation of social bonds, highlights the link between emotion regulation, attachment and the invalidating environment in over-controlled EDs (AN-R) as well as under-controlled EDs (AN-B/P, BN, BED). Additionally, the invalidating environment and its impact on emotion regulation has been used earlier in literature in a transdiagnostic way to explain emotion dysregulation in EDs (Fox & Power, 2009). Studies adopting the transdiagnostic approach indicate that characteristics of the invalidating environment, such as parental conflict, criticism from parents, comments about eating and weight while growing up, are associated with lifetime ED behaviours (Wade et al.,

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2006). A further interpretation of this finding is that non-physical abuse is a common factor influencing lifetime ED behaviours (this interpretation is discussed in depth in Chapter 3). This again highlights the fact that there are common psychological factors associated with the onset and maintenance of EDs, regardless of the subtype, and highlights the value of empirically examining ED subtypes.

The transdiagnostic nature of EDs, such as BN and AN, is supported by leading researchers and empirical findings in the field (Fairburn et al., 2003), which focus on the diagnostic crossover between AN and BN. In support of the diagnostic flux of the transdiagnostic model, studies have suggested that stability in ED diagnoses is low and that patients have migrated between AN and BN (Fairburn & Harrison, 2003; Milos et al., 2005). However, BED was not considered in the studies despite being a provisional category in the DSM-IV at the time of this cited research and researchers worldwide were using the BED diagnosis in their studies with their results leading to BED's acknowledgement in DSM-V. Contrary to the AN and BN migration results, other studies found a low crossover between AN and BN and differences in treatment outcome, supporting their diagnostic distinction (Eddy et al., 2008; van Son et al., 2010). Additionally, the findings that support diagnostic migrations (Fairburn & Harrison, 2003; Milos et al., 2005) do not explain why some patients shifted between subtypes and others did not. For instance, it could have been of explanatory significance if these studies attempted identifying the presence of comorbid BPD or at least borderline features in these patients to see if this was the contributing factor to diagnostic movement. Review studies have found that BPD or its traits can be found in AN and BN (Kelly & Davies, 2019; Torres Pérez et al., 2008). Given their personality structure, people with BPD experience a chaotic way of living, impulsive behaviour (e.g. binge eating), instability in their personal relationships and beliefs, and fluctuating self-esteem and self-boundaries (Vaslamatzis et al., 2004). It is expected, therefore, that individuals with BPD

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might experience instability in their eating behaviour. In other words, diagnostic migration is common to ED patients, particularly between AN and BN, due in part to underlying comorbid BPD (Zanarini et al., 2010). In further support of this argument, other longitudinal studies on ED symptom shifting have demonstrated that people who displayed symptom movement were more prone to engage in deliberate self-harm and shifted to both deliberate self-harm and substance use over time (Garke et al., 2019). This further indicates that the instability that characterises BPD could explain diagnostic flux rather than the argument that EDs do not have a diagnostic distinction.

Another possible reason for migration in AN patients could be the presence of autistic traits, which have been associated with a shift towards orthorexia (Herzog et al., 1992). Moreover, it is expected that in people undergoing treatment, some symptoms may slightly change due to the psychotherapeutic process, and this could be over-attributed to a diagnostic crossover. However, Milos and colleagues' findings (2005) regarding low overall remission led them to suggest that ED patients probably share common psychological causal and maintenance factors. Other studies that did consider BED found a diagnostic flux, mostly between BN and BED, while AN in this study seemed relatively stable (Fichter & Quadflieg, 2007). That said, in this study, BPD features were not researched as a possible factor for diagnostic migration. Moreover, the diagnostic flux found between BN and BED contradicts the finding that BN and BED are different (Fairburn et al., 1998; Fairburn et al., 2000). Also, AN being stable contradicts the findings of AN and BN diagnostic migration (Fairburn & Harrison, 2003; Milos et al., 2005). These authors also concluded that there could be common psychological maintenance factors in BN and BED (Fichter & Quadflieg, 2007).

In summary, it is evident that the dimensional approach to EDs, regardless of the way these dimensions are formed (e.g. diagnostic flux, over-controlled/under-controlled), is not

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strong enough to abolish diagnostic categories. Both the dimensional and categorical approaches have been characterised as simplistic (Williamson et al., 2005), and the proposal that both should be considered in clinical practice (Castellini et al., 2011; Kessler, 2002 as cited in Wade et al., 2006) is valid. Additionally, the disagreement in ED treatment with the existence of different psychotherapy models and different results for each subtype favours diagnostic distinction, which simultaneously indicates that there is something missing from therapy, such as the identification and target of common causal and maintenance factors. A significant contribution of the studies dealing with the dimensional classification of EDs is the identification of common causal factors, and at this point, it could be stated that it is the common causal and maintenance factors that give EDs their transdiagnostic character. The hypothesis of common psychological causal onset and maintenance factors aligns with the perspective of this thesis, which adopts a transdiagnostic perspective regarding the factors of eating pathology onset and maintenance upon which the studies were developed. That is, the clinical population of this thesis's studies consisted of AN, BN and BED patients, who formed the ED group, because the research aimed to identify common risk factors regarding the development and maintenance of EDs. This thesis's studies were designed to agree with the proposed concept of a transdiagnostic underlying mechanism in mental health development (Dalglish et al., 2020). However, this thesis acknowledges the differences between the subtypes, as stated in Section 2.2.6, maintaining the position that future research is required in the field of EDs to identify the factors that lead to the development of a specific subtype instead of another. Such research would benefit from the use of ED subtypes in the research design.

2.3. Aetiology of Eating Disorders

During the past two decades, thorough research has been generated in the field of EDs regarding their prevalence, possible aetiology and risk factors, prevention and treatment. EDs used to be viewed as a Western culture disorder, emphasising the role of socio-cultural factors (Nasser, 1988), but this notion is no longer valid as they are evidently present in non-Western cultures as well, indicating a global phenomenon (Chang et al., 2015; Nasser, 2009; Soyoung, 2018). This could be attributed to the fact that societies are growing economically at a fast pace, and there is access to other cultures through travel and the media, especially via the internet (Chang et al., 2015; Soh & Walter, 2013). This could be also attributed to a misconception in the literature due to methodological limitations (e.g. translation of screening tools from English into other languages) that could not allow data for comparison. There is also the belief that Mediterranean families emphasise food as a means of bonding and socialising, and much of the research has focused on dietary habits in these countries rather than on developmental factors with respect to EDs (Janicic & Bairaktari, 2014). However, studies indicate that ethnic identity is unrelated to ED symptomatology (Bhargova, 2007). This suggests that other factors related to human development are more important than the country of origin and societal messages about thinness. In conclusion, several theories have been developed over the years to explain the onset of eating pathology.

2.3.1. Theories and Models of Eating Disorders

A detailed description of ED theories is beyond the scope of this thesis. For the present purposes, the summary below is a brief description of the dominant theoretical approaches and some of the leading empirically supported models.

Psychodynamic theory: Psychodynamic theorists hold the position that EDs are the result of the disruption of self-concept, which is manifested as a *false self* (Bruch, 1973;

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Goodsitt, 1997; Strober, 1991). The concept of the false self (Winnicott, 1965) involves having an idealised public self that is the result of withdrawal from the aspects of the true self that could be considered unacceptable if displayed (anger, trauma, vulnerability, negative emotions). The public false self that needs to always be perfect forms 'idealised' relationships with significant others, leading to feelings of loneliness and disappointment as the true 'not so perfect' self suppresses its real needs and expressions causing severe distress. The individual becomes a self-object in their effort to please others, negating their true self and own needs (Goodsitt, 1997). Bruch (1973) proposed that families of ED patients display the same characteristics of Winnicott's (1965) split of the true and false selves. Even though this is also observed in other mental disorders, it is believed that the distinctive characteristic between ED and non-ED disorders is that parents of people with ED emphasise food and external appearance (Jones, 1985). That is, when parents are too involved in their child's eating behaviour and simultaneously disallow their child's expression of inner needs, they may provoke emotional relief of the child's true self through food. If, at the same time, parents emphasise the child's external appearance, then the child will view food both as a friend and an enemy (Jones 1985). Criticising body appearance can also lead to shame about one's physical looks (Goss & Allan, 2009), which lowers self-esteem, and diet may come along in order to restore it. The false self is the result of insecure attachment that fails to create a holding environment (Winnicott, 1965) in order to contain the individual's first feelings and experiences of the world, thus affecting social cognition and self-esteem. Attachment is closely related to family, as it involves emotionally bonding with primary caregivers. Thus, EDs are the coping mechanisms of the false self. This theory addresses the disruption of the development of the self, and if extended, it theoretically connects the invalidating environment with attachment, emotion regulation and self-esteem, but it has not been researched in a concise developmental model in ED patients.

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Family dynamics theory: Family dynamics have been extensively researched to understand the onset and development of EDs, especially in women with AN. Family therapists emphasise dynamic issues, like family enmeshment, assuming that ED patients' symptoms are expressions of underlying and unresolved family conflicts (Bruch, 1973, 1982; McDermott et al., 2002; Minuchin et al., 1975). Family enmeshment refers to a dysfunctional familial environment in which family members display unhealthy behaviour patterns and rigid roles, and each individual is enmeshed with the other family members, leading to suppressed individuality and unhealthy relationships (Minuchin et al., 1975). Bruch (1973) noted that AN was more frequent in girls who were 'too good' as daughters and who tried to please their parents and acquaintances by being 'perfect' in everything they did. These girls had parents who overinvested in their daughters' compliance and achievements (school, sports). They were over-controlling and did not allow the expression of negative feelings. These families have been characterised as enmeshed families or psychosomatic families in which interactions are characterised by interdependence and intensity, and the boundaries of individual family members' identities are weak and easily crossed (Minuchin et al., 1978). Parents are trying to satisfy their own needs through controlling their daughters, and their daughters do not learn to identify and accept their own feelings, needs and desires (Bruch, 1973). Even though the biggest part of this theory focuses on the role and behaviour of the mother, recent findings suggest that the paternal authoritarian parenting style has been linked to ED symptomatology (Enten & Golan, 2009), that there is a direct link between fathers' parenting and ED symptoms (McEwen & Flouri, 2009) and that paternal maladaptive behaviour is more influential for the disorder's development than the maternal one (Johnson et al., 2002). The contribution of family dynamics in EDs has been supported by research, despite criticism of Minuchin's model for blaming the family (Le Grange et al., 2010). However, there could be other factors involved in the relationship between family and EDs,

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and family dysfunctional dynamics should be reconceptualised into a more specific term, such as childhood non-physical abuse (Study 2).

Cognitive-behavioural theory: This theory proposes that eating pathology development is due to a perceived ineffectiveness in other areas of functioning, thus turning to the body as the only successful experience (Fairburn et al., 1998). From this perspective, four maintaining mechanisms have been identified that are believed to underpin EDs (Fairburn et al., 2003): a) clinical perfectionism, which means that someone has excessively high standards for themselves; b) interpersonal difficulties, like conflicting family dynamics or a pattern of failed relationships; c) mood intolerance, which refers to the difficulty of managing strong emotions and mood alterations and d) pervasive low self-esteem. All of these maintaining mechanisms have been found to relate to EDs, as well as the concept of the false self (Patterson, 2008). Even though this model refers to self-esteem, emotion regulation and attachment (implicitly) as maintaining factors, it does not directly link onset and maintenance with the aetiology of these factors' impairment. Reviews of the cognitive-behavioural model indicate that not all factors proposed are relevant for all EDs, but low self-esteem and mood intolerance are relevant for all ED subtypes and should be considered in treatment (Lampard et al., 2013).

Transdiagnostic model of EDs: Based on the abovementioned cognitive-behavioural model of EDs, the transdiagnostic model proposes that the basic elements of eating pathology are the same across the diagnostic categories as they all emphasise control of shape, weight and food intake (Fairburn et al., 2003). Based on this, enhanced CBT (CBT-E) has been developed to treat ED patients regardless their subtype. As a result, this model is not in line with the DSM-V as it does not distinguish between clinical states (AN, BN, BED). The focus of this model is placed upon AN and BN, and the authors' basic argument is that body

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dissatisfaction plays a significant maintaining role in both disorders. An interpretation of this argument, however, could be that self-esteem is the transdiagnostic factor here as body dissatisfaction is connected with low self-esteem. This interpretation could be extended even further by stating that a high self-esteem and body dissatisfaction (at least to the degree of taking psychopathological measures to deal with it) do not belong in the same sentence. In other words, body dissatisfaction is the product of low self-esteem. In support of this argument, it has been suggested that low self-esteem leads to weight and shape control in order to increase self-worth (Fairburn et al., 2008; Fairburn et al., 2009).

Fairburn et al. (2003) also argue that binge eating does not distinguish between AN and BN as some AN patients display binge/purge behaviour. However, these binge/purge episodes differ significantly in size and perception as AN patients think that they binge by consuming a small portion of food. Moreover, studies on AN and BN display differences in outcome and low crossover, supporting their diagnostic distinction (van Son et al., 2010). In addition, the argument of binge eating not differentiating AN and BED is inconsistent with the same authors' finding that BED and BN differ in their risk factor profiles (Fairburn et al., 1998) and in their natural course and outcome (Fairburn et al., 2000). It is inconsistent, as one would expect that if binge eating cannot be used to differentiate AN and BN, then it certainly could not differentiate between BED and BN as their binge eating episodes have more similarities than those between AN-B/P and BN. Overall, the transdiagnostic model does not consider the reported and observed differences between the subtypes (see Section 2.2.6) and also ignores the fact that different therapeutic interventions have been developed and proposed for each subtype over the years, meaning that a single treatment did not work for all ED patients and the subtypes could not be disregarded. Moreover, the failure of standard treatment in difficult ED cases and on cases of people suffering from BPD along with an ED has led to the inclusion of DBT in ED treatment and to the development of different models

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that work differently for each subtype (Ben-Porath et al., 2020). In other words, if the transdiagnostic model was valid and the subtypes do not exist, then CBT-E would be able to successfully treat all EDs, but it is not. The transdiagnostic model has been criticised for focusing on a narrow range of clinical presentations (Dalglish et al., 2020).

The relational–cultural theory of psychological development: Relational–cultural theory (RCT) (Jordan, 2000) emphasises affirming and growth-promoting interpersonal relationships over the course of a lifetime as important to healthy human development. It originates from the self-in-relation theory, which maintains that women experience conflict between their gender-expected role and their personal needs (Surrey, 1991). Basic qualities of relational health (Liang et al., 2002) include empathy, engagement, authenticity, diversity (the ability to integrate differences) and zest (the energising experience of genuine connection), indicating the importance of relationships and social support in people’s lives on their well-being.

RCT relates to ToM (Premack & Woodruff, 1978) and social cognition. ToM refers to the part of social cognition that involves the ability to understand others’ mental states, as well as one’s own, and effectively interpret and make inferences regarding their intentions and beliefs in social situations. Social cognition refers to the set of mental operations underlying social interactions, which include perception, interpretation and the generation of responses to the intentions, dispositions and behaviours of other people (Ostrom, 1984). In relation to this, people with AN have been found to display difficulties in effective ToM independent of their clinical status (Tapajóz et al., 2013). This theory implies attachment and emotion regulation as factors associated with EDs, leaving out self-esteem as well as why these factors become impaired.

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The socio-cultural model of ED development: This theory posits that exposure to the Western thin ideal concept in economically developed industrialised societies leads to body dissatisfaction and, consequently, to dietary restraint. The thin ideal factor has been highly associated with body image concerns affecting young women's decisions to start dieting (Putterman & Linden, 2004). The thin ideal refers to the concept of a very slim female body figure with a small waist and little body fat (Brehm et al, 2002), creating a weight prejudice that is reinforced both by mass media and society (Spettigue & Henderson, 2004; Tiggemann et al., 2009), leading scientists to target it in ED prevention programmes (Stice et al., 2013). Thinness is associated with success and social desirability, prompting many women to view their body shape as a measure of social value (Calado et al., 2011; Van Vonderen & Kinally, 2012). Western culture idealises thinness in such an extensive way that women often acquire a fixed idea of an 'acceptable' body image, internalising this beauty standard (McCarthy, 1990). Pencil-thin models are constantly presented by the media as the epitome of beauty, and women's magazines have been found to host ten times more dieting articles than men's magazines (Sarafino, 2002). Studies regarding the thin ideal in female children have concluded that their exposure to Barbie dolls may have a socio-cultural impact on their body image and their desire to be thinner, indicating that the thin ideal internalisation begins at an early age (Brehm et al., 2002; Dittmar et al., 2006). Alarming research results demonstrate that the thin ideal concept is present in girls as young as three years old, but the exact process of internalisation is still unclear (Harriger et al., 2010). In line with the above, Stice (2001) proposed the dual pathway model of BN suggesting that societal pressure, along with the thin ideal internalisation, may lead to body dissatisfaction, which in turn predicts dieting and negative effects resulting in bulimic symptoms. However, in this theory, it is not explained why some women are more prone to the thin ideal internalisation than others, indicating that other factors may play a contributing role to this difference (e.g. low self-

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esteem). Nevertheless, the socio-cultural model could explain the gender differences observed in the prevalence of EDs discussed earlier in Section 2.2.

The socio-cultural theory could be linked to the feminist approach of objectification theory, which suggests that in Western societies, the female body is inspected, evaluated, and linked to sexual pleasure (Fredrickson & Roberts, 1997; Bordo, 1993). In this theory, females adopt a self-objectifying view of themselves as objects to be evaluated on the basis of their physical appearance. However, there are studies indicating that the thin ideal concept affects developing societies and non-Western cultures as well (Chang et al., 2015; Vander Wal et al., 2008). Researchers have tried connecting the objectification theory with the allocentric lock hypothesis (Gaudio & Riva, 2013; Riva, 2012; Riva et al., 2013; Riva & Gaudio, 2012), which proposes that EDs have, as an antecedent, an allocentric (i.e. objective, from outside) negative body image that cannot be updated by the egocentric (i.e. from inside) sensory input from perception. This means that even after a demanding diet and severe, obvious weight loss, the sensory inputs are not updated because ED patients are locked to an allocentric negative representation of their body. These researchers therefore believe that the internalisation of an objectified image can be explained through spatial cognition, suggesting that women remember their body from the perspective of the observer using an allocentric frame of reference instead of the egocentric one (Riva et al., 2015). Even though this theory is a respected effort to explain the internalisation of the objectified self, it does not provide an explanation as to why this process occurs in the first place, as the majority of the women in Western societies do not suffer from an ED.

Theory on emotional abuse and EDs: Waller and colleagues (2007) discussed the clinical links between childhood emotional abuse (CEA) and EDs by outlining how CEA can result in dysfunctional levels of cognition, which in turn result in dysfunctional emotional

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functioning and lead to maladaptive coping strategies (EDs). They proposed that CEA results in two types of beliefs: a) conditional assumptions about the acceptability of expressing one's emotions, such as 'If I display my anger, I will suffer negative consequences', and b) negative core beliefs about the self, such as 'I am not worthy enough to be loved'. The first belief relates to emotion regulation and the second one to self-esteem. These difficulties result in the use of protective chaotic-dissociative and/or detached-alexithymic coping strategies, which in turn predispose the individual to employ unhealthy eating behaviours. Waller and associates (2007) added that some patients may swing between the two maladaptive coping strategies, and they underlined that in some cases, such as AN-B/P patients, the affective instability and compulsive behaviours are so intense that self-injury behaviour (SIB) may also be present (Farber, 2008). Considering the above, it is suggested here that non-physical abuse may lead to eating pathology by affecting self-esteem and emotion regulation. This ED theory of emotional abuse leading to emotional dysfunction and, consequently, to maladaptive coping strategies has similarities with the biosocial theory (Linehan, 1993) regarding the psychological mechanism of abuse and could be linked to the invalidating environment. Indeed, it has been demonstrated that with theoretical and practical modifications, Linehan's (1993) DBT could improve the treatment effectiveness of ED patients (Brown et al., 2019; Kenny et al., 2019; Wisniewski & Kelly, 2003) as well as their QoL (Rahmani & Omid, 2019). However, this model does not consider attachment even though it is developmentally connected with self-esteem and emotion regulation. It also fails to consider emotional and physical neglect, and even though a link with the invalidating environment is revealed, it is not complete without all the forms of non-physical abuse in the equation. Study 2 was designed to address these limitations.

Theories of emotion regulation and EDs: The cognitive-affective regulation model (Corstorphine, 2006) is based on Linehan's DBT (1993) and focuses on the patient's

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subjective understanding of their emotional states. Corstorphine (2006) posits that current ED treatments have been unsuccessful for some groups of ED patients, notably those with BPD, a reported history of trauma, impulsivity and compulsivity. These patients experience high emotional vulnerability along with an inability to regulate their emotions. The key triggers for their eating behaviour are their emotions because emotion dysregulation is their basic deficit. This model also maintains that growing up in an invalidating environment where emotions and experiences are disregarded by caregivers eventually causes the individual to believe that emotions are bad and dangerous and should not be experienced nor expressed. When primary emotions, like anger, are experienced the individual perceives them as unacceptable and tries to suppress them through restricting or bingeing. Consequently, secondary emotions are triggered, like guilt and shame, due to the experience of the primary emotion, and this vicious cycle escalates distress that the person cannot cope with and feels the need to employ ED behaviours. According to the cognitive-affective regulation model, it is not the emotion itself that causes the problem but the interpretation of this emotion by the individual experiencing it. This interpretation will affect the level of distress and the coping strategies. This model, even though based upon Linehan's (1993) invalidating environment concept, does not take a step further to include attachment and self-esteem, which are also linked with emotion regulation, and they are all core developmental consequences of an invalidating environment.

The schematic propositional analogical associative representation (SPAARS) cognitive model of emotion (Fox & Power, 2009; Power & Dalgleish, 1997, 2008): This model proposes that messages conveyed at an early age regarding the unacceptability of emotions suppress their normal development and dissociates them from the rest of the individual's developmental processes. Consequently, the individual perceives the dissociated emotion as intolerable distress and attempts to maintain this dissociation for self-protection. This model has been extended and applied to EDs (Fox & Power, 2009), suggesting that

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anger and disgust are important emotions in EDs and the disorder serves as a barrier against these emotions to protect the self. As this model is considered relevant to this thesis it will be further discussed in Chapter 3.

The EDs continuum: Among the relevant studies in the field, researchers have also focused on the EDs continuum mainly to detect which variables differentiate people with an ED from those without one. First proposed by Nylander (1971), the concept of the EDs continuum is represented by a continuum of ED symptomatology ranging in severity from chronic dieting at one end of the spectrum to a clinically diagnosed ED at the other. Mintz and Betz (1988) expanded this model by suggesting that eating disturbances occur along a continuum composed of three groups (asymptomatic, symptomatic and eating disordered). The symptomatic group displays milder or partial forms of disordered eating without meeting the criteria in full for the disorder (Tylka & Subich, 1999). This continuum suggests that the disorder occurs when people display extreme manifestations of common behavioural attitudinal and psychological dimensions and these group differences are a matter of degree and not of kind (Scarano & Kalonder-Martin, 1994). Researchers have noted that many women indicate that they engage in disordered eating at a subclinical level (Mintz & Betz, 1988), displaying significant distress and impairment (Hertzog et al., 1993), which places them at risk of fully developing the disorder (Franko & Omori, 1999). Even though this model could be considered valuable regarding prevention, research in this domain has focused on the similarities and differences among the groups rather than focusing on specific factors regarding the onset and maintenance of eating pathology. Studies 1 and 2 were designed to compare clinical and healthy populations regarding the mechanism of risk factors, thus enabling both treatment and prevention designs.

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The multidimensional model of EDs: This model is divided into three factors: predisposing (psychological, biological, familial, cultural), precipitating (dieting) and perpetuating (the psychological and physical effects of starvation) (Garner, 1993). The model proposes that the interplay of these factors is involved in the onset and maintenance of ED symptomatology. The model also underlines that starvation leads to a deterioration in mood and increases preoccupation with food (Garner, 1993). However, this model does not explain the preoccupation with food in BN and BED, as the ED behaviours of these subtypes do not involve starvation. Even though this model includes almost all the identified risk factors for EDs, it does not explain how they work together to lead to eating pathology and how they become impaired.

The trans-theoretical model of EDs: The Maudsley model of anorexia nervosa treatment based on the cognitive-interpersonal model of AN (Schmidt & Treasure, 2006, 2013; Treasure et al., 2020) refers to four maintaining factors (Schmidt et al., 2013; Schmidt et al., 2014). The first factor is inflexible thinking style, which is related to the fear of making mistakes and extreme focus on details. The second factor has to do with difficulties in emotion regulation. The third factor deals with problems in interpersonal relationships marked by difficulties in proper emotional responses to other people and the dominant presence of negative emotions, such as shame, guilt and fear. The last factor is the belief that anorexia will help the individual overcome the abovementioned difficulties in order to function in everyday life, offering a sense of safety. Starvation is perceived as a reinforcing and supporting element of the four factors. This model offers a deeper understanding of AN, but it does not equally consider BN and BED and their common psychological factors in terms of origin and maintenance, such as low self-esteem. Additionally, it focuses on alexithymia, overlooking earlier studies on the development of poor meta-emotional skills in AN and the presence of specific emotions (Fox, 2009).

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Summary: Reviews of the main theoretical models indicate that despite the existence of many theoretical models, the question of the causal and maintenance factors in EDs is still open (Brytek-Matera & Czepczor, 2017; Rikani et al., 2013). The integration of theories and the use of complementary interventions could offer better results in the understanding and managing of such a complex psychopathological entity. When considering the main theories and models surrounding the development of EDs, one notices how they overlap and/or link in many ways and if they were combined, they could offer a more complete explanation. For example, a dysfunctional family (family dynamics theory) could lead to insecure attachment through devaluation of the child and detachment from its needs (theories of emotional abuse and neglect). The child will constantly try to please others in order to offset the risk of abandonment and rejection, making the individual prone to the internalisation of the thin ideal, which is linked to objectification in the presence of low self-esteem. Insecure attachment will not create a holding environment; thus, it will damage the true self (self-esteem), emotion regulation and the formation of effective relationships with significant others (social support), as well as the relational health qualities (e.g. empathy, engagement) and social cognition abilities. This could lead to the development of maladaptive coping strategies, like EDs.

All these theories provide a plausible explanation for the development of eating pathology, but since they complement each other, it would be of further explanatory validity to combine them in a comprehensive theoretical model that includes all the major developmental factors associated with EDs along with reasons for these factors' impairment. A number of psychological risk factors have been identified leading to the development of separate models for EDs. Although many risk factors have been highlighted and are common to many models created to explain the onset and maintenance of EDs, the previously mentioned theories include some factors disregarding others. Even though many of the

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theories come close to an explanation, there is always the sense that something is missing. Authors point out that researchers should change the way they think about the aetiology of EDs and pay attention to the fact that certain identified risk factors are also concurrent symptoms of EDs (Rikani et al., 2013). This argument is in line with this thesis's view that the factors associated with the onset of eating pathology are also responsible for its maintenance. This thesis proposes that these factors are attachment, emotion regulation and self-esteem, which are discussed in Chapter 3.

2.4. Quality of Life and Eating Disorders

2.4.1. Quality of Life

The World Health Organisation (WHO, 1997) defines QoL hermeneutically as 'individuals' perception of their position in life in the context of the culture and value systems in which they live and in relation to their goals, expectations, standards and concerns'. QoL is perceived as a multidimensional concept measured by both objective and subjective indicators because it measures the person's ability to function physically, psychologically and socially within their environment according to their expectations. Consequently, QoL is influenced by a variety of associated domains that conjoin physical health with psychosocial functioning (Abraham et al., 2006) and is viewed as a basic need in all health-related professions (Asadi-Lari et al., 2004) and as an outcome variable in therapy (Pollack et al., 2015).

The theoretical underpinnings of QoL are rooted in ancient Greek philosophy, specifically in Aristotle's (384–322 BC) concept of *eudemonia*, which is the call on people to realise their full capabilities in order to achieve a 'good life', meaning happiness, life satisfaction and well-being (Diener & Suh, 1997; Tountas, 2009). QoL is the measure of choice by health agencies worldwide and has been characterised as the ultimate goal of all

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health interventions and evaluations (Bowling, 2001; Rubin & Peyrot, 1999). When health professionals include QoL improvement in patients' treatment there is a positive influence in the course of the illness, as well as in the doctor–patient relationship (Cochran & Conn, 2008; Debono & Cachia, 2007; Zoffmann & Kirkevold, 2012).

2.4.2. Quality of Life in Eating Disorders

Empirical findings and review studies indicate that ED patients report significantly lower QoL than non-ED patients, mood disorder patients (Bamford & Sly, 2010; Hay & Mond, 2005; de la Rie et al., 2007; de la Rie et al., 2005) and people with a chronic somatic illness (Jenkins et al., 2011), with no differences between the ED subtypes (González et al., 2001; de la Rie et al., 2005). Longitudinal studies demonstrate that ED patients maintain low QoL even after years of treatment (Padierna et al., 2002). Until recently, ED treatment outcome studies have focused on the physiological symptoms of EDs with respect to QoL, rather than the psychosocial impact (Adair et al., 2010; Bamford & Sly, 2010; Pohjolainen et al., 2016).

EDs can have a serious impact on various life domains leading to physical, psychological and social impairment, which in turn will lead to poor QoL (Ura & Preston, 2015). These domains are considered to be key concepts of generic QoL and are included in the subscales of the WHO instruments (WHOQOL Group, 1995). Regarding the physical domain of QoL, disturbed eating behaviour leads to physical impairment through serious medical complications resulting from laxative misuse, starvation and self-induced vomiting (Mehler & Brown, 2015; Mehler & Rylander, 2015; Wassenaar et al., 2019). Regarding the psychological domain of QoL, ED patients display low self-esteem, body dissatisfaction and a variety of comorbid disorders, such as affective disorders, cluster B personality disorders, anxiety disorders and substance-use disorders (Nolen-Hoeksema, 2003; Ura & Preston,

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2015). People suffering from an ED use food to cope with painful emotions and stressful situations and typically have a constant need for others' approval (Corstrophine, 2006). They also display low tolerance to stress, perfectionism, rigidity in evaluating themselves, a dichotomous thinking style and an effort to please others setting aside their own needs and wishes (Polivy & Herman, 2002). The social QoL impairment in ED patients involves occupational and educational impairment, interpersonal problems, difficulties in social relationships and social adjustment and disrupted familial relationships (de la Rie et al., 2005; Ura & Preston, 2015). Social relationships are also affected by the stigma commonly associated with EDs (Mond et al., 2006). The physical, psychological and social impairment in ED patients is long-lasting since EDs are chronic disorders, thus affecting all domains of patients' QoL, even after symptom remission (Pohjolaine et al., 2016).

Several studies have tried to explain the relationship between EDs and QoL, but it is still unclear how specific ED symptoms and associated behaviours impact QoL (DeJong et al., 2013; Mason et al., 2018). Moreover, the results of these studies are inconsistent, and the majority measured health-related QoL. For example, ED symptoms and ED-related factors, such as low BMI and bulimic eating, have been associated with poor QoL (Bamford & Sly, 2010; Hay, 2003; Padierna et al., 2000). However, other studies report that BMI, illness duration and frequency of symptoms were not associated with QoL (DeJong et al., 2013). In the same vein, Bamford and Sly (2010) concluded that ED duration and diagnostic subtypes do not contribute to QoL. In keeping with this inconsistency, low BMI in AN affected QoL (Weigel et al., 2016), but in other studies it did not (Abbate-Daga et al., 2014). Other results indicate that symptoms' severity and chronicity in AN predict QoL, suggesting the inclusion of QoL in treatment goals (Bamford et al., 2015). Additionally, even though both types of AN report low QoL (Mason et al., 2018), AN-R patients report better QoL than those with AN-B/P (DeJong et al., 2013; Mason et al., 2018). Conversely, another study found that AN-

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R patients' psychosocial QoL is similar to that of individuals without an ED (Padierna et al., 2000). This is an indication that QoL can be affected by other factors rather than just the physical symptoms of an ED, because one would expect that AN-R patients would report worse QoL than any other group, since that diagnostic type's symptoms have the most severe health implications (Mehler & Brown, 2015). The explanation of this phenomenon relies on the ego-syntonic nature of restraint eating (Mond et al., 2006; Peroutsi & Gonidakis, 2011) for which patients feel proud and view it as a difficult accomplishment, thus making their resistance to treatment a challenge for clinicians (Gregertsen et al., 2017). In addition, AN patients' QoL has been found to be influenced by depressive symptoms, poor emotional awareness and an impaired sense of control (Kane et al., 2018). This finding suggests that QoL is influenced by emotion regulation and self-esteem, as low self-esteem can trigger depression, while, at the same time, depression can decrease self-esteem (Lynum et al., 2008; Schmitz et al., 2003). Furthermore, recent studies on AN indicate that poor resilience might offer a possible explanation for the chronicity and difficulty in treatment, which affects patients' QoL (Kane et al., 2019). Resilience is the adaptation process to trauma and to any adversity that can cause significant stress (APA, 2014). In that study, resilience was affected by self-dissatisfaction and rejection (social insecurity) (Kane et al., 2019). This means that resilience is affected by self-esteem and attachment, and these two factors are also linked with psychosocial QoL. In other words, addressing attachment and self-esteem in therapy would improve resilience, thus reducing chronicity and improving psychosocial QoL. Moreover, in a study in which ED patients were asked to name the most important aspects of their lives that affect their QoL (de la Rie et al., 2007), sense of belonging was the most frequently mentioned domain both by current and former ED patients, indicating that a factor of a psychosocial nature was a more important predictor of QoL than the somatic symptoms of an ED. All these indicate that the physical symptoms of EDs are not responsible for low

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QoL in ED patients, especially since QoL is not improved after symptom reduction, meaning that psychological factors are more important.

Certain studies regarding EDs and QoL are of further clinical interest. Former ED patients report almost the same levels of QoL as current ED patients, rather than their scores being closer to a normal control group's as would be expected (Bamford and Sly, 2010; Padierna et al., 2002; de la Rie et al., 2007; de la Rie et al., 2005). These scholars, in an effort to explain this result, hypothesised that although these patients have recovered from their symptoms, maybe the residual effects of ED were still present. However, it was trait self-esteem that had the highest association with QoL in current and former ED patients, leading authors to suggest that by addressing the maintaining psychological mechanism of low self-esteem in treatment, QoL will be improved where the reduction of ED symptoms has failed to improve it (de la Rie et al., 2005). Additionally, ED patients report poor QoL even when they are undergoing treatment (Mond et al., 2005). Even though QoL is used as an outcome variable in ED and non-ED studies, there is some indication of a bidirectional relationship between QoL and EDs (Mitchison et al., 2015). In this longitudinal study, ED symptoms predicted health-related QoL and psychological distress, and low QoL and greater distress predicted ED symptoms for four years. ED symptom reduction predicted QoL for a shorter period of time. This study provides an indication that poor QoL can exacerbate ED symptoms (Mitchison et al., 2015). However, the study employed a non-clinical population, and the factors maintaining low QoL and distress were not addressed or explained. Nevertheless, these results add to the importance of including QoL in treatment aims.

It is evident that perceived QoL is a subjective concept, stable enough in EDs and that does not typically change even when the ED-related factors that seem to predict QoL are no longer present. This also indicates that somatic symptoms are not strong enough on their own

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to affect the individual's global view of their QoL. These findings highlight the need for further research in order to advance understanding regarding EDs and QoL. The explanation of low QoL's stability in ED patients could rely on stable factors that affect it. ED symptoms and associated behaviours can lower QoL, but they are not stable factors as they improve with treatment. It could be hypothesised that the presence of impaired stable psychological factors, such as self-esteem, attachment and emotion regulation, may be of greater importance to QoL than ED symptoms alone. This could explain the bidirectional relationship found between EDs and QoL (Mitchison et al., 2015), meaning that these unaddressed factors maintain both the low QoL and the ED. Study 2 offers some insight into this, focusing on psychosocial QoL as an outcome variable in mediation models that include attachment, self-esteem and emotion regulation as mediators.

2.5. Summary

EDs are chronic disorders with severe medical complications and higher death rates than any other mental disorder. Their aetiology is complex and still unclear, creating difficulties in the effectiveness of prevention and treatment. As chronic disorders, they have a profound impact on psychosocial QoL even after symptom reduction, indicating that other factors might be more important for ED patients' QoL, most notably low self-esteem. These factors could be responsible for both ED onset and maintenance. Several alternative and complementary theories have been developed to explain how EDs are formed and their underlying mechanisms. The gap in the theory surrounding the development of EDs—and as a result, the identification of the core risk and maintaining factors—is due in part to the absence of an integrated theoretical model that provides a complete theoretical explanation that makes developmental sense. As seen in Chapter 1, many variables have been identified as risk factors for EDs but have usually been tested in isolation. For example, core beliefs

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about the self and body image dissatisfaction are frequently examined in relation to eating pathology, but these are aspects of self-esteem. This thesis's position and contribution to the literature is the integration of basic theories and core risk factors as a concise theoretical and clinical explanation of EDs. This chapter reviewed the outcome variables of the empirical studies (eating pathology and psychosocial QoL). Chapter 3 theoretically presents the predictor variables (family dysfunction/childhood non-physical abuse) and the mediators (self-esteem, attachment, emotion regulation, temperament) of this relationship. These variables have been observed in all ED subtypes and are used under a transdiagnostic perspective in this thesis's studies. Additionally, the similarities in psychological variables between ED and BPD patients are discussed, leading to an adaptation of the biosocial model (Linehan, 1993) regarding eating pathology. Lastly, the continuation of childhood non-physical abuse in adulthood is discussed based on the long-lasting effects of childhood traumatic experiences.

Chapter 3: Family Dysfunction/Non-physical Childhood Abuse/the Invalidating Environment and the Mediating Role of Self-esteem, Attachment, Emotion Regulation and Temperament

3.1. Overview

This chapter will present how family dysfunction and the invalidating environment can be operationalised as emotional abuse, emotional neglect and physical neglect in order to form a comprehensive psychological construct, which is non-physical childhood abuse. The necessity of focusing on these forms of abuse with respect to eating disorders (EDs) is explained in Chapter 1. The consequences of childhood non-physical abuse are then examined in relation to the eating pathology literature to identify the major risk factors involved with these disorders (Study 2a). These major risk factors identified are attachment, self-esteem and emotion regulation, and they are transdiagnostic in EDs. In addition, the similarities between ED and borderline personality disorder (BPD) patients will be discussed in the context of an adaptation of biosocial theory regarding eating pathology (Study 2b).

3.2. Family Functioning

According to the McMaster model of family functioning (Epstein et al., 1978), family functioning is a complex phenomenon that is much more related to the transactional and systematic properties of the family system than it is to individual family members' characteristics. This model posits that family functioning can be divided into six dimensions. The first dimension is problem-solving, which reflects the family's ability to effectively resolve issues within the family. The second dimension is communication, which refers to the effective exchange of information between family members, like the content and directness of verbal messages. The third dimension of roles refers to the handling of a set of family functions, such as provision of resources, support and nurturance, encouragement and

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enhancement of personal development, maintenance and management of family systems, and clear delegation of tasks among family members. The fourth and fifth dimensions of affective responsiveness and affective involvement are concerned with the ability of family members to experience and display the appropriate affect according to the stimulus and the extent to which they get involved in each other's activities and concerns, respectively. The last dimension of behaviour control refers to the way the family expresses and maintains its members' standards of behaviour in various situations. Difficulties in relation to the six dimensions have been associated with family functioning around EDs, and the model, therefore, has therapeutic value (Dimitropoulos et al., 2013; Erol et al., 2007; Ma & Lai, 2009; Waller et al., 1989). Additionally, reviews indicate that areas identified by the McMaster model (Epstein et al., 1978) showed high relevance to ED families (Holtom-Viesel & Allan, 2014).

The familial context and its dynamics are considered important for psychological development and the formation of relationships. Family dysfunction can negatively affect children's psychosocial development and secure attachment (Waters & Cummings, 2000; Waters et al., 2000). Systematic reviews associate aspects of family functioning (poor communication, poor behaviour control, high levels of family conflict, low family hierarchy values) with an increased risk of child and adolescent obesity (Halliday et al., 2014). These aspects of family dysfunction could be further translated into emotional abuse and neglect within the family.

3.2.1. Family Dysfunction and Eating Disorders

Family dysfunction has been found across all ED subtypes (Cerniglia et al., 2017; Holtom-Viesel & Allan, 2014), indicating that it is a transdiagnostic factor in eating pathology. Yet, even though early findings (Latzer et al., 2002) and a systematic review of 17

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studies (Holtom-Viesel & Allan, 2014) suggest no consistent pattern regarding family dysfunction, other empirical results identify different areas of family dysfunction between ED subgroups (Cerniglia et al., 2017). These contradictory findings indicate the presence of family dysfunction in anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED), but they do not explain the specifics of this relationship. Early studies on this field proposed the presence of intrusive mothers and passive fathers in AN and BN family environments and that these families prohibit emotional expression, are less cohesive, more conflictual and less supportive compared to control families (Bruch, 1982; Kog & Vandereycken, 1989; Strober & Humphrey, 1987). Later studies confirm that women with an ED report high levels of family dysfunction and that there is a link between an abusive familial environment and emotion dysregulation and eating pathology (Cao et al., 2013).

Strober and Yager (1985) (as cited in Schmidt et al., 1997) identified two family patterns regarding AN patients. The first is characterised by a centripetal process dominated by excessive cohesion, a lack of permissiveness, reduced emotional expression and poor extra familial contacts. The second pattern is characterised by a centrifugal process. These families lack cohesion and attachment and display high levels of conflict. Families of BN patients have also been described as chaotic, having high conflict levels, low cohesiveness and low attachment. Additionally, clinical studies report that the most prominent characteristics of what Minuchin and colleagues (1978) described as ‘psychosomatic families’ to be enmeshment and overprotection, resulting in low self-esteem, rigidity and lack of conflict resolution. Specifically, the families of AN patients have been extensively proposed as *enmeshed*, meaning that there is such an extreme interdependence among family members that the boundaries between individual identities are very weak and frequently violated (Minuchin et al., 1978).

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The psychosomatic model (Minuchin et al., 1978) has received criticism due to the complexity of the design required in order for the model to be empirically tested (Eisler, 2005). Neither early nor recent literature reviews (Eisler, 2005; Holtom-Viesel & Allan, 2014; Kog & Vandereycken, 1985) support the psychosomatic family model (Minuchin et al., 1978), as no consistent pattern of family functioning in ED families has been empirically identified. The criticism also posits that studies that detail associations between family dysfunction and EDs falsely assume that their findings support the psychosomatic model (Eisler, 2005). Even though reviews indicate that ED families report higher dysfunction than control families (Holtom-Viesel & Allan, 2014), several authors propose that many aspects of problematic dynamics within those families could be the result of dealing with a serious chronic illness, such as an ED, rather than the cause (Eisler, 2005; Jack, 2001; Le Grange et al., 2010). Empirical studies and reviews support the idea that having a family member who struggles with any psychiatric illness is a serious family stressor (Friedmann et al., 1997; Theodoropoulou et al., 2011). Family dysfunction is higher in families containing a member with mental illness compared to those with a member facing a somatic illness (Gordon, et al., 1989; Kabacoff et al., 1990). However, the causal direction between family dysfunction and psychopathology is unclear (Friedmann et al., 1997). Literature reviews and studies indicate that dysfunction in the families of ED patients pre-existed before the ED onset, and the diagnosis only added to the dysfunction (Duclos et al., 2014; Holtom-Viesel & Allan, 2014). Furthermore, parental psychopathological risk/symptoms and the dynamics between parent–child relationships have been associated with eating pathology development in children (Atzaba-Poria, 2010; Tafà et al, 2017), indicating that parental psychological deficiencies can affect the family dynamics prior to eating pathology onset. Moreover, studies exploring family perceptions between adolescent AN patients and their unaffected sisters conclude that the patients and their unaffected sisters did not differ in their perceived relationships with

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parents regarding emotional connectedness, but they did differ in their perception of individual autonomy with patients reporting less autonomy (Karwautz et al., 2003). This finding regarding emotional connectedness indicates a familial characteristic that is not the result of ED presence as both siblings offered the same evaluation. In addition, studies on identical twins have found that BN patients reported higher parental expectations compared to their unaffected co-twins (Wade et al., 2007), indicating a parental behaviour perceived only by the patient. This finding possibly suggests a difference in the behavioural patterns of the parents towards their children that might have stressed this study's patients. Furthermore, studies on child eating pathology reveal that mothers' concerns about child weight, children's level of family satisfaction, family exposure to stress, as well as the mothers' level of education, were predictors of child eating disorder symptoms, with mothers' concerns about the child's weight the most influential factor (Allen et al., 2014). This supports findings from other studies that suggest that negative comments regarding physical appearance expressed by family members can predict ED symptomatology (Crowther et al., 2002). Elaborating on these findings, it could be hypothesised that the manner in which mothers communicate concern about their child's weight is the telling factor, rather than the concern itself. In other words, if their concern is overexpressed, demeaning and disparaging (i.e. emotionally abusive), then self-esteem could be affected, which is a risk factor for ED onset (Adamson et al., 2019; Biney et al., 2019).

In summary, even though the psychosomatic model has garnered mixed empirical support, there is enough indication from empirical findings to suggest that for some individuals, dysfunction was present in the family prior to ED onset. Furthermore, a number of prospective, non-prospective and review studies have empirically associated family dysfunction with the development of EDs, which also predicts their course (Cerniglia et al., 2017; Epstein et al., 1978; Friedman et al., 1997; Kog & Vandereycken, 1989; Kog et al.,

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1985; Kroplewski et al., 2019; Minuchin et al., 1978; Minuchin et al., 1975; Pearlman, 2005; Tetzlaff et al., 2016). A recent narrative review supports the association between family factors and ED onset and maintenance, suggesting that family dysfunction's role in eating pathology should be better understood from a developmental psychopathology perspective (Erriu et al., 2020). This suggestion is in line with this current thesis's perspectives. There is no question that the aetiology of EDs should be regarded as a multifactorial, which is what makes them so complex and difficult to treat. It is also clear that early negative life experiences affect human psychological and neurological development in numerous ways that may lead to psychopathology, with attachment and neglect being important factors (Nelson et al., 2019). Yet, even though there has been an empirically established association between family dysfunction and eating pathology, the specifics of this relationship have not been clarified (Friedman et al., 1997), something that Studies 1 and 2 will attempt. Moreover, Study 2 will try answering the family role criticism by reconceptualising family dysfunction as childhood non-physical abuse and focus on these different variables that seem to play a bigger role in ED development.

3.2.2. Factors Mediating the Relationship between Family Functioning and Eating Disorders

It has been proposed that family dysfunction does not directly cause disordered eating, but it severely affects emotion regulation (Sheffield-Morris et al., 2007) and core beliefs about the self (self-esteem), which in turn influence disordered eating development (Vajda & Láng, 2014; Wade et al., 2000). Both low self-esteem and emotion dysregulation are identified risk factors in EDs.

The familial environment is considered important in the development of emotion regulation (Morris et al., 2007). Children learn to regulate their emotions through modelling

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by observing their parents' behaviour. Modelling is a classic concept of social learning theory (Bandura, 1977), which is used to explain children's behaviour throughout their developmental stages. Modelling theory suggests that children's observation of their parents' emotional profiles, emotional interaction and expression teaches them which emotions are acceptable and unacceptable and how they *should* be expressed and managed. They also learn that specific situations trigger specific emotions so they may imitate others' responses in similar conditions. If parents display a wide range of emotions without inhibition then the child will learn that there are many different appropriate emotions for various situations (Denham et al., 1997). The familial context can also influence the development of emotion regulation through parenting practices (i.e. the direct behaviour of the parents towards the child). The way parents react to their children's emotional expressions defines the way children regulate these emotions and the cognitions they develop around these emotions (e.g. sadness or anger should not be expressed, or it is acceptable to cry in front of others) (Eisenberg et al., 1998). Moreover, the parent-child attachment is a reflection of the emotional climate between the child and the parent (Morris et al., 2007), and attachment affects emotion regulation (Cassidy, 1994). In other words, parenting style affects the overall emotional climate in the family and influences the behaviour of the child through attachment (Morris et al., 2007). Attachment shapes both self-representations and emotional consequences of self-confidence and self-doubt (Thompson, 1998). Self-concepts are fragmented in ED patients (Stein, 1996; Stein & Corte, 2003, 2008; Rorty & Yager, 1996).

A critical review of the available literature also suggests that family dysfunction may lead to the development of an ED by lowering self-esteem, which leads to body dissatisfaction/drive for thinness/dieting and associated behaviours (use of diuretics, laxatives, self-induced vomiting). For example, studies indicate that people with dysfunctional families report higher levels of body dissatisfaction and drive for thinness,

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which are both risk factors for ED development (Knobloch-Westerwick & Crane, 2012; Leung et al., 1996; Ordman & Kirschenbaum, 1986; Stice, 2001). In turn, body dissatisfaction is associated with low self-esteem (Balcetis et al., 2013; Tiggemann et al., 2009). Body image and appearance along with self-esteem belong to the psychological domain of quality of life (QoL), as measured by the World Health Organisation's (WHO's) relevant instruments (WHO, 1996), indicating that family dysfunction could have a negative effect on the psychological domain in ED patients. Family dysfunction has been associated with psychosocial QoL for adult and adolescent ED patients (Ciao et al., 2015; Theodoropoulou, 2014), indicating that it can affect psychosocial QoL in this population. Overall, dysfunctional relationships within the family could have an impact on patients' self-esteem (Ha et al., 2006) and their general psychological well-being (Bell & Bell, 2005), impairing psychosocial functioning. Attachment is negatively affected by childhood abuse (Freyd, 1996), and family dysfunction has been empirically associated with childhood abuse (Mullen et al., 1996). Negative parenting, such as hostility, lack of sensitivity and psychological control, can negatively affect the child's emotion regulation and adjustment to new situations and social environments (Calkins et al., 1998; Spinrad et al., 2007). These behaviours could be defined as non-physical childhood abuse and an invalidating environment. Family dysfunction has been used in the literature to imply emotional abuse (Kent & Waller, 2000) and has been linked with the invalidating environment (Crowell et al., 2009). The concept of the invalidating environment (Linehan, 1993) has been also examined in relation to EDs (Corstorphine, 2006; Fox, 2009; Haslam et al., 2008; Haslam et al., 2012). In addition, studies that combined family therapy with dialectical behaviour therapy (DBT) for ED patients indicate a link between family dysfunction and the invalidating environment (Johnston et al., 2015).

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Considering the abovementioned studies, family dysfunction has been associated with emotion dysregulation (Farber, 2008; Latzer et al., 2002; Martin et al., 2004; Pearlman, 2005; Ringer & McKinsey Crittenden, 2007; Ross & Green, 2011), low self-esteem (Ha et al., 2006; Lau & Kwok, 2000; Smets & Hartup, 1988) and with the failure to form stable and secure attachments (Kenny & Hart, 1992; Latzer et al., 2002; Schmidt et al., 1997). Thus, dysfunction in the family could be better understood as a form of attachment disturbance that could lead to ED development through emotion dysregulation and low self-esteem.

Although there are studies indicating the negative effect of low self-esteem on QoL in ED patients (de la Rie et al., 2005), the role of family functioning in this sequence of relationships is unclear, and so far, it comes merely as a conclusion through the interpretation of results, suggesting an association between family dysfunction and low self-esteem with respect to ED development and psychosocial QoL. Considering that low self-esteem has been identified as a major risk factor, as well as an aetiological factor for EDs (Adamson et al., 2019; Biney et al., 2019; Holston & Cashwell, 2000; Stice, 2002), it is evident that it could be a mediating variable for the association between family functioning, ED development and psychosocial QoL. These concepts need to be examined within one model, using structural equation modelling in order to extend the existing literature and theory, which is the aim of Study 1.

In sum, the literature review suggests that family dysfunction is connected with ED development through attachment, emotion regulation and self-esteem. It has also been linked with aspects of non-physical abuse and with the invalidating environment. These two lines combined indicate that the reconceptualisation of family dysfunction is theoretically possible, and it is explored in Study 2. Moreover, by exploring family functioning in Study 1, some elements of key theories used in Study 2 are also tested, theoretically linking the two studies.

3.3. Non-physical Childhood Abuse

The most widely used and accepted definition of childhood emotional abuse (CEA), which has been characterised as murder of the soul (Garbarino et al., 1987), specifies the phenomenon as ‘the sustained, repetitive, inappropriate emotional response to the child’s experience of emotion and its accompanying expressive behaviour’ (O’Hagan, 1995, p. 456). Other researchers use the term psychological maltreatment (Briere et al., 2012; Glaser, 2002; Witkiewitz & Dodge-Reyome, 2000), defined by the American Professional Society of the Abuse of Children as ‘a pattern of caregiver interaction that makes children feel unworthy, unwanted, blamed, threatened or useful for other’s needs’ (APSAC, 1995). CEA is also found in the literature as psychological abuse when childhood emotional neglect (CEN) is present (Baker & Maiorino, 2010) or as emotional maltreatment to indicate emotional abuse and emotional neglect (van Harmelen et al., 2010; Shaffer et al., 2009). Kent and Waller (1998) added the emotional abuse scale to the Childhood Abuse and Trauma Scale (CATS) questionnaire, thereby acknowledging its separate importance with respect to childhood abuse. Childhood neglect has been defined as ‘behaviour by a caregiver which is characterised by failing to engage in behaviour which is needed to meet the developmental needs of a child and which is the responsibility of the caregiver to provide’ (Straus et al., 1995, p. 2). This definition incorporates both emotional and physical childhood neglect.

Non-physical forms of abuse have been examined far less than physical ones, even though it has been identified that some degree of psychological abuse and neglect is common in the general population (Briere et al., 2012). Scholars have been arguing for years that CEA is the most prevalent and catastrophic form of abuse (Binggeli et al., 2001; Brassard et al., 1987; Doyle, 1997; Hart & Brassard, 1987; Iwaniec, 1995), leading to damaging psychological consequences, above and beyond the physical forms, across childhood and

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throughout adulthood (Crawford & Wright, 2007; Greenfield & Marks, 2010; Higgins & McCabe, 2001; Sachs-Ericsson et al., 2006; Shaffer et al., 2009; Spertus et al., 2003). The fact that CEA has been disregarded could be attributed to the misconception that it is less prevalent and damaging than physical forms, combined with the fact that it is under-reported, thus less researched (Barnett et al., 2005; Engeland, 2009). However, emotional abuse is the most frequent type of abuse that occurs either alone or alongside physical forms, followed by emotional neglect (Gross & Keller, 1992; O'Dougherty Wright et al., 2009). Emotional abuse and neglect can occur independently from physical forms of abuse, while physical abuse does not occur without the presence of non-physical abuse (Glaser, 2011). It has also been suggested that non-physical abuse can be conceptualised as an abuse form both distinct from and a consequence of physical abuse (Morgan & Wilson, 2005). The argument is that the physical marks heal quicker than memory, and as a result, non-physical abuse is more catastrophic in the long term because it is the sentiment behind the physical abuse that actually hurts the victim (Hart et al., 2002; Vissing et al., 1991). Expanding this argument, it could be suggested that repeated non-physical abuse in the early years of life perpetrated by significant others can affect the development of the self.

The problem with childhood non-physical abuse does not lie only in the problems of definition and distinguishment of abuse typologies, but also on how severity can be measured (Glaser, 2011). It is proposed that severity could be measured considering the chronicity and intensity of the maltreatment by the caregiver (Glaser, 2011). This means that it should be measured as a repeated pattern rather than a single-case incident, as a repeated pattern of such behaviour can harm the child's development on many levels with long-term consequences (e.g. dose-effect relationships). For instance, the study by Mahon and colleagues (2001a) found a dose-effect relationship in BN patients between childhood abuse and treatment dropout. However, this study only considered physical forms of abuse and one subtype of

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EDs, but it can indicate a dose–effect relationship between childhood abuse and adult behaviour.

Repeated CEA has been associated with a significant reduction in predominantly left dorsal medial prefrontal cortex volume, despite the absence of physical forms of abuse during childhood, and this finding was present in both genders, independent of concomitant psychopathology (van Harmelen et al., 2010). These results indicate the important effect of repeated CEA in human development and simultaneously suggest a dose–effect relationship between CEA and executive functions, as the prefrontal cortex is responsible for the organisation of behaviour and cognitive activities (Siddiqui et al., 2008). Neurological development is incomplete during the early childhood years, and the prefrontal cortex is not fully developed. Repetitive adverse experiences during this developmental period can influence brain development, and early response styles may be incorporated into one’s personality (Thompson et al., 2014). Personality patterns in adulthood are thought to be resistant to change (Caspi et al., 2005; McCrae & Costa, 1994), and this can also be reflected in the difficulty in treating personality disorders. One such disorder, BPD, is characterised by both emotion dysregulation and reports of childhood maltreatment (Ford & Courtois, 2014; Herr et al., 2013; Hughes et al., 2012; Linehan, 1993; Pietrek et al., 2013; Rogosch & Cicchetti, 2005; Scott et al., 2014). In that sense, BPD could be regarded as evidence of the connection between childhood non-physical abuse, and effects on personality, cognitive development and emotion dysregulation. Furthermore, there are study results indicating that sexual abuse is neither necessary nor sufficient for the development of BPD, and that other childhood experiences, particularly neglect by both gender caregivers, represented significant risk factors (Zanarini et al., 1997). In addition, reviews on childhood neglect indicate that depression, anxiety, distress, EDs, behavioural problems, self-harm and addictions are consequences of neglect (Norman et al., 2012; Pignatelli et al., 2017). The abovementioned

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studies indicate that childhood non-physical abuse's consequences are strong enough to disrupt personality development, leading to a number of mental disorders, including EDs. There is also sufficient evidence to suggest that non-physical forms of childhood abuse can act in a detrimental way without the presence of physical abuse.

The impact of emotional abuse is stronger during childhood because the development of core beliefs about the self and others is easily influenced by external experiences, due to the less advanced cognitive way information is processed (Waller et al., 2007). Negative self-beliefs formed in childhood tend to be robust and difficult to change (Young, 1994). These core beliefs in childhood are perceived as intolerable by the child and create the need to develop protective coping strategies to survive in the invalidating environment. Since the child has limited cognitive resources to adapt to their situation, the coping strategies are more likely to be of a detaching and avoidant nature, rather than focusing on thoughts and emotions, the latter of which would allow proper elaboration and resolution (Waller et al., 2007). Results indicate that the acknowledgement of being abused in childhood is associated with depression, anxiety and dissociation, and can explain why it is difficult for children to acknowledge that their caregivers have been abusive to them (Goldsmith et al., 2009). Additionally, authors argue that difficulties in emotion regulation result from neurological changes after chronic childhood maltreatment by causing hypersensitivity to threat cues and a tendency to respond to non-hostile situations as threatening ones (Cohen et al., 2013; Thompson et al., 2014). The latter is in line with the explanation of anger in ED patients offered by Fox and Power (2009), who argue that certain early life experiences make these patients feel as though the world is unfair, thus they are prone to high levels of anger in their interpersonal relationships. At the same time, anger is perceived as dangerous by ED patients, as it can destroy relationships, so, in an effort to inhibit anger and lacking effective anger-management skills, they develop ED symptoms to handle this threatening emotion (Fox &

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Power, 2009). Considering the above, there is a link between CEA, attachment, self-esteem and emotion regulation, as CEA affects the stages and developmental process of these psychological variables.

CEA is also considered to be responsible for the impairment of self-evaluation, having a direct relationship with feelings of inferiority and helplessness (Schaaf & McCanne, 1994; Waller et al., 2007), thus affecting self-esteem (Finzi-Dottan & Karu, 2006). It could be argued here that feelings of inferiority and helplessness are factors that could maintain abuse in later life, making the individual seem vulnerable to others. In accordance with this argument, CEA has emerged as a risk factor for sexual victimisation in adolescent intimate relationships (Zurbriggen et al., 2010) and as a predictor of adolescent dating violence (Rich et al., 2005). Furthermore, people who experience feelings of inferiority and helplessness may choose to regulate/control their body to regain the lost control of their environment (Root & Fallon, 1986; Vajda & Láng, 2014), indicating a link with low self-esteem, emotion dysregulation and EDs. Moreover, individuals who have experienced emotional abuse may develop a negative self-image, believing that they do not deserve their parents' attention (Frankel, 2002; Loos & Alexander, 1997), further suggesting a link between CEA, attachment and self-esteem. Thus far, a link has been demonstrated between childhood non-physical abuse, attachment, self-esteem and emotion regulation.

3.3.1. Non-physical Childhood Abuse and Eating Disorders

Many authors consider CEA the most influential factor that deserves further empirical exploration with respect to EDs (Fosse & Holen, 2006; Groleau et al., 2012; Kent et al., 1999; Kong & Bernstein, 2009; Vajda & Láng, 2014; Waller et al., 2007; Witkiewitz & Dodge-Reyome, 2000). A study exploring the experiences of all forms of childhood abuse in ED patients with comorbid disorders concluded that CEA was the most common traumatic

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experience followed by emotional neglect (Olofsson et al., 2020). Moreover, physical neglect has been found to maintain ED symptoms in all ED subtypes and affect their severity (Bou Khalil et al., 2020). However, very few studies have explored childhood neglect in EDs even though reviews suggest a strong link between them (Pignatelli et al., 2017). The literature review confirms that non-physical forms of childhood abuse have a significant effect on the development of EDs. For instance, emotional abuse and neglect have been found in higher rates in BED patients than in non-clinical populations (Alison et al., 2007) indicating a link between non-physical childhood abuse and eating pathology. Studies exploring all forms of childhood abuse and eating pathology have found CEA to be the strongest predictor of EDs (Burns et al., 2012; Kent et al., 1999; Witkiewitz & Dodge-Reyome, 2000). Furthermore, research has shown that other forms of abuse that appear to predict EDs have a greater impact when an emotional component is also involved (Kent & Waller, 2000; Kent et al., 1999). This supports the notion that emotional abuse unifies all forms of childhood abuse (Hart & Brassard, 1987) and that non-physical forms of abuse have a greater impact than physical ones (Hart et al., 2002; Vissing et al., 1991). It has been maintained that CEA contributes to ED development by impacting attachment, self-esteem and emotion regulation (Groleau et al., 2012; Hund & Espelage, 2006; Waller et al., 2007). The following review of studies reveal a link (either direct or indirect) between non-physical abuse forms and EDs, explained either by these three mediators or their associated components.

Kent and Waller (2000) proposed two models to explain the relationship between CEA and EDs. In Model 1, CEA plays a direct causal role in ED development, but this relationship could be mediated by other psychological or physiological variables. In Model 2, CEA does not directly affect ED development but moderates the influence of other forms of childhood abuse. The first model was empirically tested in a non-clinical sample (Kent et al., 1999), and CEA emerged as the only form of abuse to predict eating pathology. The

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mediators used were depression, anxiety and dissociation, with the last two being statistically significant between CEA and EDs. The age of abuse onset served as a moderator and was not statistically significant. The authors proposed that emotion regulation could be the link between CEA and EDs. The second study of Model 1 came from Kennedy and colleagues (2007), who found that CEA and physical abuse had a direct unmediated effect on ED pathology in their non-clinical population. Regarding their three mediators, self-esteem and anxiety mediated neglect and ED development but depression did not. Self-esteem did not mediate CEA and ED development in this non-clinical sample. Model 2 was empirically tested as well (Kennedy et al., 2007), and CEA did not moderate the influence of the other forms of abuse in the same non-clinical sample. Based on these findings, the authors suggested that future research should determine whether the effects of CEA on ED are direct or mediated by other psychological experiences. They also proposed that Kent's and Waller's two models (2000) should be expanded in order to emphasise the aetiological role of non-physical forms of abuse as they may result from low self-esteem and body dissatisfaction, both of which are well-known risk factors for ED development. The researchers who tested the two models (Kennedy et al., 2007; Kent et al., 1999) maintain that their results require validating in a clinical population, and they also suggest that non-physical forms of childhood abuse could be better predictors of later disordered eating development. These studies have other limitations beyond only studying a non-clinical population. For instance, Kennedy and colleagues' (2007) sample consisted of both men and women of various ethnicities. This means that their results could have been affected by either gender or cultural factors regarding both childhood abuse and eating pathology. Moreover, the neglect scale used in these studies does not thoroughly capture all aspects of neglect. Additionally, the use of SEM could have provided better comprehension of the proposed models.

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Study 2a in this thesis addresses these limitations by examining the effects of childhood non-physical abuse on EDs and psychosocial QoL in both clinical and non-clinical populations through SEM, using self-esteem, emotion regulation and attachment as mediators, rather than their subcomponents and associated emotions. Neglect is also thoroughly used in Study 2a through a better conceptualisation (emotional neglect and physical neglect) than the one measured in the abovementioned studies, in order to accurately capture all forms of childhood non-physical abuse. Various studies have used elements of these theories, displaying a link between the current thesis's proposed variables; however, there are no studies providing a comprehensive empirical test of all the factors, as reviewed below.

The literature suggests that non-physical forms of childhood abuse can result in maladaptive cognitions that could influence ED development (Hartt & Waller, 2002). The clinical links between CEA and EDs have been discussed by Waller and colleagues (2007), outlining how CEA can result in low self-esteem and dysfunctional levels of cognition, which in turn result in dysfunctional emotional functioning and maladaptive coping strategies (see Section 2.3.1.). Further to this, CEA can also result in emotional impairments, such as emotional inhibition and alexithymia (Waller et al., 2007), which are both related to EDs (Bekker & Spoor, 2008; Westwood et al., 2017). In other words, aspects of emotion regulation are linked with non-physical abuse and EDs. Emotional inhibition is the excessive inhibition of spontaneous actions, feelings or communications, and it serves to avoid others' disapproval, feelings of shame and loss of impulse control (Young et al., 2003). Alexithymia is defined as the inability to express emotions due to a lack of emotional awareness, and it is perceived as an unconscious process (Sifneos, 1973). Both emotional processes are in accordance with trauma-related theories, which suggest that traumatic experiences in the early years of life lead to violation of boundaries, trust issues and poor distress tolerance

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(Goldsmith et al., 2004; Waller et al., 2007). As a consequence, traumatic experiences make the individual vulnerable to later psychopathology development by damaging self-concepts and self-esteem, which result in the inability to manage strong affect and general distress, thus leading to the use of maladaptive coping strategies in adulthood (Follette et al., 1998; Goldsmith et al., 2004; Rorty et al., 1994; Schwartz & Gay, 1996 as cited in Hund & Espelage, 2005; Waller et al., 2007). These maladaptive coping strategies are employed by the abused individual to survive in emotionally invalidating environments, and they are maintained during adulthood even when the invalidating environment may no longer be present (Waller et al., 2007). The explanation of the latter could be that self-esteem and emotion regulation are formed early in life, and when chronic abuse occurs in childhood, it permanently impairs psychological factors.

In line with the above, Hund and Espelage (2006) found a significant relationship between CEA and EDs that was partially mediated by alexithymia and general distress. The authors concluded that ED behaviours are efforts to cope with overwhelming affect coming from traumatic experiences. However, this study neglected to consider self-esteem's impairment as an important casualty of CEA that could lead to distress. In other words, an invalidating environment will lead to low self-esteem and emotion dysregulation, which will then lead to maladaptive coping strategies, such as ED symptoms, to handle intolerable affect. Hund's and Espelage's results (2006) indicate that people who have experienced CEA and people at risk of developing an ED could benefit from interventions regarding adaptive ways to cope with distress and negative affect, such as DBT (Linehan, 1993). Difficulty in managing strong emotions can cause distress (Hund & Espelage, 2006; Krause et al., 2003), and this could possibly explain the depression and anxiety observed in people with emotion regulation difficulties, such as ED patients, as well as general distress's association with alexithymia in Hund's and Espelage's study (2006). Mountford and colleagues (2007)

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concluded that poor distress tolerance partially mediates the relationship between the invalidating environment and eating pathology. The symptoms of ED, therefore, serve as means to block emotions (Corstorphine et al., 2007; Meyer et al., 1998). In other words, there is a link between the invalidating environment, emotion dysregulation and ED symptoms, which has been further empirically supported (Fox, 2009). Studies also indicate that BN patients rely on maladaptive coping strategies and report negative self-perceptions (Wallace, 2013). The majority of the studies emphasise emotion dysregulation coming from an invalidating environment, overlooking the role of self-esteem in this equation. These conclusions are in harmony with Study 2a, which proposes that non-physical abuse may lead to EDs through emotion dysregulation among other important psychological factors examined as mediators, including self-esteem. The aforementioned are also in line with Linehan's (1993) DBT, which hinges on the understanding that invalidating environments lead to a lack of emotional awareness, thus the main goals of DBT include identification, validation and management of emotions and the development of trust (Linehan, 1993). There seems to be a common link between ED and BPD patients regarding invalidating familial environments and emotion dysregulation, which is reflected in the aims of Study 2b.

There is also a connection between childhood non-physical abuse and ED development through attachment. Witkiewitz and Dodge-Reyome (2000) propose that attachment problems may be a mediating factor in the relationship between ED pathology and psychological maltreatment. Clinical experience from sessions with ED patients suggests that a history of CEA increases the likelihood of ED symptoms' severity, comorbidity and dropping out of treatment (Mahon et al., 2001a, 2001b; Waller et al., 2007), making it a variable of interest for researchers and therapists. Specifically, Mahon and colleagues (2001b) concluded that experiences of childhood trauma were associated with dropping out of therapy in adult women with BN, and that CEA had an additive effect on dropping out,

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with these findings remaining the same in replication (Mahon et al., 2001a). They discussed that an impaired ability to trust others, due to disturbed attachment, may explain the finding that links childhood trauma with discontinuing therapy. These findings indicate the importance of addressing attachment in ED patients' psychotherapy. Moreover, attachment insecurity has emerged as a mediator in the relationship between childhood maltreatment and eating disorder psychopathology (Tasca et al., 2013). Scholars have concluded that attachment insecurity, characterised by affect dysregulation and interpersonal sensitivities, could explain why ED symptoms may be one consequence of childhood maltreatment in a clinical sample. Waller and colleagues (2007) proposed that a validating relationship with at least one significant other, or else positive attachment, may act as an important protective factor even within an invalidating familial environment. Extending the argument that CEA is linked to ED development via attachment, along with the fact that attachment affects treatment discontinuation, it could be hypothesised that there is a link between attachment and ED maintenance as well.

CEA has also emerged as a predictor of ineffectiveness, interoceptive awareness, EDs and impulse regulation, which is consistent with the notion that trauma survivors may employ self-harming behaviours, like purging and vomiting, to seek relief from emotional tension, as they cannot otherwise regulate their internal emotional state (Corstorphine, 2006; van der Kolk, Perry & Herman, 1991). These findings further support the link between CEA and emotion regulation, as well as between CEA and self-esteem. In line with these studies, CEA has predicted ED development through the mediating effects of ineffectiveness and affective instability in women with BN, further supporting that CEA is linked with ED development through self-esteem and emotion regulation (Groleau et al., 2012). Childhood physical neglect (CPN) has emerged as a predictor for the drive for thinness, bulimia and body dissatisfaction (Kent et al., 1999; Kong & Bernstein, 2009), indicating a link between neglect

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and self-esteem. Additionally, CEA has been associated with body dissatisfaction (Kircaburun et al., 2019) and has been linked with self-esteem regarding eating pathology, as it has emerged as a risk factor for symptoms related to ED, such as low self-esteem, discontentment with one's body and food restriction (Gross & Keller, 1992). Combining these findings, it could be stated that CEA is associated with body dissatisfaction through a lowered self-esteem, triggering ED behaviour. A possible link between CEA and low self-esteem could be that repeated negative messages conveyed at an early age from significant others become internalised and accepted as true, thus shaping self-perception through dysfunctional attachment. Both CEA and neglect were associated with higher rates of depression and low self-esteem scores in obese male and female bariatric surgery candidates (Grilo et al., 2005), but they were not associated with BMI and ED symptoms. Elaborating on this result, it could be argued that childhood non-physical abuse leads to low self-esteem and depression regardless of ED behaviour's presence and that low self-esteem could be responsible for depression. Physical neglect has been also associated with ED, as Grilo and Masheb (2001) reported that physical neglect is associated with dietary restraint in BED patients, highlighting the need for further exploration of this association (Kong & Bernstein, 2009). This further indicates that neglect also plays a role in EDs. In the same study by Grilo and Masheb (2001), only CEA was significantly associated with body dissatisfaction, depression and low-self-esteem. They concluded that childhood maltreatment may be associated with increased psychological distress in general, but not with the features of ED in people suffering from BED. By extending this argument, it could be further proposed that general psychological distress is due to low self-esteem, resulting from childhood non-physical abuse.

Several studies posit that non-physical abuse could be more significant than physical abuse, as discussed in the beginning of this section. For instance, CEA and CEN were

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significantly associated with eating pathology in a non-clinical sample, and the relationship was mediated by emotion regulation and dissociation (Moulton, 2013). In the same study, when female participants were examined separately from men, CPN along with childhood physical abuse (CPA) were added to CEA and CEN. In this new significant association with EDs, the relationship was mediated by emotion regulation and dissociation as well, except in the case of CPA. Moulton (2013) concluded that childhood trauma has an indirect impact on ED development through an enduring effect on emotion regulation and dissociative processes. Elaborating on this result, one could argue that non-physical abuse is actually responsible for this effect, since the relationship between CPA and EDs was not mediated by the study's mediators.

In Messman-Moore's and Garrigus's study (2007) involving a non-clinical female college students sample, it was determined that women who reported experiencing multiple forms of abuse (revictimisation), regardless of the type, experienced more eating problems and deficits in interoceptive awareness than those who had experienced a single form of abuse. The authors concluded that the link between CSA and EDs found in other studies might be caused by the combination of CSA and an undetected form of abuse. CEA could be the undetected abuse form that exacerbates the impact of CSA, as discussed earlier (Hart et al., 2002; Vissing et al., 1991). Indeed, the authors hypothesised that CEA appears to impact EDs indirectly by affecting the individual's ability to distinguish emotional states and act upon emotional cues, and this could lead to disordered eating (Messman-Moore and Garrigus, 2007). They also maintained that if these underlying mechanisms do in fact explain this link, then intervention programmes could be developed addressing deficits in affect regulation and coping that would help college students resolve many behavioural problems besides disordered eating, including binge drinking, self-injury behaviours (SIB) and sexual behaviour (Messman-Moore and Garrigus, 2007). Another study that investigated the

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influence of all child abuse forms on ED symptoms and general psychopathology using a clinical population concluded that there was an association between physical neglect and current psychiatric comorbidity (Klopfer, 2011), further suggesting that physical neglect is an important form of non-physical abuse. The last two studies reflect this thesis's theoretical assumption that the impact of childhood non-physical abuse on important developmental factors is the foundation of any psychopathology development, and other psychological factors (e.g. parental emphasis on food and physical appearance in the case of EDs) or physiological factors (e.g. genetic predisposition) define the specifics of the disorder.

Another study examined the impact of CEA on emotion regulation and EDs, while controlling for the effects of physical and sexual abuse (Burns et al., 2012). CEA evidenced the strongest association with ED symptoms, and emotion dysregulation was positively associated with ED symptoms and mediated the effects of CEA on them. The authors proposed that emotion regulation is a good mediator between non-physical abuse and EDs, a notion examined in Study 2a, along with self-esteem and attachment as mediators and the inclusion of all forms of non-physical abuse. The only study that particularly focused on emotional abuse and neglect, examining their relationship with EDs, was conducted with a sample of adolescents suffering from AN and BN, and concluded that there is a significant relationship between non-physical abuse and emotion regulation (Vajda & Láng, 2014). The results of this study indicate that non-physical abuse constitutes a risk factor in the development of eating pathology through its effect on emotion regulation. The authors posited that these adolescents grew up in dysfunctional families in which emotions were rejected and inhibited and, as a result, disordered emotion regulation strategies (binge eating, food restriction) are employed to split, block or escape from painful emotions (Vajda & Láng, 2014). Yet, further studies are needed to explore emotion regulation along with other mediators that seem to play an important role in EDs and that are disturbed by childhood non-

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physical abuse, such as self-esteem and attachment. Moreover, BED should be considered as well in the clinical sample, and an adult population would better provide a link with chronicity and childhood non-physical abuse, providing an explanation regarding maintenance.

It is clear from the literature reviewed thus far that the studies conducted have used non-clinical populations and/or pre-adult ED patients (e.g. Hund & Espelage, 2006; Kent et al., 1999; Vajda & Láng, 2014; Wiederman et al., 1998; Witkiewitz & Dodge-Reyome, 2000). This is useful in terms of prevention but problematic in terms of specific therapeutic interventions and factors that should be targeted in therapy for ED patients. Regarding the two forms of neglect, these types of childhood trauma have been examined by only a handful of studies in the ED literature (Hartt & Waller, 2002; Johnson et al., 2002; Kong & Bernstein, 2009; Mitchell & Mazzeo, 2005; Vajda & Láng, 2014; Wonderlich et al., 2007). Moreover, they are based on non-clinical samples or mostly adolescents (Kong & Bernstein, 2009; Vajda & Láng, 2014). Research into the association between non-physical child abuse and EDs is still considered to be scarce even though reviews conclude that there is an aetiological link (Rai et al., 2019). The aetiological link appears to rely on specific developmental factors, such as attachment, self-esteem and emotion regulation, which are all affected by non-physical abuse and are all related to EDs. Yet, there are no studies exploring all childhood non-physical abuse forms in EDs using these factors as mediators. The use of adult ED populations, which is lacking in the relevant literature, would have provided empirical support about maintenance rather than implying it. The use of non-clinical samples or underage clinical samples does not provide enough distance between childhood experiences and adult active psychopathology to assume maintenance. It is also evident that the complexity surrounding EDs is reflected in the research as well. A basic, concise

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developmental and comprehensive model that could explain the development and maintenance of the majority of EDs is missing from the literature.

In summary, research to date has focused mainly on the physical forms of abuse with respect to ED development, and whenever non-physical childhood abuse has been merited with empirical attention, a non-clinical sample or an underage clinical sample has usually been employed. Study 2a aimed to rectify this by testing pertinent aetiological correlates within one theoretical model. The major developmental consequences of non-physical childhood abuse are on attachment, self-esteem and emotion regulation—three factors strongly associated with EDs. Further to this, Study 2b adapted the biosocial theory (Linehan, 1993), defining the invalidating environment as childhood non-physical abuse and using different psychological constructs that are important for ED patients. Both childhood non-physical abuse and the invalidating environment have been found in all ED subtypes, suggesting that they are transdiagnostic factors.

3.4. The Invalidating Environment Conceptualised as Childhood Non-physical Abuse

Linehan (1993) theorised that an invalidating environment, along with a genetic tendency to be over-emotional (temperament), is one of the two major causes of BPD via its effects on emotion regulation. Some authors include family dysfunction and childhood trauma when referring to an invalidating environment (Cha & Nock, 2014). Linehan's (1993) concept of the invalidating environment has been very useful in modelling the impact of CEA and in understanding the psychological interpretations of ED patients' individual experiences (Waller et al., 2007). Neglect could also be added since Linehan's *chaotic environment* involves physically and emotionally unavailable parents (Linehan, 1993), and the invalidating environment has been related to negative core beliefs in females with EDs (Ford et al., 2011). With this understanding and for the purposes of the thesis studies, the invalidating environment is represented by non-physical childhood abuse. That is, dysfunction in the

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family is examined through the consequences of CEA, CEN and CPN, which from now on will constitute the term *non-physical childhood abuse*, to understand its developmental consequences regarding ED development and the psychosocial QoL of ED patients.

Linehan (1993) defines the invalidating environment as a context in which communication attempts are negatively responded to (emotional abuse) or ignored (neglect). In such an environment, everyone should look happy despite their true feelings, and the expression of negative feelings and individuality are not allowed. Consequently, the child uses blocking mechanisms to reduce awareness (Root & Fallon, 1989; Waller et al., 2007). Invalidating relationships with caregivers can lead to deficits in emotion regulation and social skills. These deficits lead to the development of maladaptive coping strategies to deal with distress intolerance. The ability to tolerate distress is the ability to endure and accept negative affect in order to develop effective problem-solving (Linehan, 1993). Individuals avoid emotional triggers or use impulsive (binge eating, purging, self-injury) and compulsive (food restriction, compulsive exercise, obsessive–compulsive features) behaviours to cope with negative affect (Haslam et al., 2008; Linehan, 1993). Studies on eating pathology and emotion dysregulation already support the basic concept behind the biosocial theory (Fox, 2009).

Relationships with invalidating caregivers are likely to be characterised by emotional and/or physical neglect, poor overall relationship quality and disrupted attachment relationships (Martin et al., 2011). As Mountford and colleagues (2007) proposed, poor distress tolerance resulting from an invalidating childhood environment seems to be central in ED development and could mediate this relationship. In other words, emotion regulation could be a mediator between the invalidating environment and EDs. In addition, an association has been established between an invalidating childhood environment and eating

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pathology (Haslam et al., 2008). The attitudes towards emotional expression have been reported to mediate the relationship between a childhood invalidating environment and adult eating concerns (Haslam et al., 2012). Moreover, attachment has emerged as a mediator between the invalidating environment and EDs (Gonçalves et al., 2019). However, authors suggest that it is unclear how the invalidating environment influences the development of EDs (Haslam et al., 2012). Therefore, the pathway that leads to this clinical consequence is explored by this thesis in Study 2.

Experiences of invalidating environments can have a tremendous psychological impact, such as difficulty in tolerating and effectively handling distress, emotional inhibition and alexithymia (Waller et al., 2007). These are traits observed in ED patients, who cannot tolerate negative affect, and they are also found in people with BPD (Linehan, 1993). People coming from such an environment learn that the communication of their thoughts and feelings will be rejected or punished by significant others. Moreover, the emotional abuse literature indicates that emotional invalidation results in difficulties regarding the identification, expression and management of emotions (Waller et al., 2007). These are the functions of emotion regulation (Fox & Power, 2009; Gupta et al., 2008; Ioannou & Fox, 2009), and EDs have been characterised by some researchers as a disorder of emotion regulation (Bydlowski et al., 2005). However, there may be other reasons for alexithymia's presence, such as autism (Kinnaird et al., 2019) or depression (Honkalampi et al., 2000). Nevertheless, the emotion regulation seems to be an independent trait that is not linked with a depressive mood state (Honkalampi et al., 2000). Additionally, it is possible for an individual to be alexithymic regarding certain emotions but able to express and regulate others (Power & Dalgleish, 1997, 2008). Combining these notions with the problematic measurement of alexithymia and the proposal of measuring emotional awareness instead (Suslow et al., 2001), it could be argued that measuring emotion regulation is more conceptually relevant in EDs.

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Various therapies target emotion regulation, such as emotion-focused therapy (Greenberg, 2004), DBT (Linehan, 1993) and enhanced cognitive-behavioural therapy for EDs (Fairburn, 2008).

Combining all these arguments to draw an overall conclusion, it is hypothesised that childhood non-physical abuse could affect eating pathology and psychosocial QoL via self-esteem, attachment, emotion regulation (Study 2a). These variables, which comprise this thesis's studies' mediators, are discussed next, along with temperament. Temperament, as a basic element of the biosocial theory (Linehan, 1993), was retained in Study 2b's adapted model because temperament has also been associated with eating pathology (Atiye et al., 2015).

3.5. Mediating Factors Between Childhood Non-physical Abuse and Eating Disorders/Quality of Life

When a mediational relationship is hypothesised, there must be both theoretical and empirical relationships between the predictor and mediator (e.g. CEA and self-esteem) and mediator and outcome (e.g. self-esteem and ED). The theoretical rationale was reviewed in Section 3.3. The sections below review four mediators (self-esteem, attachment, emotion regulation and temperament) and the evidence for the relationships with both predictor (childhood non-physical abuse) and outcome variables (EDs and psychosocial QoL).

3.5.1. Temperament

The word temperament comes from the Latin word *temperamentum*, from *temperāre*, which means *to temper*, that is, to mix (Rothbart et al., 2000). The concept of temperament originates in the four fluids of Hippocratic medicine (black bile, yellow bile, phlegm and blood), positing that an imbalance in these fluids is responsible for the development of

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diseases, as well as the expression of human behaviour (Christopoulou-Aletra, 2002). Hippocrates (c. 460–c. 370 BC) explained that humans are part of, and thus affected by, their environments (Christopoulou-Aletra, 2002), indicating that temperament can be responsive to environmental influences. The theory of fluids is considered the root of modern endocrinology and Hippocrates's concept of their correct analogy in the body introduced mathematics and geometry into medicine. Hippocrates's view on the four humours in relation to human mood and behaviour is consistent with current views on temperament (Clark & Watson, 2008; Zuckerman, 1995).

In modern psychology, temperament is perceived to be an aspect of personality and is believed to be biologically based (Tortela-Feliu et al., 2012) with emotion being important for temperament (Goldsmith et al., 1987). It is the tendency to express specific emotions with a certain intensity that is unique to each individual (Fox, 1998). Personality is further extended to include cognitions, beliefs and values (Evans & Rothbart, 2007a). McCrae and colleagues (2000) suggested that the Big Five personality model has a temperamental basis that could be inherited, and research indicates that the Big Five adult personality traits have their origins in early temperament (Shiner & De Young, 2013). Contemporary views of temperament also suggest that even though there seems to be a biological predisposition for emotional skills, temperament can also be influenced by the environment (Wilson et al., 2000; Zetner & Bates, 2008), such as from attachment (Fox, 1998), instead of just being an inherited trait (Martins et al., 2012). Additionally, modern perspectives on temperament suggest that it includes dispositional attentional processes but not specific cognitions (Evans & Rothbart, 2007a). This means that cognitions can be influenced by temperament, but the temperamental and non-temperamental personality domains are distinct (Evans & Rothbart 2007a). Moreover, both temperament and personality have shown stability and change over one's lifespan (Roberts & Del Vecchio, 2000). Several models of temperament have been

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produced over the years, but still, there is no consensus on what defines temperament. It has been argued that one of the problems is that researchers continue to rename the temperament constructs, even though the new terms do not differ from the previous ones (Rothbart et al., 2000).

Thomas and Chess (1977) defined nine dimensions of temperament appearing in infancy: activity level, regularity of biological functions, initial approach or withdrawal from new stimuli, adaptability to new situations following the initial response, threshold of sensory responsiveness, intensity of emotional reactions, general positivity versus negativity of mood, distractibility or capacity for external stimuli to alter behaviour and attention span or persistence in the face of obstacles. In this model, temperament is conceptualised as the stylistic component of behaviour, meaning how behaviour is distinguished from motivation—the why and what of behaviour and abilities. This model is limited because of the difficulty in differentiating behaviour from motivation (Zelazo, 2013).

Buss and Plomin (1975, 1984) argued that temperament is inherited and stable throughout one's life. They identified four traits in their initial model on childhood temperament: emotionality, activity, sociability and impulsivity. The model changed later to differentiate between sociability and shyness, and by removing impulsivity from the model and then re-adding it in the face of new evidence. These back-and-forth alterations indicate that it is not a well-perceived and robust model.

Cloninger (1987) focused on the manifestation of temperament in adults, defining four temperament traits: novelty seeking, harm avoidance, reward dependence and persistence. He also specified three character traits: self-directedness, co-operativeness and self-transcendence. His theory proposes that temperament traits manifest early in life and are biologically determined, while character traits appear later in life because they are affected by

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social learning and environmental experiences (Cloninger et al., 1993). Cloninger's theory has not received much support from other research due to the fact that character traits have been found to be as heritable as the proposed temperament traits, the temperament traits are not clearly distinguishable and the measures used to assess these traits have demonstrated psychometric weaknesses (Farmer & Goldberg, 2008; Zelazo, 2013).

Kagan (2008) suggested that temperament refers to a biological foundation for clusters of feelings and actions that manifest in early childhood, stating that human temperaments are heritable neurochemical profiles. Kagan (2008) focused on the predisposition of high or low reactivity to new situations, which is sometimes termed inhibition to the unfamiliar. It refers to the tendency to withdraw and express fear in the face of new stressful situations (Fox et al., 2005). Kagan and colleagues reported research outcomes pointing towards the biological component of this temperamental trait and its stability (Kagan et al., 2007). However, this model overlooks the possibility of temperament being responsive to environmental influences.

Rothbart and Derryberry (1981) created a model that assumes that temperament traits consist of 'constitutional differences in reactivity and self-regulation' (Rothbart & Derryberry, 1981, p. 65). In this model, *constitutional* is viewed as the relatively enduring make-up of the organism influenced over time by heredity, maturation and experience; *self-regulation* is viewed as neural and behavioural processes that function to modulate the underlying reactivity; and *reactivity* refers to the excitability, responsivity or arousability of the organism's behavioural and physiological systems. According to the authors (Rothbart et al., 2000), this definition was offered as an alternative to the behavioural style definition of Thomas and Chess (1977) and Buss and Plomin (1975). This model suggests that both heredity and environmental influences can play a developmental role on temperament. While

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temperament is regarded as reactivity to stimulation, Posner and Rothbart (2000) added self-regulation as a variable that modulates this reactivity. The authors have been influenced by Allport (1961), who defined temperament as ‘the characteristic phenomena of an individual’s emotional nature, including his susceptibility to emotional stimulation, his customary strength and speed of response, and the quality of his prevailing mood, these phenomena being regarded as dependent upon constitutional make-up’ (p. 34). Rothbart and colleagues (2000) went beyond mood and emotions to include motor tendencies and attention in order to study self-regulation and the reactive process. They developed temperament measures from infancy through to adulthood (Evans & Rothbart, 2007a; Putnam et al., 2001). The five adult temperament factors of Rothbart’s model (affiliativeness, negative affect, orienting sensitivity, effortful control and extraversion/surgency), as measured by the adult temperament questionnaire (Evans & Rothbart, 2007a), correspond to the Big Five personality factors (Evans & Rothbart, 2007a), and this work on temperament conceptually links with the personality traits observed in children and adults (see Section 4.3.3 in Chapter 4). According to the authors, the distinction between motivational, emotional and attentional constructs is an advantage of their model, which has influenced the majority of studies on temperament (Putnam & Stifter, 2008; Shiner & DeYoung, 2013). The strengths of the model include recognising the developmental pathways of the temperament dimensions along with their variations during development, possible links with biology and correlation with the Big Five personality model (Laverdière et al., 2010). Moreover, this model links temperament to self-regulation, simultaneously suggesting that both are flexible over one’s lifespan and could be affected by environmental factors, leading to changes in emotions, behaviour and autonomic responses (Putnam & Stifter, 2008). Thus, this model is considered more robust, has influenced research on temperament (Zelazo, 2013) and is relevant to this thesis.

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Non-physical abuse and temperament. Childhood abuse—specifically, neglect—has been found to affect temperament (Kanai et al., 2016), indicating that temperament can be influenced by the environment, supporting Rothbart and Derryberry's (1981) model. It has also been suggested that caregivers' behaviour could affect children's temperamental vulnerability (Rothbart & Derryberry, 1981), which further supports the notion that temperament could be flexible, developing and open to change upon social interaction (Putnam et al., 2001). Moreover, studies have found that temperamentally irritated children display avoidant attachment to their mothers (van den Boom, 1989, 1994; van den Boom & Hoeksma, 1994) and that infant irritability affects the mother–infant relationship during the first year of life (van den Boom, 1994), indicating that temperament may affect the quality of infant–mother attachment. Expanding this point of view, it could be hypothesised that a child's difficult temperament could trigger physical and emotional neglect and abuse from their parents, as the parents may experience frustration in the face of their child's irritability and lose their own temper or give up on the child. On the other hand, childhood non-physical abuse has been associated with parents' temperamental characteristics (Morgan & Wilson, 2005). In other words, a difficult temperament, in either the caregiver or the child, could trigger abusive behaviour. In both cases, temperament seems to be affected by the dynamics of social interaction, and its expression through emotion regulation will affect the relationship.

Wilson and colleagues (2000) argued that infant temperament can be influenced by family dynamics and attachment, as the last two concepts correlated with infant temperament in their study. They discovered that there is a relationship between rhythmicity and the mother's reported stability. Role ambiguity experienced by expectant fathers, as well as mothers' burden from pregnancy itself, the adjustment to their new roles and the mismatch of their expectations in contrast to reality can influence family dynamics and affect both

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temperament and attachment (Wilson et al., 2000). Additionally, Matheny (1986 as cited in Wilson et al., 2000) determined that noise, confusion and disorganisation in the familial environment predicted a change towards difficultness in the second year of life. It seems that temperament can affect others' behaviour towards us and vice versa. Scholars argue that a temperamental trait cannot by definition create problems in social interactions, but interaction with significant others will determine the acceptability of this trait's expression, thus parenting style can influence children's behaviour (Butler, 2010; Hong & Park, 2012). This point of view could have merit considering the fact that parents, who are adults, have more advanced and mature cognitive and emotional skills than their children, meaning that they can better handle and process issues. Also, authors perceive individual differences in attachment style to be related to both the attachment figure's behaviour and the child's temperament (Crowell & Treboux, 1995). Additionally, studies into the influence of temperament on attachment have concluded that this relationship works both ways: the interaction of the parent with the child will determine the quality of the relationship, as they both are active participants (Butler, 2010).

There is also the assumption that attachment stability is influenced by genetic factors rather than solely resulting from early representations (Fraleay, 2002). This assumption has been triggered by studies showing that dynamic models of attachment stability containing genetic/trait components were able to produce the same predictions on attachment stability as the prototype perspective of attachment (Fraleay, 2002). In that sense, maybe temperament could be an influential factor in the parent-child relationship. Longitudinal studies show that temperament, along with psychological maltreatment in childhood, predicts the onset of BPD and antisocial personality disorder symptoms in adolescence (Jovev et al., 2013), indicating that the combination of temperament and maltreatment can play a role in psychopathology development. In addition, studies on genes and environment claim that the environment

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influences genes' activity (Manuck & McCaffery, 2014). This means that being raised in an adverse environment may activate the genes that increase the risk of impulsive temperamental traits, but being raised in a loving and safe environment may result in a calm temperament because different genes will be activated.

Moreover, review studies (Barros et al., 2015) suggest that parental self-regulation, emotion regulation and temperament can influence children's self-regulation, emotion regulation and temperament through attachment, indicating that temperament can be influenced by early environmental experiences. In support of this argument, inherited temperamental traits are believed to be the raw antecedents of later personality disorders, being shaped and formed by parenting style and early life stressors (American Psychiatric Association, 2000; Sansone & Sansone, 2011). The notion that temperament is influenced by the environment is in line with the position adopted by this current thesis.

Temperament and QoL. It has been suggested that temperament can be an important factor in the socialisation of children, since since children's temperament affects how they interact with their social environments in multiple ways (Rothbart et al., 1994). Furthermore, it has been suggested that parenting and child temperaments interact to predict adjustment (Rothbart & Bates, 1998). Empathy, an important factor in human relationships, is strongly related to effortful control, indicating that children with high levels of effortful control (a dimension of temperament) display greater empathy (Rothbart et al., 1994). Authors maintain that effortful control is related to children's emotional, cognitive and social development (Posner & Rothbart, 2000), thus affecting psychosocial QoL. Studies also indicate that maternal parenting style can influence the effortful control temperamental trait, which is considered a necessary skill for children's later adjustment and social competence (Spinrad et al., 2007), indicating that temperament is important for socialisation (Rothbart & Derryberry, 1981). The temperamental ability to adapt to new situations and effectively react to adverse

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stimuli could affect relationships with others. It could be hypothesised at this point that temperament could be related to psychosocial QoL.

Temperament and EDs. It has been argued that temperament per se cannot lead to ED development, but temperamental traits can increase an individual's vulnerability to disordered eating in the presence of other risk factors (Martin et al., 2000). Effortful control and attentional control have been linked with various types of psychopathology (Posner & Rothbart, 2000), as well as with emotional eating (Messerli-Bürgy et al., 2018). Studies suggest that people with BED display low levels of self-directedness and that they differ in novelty seeking, harm avoidance and co-operativeness when compared to obese people without BED (Fassino et al., 2002). Moreover, reviews indicate that AN is associated with an inflexible temperamental style marked by perfectionism, harm avoidance, need for order and sensitivity to praise and reward, while BN is associated with impulsivity and novelty seeking (Mazzeo & Bulik, 2009). Studies also indicate a relationship between temperament factors associated with EDs. For example, a relationship was found between self-esteem and effortful control, concluding that adolescents with high self-esteem display high levels of effortful control (Robins et al., 2010). Other studies indicate that self-esteem and depression are both affected by the temperamental trait neuroticism (Mu et al., 2019). As discussed earlier in this chapter, temperament is affected by childhood non-physical abuse, meaning that childhood non-physical abuse could be the primary common factor between self-esteem and depression. If we accept that temperament could have some effect on attachment, as has been suggested, it could be further examined (in Study 2b) whether temperament affects self-esteem, which is an important factor in EDs. The combination of these concepts and the potential links among them elucidate the need for further exploration of a model designed to address the factors underlying the development of eating pathology, since there are suggestions of a potential association between EDs and temperament (Atiye et al., 2015; Burt et al., 2015; Kaye et al.,

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2015; Rotella et al., 2015; Rotella et al., 2016; Segura-García et al., 2013), as well as a link between temperament and emotion regulation that affects social development (Calkins & Mackler, 2011) and, thus, the social aspect of QoL. The expansion of the already proposed model by Linehan (1993) (Study 2b) could offer a better and more sophisticated understanding of eating pathology, taking into consideration ED and BPD patients' common traits. In support of this, temperament has been empirically linked with BPD and EDs, as perfectionism has mediated the relationship between BPD, EDs and self-esteem (Mas et al., 2011). This indicates that in the case of BPD, a temperamental factor seems to be important to its relationship with ED and self-esteem. In this sense, Study 2b further explores and expands the results of the model proposed in Study 2a, while also testing the biosocial theory (Linehan, 1993). The inclusion of psychosocial QoL in the outcome variables offers a complete understanding of the long-term effects of the factors projecting their influence on basic aspects of human life.

In summary, temperament seems to be affected by attachment through the parents' ability to regulate their own emotions. There is also the suggestion that the child's temperament could affect the attachment pattern, but upon further elaboration, it could be stated that it is still the parental emotion regulation that determines the response (attachment) towards the child. There is a connection between temperament, attachment and emotion regulation, and it could be implied that emotion regulation is the behavioural expression of temperament, which is affected by attachment.

3.5.2. Attachment

Attachment theory refers to the bonding process between mother and infant and the consequences the infant suffers when this bond is disrupted. The basic concept of attachment

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theory assumes the existence of a biological predisposition in species, created by natural selection, that attaches the infant to the caretaker in order for the infant to be protected from environmental dangers and, thus, survive (Bowlby, 1969). Security, therefore, is a crucial construct in attachment theory (Waters & Cummings, 2000).

The core concept of adult attachment theory is that an individual's attachment pattern in adulthood is a reflection of their attachment history, originating from their earliest attachment relationships (Fraley, 2002). People are believed to construct mental representations (working models) of their self and significant others based on their interpersonal experiences (Fraley et al., 2011). These representations are thought to play a crucial role in the way people interpret and understand their social environment. Therefore, assessing the security of working models is crucial for comprehending personality dynamics, emotions and interpersonal relationships (Fraley et al., 2011). The quality and type of attachment children have with their parents has been found to strongly predict the quality of attachment in intimate relationships the child experiences as an adult (Collins & Read, 1990; Hazan & Shaver, 1987; Homes & Johnson, 2009; Mikulincer, 2004; Vorria et al., 2007). Some scholars hold the view that cumulative experiences, along with current stressors and life events, may better determine stability or change in attachment, rather than early experiences alone (Soufre et al., 2005).

Ainsworth and colleagues (1978) defined three types of attachment according to their strange situation study: if the mother pays attention to the child's signals, interprets them correctly and responds in a caring way, the child will form a secure attachment. These children will approach their mother upon reunion without signs of anger and then return to their play. If the mother is ignorant and unresponsive to the child's signals, the child will develop the anxious-avoidant attachment style. Children with this attachment type will

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ignore the mother after separation, without crying. If the mother tends to sometimes respond in a caring way and sometimes disregard the child's needs, the result will be the anxious-ambivalent attachment style (or else insecure-resistant). In this style, the child will cry during separation and their play will be disrupted, and the emotional distress continues after the mother returns. This theory explains the link between attachment and emotion regulation formation, indicating that neglect has a negative impact.

Bowlby (1969, 1973, 1979) proposed that the relationship between the child and the parent serves as an internal working model for future close relationships, and that attachment behaviour characterises people throughout their lives. The internal working model is a cognitive framework that involves mental representations for the understanding of the self, others and eventually the world, and through this cognitive framework, the primary caregiver functions as a prototype for the formation of future relationships (future attachment) and responsiveness (emotion regulation) to others (Bowlby, 1969). It has three central features: a) a model of others as being trustworthy, b) a model of the self as valuable and c) a model of the self as effective when interacting with others. Secure attachment, or else, a secure internal working model, allows the child to develop a positive view of the self and a basic sense of trusting others, which helps them tolerate separation and rejection, develop an effective emotion regulation process, produce mature defence mechanisms and continue to form secure attachments with others (Bowlby, 1973). Conversely, the development of insecure attachment to the caregiver leads to impaired social relationships and affect dysregulation (Bowlby, 1973). By the age of three, the internal working model becomes part of the child's personality, affecting how they understand the world (Schorer, 2000). Neuroscience supports this theory by demonstrating the link between early attachment and obvious difficulties in affect regulation (Schorer, 2001), a common deficit in people suffering from EDs. Brain imaging studies demonstrate that the communication pattern between the parent and the child

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shapes the way the child's attachment system adapts to experiences with the attachment figure, hardwiring the child's brain (Farber, 2008). Neurobiological findings also indicate that the primary caregiver can psychobiologically regulate the infant's developing limbic system, the area of the brain responsible for adapting to a rapidly changing environment (Schoore, 2001). In other words, attachment seems to play a significant role in temperament and emotion regulation. In accordance with this, Bowlby maintained that secure attachment with at least one adult is very important for the individual's overall healthy development, suggesting that failure or trauma in this relationship will negatively shape the child's psychological development and permanently and severely disturb social and emotional behaviour, leading to poor mental health, low self-esteem, antisocial behaviour and disturbance in emotion regulation (Bowlby, 1944, 1969, 1973, 1988). Bowlby (1988) linked the invalidating environment with emotion dysregulation by proposing that when parents forbid their children from crying or expressing negative emotions, they exclude emotional experiences from awareness as children learn to deny their feelings in order to preserve attachment and survive. Continuing from this, Linehan (1993) maintained that consequently, individuals do not learn to accurately label and recognise their emotions, so they do not learn how to effectively modulate them. Longitudinal studies support the notion that early disorganised attachment may lead to unresolved representations, which have been linked to psychopathology, low self-esteem, poor peer relationships and low self-regulation (Aikins et al., 2009).

Bion (1962) introduced the concept of *containment*, proposing that the infant projects their unmanageable feelings onto the primary caregiver, who then reflects them back to the infant in a more tolerable way, describing this reflection as the essence of emotion regulation formation through the attachment process. In the early stages of life, the continuous process of hearing and absorbing cries of hunger, discomfort, fear and anger, and responding

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accordingly, creates the early experience of containment. An optimal containment enables the development of thought and emotion management. This requires a goodness of fit between the mother's and the infant's temperaments (Belsky, 1984). On the contrary, an inadequate or disturbed containment affects cognitive and emotional development. Infants are capable of affective monitoring, which means that they can interpret an experience as pleasurable or not and engage in actions to modify their caregiver's response in order for the caregiver to meet their needs and make them feel safe and worthy (Thayer & Hupp, 2010). This early relationship is crucial for the formation of an experience of a trustworthy caregiver, as this relationship is later internalised in what Bowlby described as the internal working model (Thayer & Hupp, 2010). In line with this theory, Winnicott's (1965) *holding environment* has strong parallels with attachment theory. The holding environment refers to the experience of being physically and emotionally held by caregivers (Ogden, 2004b), indicating that emotional and physical neglect will disrupt this developmental process. A holding environment provides safety and enables emotional development as it accounts for the infant's first experiences with life outside of the uterus (Winnicott, 1987). Within such an environment, the child will learn to identify thoughts and feelings, and develop the capacity to think, trust, symbolise and play. Holding environments create safe boundaries, represent a protective space and enable children to experience themselves as valued and safe within a secure base. Interpreting this, it could be argued that a holding environment shapes self-esteem in order to help the child face the world. Both Bion (1962) and Winnicott (1965) explained how attachment shapes emotion regulation and applied their models to the client-therapist relationship, highlighting the importance of metaphoric *containment* (holding) as part of the healing process. Containment occurs when a person receives and understands the emotional communication of another—without being overwhelmed by it—processes it, and then communicates understanding and recognition (empathy values) back to the other person

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(Douglas, 2007). This psychodynamic-orientated therapeutic suggestion is valued in modern psychology of emotion regulation, which proposes that ED patients' therapy should help them accept their painful emotions and contain them within the self (Fox & Power, 2009). This way, the real self will be accepted and tolerated, and there will be no longer the need for the ED to take control of the self (Williams et al., 2016). These theories complement each other, offering a valuable explanation on the formation of emotion regulation and self-esteem through attachment, and indicate that the effects of attachment are long-lasting.

Authors maintain that our expectations define our behaviour, cognitions and perceptions, like in the case of the self-fulfilling prophecy (Thomas & Thomas, 1928). Therefore, it is concluded that internal working models developed in the early years of life have a long-lasting impact on how we perceive and interpret ourselves and the world (Kamenov & Jelić, 2005). Research indicates that adults with high attachment anxiety are more likely to perceive earlier the offset of others' emotions, and this could lead them to defensively respond rashly before properly evaluating the situation, affecting their personal relationships (Fraley et al., 2006). This could be another indication that attachment plays a role in social relationships QoL. Fraley et al. (2006) explain that individuals with high levels of attachment anxiety are sensitive to changes in both positive and negative emotions, meaning that their attachment systems do not differentiate between emotional signals. Other researchers have concluded that people with an avoidant attachment style are less responsive to their partners' needs (Simpson et al., 2002), indicating the role of attachment avoidance in adults' social interactions. In addition, adult loneliness has been associated with insecure attachment (Bartholomew & Shaver, 1998). Farber (2008) noted that the attachment process also impacts the child's perception of whether they are worthy of being cared for by others, meaning that attachment may have a direct effect on self-esteem formulation. Expanding

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these points of view, it could be argued that attachment affects the child's self-esteem (model of self) and social relationships (model of others), affecting psychosocial QoL.

Evidence also suggests that conversations between the parent and the child in the early years of life shape early autobiographical event representation, as the parent offers a narrative structure for the child's recollection of events (Nelson, 1993; Snow, 1990). Through this procedure, parents inevitably shape children's working models (representations) of self, morals, emotions and relations (Thompson, 1998). In line with this, clinical populations with emotion regulation deficit, display poor autobiographical memory, including AN-R patients (Kovács et al., 2011; Nandrino et al., 2006), who display general difficulty in accessing emotional memories. Moreover, self-referent emotions, such as guilt, embarrassment, pride and shame, start developing around the second year of life, and parental reactions, such as approval or disapproval, are important for the formation of this type of emotion, along with basic emotions, like fear, anger and sadness (Barrett, 1995; Emde et al., 1987). These basic emotions have been discussed and explored in EDs (Fox, 2009; Fox & Froom, 2009; Fox & Power, 2009), linking attachment and emotion regulation with eating pathology. Handling basic and self-referent emotions in a culturally appropriate manner—since they usually occur in a social context (Thompson, 1998)—and effectively coping with emotional arousal produced by these feelings are virtues of emotional competence affected by attachment.

According to Bretherton and Munholland (1999), the interaction of individuals with others is influenced by their memories and expectations of their internal working model, which shapes and forms their relationships with significant others throughout their lives. Empirical evidence from longitudinal studies indicates that the effects of attachment are evident in our relationships with significant others (Bartholomew & Shaver, 1998). Thus, it is generally argued that attachment theory is very helpful in understanding how people attach

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and relate to significant others later in life through understanding the attachment with their caregivers (Briere et al., 2012; Farber, 2008; Fraley, 2002; Fraley et al., 2011). According to Scharfe and Bartholomew (1994), social theorists have expanded the concept of internal working models to include friendship and partner relationships. It has also been suggested that romantic love is an attachment process as well (Hazan & Shaver, 1987), highlighting the influential role of attachment across one's lifespan, a notion expressed by Bowlby (1979) when he proposed that attachment behaviour characterises people throughout their lives.

Meta-analytic studies support the argument that early models of attachment could influence security in adult romantic relationships (Fraley, 2002). Hazan and Shaver (1987) maintained that the emotional and behavioural dynamics of parent–child attachment and adult romantic relationships are driven by the same biological system. They proposed that, generally, adults feel secure and safe when their partner is responsive and accessible, and, in that sense, they use the partner as a secure base from which to explore the environment (e.g. being creative in work or academia), like children do with their parents. They also argued that patterns of attachment (secure, anxious–ambivalent, anxious–avoidant), as described by Ainsworth and colleagues (1978) in the strange situation study on infants, resemble love styles in adult romantic relationships. They suggested that individual differences in adult attachment are reflections of the beliefs and expectations people have formed of themselves and others based on their internal working models, which are relatively stable over time. Some differences include the sexual component of adult romantic attachment and the fact that in romantic relationships the caregiving role is interchanged. Hazan and Shaver (1987) suggest that even though attachment and sex are regulated by different systems, they can influence each other. For example, sexual desire can diminish because the individual does not feel secure with the partner or it can be used to inhibit the development of deep emotional attachment (e.g. people who want to avoid intimacy may engage in short-term promiscuity,

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projecting their sexual behaviour). However, according to authors, attachment theory reveals that social development is a continuous process of construction, revision, integration and abstraction of mental models. They propose that adult romantic love could be more complex than the infant–caretaker attachment (Hazan & Shaver, 1987). Others also add that though the primary source of security for an infant is the physical presence and responsiveness of the caregiver, this could be broader for adults, including social relationships, physical assets, health and religious values, among other things (Waters & Cummings, 2000). This means that adults probably have a broader range of options from which they can derive security than they had as children.

Results on adult attachment are not always straightforward, mainly because researchers have developed different measures for assessing adult attachment (Crowell & Treboux, 1995), so it is not easy to compare results. Reviews on adult attachment measures (Bartholomew & Shaver, 1998) conclude that they differ on domain (attachment target), method (interview versus self-reporting) and dimensionality (attachment categorisation systems). However, when reliability and statistical power are high, these measures converge to some degree and reveal empirical findings compatible with Bowlby’s and Ainsworth’s attachment theories (Bartholomew & Shaver, 1998). Fraley and Shaver (2000), revisiting these views, concluded that Hazan’s and Shaver’s classic theory (1987) needs expansion and improvement, as it should not be assumed that all romantic relationships are attachment relationships. In support of Fraley and Shaver (2000), longitudinal studies demonstrate that negative life events are associated with changes from secure to insecure attachment within twenty years of study (Waters et al., 2000). This confirms Bowlby’s (1953) hypothesis that attachment can be stable across one’s lifespan, yet open to alterations in the face of life experiences. However, there were cases of people who experienced negative events, but their attachment style was not affected, and there were others who changed attachment pattern

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without reporting any stressful life event (Waters et al., 2000). This may indicate that stability and change are related to other psychological factors, such as self-esteem. For instance, if people display some flexibility in their attachment relationships to different people, coming across a potential significant other (e.g. a partner) who is rejective and abusive could create a change in the attachment style because self-esteem will be jeopardised.

Bartholomew (1990), in terms of adult attachment, further divided the avoidant attachment style into dismissive (motivated by the defence mechanism of self-worthiness) and fearful–avoidant (motivated by the fear of rejection) types, suggesting that the four types could be organised along two orthogonal dimensions based on the model of self and model of others. These categories are based on Bowlby’s theory, unlike Hazan and Shaver (1987), who based their theory on Ainsworth’s model (Kamenov & Jelić, 2005). According to Bartholomew’s two-dimensional four-category model, people with a positive view of both the self and others will form secure attachments. People with a positive view of the self but a negative view of others will develop dismissing attachment denying closeness with others due to negative expectations, while maintaining a sense of self-worth by defensively devaluing close relationships. People with a negative view of both the self and others will develop the fearful attachment type, desiring social acceptance and depending on others but at the same time being inhibited due to their fear of rejection. People with a negative view of the self but positive view of others will develop the preoccupied–anxious attachment type, always seeking others’ acceptance due to their feeling of unworthiness. Authors using adult attachment measures with two dimensions explain that attachment anxiety reflects the model of the self, while attachment avoidance reflects the model of others (Briere et al., 2012). This conceptualisation of attachment is reflected in the attachment measurement used in Study 2.

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In sum, it is clear that attachment may affect the individual's psychological health, self-esteem and emotion regulation. Early attachment patterns also affect people's social bonding throughout their lives and, consequently, their psychosocial QoL (Feldman, 2012; Feldman et al., 2011; Gordon et al., 2008; Gray, 2011). Literature reviews suggest that significant others play an important role in people's mental health and positively affect prognoses in serious mental disorders (Theodoropoulou et al., 2011), indicating the importance of attachment across the course of a lifetime.

Non-physical Childhood Abuse and Attachment. According to betrayal trauma theory (Freyd, 1996), the interpretation of a traumatic event is defined from the nature of the relationship between the perpetrator and the victim. If the abuser is someone the victim cares for, relies upon and trusts, then the trauma is high in betrayal, indicating a strong link between attachment and abuse. Betrayal trauma theory also argues that the violation of trust by the perpetrator can be more damaging than the actual act of abuse. Betrayal blindness is also proposed in this theory (Freyd, 1996) as an effort to maintain attachment by distancing themselves from the source of trauma through dissociation. Research supports this process by indicating that victims of traumatic events are less likely to remember incidents of high betrayal compared to low betrayal ones (Freyd et al., 2001). This could explain the under-reporting of non-physical abuse, combined with the fact that there is no physical evidence. It could also explain the dissociation observed in people coming from invalidating environments and the pathological ways they use to cope with the anger produced, as the anger can destroy the relationship and is thus highly dangerous (Fox & Power, 2009).

Research confirms that abuse in the early years of life can affect attachment and that poor attachment is present in dysfunctional families, disturbing children's psychological development (Briere et al., 2012; Egeland & Sroufe, 1981; Finzi et al., 2000; Mothersead et

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al., 1998). In turn, attachment affects the individual's self-regulatory process (Bowlby 1988; Ford, 2005). Emotions and attachment are strongly connected, as the realisation and expression of emotions serve the purpose of preserving the relationship to the attached object (Scheidt & Waller, 2002). People who have experienced traumatic early relationships probably have not had the opportunity to experience the full range of emotions produced by the parent-child relationship, and as a consequence, they may not have learned to effectively interact with others, and experience and interpret the full range of emotions (Ford, 2005). Considering the attachment-related literature, neglect is a considerable factor for emotion dysregulation because when children need to regulate overwhelming emotions by themselves, dysfunctional emotion regulation development can be expected. In support of this, people with emotion regulation difficulties have reported CEN (Frewen et al., 2008; Zlotnick et al., 2001), and non-physical abuse forms rather than physical forms have been found to affect aspects of emotion regulation (Zlotnic et al., 2001).

In psychoanalytic terms, a disturbed experience of object relations may result in the failure to develop a psychologically proper and strong ego identity and maturity (Ross & Green, 2011), further linking attachment to self-esteem developmentally. Object relations theory in psychoanalytic psychology posits that fundamentally, humans are motivated to search for satisfying object (person) relationships (Greenberg & Mitchell, 1983). This theory emphasises interpersonal relations within the family, most notably between the mother and the child (Ainsworth, 1969). The 'object' is a person, a significant other, who is the object of the child's feelings and intentions. 'Relations' refers to the interpersonal relationships. The theory conceptualises that experiences of primary relationships with significant others in the past affect a person's present relationships.

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It is also argued that attachment theory offers insight into the comprehension of how people become attached to pain and how the trauma-related dissociative process connects to the brain, producing self-harm behaviour, such as EDs. Self-harm patients have difficulty identifying, expressing and regulating emotion, experiencing them as somatic events (Taylor et al., 1991), like ED patients do. When they feel anxious, angry or numb, they may turn to self-harm to regulate emotions, as toddlers may turn to their transactional object when feeling lonely or anxious (Winnicott, 1953). The transactional object in the case of EDs is the body. Farber (2008) connects traumatic attachment with self-harm based on the notion that traumatic attachment leads to dissociation, and dissociation then leads to self-harm. He proposed that self-harm manifestations, such as SIB and EDs, are dissociated attempts to regulate affect and ultimately master the trauma through re-enactment. He suggested that self-harm behaviours develop due to the child's disorganised attachment to those who have caused them pain, and the person maintains this disorganised attachment by deliberately causing themselves pain. This means that trauma can cause dissociation, and when the child internalises a dissociated identification with the aggressor, they become attached to the figure that caused the trauma (Siegel, 1999 as cited in Farber, 2008). Continuing this line of thought, it has been proposed that betrayal trauma damages the cognitive functions responsible for the identification of betrayal and trauma signs, so people are less likely to protect themselves by leaving an abusive relationship later in life (DePrince, 2005; Gobin & Freud, 2009). This could explain why people who have experienced traumatic attachment find it difficult to avoid abusive relationships and why ED patients continue to face emotional abuse in adult life in an effort to establish attachment, as this is the only type of relationship they are familiar with. Supporting this argument, anxious attachment has emerged as a mediator between childhood maltreatment and the experience of trauma betrayal in adult life (Hocking et al., 2016), possibly indicating the reason abuse continues in adult life.

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Winnicott (1965) introduced the concept of the *true* and *false* selves to discuss the developmental consequences of a successful and unsuccessful holding environment accordingly. While Freud's ego intervenes between the primitive 'id' and the moral 'super ego' in order to create balance in the self (Bee, 1999), Winnicott's false self takes control, manifested as a defence mechanism when the true self cannot create the desired safe environment (Winnicott, 1960). In other words, the false self is a defence mechanism designed to protect the real self by hiding it in order to comply with environmental demands and remain secured (Daehnert, 1998). The organisation of the false self is based on what the infant receives as a message from their primary caregiver (most notably, the mother) regarding which aspects of their inner self can be shared with another person (Daehnert, 1998). The false self develops during the first stages of object relationships if the mother fails to create a holding environment and she is not contained enough to allow the true self to develop (Kernutt, 2007). Winnicott focuses on the relationship between a caregiver (usually the mother or a mother figure) in defining the true self from which emerges the need to develop a false self. In terms of Winnicott's theory, as the infant matures, they are able to mask their true self in order to maintain connection with the mother and protect the true self from the mother's anger or neglect, to relieve their anxiety and attain the ability of disidentifying from their mother, as well as protect the mother from the infant's destructiveness (Daehnert, 1998). In the case of EDs, the false self, who cannot regulate emotions, releases the unexpressed anger to the body in order to preserve the relationship with significant others. At this point, it could also be argued that the child, through the development of the false self, is trying to avoid non-physical abuse (emotional abuse, emotional neglect and physical neglect) from the primary caregiver and preserve attachment at any cost. A modern approach resembling the false and true self comes from Wonderlich and colleagues (2001) in the integrated cognitive affective theory model, which proposes that

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there is a discrepancy in BN between the ideal and perceived self that creates negative emotions, thus, ED strategies target the self in order to change the self.

Attachment and EDs/QoL. Attachment has been linked with all ED subtypes, mediating childhood traumatic experiences and EDs (Tasca et al., 2013), and the invalidating environment and EDs (Gonçalves et al., 2019), indicating attachment's transdiagnostic role in eating pathology. Women suffering from an ED report insecure attachment to their parents (Armstrong & Roth, 1989; Chassler, 1997; Cunha et al., 1992; Gander et al., 2015; Orzolek-Kronner, 2002; Turner et al., 2009; Witkiewitz & Dodge-Reyome, 2000), most notably to their mothers (Ward et al., 2000). This leads therapists to believe that the intense focus on food replaces the inadequate regulatory functions of early attachment (Farber, 2008; Pearlman, 2005). Moreover, high levels of attachment insecurity are linked with ED symptoms' severity and high dependence on the disorder (Forsén Mantilla et al., 2019; Tasca, 2019). There are also studies that link childhood traumatic experiences and EDs with attachment (Hewett, 2014; Tasca et al., 2013), and ED patients also report childhood separation anxiety (Troisi et al., 2005), which, in turn, has been linked with body dissatisfaction (Troisi et al., 2006). Additionally, BN patients offer accounts of a hostile home environment in which threats and a tense atmosphere occurred more frequently compared to depressed and healthy control samples (Stuart et al., 1990). Furthermore, a study with AN patients revealed that insecure attachment is associated with eating pathology, as well as with alexithymia (Peters, 2010). Moreover, case studies indicate that attachment intervention could help BED patients (Szalai, 2020). Literature reviews suggest that attachment has been overlooked in the dominant models of eating pathology, even though these models are based on psychological components and behaviours that are driven by attachment (Tasca, 2019). In addition, patients' resistance to treatment has been empirically linked with attachment insecurity, whereby patients use the disorder as an attachment figure

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(Forsén Mantilla et al., 2019). In view of this, it is important to understand the underlying mechanism of attachment in eating pathology.

For ED patients, who have developed a distorted body image, self-harm, such as ED symptoms, can serve to differentiate boundaries, such as inner from outer, and self from others (Krueger, 1989). It is believed that dissociation underlies their projective identifications, as well as their re-enactments of early attachment trauma, both in the transference/countertransference and on their bodies (Bromberg, 1998; Farber et al., 2007; van der Kolk, 1988, 1989). When a significant other is really there for them, they do not need to turn to their bodies to feel better (Farber, 2000). If one accepts that an ED is a metaphoric mask that protects the individual from being exposed to painful real feelings, thoughts and experiences of the true self, then one could assume that this mask could be perceived as Winnicott's (1960) false self. It is believed that EDs are a defined feature of the false self (Bruch, 1973; Jones, 1985), something that has been empirically supported in a qualitative study with AN patients, who stated that the disorder has taken over the real self in order to protect it (Williams et al., 2016). Additionally, reviews explain AN as the result of the loss of the emotional self due to attachment disturbance (Oldershaw et al., 2019).

According to Jones (1985), many parents of ED patients emphasise food and external appearance. They want successful, compliant model children. They are also socially isolated and highly enmeshed. At the same time, they have not prepared their children to be autonomous, nor have they been responsive to and tolerant of their children's needs. In this sense, the false self develops as a defence mechanism against parental demands for super performance and zero tolerance to expression of needs and emotions (Jones 1985). Thus, therapists believe that treating the symptoms manifested in the false self has no value because only the true self can be analysed and cured (Jones, 1985; Williams et al., 2016; Winnicott,

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1965). The true self includes not only the person in need of being understood and respected for who they really are, but also the person who needs to express, experience, accept and tolerate negative emotions and autonomous characteristics without the help of ED symptoms. ED patients may not always be able to identify or verbally express with accuracy how they feel when it comes to negative emotions. Reduced emotional awareness and dissociation could be viewed under this theory as maladaptive coping strategies employed by the false self to protect the real self from potential loneliness and abandonment by setting aside its needs and suppressing its emotions. In that sense, the false self uses the symptoms of an ED to emotionally survive. But at the same time, it is self-destructive, affecting people's physical and psychological health, as well as social relationships, thus, impacting their psychosocial QoL. In a holding environment created in therapy, the ED patient has the opportunity to experience and unfold all aspects of the true self, including those that are *imperfect* and *flawed*. Thus, attachment is an important factor to research in EDs and target in psychotherapy, as the unaddressed true self could possibly explain treatment resistance and chronicity in eating pathology. Reviews on EDs and attachment conclude that insecure attachment is insufficient on its own to cause an ED, but it causes the impairment of other factors that are responsible for ED development, and this underlying mechanism has not been sufficiently explained (Cortés-García et al., 2019). As has been discussed so far, attachment is responsible for shaping self-esteem and emotion regulation, which are both major risk factors for EDs.

3.5.3. Emotion Regulation

Emotions provide essential information for human survival and environmental adaptation (Drvaric et al., 2013; Campos et al., 1989; Nesse & Ellsworth, 2009). Emotion regulation is the ability to manage emotions in the self and others (Mayer, 2001), and it

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includes the moderation and expression of the experience of emotions, as well as their evoked psychological responses (Gross & Feldman-Barrett, 2011; Izard et al., 2011). Failure to do so can result in psychopathology (Cole et al., 1994). Positive emotion regulation refers to the ability to accurately recognise emotions and respond to them in an adaptive and flexible way (Paivio & Pascual-Leone, 2010). It develops through positive interaction with responsive parents in the early years of life (Field, 1994) and through interaction with peers in childhood (Ford, 2005). It is also suggested that parents' beliefs about emotions can affect their children's emotion regulation development (Morris et al., 2007) as these beliefs determine children's behaviour regarding particular emotions.

Difficulties in emotion regulation can result from emotionally and physically unavailable parents (Field, 1994), indicating the role of emotional abuse and neglect in this developmental process and the link with attachment. The concept of emotion dysregulation has received growing attention for its possible contribution to numerous symptom manifestations (Gross & Munoz, 1995). Difficulties in emotion regulation may lead to many mental disorders and maladaptive behaviours (Aldao et al., 2010), and there are no gender differences regarding emotion regulation and psychopathology (Nolen-Hoeksema, 2012).

Gratz and Roemer (2004) proposed six theoretically derived and empirically validated factors in their conceptualisation and measurement of emotion regulation (see Chapter 4, Section 4.3.3), maintaining that the absence of a factor will result in emotion dysregulation. These factors are composed not only of the experience of negative affect, but also of one's ability to label emotions accurately, tolerate the experience of distress, and engage in goal-directed and adaptive behaviour while facing distress. Thus, the construct of emotion regulation includes the ability to adaptively identify and cope with negative emotional states, not just the experience of a negative emotion itself. Due to high levels of physiological arousal produced while experiencing negative affect, emotion dysregulation is regarded as an

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important risk factor for psychopathology development (Bradley, 2000; Gratz & Roemer, 2004; Linehan, 1993; Robertson et al., 2014).

According to Saarni (1999), emotion regulation is a component of emotional competence, which is defined as the ability for self-efficacy in emotion-eliciting social transactions. Emotional competence is believed to have an important effect on the development of the self (Thompson, 1998), linking emotion regulation to self-esteem. Its components include emotional awareness, the ability to use emotion-related vocabulary and expressions, empathy, the ability to distinguish between internal subjectivity from external expression, emotion regulation and coping skills, and adaptive emotional communication with others (Saarni, 1999). Emotional competence is connected with social competence, self-understanding, relational security, social cognition and moral understanding (Denham et al., 2007; Eisenberg et al., 1998; Spinrad et al., 2006), indicating an association with psychosocial QoL. A very important element of competent emotional functioning is the capacity to use effective coping strategies (Saarni, 1999).

An explanatory integrated cognitive model of emotion is the schematic, propositional, analogical and associative representational systems (SPAARS) model (Power & Dalgleish, 1997, 2008), which proposes that messages conveyed at an early age regarding the unacceptability of emotions suppress their normal development and dissociates them from the rest of the individual's developmental processes. Consequently, the individual perceives the dissociated emotion as intolerable distress and attempts to maintain this dissociation for self-protection. It further originally proposes that people have different relationships with their individual emotions due to their learning histories, linking early life experiences with emotion regulation. Further to this, it is the only model of emotion regulation to date that actually considers the impact of an invalidating environment on self-esteem and attachment, linking these factors to emotion dysregulation. According to Fox and Power (2009), the

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SPAARS model presents a schematic model of a bad and unaccepted by significant others self when this self is emotionally expressed. The message conveyed is that this bad and worthless self will be rejected and abandoned by others if expressed, so emotions should be inhibited because others are more important, and the world is not safe. In other words, an invalidating environment will destroy attachment and self-esteem, and the only way to survive is through emotional suppression, which is also reminiscent of the false self defence mechanism (Winnicott, 1965) discussed earlier in this chapter. The dominant emotion is anger, but low self-esteem does not allow its expression as anger can hurt interpersonal relationships within an unfair and unpredictable world (Fox & Power, 2009). The SPAARS model specifically proposes that emotions are generated through two cognitive routes. The first is the appraisal route, which reflects the triggering of emotions as a result of the individual's processing of internal and external stimuli. In the second route, the emotional reaction to a stimulus becomes automatic. This model adopts a basic emotions perspective that includes anger, sadness, fear, disgust and happiness, and proposes that emotions may become coupled with each other. This coupling may be facilitating or inhibitory.

Emotion Regulation and EDs/QoL. Difficulties in emotion regulation are believed to be significantly involved in the development and maintenance of EDs (Markey & Vander Wal, 2007), and reviews on EDs and emotions support the notion that people use EDs to alleviate negative affect (Henderson et al., 2019). Emotion dysregulation has been found to be an important transdiagnostic characteristic of EDs (Monell et al., 2018).

The SPAARS model (Power & Dalgleish, 1997, 2008) has been extended and applied to EDs (Fox & Power, 2009), adopting a transdiagnostic perspective, suggesting that anger and disgust are important emotions in EDs, and the disorder serves as a protective barrier between these emotions and the self. Specifically, the SPAARS-ED model proposes that, as a

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result of developmental factors, anger becomes ego-dystonic and is coupled with disgust because disgust is the result of preparedness, thus, disgust is an automatic emotion (Fox & Power, 2009). Empirical evidence supports this coupling of emotions regarding bulimic symptomatology, theorising that disgust is employed to cope with the ego-dystonic feeling of anger (Fox & Harrison, 2008). However, in another study on coupled emotions (Fox & Froom, 2009), only anger and sadness emerged as significant predictors of eating pathology. It was hypothesised that disgust probably did not have an effect due to the small sample size, or that disgust is so closely linked to anger and sadness that it disappears in their presence (Fox & Froom, 2009). Using a further interpretation, this result could also rely on the participants' differences in eating symptomatology because in Fox and Harrison (2008) the symptomatology was of a bulimic nature, while in Fox and Froom (2009), participants with both BN and AN symptoms were recruited. It could be stated that anger resulting from early attachment betrayal (Freyd, 1996) is linked with sadness for not being able to react and express anger in restrictive symptomatology, while in bulimic symptomatology (which includes vomiting), this sadness is expressed through disgust (mainly towards the self rather than food), leading to vomiting, just as disgust prompts vomiting in everyday life. Continuing this further interpretation, both sadness and disgust are directed towards the vulnerable self, who cannot adequately understand and express anger, so uses food to cope. Even though anger in early life is caused by non-physical abuse, resulting in disorganised attachment and emotion dysregulation, at a later developmental stage, when the individual is able to understand the presence of low self-esteem, the anger is also directed towards the vulnerable self, who is not strong and confident enough to react. This creates feelings of sadness (probably related to helplessness, which is associated with low self-esteem) and disgust. Some authors distinguish between food restriction and bingeing/purging by proposing that restriction is used to prevent the experience of negative emotion, and bingeing/purging is

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employed to suppress an already activated emotion (Waller et al., 2007). This concurs with Corstorphine's (2006) proposal of primary (anger) and secondary (shame, guilt) emotions, arguing that the primary emotion is not the real issue, rather, its interpretation as intolerable, dangerous and toxic (Fox, 2009), and if expressed, it could destroy interpersonal relationships, thus, anger is inhibited by ED behaviour (Fox & Power, 2009). Borrowing elements from the trauma betrayal theory (Freyd, 1996), it could be argued that anger at a betrayal (primary emotion as a result of non-physical abuse that disrupted attachment) is suppressed, and then, the individual feels guilty and ashamed for the anger towards the caregiver(s) (secondary emotion). ED behaviours are used in order to dissociate the self from the dysphoric emotions in order to maintain attachment and not destroy important relationships. This dysfunctional procedure that forbids effective self-expression creates sadness and disgust towards the self, who is perceived as weak (low self-esteem). The view that depression could be created from sadness and disgust directed at the self (Power & Dalglish, 1999) is in line with this thesis's position regarding the role of depression in EDs. This means that depression in EDs is caused by low self-esteem, which prohibits the self to express and handle negative emotions, fearing the consequences of a disrupted relationship. It could be stated at this point that negative emotions from early traumatic experiences are not only coupled but work together in a catastrophic and maintaining way. Regarding happiness, it cannot be said that it is a feeling expressed by ED patients as they frequently report in therapy that they cannot enjoy anything. This is self-explanatory as, given the abovementioned emotions, it could be rather difficult to experience happiness. However, happiness—as a basic emotion vital for human health—can take the form of pride in EDs, in the sense that the ED patient takes control of their body through thinness (Goss & Allan, 2009; Skarderud, 2007) to compensate for the lost control in their life and to avoid rejection. In other words, by maintaining a desirable shape and weight, they are elevating their self-

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esteem (Vitousek, 1996). Reviews on pride in EDs conclude that weight control is the only way ED patients increase their self-esteem (Goss & Allan, 2009), indicating its important role in the development of eating pathology. At the same time, this pride can maintain the ED symptoms, as in AN, because the feeling produced by restraint eating is ego-syntonic (Faija et al., 2017).

The SPAARS-ED model's notion of preparedness is directly linked with attachment, as Fox and Power (2009) propose that certain life experiences make people believe that the world is unfair (an attachment process), and they are more prone to experiencing high levels of anger in their social interactions. This agrees with the suggestion that emotional sensitivity is the result of an invalidating environment (Linehan, 1993), meaning that some people display a low emotional threshold, reacting quickly to even mild emotional stimulus, such as disapproval (Linehan, 1993). The preparedness concept also resembles the general adaptation syndrome (Selye, 1950) reaction to stress upon a perceived threat. It could be hypothesised that a disrupted attachment makes people prone to an early offset towards a threatening world, an exhausting procedure by definition. In support of this argument, attachment and reaction to stress have been empirically connected (Higgenbotham, 2016), and, as discussed earlier, attachment theories explain how early experiences with caregivers impact our adaptation to the environment, formulate our behaviour towards others and dictate our interpretation of potentially stressful events, plus our reactions to them. Empirical findings regarding emotions in AN offer support to both the invalidating environment perspective (Linehan, 1993) and the SPAARS-ED model (Fox, 2009), indicating that important theories can be linked to offer a concise explanation of disorders where self-harm has been observed, like EDs and BPD.

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Given that attachment has been successfully used as a transdiagnostic mediator between childhood abuse and EDs (Tasca et al., 2013), considering also the transdiagnostic perspective of emotion regulation in the SPAARS-ED model (Fox & Power, 2009), it is rather clear that we are heading towards a transdiagnostic explanation of ED origins and maintenance. The SPAARS-ED model is considered relevant to this thesis as it combines the invalidating environment (Linehan, 1993), attachment and self-esteem with emotion regulation, emphasising the protection of the self and attachment preservation through emotional suppression, in accordance with the trauma betrayal theory (Freyd, 1996), Bowlby's (1969) internal working model and Winnicott's (1965) false self.

Numerous studies have explored the role of emotion regulation in EDs. For example, a link between disordered eating behaviour and retrospective reporting of parental responses to emotions in the early years of life has been established (Buckholdt et al., 2010), as has an association between maladaptive coping methods and eating pathology (Janzen et al., 1992) and the mediating role of emotion dysregulation between childhood abuse and AN (Racine & Wildes, 2015). In other words, there seems to be a link between invalidating environments, attachment and emotion dysregulation in eating pathology.

ED behaviours in the emotion regulation models are conceptualised as maladaptive efforts to cope with intolerable negative affect (Cooper et al., 2004; Waller et al., 2004), indicating the poor emotion regulation skills of the individual who employs these behaviours. Patients suffering from EDs are clinically supposed to endure impairment in emotional processing, awareness, identification and expression. The emotion regulation hypothesis of ED development maintains that symptoms, such as binge eating, purging and food restriction, are employed in order to distract oneself from negative emotions and self-soothe by regulating adverse emotional arousal in the absence of adaptive coping strategies (Aldao et

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al., 2010; Becker-Stoll & Gerlinghoff, 2004; Burns et al., 2012; Bydlowski et al., 2005; Clyne & Blampied, 2004; Corstorphine, 2006; Dawson, 2012; Evers et al., 2010; Gilboa-Schechtman et al., 2006; Harrison et al., 2009; Heatherton & Baumeister, 1991; Holliday et al., 2006; Ioannou & Fox, 2009; Krystal, 1977; Kucharska-Pietura et al., 2004; Lane et al., 1990; Mills, 2011; Nolen-Hoeksema, 2012; Overton et al., 2005; Polivy & Herman, 1993; Power & Dalglish, 1997, 2008; Root & Fallon, 1989; Vajda & Láng, 2014).

It has been suggested that restriction is used to suppress negative emotions and binge/purge behaviour is employed when the negative emotions have reached consciousness (Waller et al., 2007). This proposed difference in the ED symptoms' expression could rely on the degree upon which the individual can explain and understand the origins of their anger. For example, in Fox (2009), AN patients seemed confused regarding who to blame for their overwhelming anger. It could be that restriction is linked with low consciousness of negative emotions resulting from an invalidating environment due to betrayal blindness (Freyd, 1996). Empirical data from Fox and Harrison (2008) showed that women with bulimic symptoms reported higher states of anger and disgust sensitivity than controls, leading to the assumption that people with bulimic symptomatology experience anger and disgust simultaneously. This is in accordance with the SPAARS-ED model (Fox & Power, 2009), which states that due to developmental factors, anger becomes ego-dystonic, while disgust becomes an automatic response to the intolerable negative affect and could explain the findings that demonstrate a relationship between emotional inhibition and body dissatisfaction in women with EDs. It could be proposed that in people with bulimic symptomatology, the origins of anger are clearer and directed at themselves, leading to disgust. Ioannou and Fox (2009) suggested that there may be new routes for emotions within EDs, where the patients focus their emotion upon the body. This could mean that disgust is expressed via food and the body even though

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it is aimed at the weak self. They also proposed that people have different relationships with different emotions, and empirical findings support this notion (Fox & Froom, 2009).

Difficulties in emotion regulation have been observed in people with subclinical ED features as well (Ridout et al., 2010), indicating the role of emotion regulation in the development of the disorder. Furthermore, emotional eating and restrained eating have been identified in the general population as a means to regulate anger, stress and anxiety (Goossens et al., 2009; Nguyen-Rodriguez et al., 2009; Norwood et al., 2011), indicating that ED prevention could benefit from the inclusion of emotion regulation strategies. In addition, emotion regulation has been associated with QoL with no differences between men and women (Manju & Basavarajappa, 2017), signifying that emotion regulation strategies in therapy can also improve QoL.

Non-physical Childhood Abuse and Emotion Dysregulation. An important consequence of emotional abuse is the difficulty of emotion regulation (Corstorphine et al., 2007; Hund & Espelage, 2006; Vajda & Láng, 2014; Waller et al., 2007), because an emotionally threatening environment with criticism, punishment and forbidden emotional expression may lead to emotional repression (Bowlby, 1988; Linehan, 1993), which is one of the maladaptive emotion regulation strategies, known as avoidant coping (Hayaki, 2009). Combining these lines of thought, it can be concluded that Kent and Waller's (2000) theory on the consequences of emotional abuse and Linehan's (1993) biosocial theory of the invalidating environment are similar psychological mechanisms, which could be further expanded and tested more specifically as childhood non-physical abuse. In addition, Corstorphine (2006) and Linehan (1993) are in line with Bowlby (1988), who proposed that parenting promotes the exclusion of certain emotions from awareness, leading children to believe that they must deny their feelings and needs in order to preserve attachment. In

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accordance with this, the betrayal trauma theory model (Freyd, 1996; Freyd et al., 2001) argues that children separate abuse experiences from memory and consciousness to preserve attachment to their caregivers, explaining that, thus, disassociation comes from traumatic attachment. These theories connect childhood non-physical abuse, attachment and emotion regulation with empirical findings to support and expand this link.

Research has demonstrated that CEA is associated both with emotional inhibition and avoidant coping (Gratz et al., 2007), and researchers also believe that CEA plays a role in ED development by impacting emotion regulation and self-esteem because ineffectiveness and affective instability mediate CEA and EDs (Groleau et al., 2012). Emotion regulation has also emerged as a mediator of the relationship between emotional abuse, neglect and EDs in adolescents with AN and BN (Vajda & Láng, 2014), as well as in non-clinical samples (Mills, 2011; Moulton, 2013; Burns et al., 2012). The hypothesis here is that emotional invalidation, a consequence of non-physical child abuse (Waller et al., 2007), affects emotion regulation (Hund & Espelage, 2006; Linehan, 1993; Reeves, 2007), which in turn leads to disordered eating (impulsive behaviour) as a means to cope with intolerable affect.

People with an ED experience adverse emotional environments during childhood characterised by the presence of negative emotions and poor attachment (Henderson et al. 2019). Chronic childhood maltreatment is considered to be an identified factor of emotion dysregulation (Ehring & Quack, 2010). This is also corroborated by Thompson's and colleagues' research (2014), which concluded that people who had endured chronic maltreatment in childhood reported greater fight-flight-freeze brain sensitivity and increased emotion dysregulation compared to controls with no abusive history, supporting the SPAARS-ED model's concept of preparedness (Fox & Power 2009). Growing up in an emotionally abusive and neglectful environment may therefore result in the development of

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dysfunctional emotion regulation skills (Linehan, 1993). In children, caregiver criticism, punishment and disallowance of emotional expression, emotion invalidation and disregard of emotions are linked to emotional suppression, avoidant coping and failure to seek support, which may be conceptualised as maladaptive emotion regulation strategies (Berlin & Cassidy, 2003; Shipman et al., 2007; Spinrad et al., 2004). In adults, CEA has been associated with emotional inhibition and avoidant coping (Kraus et al., 2003), emotional non-acceptance and experiential avoidance (Gratz et al., 2007). In brief, emotional abuse is related to maladaptive emotion regulation strategies in both childhood and adulthood.

Emotion regulation is not present at birth but develops across one's lifespan (Dodge & Garber, 1991; Morris et al., 2013) and is mostly affected by attachment (Malekpour, 2007; Sroufe, 2005), among other factors. Following this notion, which is also supported by studies in adopted orphan children (Bátki, 2009; Cohen et al., 2013), early interactions with caregivers and significant others, shape and influence the development of emotion regulation through the pathway of attachment (Bee, 1999; Calkins & Hill, 2007; Cohen et al., 2013; Fries et al., 2008; Schuengel et al., 2009). If the relationship with the caregivers is abusive and the environment is perceived as hostile and threatening, the development of adaptive emotion regulation is disrupted, leading to maladaptive emotion regulation skills (Morris et al., 2007). Following attachment theory, emotion regulation is learned through the individual's interaction with attachment figures, and this interaction defines how the individual will cope in the face of distress cues (Cole-Detke & Kobak, 1996). This indicates a clear link between family functioning, attachment and emotion regulation.

Rorty and Yager (1996) hold the position that the self-destructiveness and persistence of abused women's ED symptoms could be better understood as desperate efforts to regulate negative affect and compose self-coherence. They maintain that psychologically traumatic

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experiences lead to the dysregulation of the arousal system, and as a consequence, self-coherence is damaged, and the world is perceived as threatening, just as in BPD (Jørgensen, 2006; Lafleur, 2013; Linehan, 1993; Nolen-Hoeksema, 2003). The authors explain that abused people learn that being in need equates to being exposed to pain, abandonment and betrayal from the individuals that could satisfy that need. Thus, their psychological and physiological needs must be controlled because interaction with others is dangerous and painful. This leads to controlling their needs instead of expressing them and finding effective ways to satisfy their needs. Root and Fallon (1989) suggested that restrictive eating and bulimic behaviour are attempts to control the body and, thus, control the environment. It is believed that CEA in particular is linked with ED development because this type of disorder serves the function of managing strong emotions through suppression or avoidance (Fox & Power, 2009; Kent et al., 1999; Overton et al., 2005). However, there is only the hypothesis that there is an interaction of factors, including the individual's early environment experiences and their temperament (Rorty & Yager, 1996; Waller et al., 2007), to explain how this relationship works and how the different types of invalidating environment caused by non-physical abuse can impact the individual. Rorty and Yager (1996), in their stress–diathesis model, proposed that the interplay between the negative consequences of all forms of childhood abuse with the intrinsic and extrinsic risk factors of ED development, such as temperament or being reared in an invalidating environment, could explain the pathway of eating pathology development. Emotional invalidation is viewed as the most important consequence of CEA, and it is regarded as a form of abuse as well (Mountford et al., 2007; Waller et al., 2007).

Fox (2009), using qualitative methodology, concluded that AN patients experience significant negative affect while growing up as their needs are not met, resulting in the inability to express emotions, most notably anger. The origins of this overwhelming anger

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confuse patients and is perceived as dangerous and toxic to their interpersonal relationships. The author used grounded theory to propose the development of poor meta-emotional skills in AN in order to describe the early experiences of overwhelming affect within poor emotional environments, in which the patients grew up, revealing the dynamics of *too much emotion* that cannot be expressed. This study offers value as it presents the birth of emotion dysregulation in AN. It is important to note that according to the author (Fox, 2009), the accounts of physical abuse in his study were minimal, leaving more space for the non-physical aspects of an invalidating environment. One of the most important assets of this study is the unique display of the continuation of anger and its inhibition from childhood to adulthood—that is, the continuation of emotion dysregulation, which is line with this current thesis's views.

It could be hypothesised that in the early years of life, anger is aimed at the self because the true self is not good enough to be cared for and loved. Later in life, this anger remains directed against the self because the self is perceived as weak and unable to react (low self-esteem). As a result, the self is punished through self-harm behaviours (e.g. food restriction). On top of that, when emotional expression is prohibited in the family, the only way this overwhelming emotion can be expressed and relieved is through the self (body). The overwhelming, suppressed anger is perceived by the individual as dangerous not only for the self (if expressed, the self will be rejected by significant others) but for others, as it will destroy interpersonal relationships, and the self will be abandoned. Suppressed emotions usually appear magnified when they are expressed, as the person has been under prolonged psychological pressure in order to inhibit them, along with the fact that there is lack of training in emotional expression (invalidating environment), thus, emotional expression is perceived and experienced as an unknown and frightening procedure with unpredictable and unmanageable results. Except for the SPAARS-ED model (Fox & Power, 2009), which

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considers schemas for the self and attachment, self-esteem and attachment have been overlooked by affect regulation theories, even though attachment is responsible for the formation of self-esteem and emotion regulation in early life. Low self-esteem is defined as the inability to react for fear of rejection, abandonment and loneliness. It could also be proposed that the effort of inhibiting emotions causes the automatic response of anger in adulthood, when feelings and their management (or lack of) is a rather more conscious procedure compared to childhood.

Summary. An argument for a strong link between childhood non-physical abuse, emotion regulation, psychosocial QoL and eating pathology development has been presented. Some of these factors are included in the biosocial theory of BPD (Linehan, 1993), which has been successfully linked with EDs (Fox, 2009). Therefore, the next section discusses the psychological similarities between ED and BPD patients and how biosocial theory (Linehan, 1993) could be adapted for EDs. According to the proposed model, ED symptoms serve as a means to regulate intolerable emotions, and they are regarded as self-harm behaviours (in EDs, food restriction and bingeing/purging physically harm the body). Some individuals with BPD manage their emotion dysregulation through self-injury and other forms of self-harm, indicating a common pattern with eating disorders. The theory also stipulates that emotion dysregulation is the result of family dysfunction/invalidating environment/emotional abuse. In other words, it is the result of childhood non-physical abuse.

Effects of Childhood Non-physical Abuse on Emotion Regulation in Eating Disorders: Relevance of the Biosocial Theory of BPD. As discussed earlier, Fox's study (2009) thoroughly demonstrated and explained the origins of emotion dysregulation in AN due to an invalidating environment, offering value to the DBT perspective regarding emotion dysregulation in invalidating environments (Linehan, 1993). The literature about

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psychological features observed separately in BPD and ED patients of all subtypes indicates that many of them are common. These include history of childhood abuse, impaired self-perceptions, emotion dysregulation, low self-esteem, alexithymia, dissociation, shame, anger, guilt, disturbed body image, disgust, self-harm behaviours and poor attachment (Agrawal et al., 2004; Becker-Stoll & Gerlinghoff, 2004; Black, 2010; Bydlowski et al., 2005; Claes & Vandereycken, 2007; Corstorphine, 2006; Favazza et al., 1989; Fox, 2009; Fox & Froom, 2009; Fox & Power, 2009; Gupta et al., 2008; Jacob et al., 2010; Jones et al., 1999; Jørgensen, 2006; Ioannou & Fox, 2009; Kleindienst et al., 2014; Lafleur, 2013; Linehan, 1993; New et al., 2012; Paul et al., 2002; Peters et al., 2014; Rüsçh et al., 2007; Rüsçh et al., 2011; Sansone & L. Sansone, 2007; Soloff et al., 1994; Svirko & Hawton, 2007; Vanderlinden et al., 1993; Zanarini et al., 1997; Zonnevijlle-Bendek et al., 2002). In sum, the core common factors are childhood non-physical abuse, attachment, emotion dysregulation, self-esteem and maladaptive coping strategies. This may indicate that there could be a link in the aetiology of eating pathology and BPD or at least common influential factors. Some authors argue that commonalities presented between EDs and BPD could be attributed to mood changes as a result of malnutrition (Vitousek & Stumpf, 2005). This suggestion is an important consideration regarding the comorbid diagnosis of eating pathology and BPD, but it cannot explain the presence of the invalidating environment, poor attachment, low self-esteem, emotion dysregulation and self-harm in both disorders, as these factors are irrelevant to starvation. Moreover, starvation is only relevant in the case of AN-R and could definitely account for mood fluctuations and irritation.

Furthermore, SIB or else parasuicidal behaviour, frequently observed in BPD (Linehan, 1993), is often present in ED patients too (Favazza et al., 1989; Paul et al., 2002; Sansone & Levitt, 2002b; Svirko & Hawton, 2007; Vieira et al., 2016). SIB is defined as any direct and deliberate damage of one's body tissues without lethal intent (Claes &

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Vandereycken, 2007). Its presence has been associated with childhood traumatic experiences that led to emotional suppression and low self-esteem (Morris et al., 2015). Emotional suppression and negative self-beliefs were reinforced through later experiences (Morris et al., 2015), clearly indicating that emotion dysregulation and low self-esteem maintain SIB, and the subsequent experiences could be the continuation of abuse in adult life.

EDs, especially the purging subtypes, have been interpreted by many as a self-harm behaviour in an effort to impulsively regulate intolerable affect (Anderson et al., 2002; Farber, 2008; Favaro & Santonastaso, 2002; Favazza et al., 1989; Claes & Vandereycken, 2007; Claes et al., 2003; Claes et al., 2005; Sansone & Levitt, 2002a; Sansone & Levitt, 2002b; Sansone & Sansone, 2007; Solano et al., 2005; Svirko & Hawton, 2007; Wildman et al., 2004; Wright et al., 2009). This notion could provide a further supportive link between emotion dysregulation and eating pathology, plus highlight a link between EDs and BPD, as self-injury is a common behavioural process. BPD is known to involve disturbances in body image, emotion regulation and self-concepts, leading to self-harm behaviour, thus making researchers believe that the interaction of these psychopathological features could be the reason for the link to ED development in people with BPD (Sansone & Sansone, 2007). Interpreting the abovementioned, it could be argued that BPD patients employ ED behaviours as a form of SIB, explaining the comorbidity that is often observed. In other words, the presence of EDs in BPD patients could be regarded as a form of SIB and not as a separate disorder. Targeting emotion dysregulation and low self-esteem in therapy could benefit both disorders, as these factors are common.

Many theoretical models of EDs and much relevant research have focused on the role of disordered eating as a means to distract from negative self-beliefs and alleviate emotional distress, thereby further highlighting the aetiological overlap between BPD and EDs

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(Corstorphine, 2006; Gratz & Roemer, 2004; Ioannou & Fox, 2009; Power & Dalgleish, 1997, 2008). The hypothesised common aetiological mechanism is also reflected by the efforts of the scientific community to design treatments for ED patients incorporating variations of the DBT tenets. The effectiveness of DBT in treating BN and BED (Safer et al., 2001) further supports this assumption, because DBT teaches patients to effectively tolerate distress (Linehan, 1993). DBT was designed for BPD patients (Linehan, 1993), but evidence indicates that it also works for ED patients (Bankoff et al., 2012; Safer et al., 2001; Wisniewski & Kelly, 2003), implying that both disorders may share similar aetiological and/or maintenance mechanisms. Recent reviews of DBT's effectiveness in ED patients (Ben-Porath et al., 2020) reveal that the majority of studies employ three adaptations of DBT for EDs: the Stanford model, radically open-DBT (RO-DBT) and multidagnostic ED-DBT (MED-DBT). The Stanford model has been proven to be highly efficient in BED, yet more research is needed regarding its efficacy in AN and BN, and the same applies to the other models: there are positive outcomes, but further research is required (Ben-Porath et al., 2020). The RO-DBT model has positive effects in AN (Lynch et al., 2013), and MED-DBT has been found to be effective in difficult ED cases with BPD comorbidity (Federici & Wisniewski, 2013). The inclusion of DBT in ED treatment is not included in the National Institute for Health and Care Excellence (NICE) guidelines for the standard treatment of EDs (NICE, 2020). Current NICE-recommended treatments for EDs include individual ED-focused cognitive-behavioural therapy (CBT-ED) for AN, BN and BED. For AN, the Maudsley anorexia nervosa treatment for adults (MANTRA) and specialist supportive clinical management (SSCM) are also recommended (NICE, 2020). For a treatment method to be included in the guidelines, empirical evidence is required, thus, this is something that could change in the face of sufficient evidence (Ben-Porath et al., 2020).

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Review studies on the use of DBT in ED patients conclude that further research is needed as the effectiveness of emotion regulation strategies on reducing ED symptoms was not fully demonstrated (Bankoff et al., 2012; Ben-Porath et al., 2020). This means that a more comprehensive adaptation of the biosocial theory regarding EDs is needed as emotion regulation, though a significant factor, does not seem to play a crucial role in ED treatment. In support of this argument, studies indicate that self-image is an intervening factor between emotion dysregulation and ED symptoms (Monell et al., 2015). Specifically, women with emotion regulation difficulties were vulnerable to ED symptoms only if these difficulties affected their self-image. Emotion regulation per se did not lead to ED symptoms, and it was not an intervening factor between self-image and ED symptoms. This study was expanded to include women diagnosed with all ED subtypes and drew the same conclusions (Monell et al., 2020). In addition, in the participants with no binge eating behaviour, emotion dysregulation was associated with less self-love and more self-attack, while in the participants with binge eating behaviour, emotion dysregulation was associated with less self-affirmation and more self-blame (Monell et al., 2020). An interpretation of these results in both cases could be that emotion dysregulation is associated with self-esteem that defines self-directed behaviours. Thus, including self-esteem in both BPD and ED models could possibly make a difference both in the explanation of the disorders and in their treatment.

Self-image is created through attachment with significant others defining the way we treat ourselves (Monell et al., 2015). Moreover, self-image guides how our social interactions will be perceived and interpreted (Monell et al., 2015). Self-image is represented by self-directed behaviours—how we treat ourselves—(Monell et al., 2015) and is considered responsible for the development of these self-directed behaviours (Cloninger et al., 1993). It could be argued that the way we were treated early in life (attachment) affects and shapes the way we treat ourselves (e.g. a good, capable self that is worthy of love and care or a bad,

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vulnerable self that is neglected). The view of the self will affect the way emotions are going to be experienced and expressed, thus affecting the development of emotion regulation strategies (Monell et al., 2015). This means that a devaluated self (low self-esteem) will lead to difficulties managing strong affect (emotion dysregulation). In support of this, trait self-esteem has been found to modulate attentional responses to emotional stimuli (Dandeneau & Balwin, 2004, 2009; Downey & Feldman, 1996; Gyurak & Ayduk, 2007; Li et al., 2012; Li, Zeigler-Hill et al., 2012; Murray et al., 2002; Sommer & Baumeister, 2002; Somerville et al., 2010). Furthermore, trait self-esteem greatly influences how and with what intensity the brain responds to positive and negative stimuli (Wang & Wu, 2019). In summary, trait self-esteem can affect emotion regulation.

3.5.4. Self-esteem

The word 'esteem' originates from the Latin *aestimare*, which means to estimate or appraise; thus, self-esteem refers to positive and negative evaluations of oneself (Brehm et al., 2002). Early influencing theories on self-esteem include Cooley's (1902) *looking glass self*, according to which other people serve as a mirror in which we see ourselves, and Mead's (1934) concept that states that we form knowledge of ourselves by imagining what significant others think of us and then incorporating this perception into our self-concept. These theories suggest that self-esteem is formed early in life through interaction with significant others, that is, through attachment. Longitudinal studies support this concept, indicating that the early childhood environment has a long-term effect on self-esteem that can be observed in adulthood (Orth, 2018). Additional longitudinal studies indicate that the familial environment continues to affect self-esteem during childhood and adolescence (Kraus et al., 2020). It is evident that the relationship between the familial environment and the development of self-

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esteem is undisputedly strong, confirming attachment theories (see Section 3.5.2. of the current chapter).

There has been much debate regarding the trait or state nature of self-esteem (Donnellan et al., 2012). Traits and states are concepts used to describe the self and its characteristics (Chaplin et al., 1998). Traits are stable and long-lasting, caused by internal developmental procedures early in life, while states are temporary and brief, caused by external circumstances (Chaplin et al., 1998). Empirical evidence from longitudinal studies that used a latent variable trait–state model from adolescence to adulthood indicate that self-esteem displays both trait-like and state-like components, but the state components were comparatively smaller than the trait ones and that the consistency of trait self-esteem increased with age (Donnellan et al., 2012). Meta-analysis results suggest that psychological traits become more consistent with age and that there is a cumulative continuity with respect to personality development (Caspi et al., 2005). Meta-analysis on traits self-esteem’s stability identified a pattern of increasing stability from adolescence to adulthood (Trzesniewski et al., 2003). This finding is consistent with other results that have identified an increasing consistency of personality traits from childhood to adulthood (Ferguson, 2010; Roberts & Del Vecchio, 2000). Thus, authors believe that there is a developmental aspect involved in the stability of self-esteem (Donnellan et al., 2012). This idea is in line with the early literature suggestion that the organisation of self-esteem is linked with an individual’s developmental periods (Damon & Harts, 1982). Therefore, global self-esteem is best conceptualised as a relatively stable psychological trait that becomes increasingly consistent with age, fitting within the cumulative continuity concept of personality development, as the consistency of self-esteem increases with age (Donnellan et al., 2012).

Studies indicate that trait self-esteem relates to the Big Five personality characteristics, and these factors are researched together in various studies of human

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behaviour (e.g. Bojanić et al., 2019; Joshanloo & Afshari, 2011; Skorek et al., 2014; Weidmann et al., 2017). The correlation between self-related concepts and the Big Five personality traits (e.g. Judge et al., 2002; Robins et al., 2001) has led to the view that self-concepts, such as trait self-esteem, are outcomes of the core personality traits (Headey, 2010). Authors (Asendorpf & van Aken, 2003) distinguish between core (e.g. the Big Five) and surface personality characteristics (e.g. global self-esteem), suggesting that unlike surface characteristics, core characteristics are stable and unaffected by relationship experiences. However, they acknowledge that relationship experiences in early childhood could affect core characteristics and their stability, but more research is needed in this area in order to distinguish between surface and core characteristics (Asendorpf & van Aken, 2003). Research supports the notion that the Big Five adult personality traits have their origins in early temperament (Shiner & De Young, 2013). Research also indicates that temperament can be influenced by environmental experiences, such as attachment (see Section 3.5.1 in this chapter). Combining these lines of thought, it could be suggested that the Big Five traits are also susceptible to relationship experiences, as Asendorpf & van Aken (2003) accepted. Other scholars (McCrae & Costa, 1996, 1999) are less flexible and divide personality characteristics according to their stability (basic tendencies versus characteristic adaptations), suggesting that core and stable personality characteristics, such as the Big Five and temperament, are based on genetic differences and are less affected by life experiences (basic tendencies), while the self-concepts are more susceptible to life experiences (characteristic adaptations). It is believed that the surface characteristics, such as self-esteem, are the developmental result of the interaction between core characteristics and environmental experiences (McCrae, 2009) and develop after the core characteristics are established (McAdams & Olson, 2010). Reviews on the differentiation between core and surface personality traits (Kandler et al., 2014) conclude that self-related concepts, such as self-

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esteem, could be surface characteristics and manifestations of core personality traits, such as neuroticism, extraversion and conscientiousness. Additionally, self-related concepts seem less stable and less genetically oriented than core personality traits, and the genetic variance in measures of self-related concept could be attributed to the genetic variance in the Big Five personality traits (Kandler et al., 2014). However, the direction of causation between core and surface characteristics has not been examined, and these studies have methodological issues (Kandler et al., 2014). For instance, in some studies, self-esteem longitudinally predicted neuroticism (Weidmann et al., 2017). Despite the debate regarding the stability and instability of traits, there is a strong indication in the literature that self-esteem is a trait flexible to relationship experiences. Given the formation of self-esteem early in life, it could be that self-esteem's formation is affected by attachment, as suggested by attachment theories (see Section 3.5.2 in this chapter). Moreover, the trait–state argument could be answered by the principle of cumulative continuity in personality development, as trait self-esteem becomes more stable with age, which is in accordance with its developmental nature. The position of this thesis regarding trait and state self-esteem is that further research is needed in order to revisit the state self-esteem concept. Based on the aforementioned personality theories that regard trait self-esteem as flexible to environmental influences, most notably early relationship experiences, longitudinal studies could be very helpful in elucidating self-evaluation in different social contexts, as the state self-esteem concept proposes. In other words, a comparison of longitudinal design between people with high and low self-esteem regarding their self-evaluation after accomplishments and failures would clarify if the concept of state self-esteem is valid. Research focusing on state self-esteem has not clarified what level of trait self-esteem the participants had prior to their participation in the research. For instance, people with low trait self-esteem are expected to be significantly influenced by a situational failure and generalise it with respect to their overall self-worth, leading to

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analogous mood states (Rosenberg, 1965; Vandellen et al., 2012), and trait self-esteem is even capable of affecting how people adapt to a chronic illness (Juth et al., 2008).

The classic conceptualisation of trait (also termed global) self-esteem, viewed as a cognition about the self (Swann Jr et al., 2007), has been offered by Rosenberg (1965), who described it as a global and unidimensional construct of one's positive or negative attitude toward oneself and one's evaluation of one's own thoughts and feelings of overall worth in relation to oneself. Trait self-esteem is a good predictor of psychological well-being and strongly empirically related to anxiety expressed either as somatic symptoms or psychological manifestations (Rosenberg et al., 1995). It is crucial for psychological health because the way people feel about themselves influences all aspects of life and defines their behaviour (Donnellan et al., 2011; Trzesniewski et al., 2006). The explanation of trait self-esteem affecting psychological health relies on the self-enhancement theory (Baumeister, 1982), which proposes that self-esteem is a core human motive. Trait self-esteem as a basic human motive has been also labelled as the self-maintenance motive (Tesser & Campbell, 1983) and motive for self-worth (Covington, 1984). These theories propose that individuals have a universal need to protect and enhance their feelings of self-worth, and this desire and effort can generate psychological distress (Maslow, 1970). The effort to maintain self-esteem leads to self-protective motives, self-enhancement processes and various coping strategies. In other words, global self-esteem is regarded as one of the most important factors for mental health (Chamberlain & Haaga, 2001) and a significant determinant of human behaviour (Fox, 2003).

Contrary to state self-esteem (e.g. academic, athletic), which varies and depends upon self-evaluation in different social contexts (Brown & Marshall, 2006), trait self-esteem is considered to be a relatively stable trait and a robust developmental characteristic across life

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situations with no gender differences (Brehm et al., 2002). This indicates that its formation in the yearly years of life can be crucial for the way individuals evaluate themselves throughout their lifetimes (Heatherton & Polivy, 1991; Heatherton & Wyland, 2003; Kuster & Orth, 2013; Orth, 2018; Trzesniewski et al., 2003). Studies confirm that trait self-esteem is a stronger predictor of psychological well-being (depression, anomie, general anxiety resentment, anxiety, irritability, life satisfaction, guilt, happiness, negative affect states) than state self-esteem and is possibly the only predictor (Rosenberg, 1995).

The cognitive model of trait self-esteem (McKay & Fanning, 2016) focuses on the pathological critic (Sagan, 1967), which is the inner voice that attacks and judges the self. It is suggested that everyone has a self-esteem-related inner voice, but in those with negative self-esteem this voice is pathological and highly critical, focusing on failures, disregarding accomplishments, negatively labelling the self, generalising a failure to all aspects of life and negatively comparing the self to others (McKay & Fanning, 2016). The critic is formed in the early years of life as a result of the interaction with parents and shapes the way people see themselves, the world and others. Abused and neglected individuals develop a pathological inner voice, which is highly toxic for the self and social relationships (McKay & Fanning, 2016). Thoughts about ourselves impact our emotions, behaviour, the way we perceive the world and our social relationships and are able to prompt the development of somatic symptoms and mental disorders (Kennerley et al., 2007). Negative thoughts and emotions of the self and others lead to unconscious destructive patterns that create distress and emotional upheaval (Kolts & Hayes, 2016; Linehan, 2014; McKay & Fanning, 2016; Young & Bernstein, 2011).

Several clinicians have talked about a critical voice in AN that negatively criticises the patient, dictating and controlling their behaviour (Aya et al., 2019; Pugh & Waller, 2016;

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Tierney & Fox, 2010). Reviews on this matter indicate that it is an ED voice, as it has been reported in all subtypes, but there is controversy regarding the specifics of this voice, as it has not been adequately differentiated between an auditory hallucination and internal intrusive thoughts (Aya et al., 2019). The ED voice is defined as a second or third person's comments on shape, weight and eating and their implications on the patient's self-worth (Pugh, 2016). The inner voice in EDs has been associated with low trait self-esteem (Noordenbos et al., 2014). It could be proposed that what is called an ED voice could be the pathological critic of a very low self-esteem, which, upon the development of the disorder, connects the individual's self-worth with eating patterns and their reflection on body image.

The cognitive model of self-esteem (McKay & Fanning, 2016) is in line with Beck's (1979) cognitive triad of depression, according to which, cognitions about the self, the world and others, along with their expectations from the future are negative in people with depression, defining their behaviour accordingly. In mental disorders, including depression, there exist negative cognitions about the self, which are very strict and stable (Beck, 1987). People with a very low self-esteem feel worthless, hopeless and helpless to such a degree that they cannot tolerate the emotional pain caused by this low self-esteem, leading to suicide attempts (Beck, 1967). Given the developmental fact that the formation of trait self-esteem predates the development of depression, it could be argued that low self-esteem is the major risk factor for depression.

As discussed, trait self-esteem is not only composed of beliefs about oneself but also of associated emotions (Hewitt, 2009). Self-discrepancy theory (Higgins, 1987) further links the self with affect, proposing that the result of the match or mismatch between how we see ourselves and how we want to see ourselves is reflected in self-esteem, causing associated mood states and emotions, most notably depression (Higgins, 1987; 1989). If there is a

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discrepancy between our actual and ideal selves, self-esteem will be low and negative mood states and emotions develop, such as anxiety, depression, guilt, inadequacy, feelings of failure, shame, sadness, resentment and fear of rejection, affecting our behaviour and psychosocial life (Beck, 1967; Hewitt, 2009; Higgins, 1987; Maric et al., 2019). In other words, self-esteem is both the evaluation of ourselves and how we feel about it (Smith & Mackie, 2007). The way we see ourselves and the way we want to see ourselves is affected by how our parents see us, and what they want and expect from us. If we feel that we do not meet their standards, we may feel unloved, emotionally abandoned and rejected (Higgins, 1987). This means that the way we perceive ourselves and the way ourselves are mirrored through significant others create a mental combination that is reflected upon our trait self-esteem. In turn, trait self-esteem is the mirror that reflects our perceptions about ourselves, the world and others. Moreover, people's emotions and behaviour in future relationships with different significant others (e.g. a partner) are continuously based on the self-discrepancy system (Higgins, 1987). The abovementioned combined indicate that attachment, self-esteem and affect are connected in a continuous pattern throughout life, and once self-esteem is created it affects if and how we relate to significant others and how we feel about these relationships. Upon further elaboration, it could be argued that the formation of the false self (ideal) in order to hide the true self (actual) in EDs is the result of damaged self-esteem created by the previously mentioned discrepancy in the presence of early life events that have jeopardised attachment. This discrepancy between the actual and ideal self is able to affect mood states regardless of the presence of psychopathology.

All the above theories on trait self-esteem's development are connected with attachment theories and associated research that place self-esteem's formation within the attachment process. Global self-esteem is considered to be influenced and shaped by both parents' behaviour towards the child and has been proven to be highly sensitive to CEA,

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mediating CEA and psychopathology in adulthood (Finzi-Dottan & Karu, 2006). Reviews on self-esteem further indicate that the way people view themselves influences the way they respond to others' feedback (Swann Jr. et al., 2007), highlighting an empirical link with self-esteem and emotion regulation. Scholars (e.g. Luhtanen & Crocker, 1992) have also broached the experience of collective self-esteem, arguing that people also base their self-esteem on their social identity as they belong to certain groups (e.g. race/ethnicity). In line with this, the sociometer model of self-esteem suggests that people try to maintain high self-esteem because it monitors others' reactions, thus alerting them to potential social exclusion and saving their interpersonal relationships (Leary et al., 1995). In other words, self-esteem serves as a measure (sociometer) of the quality of current and future social relationships (Leary et al., 1995). In this theory, the self-esteem system is composed of both trait and state self-esteem (Leary, 2004). The sociometer definition of global self-esteem states that specific experiences of acceptance and rejection are internalised to form a stable and global perception of one's worth as a social partner (Leary, 2004). A revisit of sociometer theory further explains that self-esteem defines people's social relationships as individuals: those with high self-esteem perceive themselves as being valued by others, and this makes them confident enough to socialise, while those with low self-esteem question their self-worth, feeling insecure to engage in future relationships (Cameron & Stinson, 2017). Elaborating on the early sociometer theory (Leary et al., 1995), self-esteem serves as a means of preserving attachment to significant others across one's lifespan, thereby affecting the social relationships QoL aspect. Further extending its contemporary evaluation (Cameron & Stinson, 2017), self-esteem formed in the early years of life, defines attachment later in life.

Self-esteem is strongly related to the way we emotionally experience life events, as it increases or decreases positive emotions when experiencing success or influences negative emotions following a perceived failure (Greenberg, 2008). This indicates that self-esteem is

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related to and can, therefore, affect emotion regulation development (see Section 3.5.3 in this current chapter). Self-esteem is also believed to play an important role in social identity development and maintenance (Allport, 1955). This has been empirically supported by Cvencek and colleagues (2016), who found that self-esteem is formed and stable enough to be measured by the age of five years old, serving also as identity maintenance (gender identity, attitudes towards groups, me–not me, bad me–good me). Empirical findings also suggest that trait self-esteem modulates the degree of both affective processes in the orbitofrontal cortex during self-reflection and cognitive processes in the medial prefrontal cortex during evaluation of social feedback, indicating both its stability and its effects on state self-esteem and emotions (Yang et al., 2016). Moreover, trait self-esteem, has been found to be very distinct from academic achievements and socio-economic status (Blascovich & Tomaka, 1991), further indicating that is a quite robust psychological construct developed early in life.

Reviews on global self-esteem conclude its important role as a protective and risk factor in a diversity of mental and physical disorders, as well as social functioning, highlighting the importance of self-esteem's inclusion as a core variable in mental health implementation and prevention programmes (Mann et al., 2004). Empirical findings from prospective studies showed that adolescents with high global self-esteem suffer less attention problems, and fewer anxiety and depression symptoms (Henriksen et al., 2017). In line with these results, other studies on adolescents concluded that global self-esteem is associated with academic stress, suicidal ideation, depression and anxiety development, and low QoL (Nguyen et al., 2019). These results indicate a strong link between low trait self-esteem and mood disorders, and they complement previous studies on trait self-esteem's positive and negative outcomes on mental and physical health. In general, people with high self-esteem tend to be happy, healthy, productive and successful, reporting high psychological well-being

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(Baumeister et al., 2003; Dogan et al., 2013). They also persist with difficult tasks for longer (Baumgardner, 1990) and sleep better at night (Brehm et al., 2002). Moreover, high trait self-esteem buffers against stress and negative emotions, enhancing personal adjustment (Leary et al., 1995). It has also been suggested that it alleviates existential stress by functioning as a buffer against the fear of fragility and mortality (Greenberg et al., 1986 as cited in Leary et al., 1995). In contrast, people with low trait self-esteem tend to be more depressed and pessimistic about the future, and are prone to failure, poor health, criminal behaviour and maladjustment (Baumeister et al., 2003; Trzesniewski et al., 2003), and thinking and behaving in ways that impoverish their QoL (Swann Jr et al., 2007). This indicates that low trait self-esteem can affect mood states. Even though trait self-esteem has been empirically linked with physical and psychological health, including psychopathology development, it has been overlooked as a factor in treatment and prevention programmes (Swann Jr et al., 2007). Despite the fact that there is a connection between trait self-esteem, emotions, adjustment and health, it has not been explained why trait self-esteem has this considerable impact (Leary et al., 1995) as it has not been clarified why self-esteem has a significant impact on EDs.

Self-Esteem and EDs/QoL. People with ED symptoms and behaviours report significantly lower self-esteem than those without such problems (Beren & Chrisler, 1990; Fisher et al., 1994; Thomas et al., 2002; Wallace, 2013). Silverstone (1992) considered EDs to be a symptom of low self-esteem, and his view is supported by research (Karpowicz et al., 2009; Newns et al., 2003). Low self-esteem is a well-known transdiagnostic trait in eating pathology (Fisher et al., 1991; Kästner et al., 2019; Newns et al., 2003; Sassaroli & Ruggiero, 2005) and a major risk factor for ED development and maintenance (Adamson et al., 2019; Biney et al., 2019; Cervera et al., 2003; Giovazolias et al., 2013; Gual et al., 2002; Holston & Cashwell, 2000; Katsourani, 2009; Leon et al., 1999; Stice, 2002). There is much empirical support for a link between self-esteem and disordered eating (Fisher et al., 1994; Mintz &

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Betz, 1988; Peck & Lightsev Jr, 2008; Woodward et al., 2019), but the explanation of the relationship between self-esteem and EDs remains unclear (Adamson et al., 2019).

Studies have identified the significant mediating effects of self-esteem when eating pathology is an outcome. For instance, self-esteem is a mediator in the relationship between interpersonal problems and ED symptoms (Lampard et al., 2011). Interpersonal problems refer to difficulties in social relationships marked by issues in bonding and affect (Horowitz et al., 2000) and have been found to result from family dysfunction (Mothersead et al., 1998) with adverse effects on self-esteem (Fairburn et al., 2003). This indicates a link between family dysfunction, self-esteem and EDs, further supported by the mediating role of self-esteem between family dysfunction and the risk of developing an ED (Kroplewski et al., 2019). A further extension of the findings pertaining to interpersonal problems and self-esteem (Lambard et al., 2011) suggests that there is an association between self-esteem and attachment, and emotion regulation and psychosocial QoL. Moreover, studies examining personality traits and EDs concluded that low trait self-esteem was a mediating variable between schizoid, paranoid, self-destructive and borderline personality traits and EDs (Mas et al., 2011). This further indicates trait self-esteem's role in EDs. Additionally, trait self-esteem was the strongest predictor for body image esteem compared to personality characteristics (Skorek et al., 2014), indicating the way trait self-esteem can disturb the relationship with food, leading to EDs.

Self-esteem has been also linked with QoL in EDs, as low trait self-esteem has been determined to negatively affect the QoL of ED patients with the highest association of low trait self-esteem and QoL shown in current and former ED patients (de la Rie et al., 2005). It has been also found to be a predictor of post-treatment QoL in ED patients undergoing enhanced cognitive-behavioural treatment (Watson et al., 2012). Longitudinal studies indicate

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that low trait self-esteem is linked to overall well-being in AN, simultaneously highlighting the chronic and stable presence of low self-esteem (Button & Warren, 2002). Moreover, it has emerged as a strong predictor of QoL in ED patients, even stronger than family functioning, especially in the psychological domain of QoL (Theodoropoulou, 2014). Self-esteem's link to ED patients' QoL further highlights its important role in eating pathology, in that it also affects the treatment outcome.

It has been proposed that eating attitudes will be improved by addressing self-esteem instead of the eating pathology (Adamson et al., 2019; Biney et al., 2019; Geller et al., 1997; Silverstone, 1992), supporting Yellowlees's (1997) argument that low self-esteem is also a maintaining factor of EDs. In support of this, decreases in self-esteem have been associated with increases in ED attitudes (Mora et al., 2017), indicating that low self-esteem can maintain the disorder. Moreover, studies indicate that high self-esteem protects people from EDs, while low self-esteem was found in girls who subsequently developed EDs (Cervera et al., 2003). This means that low self-esteem pre-existed the EDs' development, strongly indicating that it could be a causal factor. The impact of self-esteem on the development of EDs has not been clarified yet (Adamson et al., 2019; Mora et al., 2017), even though study reviews indicate that low self-esteem is a risk, predisposing, precipitating and maintenance factor in EDs (Adamson et al., 2019; Biney et al., 2019; Dakanalis et al., 2017).

An explanation for low self-esteem's significant role in EDs could be its link to body dissatisfaction (Balcetis et al., 2013; Button et al., 1997; Delgado-Floody et al., 2018; Frost & McKelvie, 2005; Park, 2020; Tiggemann et al., 2009). Body dissatisfaction refers to the negative perception and associated emotional responses of a person's internal representations of physical appearance (Thompson et al., 1999) and has been found to directly predict disordered eating in adolescents (Park & Epstein, 2013; Paxton et al., 2006; Sim & Zeman,

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2006). A possible explanation could be that negative self-views are projected onto the body in the form of body shame and dissatisfaction (Woodward et al., 2019), which, along with peer rejection in adolescence, leads to eating pathology (Smink et al., 2018). In this sense, body dissatisfaction and shame could be viewed as consequences of low self-esteem. Moreover, low self-esteem is also believed to be the driving force behind people's desire for thinness (Tiggemann et al., 2009) as it has been found to be lower in repeated dieters than non-dieters (Dykens & Gerrard, 1986). Research into the drive for thinness suggests that low self-esteem is the key factor behind the desire to be thin (Beals, 2004), implying that it could be a consequence of low self-esteem. AN patients display low self-esteem (Adamson et al., 2019; Biney et al., 2019; Karpowicz et al., 2009), supporting earlier findings that low self-esteem is a stronger risk factor than restrictive dieting in AN (Schmidt, 2001). The loss of weight and, ultimately, the control of the body due to low self-esteem could explain the feeling of pride observed in AN (Faija et al., 2017). Additionally, dietary restrictions and weight loss have emerged as responses to social exclusion whereby the individual tries to gain status and control through interventions to their body's appearance (Gatward, 2007; Goss & Gilbert, 2002; Stevance & Price, 2000). This is in line with the sociometer theory and concept of self-esteem serving as a means to preserve attachment and avoid rejection (Leary et al., 1995). This means that low self-esteem is the reason for restrictive dieting and compensating behaviours because self-esteem can cause body dissatisfaction; thus, low self-esteem can prompt the desire to be thin in order to improve one's self-esteem. In line with this argument, Button and Warren (2002) maintain that the need to control the body in EDs is rooted in low self-esteem caused by developmental issues in interpersonal relationships (attachment) and the development of the self in the early years of life. Further to this, AN patients report more submissiveness within their family compared to the general population (Castilho et al., 2014; Gilbert et al., 2003) Submissiveness, along with the fear of negative evaluation, have been

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causally linked with AN (Cardi et al., 2018). Elaborating on this, it could be stated that the fear of rejection, due to low self-esteem caused by poor attachment, leads to emotion dysregulation. Additionally, low self-esteem, mood intolerance, perfectionism and interpersonal differences have been evaluated as maintaining factors in AN, as they predate the onset of the ED (Aberdeen, 2013). Perfectionism is believed to be *masked* low self-esteem (Baldwin & Sinclair, 1996; Egan et al., 2014; Koivula et al., 2002). In other words, poor attachment creates low self-esteem, and the child tries to be perfect in order to please others and avoid rejection. In other words, the imperfect self (the true self) is taken over by the perfect one (the false self) (Winnicott, 1965). There seems to be a consistent link between attachment, self-esteem and emotion regulation in EDs, with self-esteem being the connecting factor. In treatment terms, it would be more valuable to address the cause (low self-esteem) than its symptom (food restriction) only. Empirical findings support this suggestion as self-esteem-focused group interventions for AN patients improved both their self-esteem and treatment outcomes (Adamson et al., 2019; Biney et al., 2019). Additionally, self-esteem has been linked with binge eating (Lo Coco et al., 2011) and has been found to be lower in current and past BN patients (Dykens & Gerrard, 1986), indicating that it is not improved by a reduction in ED symptoms, further supporting the notion that it is better to address the cause of the symptom than the symptom alone. The literature supports the argument of addressing self-esteem instead of just the ED symptoms. Empirical evidence demonstrates that ED patients can benefit from a treatment focused on self-esteem, reducing their symptoms (Adamson et al., 2019; Biney et al., 2019; News et al., 2003). Specifically, 33 adult female ED patients (eight AN, 12 BN, nine BED, four EDNOS) completed eight one-and-a-half-hour sessions of group self-esteem skills training (News et al., 2003). These patients joined the group after completing their individual therapy, and the main target of the group intervention was to break the link between low self-esteem and shape and weight

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concerns. Scores obtained before and after this group intervention indicate that eating attitudes, self-esteem and depression were significantly improved. This finding, along with the fact that in longitudinal studies low self-esteem has been found in girls who later developed an ED (Cervera et al., 2003), could be interpreted as a strong indication that low trait self-esteem plays an important role in the development and maintenance of eating pathology. One could argue that it was the combination of individual ED psychotherapy along with the group self-esteem intervention that produced this result. However, the scores were obtained prior to the group therapy, after having finished their standard treatment. That the scores had improved by the end of the self-esteem group sessions clearly indicates the significant contribution of self-esteem. Another important highlight of this self-esteem-based intervention (Newns et al., 2003) was the improvement of depression separate from ED behaviours. ED attitudes, like those observed in AN, can cause physiological changes due to being underweight, starvation and malnutrition that could negatively affect mood (Altemus & Gold, 1992; Rao et al., 2008). This has led authors to suggest that depression in AN is caused by starvation (Altemus & Gold, 1992). Based on this, it could be argued that the improvement of ED symptoms via self-esteem could explain the improvement in depression in Newns et al.'s (2003) study. However, this study did not just include AN patients: BN, BED and EDNOS patients combined accounted for the majority of this study's participants. This means that depression did not improve as a result of increased calorie consumption. Moreover, studies on AN and depression have demonstrated that depression is not a central factor in AN (García-Alba, 2004). The finding that depression symptoms improved when self-esteem increased offers grounds to revisit the role of depression in EDs.

A perspective on EDs and depression proposes that both disorders may develop from common factors (Casper, 1998). Empirical findings suggest that EDs and depression share feelings of failure as a common factor (Cooper et al., 2006; Waller et al., 2001), leading

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scholars to suggest that further exploration is needed on this topic (Fox & Power, 2009). However, feelings of failure are a consequence of low self-esteem (Hewitt, 2009; Higgins, 1987). Low self-esteem is a common identified risk factor of EDs and depression. Self-esteem is affected by attachment, which is sensitive to non-physical abuse, as previously discussed. Low self-esteem can lead to body dissatisfaction, among other things, and body dissatisfaction has been associated with depressive symptoms (Morken et al., 2019). Moreover, the burden of struggling with a chronic illness can lead to depression (Simon, 2001). If depression is viewed as an outcome of EDs and not as a predecessor, it explains why improving self-esteem's had a positive effect on depression (Newns et al., 2003), meaning that by targeting the primary risk factor (self-esteem), subsequent pathologies will also improve.

Low self-esteem is clinically considered a manifestation of depression (Lynum et al., 2008), but it is also an acknowledged risk factor for the development of mood disorders (Scmitz et al., 2003). In other words, self-esteem both affects depression's development (vulnerability model) and is affected by it (scar model), with some studies only validating the vulnerability model (Orth et al., 2008) while others confirm both (Steiger et al., 2015). A possible explanation for this relationship, regardless of the empirical findings' inconsistency, could be that low self-esteem is a risk factor for the development of depression, and when depression has manifested, it further lowers already low self-esteem. Empirical results support the vulnerability model instead of the scar one (Orth et al., 2008), suggesting that in the self-esteem–depression relationship, self-esteem is more important. Meta-analytic reviews have concluded that self-esteem's impact on depression was significantly higher than depression's on self-esteem, concluding that in the relationship between self-esteem and depression, self-esteem is the contributing risk factor (Sowislo & Orth, 2013). Empirical findings support that trait self-esteem is the risk factor for depression rather than state self-

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esteem (e.g. Orth et al., 2014). Moreover, longitudinal studies indicate that trait self-esteem affects trait depression (Takagishi et al., 2011), and findings also highlight the direct and long-lasting effects of low self-esteem on depression (Park & Yang, 2017). Furthermore, studies evince that low self-esteem is present in ED patients in the absence of depression (Silverstone, 1990), meaning that low self-esteem is not caused by depression in eating pathology. Scholars have argued that a genetic predisposition to depression (trait depression) may facilitate the development of an ED (Casper, 1998). Yet, as was previously mentioned, trait self-esteem can affect trait depression. All the aforementioned indicate that trait self-esteem might be a larger risk factor for EDs than depression.

Summarising the above, there is empirical evidence that low trait self-esteem predates EDs development, depression is not present in all ED patients contrary to low self-esteem, low self-esteem is present in depression and trait self-esteem's impact on depression is stronger than depression's on self-esteem. It can be proposed that the much-discussed relationship between depression and EDs can be explained through low self-esteem, meaning that EDs could be a consequence of low self-esteem and not of depression.

Self-Esteem and Non-physical Childhood Abuse. CEA can lead to negative core beliefs about the self, severely affecting self-esteem (Finzi-Dottan & Karu, 2006; Frankel, 2002; Kent & Waller, 1998, 2000; Waller et al., 2007). Wiehe (1990) proposed that the sufferers of psychological maltreatment often respond by internalising the expressions of maltreatment and accepting them as true, thus, impacting their self-esteem. Kent and Waller (1998, 2000) hypothesised that self-esteem could mediate the relationship between CEA and ED pathology, stating that CEA could lead to low self-esteem more than any other form of childhood abuse, as it may be interpreted as a personal attack on the self. Yates (2004) also suggested that self-harm (such as ED behaviours) may reflect a physical manifestation of the

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individual's negative self-representations, which are the consequence of childhood maltreatment.

Self-esteem has mediated the relationship between childhood neglect and eating pathology in non-clinical samples (Kennedy et al., 2007), as well as between CEA and adult psychopathology (Finzi-Dottan & Karu, 2006). This indicates that trait self-esteem is developmentally sensitive to non-physical childhood abuse with consequences in adult life regardless of both psychopathology's presence and specific psychopathology's presence. Groleau and colleagues (2012) posited that CEA might influence ED symptom severity by impacting the individual's self-esteem due to repeated insults and criticism, as well as the capacity for affect regulation. Burns and colleagues (2012) proposed that self-esteem should be examined in future research, as they believe that it could be a pathway through which CEA affects eating behaviour. Specifically, they maintain that the devaluation produced by CEA could be integrated into the individual's self-concept and contribute to low self-esteem.

As indicated so far in this chapter, research shows a strong link between self-esteem and non-physical childhood abuse, between self-esteem and eating disorder pathology, and between self-esteem and psychosocial QoL. Moreover, low trait self-esteem has been found in all ED subtypes, indicating its transdiagnostic nature. The sequence of events could be that non-physical child abuse, in the form of repeated criticism or insults, may lead to reduced self-esteem (and self-harming behaviour), body dissatisfaction (triggered by concerns about weight and shape), dietary restraint (as an attempt to offset perceptions of a negative body image), leading to severe food restriction, or bingeing and purging behaviours (i.e. a clinical diagnosis of an ED). Despite the fact that multiple studies have implicated self-esteem as possessing an important connection to both ED development and non-physical child abuse in early life, it has not been tested as a mediator between non-physical abuse and ED, nor

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between non-physical abuse and psychosocial QoL. Study 2 overcomes that limitation. All the studies mentioned in this section dealt with self-esteem as a trait, linking it to EDs. The vast majority of them, if not all, have measured trait self-esteem through Rosenberg's self-esteem scale, as do the thesis studies. This thesis's studies aim to demonstrate self-esteem's continuous developmental role in eating pathology by relating it to stable psychological factors and situations formed and occurring in early life. Therefore, it is regarded as trait rather than state self-esteem.

3.6. Non-physical Abuse and Eating Disorder Patients: Continuation into Adulthood

It has been proposed that women with AN are trapped in an abusive/toxic relationship with their disorder, resembling that of domestic violence relationship (Tierney & Fox, 2011). Expanding this view, it can be suggested that women with an ED are vulnerable to abusive experiences in a continuation from childhood to adulthood, as a link has been established with non-physical childhood abuse and adult EDs (Studies 1 and 2). It could be hypothesised that the proposed entrapment within the abusive ED relationship (Tierney & Fox, 2011) is the result of the impaired core psychological factors proposed in this thesis. This impairment does not allow ED patients to step out of abusive relationships, making them vulnerable and prone to victimisation. This hypothesis is in line with the suggestion that people who have been psychologically maltreated internalise the expressions of maltreatment and accept them as true (Wiehe, 1990). This further indicates that emotional abuse can affect self-esteem, leading to self-blame and perpetuating the feeling of victimisation. In addition, the damage to the cognitive mechanisms responsible for acknowledging trauma, due to high betrayal trauma from a caregiver in early life, makes people prone to abuse continuation (DePrince, 2005; Gobi & Freyd, 2009) in a continuous effort to establish the desired attachment at any cost.

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Children's primary experiences with their caregivers, before attending school, formulate their ability to adapt to new environments, cope with stressors and form social relationships with their peers, as parenting behaviour serves as a model upon which children base their behaviour towards others and their expectations of relationships (Ladd, 1992). Social learning theory (Bandura, 1978), family relational schema (Perry et al., 2001) and attachment theory (Bowlby, 1973) indicate that children who are brought up in a hostile and abusive environment are at risk of learning and being familiar with negative relationship patterns. Research suggests that abused children encounter multiple forms of victimisation during their lives, possibly because they display certain characteristics that portray them as easy targets by others, such as submissiveness, in order to preserve safety when coming from a chaotic familial environment, as well as feelings of helplessness and low self-confidence because they have learned that their needs are ignored, so they are probably unimportant (Duncan, 1999, 2004; Finkelhor & Browne, 1985; Koenig et al., 2000). This could lead them to generalise their cognitions and behaviour in extra familial relationships across their lifespan (Schwartz et al., 1993; Wolke & Samara, 2004), indicating the long-lasting consequences of an adverse home environment (Copeland et al., 2013). Expanding this argument, disrupted attachment and low self-esteem caused by abuse and neglect in childhood could lead to a deficiency in effective coping, adaptation and socialising later in life due to vulnerability to harm. Vulnerability to harm is a maladaptive schema defined as an exaggerated fear that imminent catastrophe will strike at any time that one is powerless to prevent (Young et al., 2003).

Interpersonal trauma survivors display vulnerability to harm (Karatzias et al., 2016), and empirical evidence indicates that there is a link between non-physical childhood abuse and the vulnerability to harm schema (Hartt & Waller, 2002; O'Dougherty Wright et al., 2009). There are also studies indicating that students from violent and dysfunctional families

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are more vulnerable to school bullying victimisation (Craig et al., 1998; Lereya et al., 2013; Mohr, 2006). This could suggest a link between being bullied in childhood and suffering non-physical childhood abuse, since CEA and neglect are present in dysfunctional and hostile families. Maltreatment has also been found to place children at risk of victimisation by their peers, and bullying victims have displayed problems with emotion regulation (Shields & Cicchetti, 2001). In the same study, the effect of maltreatment on children's risk of bullying and victimisation were mediated by emotion dysregulation. In other studies, low self-esteem was associated with violence and bullying victimisation (Delfabbro et al., 2006; O'Moore & Kirkham, 2001; Rigby & Slee, 1991; Slee & Rigby, 1993). This suggests that non-physical abuse that causes emotion dysregulation makes people susceptible to later victimisation due to the vulnerability to harm schema and low self-esteem. Furthermore, this is an enduring, lifelong effect. Moreover, low self-esteem and experiences of childhood abuse were associated with partner violence victimisation and perpetration (Papadakaki et al., 2009). According to the above, it is evident that people who have been psychologically maltreated are prone to the continuance of abuse, even outside the primary abusive environment.

Research findings indicate that there is stigmatisation against ED sufferers (Crisp, 2005; Froom, 2007; Mond et al., 2006; Roehrig & McLean, 2010; Zwickert & Rieger, 2013), which may indicate the presence of non-physical abuse in adult life, affecting their psychosocial QoL. In support of this argument, studies indicate that people differentiate EDs from other mental illnesses because they have the view that ED patients are responsible for the disorder, a point of view similar to that of substance abusers (Crisp, 2005; Mond et al., 2006; Roehrig & McLean, 2010). This could be associated both with the media presentation of the female body, along with the ego-syntonic nature of certain ED features (e.g. dietary restraint) that, in combination, make the disordered behaviour look desirable (Mond et al., 2006; Peroutsi & Gonidakis, 2011). Additionally, ED patients have control of their illness

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compared to others, such as people with schizophrenia (Roehrig & McLean, 2010). In this respect, it has been suggested that the familial and social environment may envy the patient in the early stages of the disorder for being able to lose weight and maintain a *socially desirable* female body figure, especially in cases of AN (Mond et al., 2006).

In studies examining the bullying and stigmatisation of people with ED compared to people with depression, the ED group reported higher levels of being ignored by their peers (Froom, 2007), and stigmatisation was greater for them compared to people suffering from depression (Roehrig & McLean, 2010). Evidence also suggests that there is a correlation between being bullied about one's weight and appearance as a child and later developing an ED (Sweetingham & Waller, 2008). This study provided evidence for the relationship between being bullied by family and peers about one's physical appearance and body dissatisfaction, which was mediated by shame. Shame is an emotion linked with low self-esteem, as is body dissatisfaction. Bullying can be considered a form of emotional abuse that could lead to low-self-esteem, body dissatisfaction, the drive for thinness and depression, escalating the chances of developing an ED (Eisenberg et al., 2003; Hill & Murphy, 2000; Jackson et al., 2000).

In sum, the theoretical and empirical literature suggests that individuals who have experienced childhood non-physical abuse are at risk of victimisation and trauma in later life. Yet, what is missing from the literature is empirical evidence indicating whether adult ED patients who have suffered non-physical childhood abuse continue to face non-physical abuse (i.e. victimisation, bullying, stigma) as adults from their families, employers, peers, romantic partners and health professionals due to their eating pathology or their past non-physical abuse and its enduring consequences. Study 3 will try to answer this by exploring ED patients' experiences using qualitative methodology.

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Waller and associates (2007) expanded O'Hagan's (1995) definition of emotional abuse to include adult experiences, arguing that many patients report emotional abuse in their adult years, either as a primary experience or as a revival of their relevant childhood experiences. The latter is in agreement with the psychoanalytic view offered by Freud (1920) within the term *repetition compulsion* to explain that humans seek comfort with the familiar. He famously defined it as the desire to return to an earlier state of things. Waller et al.'s argument (2007) fits perfectly with the present author's clinical experience. Since this has not been researched before, Study 3 will be a pioneer study.

3.7. Summary

In this chapter, a relationship was presented between family dysfunction, ED/QoL and self-esteem, indicating that self-esteem could be an important mediator (Study 1). Next, family dysfunction and the invalidating environment were conceptualised as childhood non-physical abuse (predictor variable), incorporating Kent's and Waller's (2000) and Linehan's (1993) theories. This chapter also demonstrated how this type of abuse affects emotion regulation, self-esteem and attachment (mediators), leading to eating pathology and poor psychosocial QoL (outcome variables in Study 2a). The common psychological presentations and aetiology between ED and BPD were then presented, leading to an adaptation of the biosocial theory for eating disorders. Temperament was retained in this new model, given the established relationship between childhood non-physical abuse and EDs, and attachment was added as a mediator since an invalidating childhood environment can disrupt attachment (Study 2b). Finally, the continuation of childhood non-physical abuse in adulthood was discussed since the effects of abuse on core psychological factors are long-lasting (Study 3).

A detailed description and criticism of the theories underpinning the cognitive models of EDs, as well as a detailed description of the basic emotions regarding EDs is beyond the

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scope of this thesis. Such detailed and thorough work already exists (e.g. Fox, 2009; Fox & Froom, 2009; Fox & Power, 2009). The aim of this thesis is to present a concise developmental model of EDs that could possibly explain a great amount about the origins and maintenance of eating pathology. This model combines psychodynamic and cognitive theories, linking non-physical abuse with emotion regulation, attachment and self-esteem, while also providing a more complete conceptualisation of the invalidating environment (Linehan, 1993) and a mediational conceptualisation of the SPAARS-ED model (Fox & Power, 2009). This developmental transdiagnostic psychopathology perspective can guide understanding of the complexity of outcomes associated with experiences of childhood non-physical abuse in relation to EDs. Chapter 4 presents this thesis's studies' methodology.

Chapter 4: Aims and Methodology

4.1 Overview

This chapter presents the thesis's aim and an overview of the research questions addressed in each of the three studies. A general discussion regarding sample selection and recruitment for all studies included in this thesis follows. Finally, the methodology and data analysis techniques used for each study are discussed.

4.2. Aims of the studies

Study 1 explored self-esteem as a mediator between family functioning, EDs and psychosocial quality of life (QoL). Study 2 included two studies that shared the same population. To acknowledge this, these studies will be referred to as Studies 2a and 2b. Study 2a built on Study 1 by conceptualising family dysfunction as non-physical abuse and testing its effects on EDs and psychosocial QoL with self-esteem, attachment and emotion regulation as mediators. Study 2b continued the conceptualisation of family dysfunction as non-physical abuse and adapted Linehan's (1993) borderline personality disorder (BPD) biosocial theory for eating disorders (EDs). Specifically, it tested the effects of non-physical abuse on EDs, QoL, emotion regulation and self-esteem, with attachment and temperament as mediators. Study 3 was qualitative and explored ED patients' experiences of the continuation of non-physical abuse in adult life, as well as patients' experiences of the potential link to ED onset and maintenance. The latter are explored through ED patients' unique experiences, using interpretative phenomenological analysis (IPA).

4.3. Methodology of the Studies

4.3.1. Mixed Methodology

A mixed methodology was employed to carry out the three thesis studies in order to answer related aspects of the same research questions (Schoonenboom & Johnson, 2017;

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Teddlie & Tashakkori, 2009). Mixed methods can provide a rigorous, robust and deeper understanding of a phenomenon (McKim, 2017; Schoonenboom & Johnson, 2017) and are important for answering multiple research questions (Azorin & Cameron, 2010). Structural equation modelling (SEM) was used for the first two studies to better understand relationships and the underlying mechanisms through which the predictor variables (X) influence the outcome variables (Y) via mediators (M). Data were analysed using IBM SPSS Statistics 21.0 (IBM Corporation, 2012) and AMOS 22 (Arbuckle, 2013) with the significance level set at .05 to conserve statistical power. IPA was employed for Study 3 (Smith et al., 2009).

Mixed methods approaches include four designs and their variants (Creswell & Clark, 2007). These designs are the triangulation design, used to expand quantitative results with qualitative data (there are four models within the triangulation design, including the convergence model, the data transformation model, the validating quantitative data model and the multilevel model); the embedded design, used to embed a qualitative component in a quantitative design particularly when developing a treatment model (experimental model and correlational model); the explanatory design in which qualitative data is used to explain quantitative results (follow-up explanations model and participant selection model) and the exploratory design, used for questionnaire development (instrument development model and taxonomy development model). The triangulation design is considered the typical framework for mixed methods research (Creswell & Clark, 2007), and the triangulation design's convergence model is considered the traditional model for a mixed methods design (Creswell, 1999).

A triangulation design was preferred for this thesis in order to further explain and expand the quantitative results with qualitative data (Creswell & Clark, 2007) with the aim of

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producing substantiated conclusions about the consequences of childhood non-physical abuse and their role as risk factors in eating pathology. Data obtained were different but complementary, offering in-depth understanding (Morse, 1991). Data for each study were collected at different times, as each study had different participants (convergence model). This approach resulted in different sample sizes for each study, notably, a smaller sample for Study 2, due to recruiting from all available clinics in Athens, Greece. In practical terms, this meant that recruitment for Study 2 began immediately after Study 1 ended; therefore, it sampled the new ED outpatients who had entered the clinics for psychotherapy.

Regarding the sequence of the studies, the quantitative studies took place first as it was important to first establish a link between childhood non-physical abuse and eating pathology (Study 2) in order to further explore this phenomenon in adulthood (Study 3). Specifically, Study 1 aimed to establish the potential link between self-esteem and EDs and clarify the much-discussed role of family dysfunction. This step was necessary in order to reconceptualise family dysfunction as childhood non-physical abuse and maintain self-esteem as a constant, important mediating factor. The results of Study 1 offered initial merit and empirically based justification to proceed with the core theoretical models of this thesis explored in Study 2. Study 2 represents the core hypothesis of the thesis regarding the role of childhood non-physical abuse in EDs. Once this link was empirically established, Study 3 served a twofold purpose. First, the study aimed to explore Study 2's findings by giving the participants a voice to see if the empirical findings are grounded in their lived experiences. Second, the study expanded these results by exploring, from the participants' perspectives, the process of continuation of childhood non-physical abuse into ED patients' adult lives. The twofold purpose realised this thesis's reasoning that certain psychological factors are responsible for ED maintenance, and these same factors are also responsible for ED onset. That is, ED maintenance could be implied from the quantitative studies, but the final

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qualitative study would elucidate the consequences of childhood non-physical abuse from the participants' point of reference, linking their experiences to the onset and maintenance of ED symptoms. Moreover, the transdiagnostic nature of the models used in Studies 1 and 2 had to be quantitatively tested before proceeding with the qualitative study that then offered a deeper insight in the transdiagnostic approach with respect to developmental factors and EDs. Additionally, only the qualitative study could explore the psychological mechanism behind the non-physical abuse continuation, as well as whether this mechanism is linked with ED onset and maintenance, as there is no research data regarding this topic in the literature and no questionnaires to measure this.

One could possibly argue that the qualitative study could have occurred second (explanatory design) or even first (exploratory design). These designs would have been appropriate if there were not identified risk factors in the ED literature, which therefore needed identifying via interview. However, the core variables causally involved in EDs are already known but to the best of my knowledge, have not been empirically examined altogether. Moreover, the purpose of this thesis was to examine the connection of these risk factors to childhood non-physical abuse, which has been overlooked in both the literature and clinical practice. Thus, this series of studies did not seek to resolve the lack of theory nor identify the range of constructs that contribute to ED development. Rather, the studies aimed to expand and empirically test specific theories and relations between variables proposed in the literature by offering a transdiagnostic perspective to explain both the origins and maintenance of EDs, focusing on the core developmental factors. This means that Study 3 is a separate, important study, but it is theoretically connected with the first two quantitative studies. Moreover, by being the last study in the sequence, Study 3 offers valuable insight into the results of the quantitative studies, as the continuation of non-physical abuse in adulthood has not been researched before, either for ED patients or any other population.

4.3.2. Sample Size and Statistical Power

The Quantitative Studies: Studies 1 and 2. The sample size was obtained by calculating the alpha level (.05), the number of predictors, the effect size (.15) and power (.80), according to Cohen (2001). The alpha level is the amount of risk the researcher is willing to take regarding the confirmation of the null hypothesis (Cohen, 2001). The widely acceptable alpha of $p < .05$ was used (Cohen, 2001) to reject the null hypothesis. The number of predictors is determined by using the independent variables (Green, 1991). The effect size is the measure of the magnitude of the relationship (Cohen et al., 2003). A medium effect size of .15 was used. Statistical power refers to the probability of rejecting the null hypothesis (Cohen et al., 2003) and the general consensus in behavioural sciences is that a power of .80 (Cohen, 2001), which was selected for the current studies, is sufficient. An a priori calculation was used to determine the optimum number of participants for the first two studies, following Fritz's and MacKinnon's guidelines (2007) combined with a thorough review of the literature's recommendations for SEM sample size.

Although there is no general consensus regarding SEM sample size (Christopher, 2010), various general rules for determining adequate sample size can be found in the literature. Typical sample sizes for SEM usually require 200 participants (Kline, 2011) to avoid a Type I error. However, Ding and colleagues (1995) argued that the minimum sample size sufficient for SEM analysis is generally 100–150 participants, with others recommending 150–300 cases (Hair et al., 2006; Hutcheson & Sofroniou, 1999). Others recommend at least 300 cases (Norušis, 2005), and some propose a minimum of 50–70 for low Type I error rates (Iacobucci, 2010; Sideridis et al., 2014). In general terms, sample sizes below 100, between 100–200 and over 200 are considered small, medium and large, respectively (Kline, 2005). Others propose that there should be 51 more cases than the number of variables used in the

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analysis (Lawley & Maxwell, 1971), or ten cases per variable (Nunnally, 1967 as cited in Wolfe et al., 2013).

Studies have indicated that samples smaller than 200 can yield accurate results, offering a robust model (Dunkley et al., 2010; Finzi-Dottan & Karu, 2006; Gardner et al., 2014). For example, in psychological research, sample sizes of between 135–150 participants are deemed acceptable, especially with the use of software, such as AMOS, that can efficiently handle small sample sizes (Mesly, 2015). Guadagnoli and Velicer (1988) determined that when researchers selected variables that were good indicators of a component, 150 observations yielded accurate solutions.

A more precise and robust method is to consider model complexity in terms of the ratio of the sample size to the number of free parameters estimated in the model. Some authors suggest that sample size should be at least 10 times the number of free model parameters (Mueller, 1996; Raykov & Marcoulides, 2006). Others suggest a ratio of 5 to 10 per free parameter (Bentler & Chou, 1987) and others, a ratio of 3 to 5 per free parameter (Bollen, 1989). Yet, there is limited agreement for estimating the sample size for adequate power regarding SEM. Quintana and Maxwell (1999) suggested that some goodness-of-fit indices could perform adequately with sample sizes of 100 participants, though 200 participants remained the safe choice. This rule is accepted by researchers with the additional caveat that 150 participants is the minimum amount required to reduce estimation error in order to obtain a correct model, adequate power and reliable results (Grall, 2011; Holbert & Stephenson, 2002; Jung et al., 2015; Sivo et al., 2006; Teo, 2009). However, review studies regarding SEM sample size have demonstrated that many of the abovementioned rules of thumb could be over-evaluated, and that sample sizes could begin from even 30 participants (Wolfe et al., 2013) or 50–70 participants for a model with 4 latent variables (Sideridis et al.,

2014). There is also the argument that fit indices are sensitive to small sample sizes, tending to over-reject valid models especially when $n < 100$ or $n < 200$ (Chen et al., 2008).

Study 1. Pre-existing data collected for the author's master's study (88 ED patients and 44 controls) were used, but the number of participants was increased to permit mediational analysis, leading to a sample of 154 ED patients and 154 controls to detect for effect sizes in the small to medium range (Fritz & MacKinnon, 2007). The desired sample for detecting paths of medium effect sizes in mediational models is 74 (Fritz & MacKinnon, 2007), but the goal was a sample size that would enable the detection of effect sizes in between the small to medium range. This number of participants satisfies Bentler's and Chou's (1987) rule of 5 to 10 participants per estimated parameter for Study 1's SEM model.

Study 2. Study 2 recruited a new population and numbered 268 Greek adult female participants (80 ED patients and 188 controls). The optimum number of participants for Study 2 in order to detect small to medium effect sizes was the same as in Study 1 (74) (Fritz & MacKinnon, 2007). Again, this number of participants satisfies Bentler's and Chou's (1987) rule of 5 to 10 participants per estimated parameter for Study 2's SEM model.

The Qualitative Study: Study 3. This study employed six adult female ED outpatients. According to Smith and colleagues (2009) there is no right answer regarding the sample size, as IPA can be conducted with as many as twenty-five participants or as few as one (Alase, 2017, Glasscoe & Smith, 2008; Smith, 2004). IPA studies are usually conducted on a small sample due to their idiographic and experiential focus (Smith & Osborn, 2003). Sample homogeneity is important in IPA in order to gain a thorough understanding of the participants' similar lived experiences (Creswell, 2013), but female ED patients with past and present experiences of non-physical abuse, who are aware of this abuse and are willing to discuss it, do not come in large homogenous numbers. Authors suggest that three participants

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is a useful number for students who use IPA for the first time, as it allows a thorough examination of the similarities and differences without an overwhelming amount of data (Smith & Osborn, 2008). A single-case analysis has also been proposed (Smith, 2004), and, consistent with the latter guidance, there are published IPA studies with one participant (Glasscoe and Smith, 2008). Published IPA studies employing ED patients can be found with four (Papathomas & Lavalley, 2010; Plateau et al., 2018; Wu & Harrison, 2019), five (Curtis & Davis, 2014; Mortimer, 2019; Watson et al., 2020), six (Fox & Diab, 2015; Towner, 2020) and eight participants (Ison & Kent, 2010; Pemberton & Fox, 2013). Additionally, IPA studies focused on overweight participants can be found with four and six subjects (Maxwell, 2019; Miles & Barrow, 2018). Overall, small sampling for IPA (3–6 participants) has been confirmed as efficient (Smith et al., 2009). Based on the above, six participants were considered sufficient.

4.3.3. Measures and materials

Study 1

1) World Health Organisation Brief Quality of Life Assessment Scale (WHOQOL-BREF)

The WHOQOL-BREF is a 26-item self-administered instrument and is an abbreviated version of the World's Health Organisation Quality of Life assessment (WHOQOL-100; World Health Organisation, 1996). The WHOQOL-BREF contains one item from each of the 24 facets of WHOQOL-100 plus two items from the "Overall QoL" and "General Health" facets (WHOQOL Group, 1998b). These facets were agreed by international consensus (Skevington, Lotfy et al., 2004; Skevington, Sartorius et al., 2004). WHOQOL-BREF is widely used to measure QoL both in clinical and healthy population and it provides data for clinical and research purposes covering many aspects of an individual's life (Esch et al.,

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2011). The WHOQOL-BREF is a multi-dimensional profile of people's QoL instead of a single index following the subjective well-being tradition in the behavioural sciences (Billington et al., 2010; Diener & Suh, 1997; Huang et al., 2006; Skevington, Lotfy, et al., 2004; WHOQOL Group, 1994; 1998a; World Health Organisation, 1996, 1997). WHOQOL-BREF has been used in ED studies (Mond et al., 2004c; Theodoropoulou, 2014).

The WHOQOL- BREF examines QoL in four domains: Physical Health (7 items: Q3, Q4, Q10, Q15, Q16, Q17, Q18), Psychological Health (6 items: Q5, Q6, Q7, Q11, Q19, Q26), Social Relationships (3 items: Q20, Q21, Q22) and Environment (8 items: Q8, Q9, Q12, Q13, Q14, Q23, Q24, Q25). These items are scored on a 5-point Likert scale with higher scores indicating better QoL and the three negatively worded items (Q3, Q4, Q26) are reverse scored. The first two items (Q1, Q2) examining the individual's perception of Overall QoL and the General Health respectively, are examined separately on a 5-point Likert scale as well, and they can either produce a mean score or be scored and interpreted individually. These raw mean scores are multiplied by 4 in order to make each domain's mean score comparable with the domain mean scores of WHOQOL-100, so after the transformation they will range among 4-20. If a domain has a missing item, the mean of the other items in this domain is substituted, but if more than 20% of data are missing from a domain, the assessment is discarded (World Health Organization, 1996). It has excellent psychometric properties performing according to international standards (Skevington, Lotfy et al., 2004; Skevigton, Sartorius et al., 2004; WHOQOL Group, 1998b).

The WHOQOL-BREF has been standardised (Ginieri-Coccosis et al., 2006), and widely used both in clinical and non-clinical populations in Greece (Ginieri-Coccosis et al., 2012; Ginieri-Coccosis et al., 2008; Hyphantis, 2007; Papanikolaou et al., 2012; Theofilou, 2010, 2012, 2013; Theofilou & Panagiotaki, 2010; Vairli, 2011).

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2) *Rosenberg Self-esteem scale (RSES)*

The Rosenberg Self-esteem Scale (Rosenberg, 1965) is a 10-item self-report measure of explicit global self-esteem (Gailliot & Schmeichel, 2006). Items are rated on a four point Likert scale (3: strongly agree, 2: agree, 1: disagree, 0: strongly disagree). Five items are positively worded (1, 3, 4, 7, 10) and five are negatively worded (2, 5, 6, 8, 9). The higher the total score, the higher the self-esteem. The total score ranges between 0-30 and if a value is missing from an item, the calculated mean score of that respondent's nine answered items can replace the missing value (Tinsley & Brown, 2000). The negative worded items are reverse scored. The RSES is reliable and valid and has been translated and adapted in 28 languages having been used in cross cultural studies in up to 53 different nations (Schmitt & Allik, 2005). The RSES has been standardized for use in a Greek population (Spanea et al., 2005) and has been used in numerous studies in Greece (Giovazolias et al., 2013; Griva & Anagnostopoulos, 2010; Koumi & Tsiantis, 2001; Koutra et al., 2010; Lyrakos et al., 2012; Michou, & Costarelli, 2011; Papadakaki et al., 2009; Stathopoulos & Sourtzi, 2013; Tsaoussis & Kerpelis, 2004; Vassilopoulos & Brouzos, 2012) with good internal consistency ($\alpha = .84$) (Spanea et al., 2005); $\alpha = .90$ (Theodoropoulou, 2014).

3) *McMaster Family Assessment Device (FAD)*

The McMaster Family Assessment Device (Epstein et al., 1983) is a 60-item self-reported screening instrument rated on a 4 point Likert scale (from strongly agree = 1 to strongly disagree = 4) that distinguishes between healthy and unhealthy family functioning (ranging from 1 = healthy to 4 = unhealthy). The items of each scale are statements that one could make about one's family and they are rated according to the individual's level of agreement or disagreement on these statements. Endorsed answers are coded 1-4 for the 25 positively worded items and the 35 negative worded items are reverse scored (R), so that the

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higher scores indicate poorer family functioning. If more than 40% of the items on a scale are incomplete, the scale score is not calculated (Grotevant & Carlson, 1989; Ryan et al., 2005).

There are seven clinically relevant dimensions, which form the following scales: Problem Solving (6 items: 2, 12, 24, 38, 50, 60), Communication (9 items: 3, 14R, 18, 22R, 29, 35R, 43, 52R, 59), Roles (11 items: 4R, 8R, 10, 15R, 23R, 30, 34R, 40, 45R, 53R, 58R), Affective Responsiveness (6 items: 9R, 19R, 28R, 39R, 49, 57), Affective Involvement (7 items: 5R, 13R, 25R, 33R, 37R, 4R, 54R), Behaviour Control (9 items: 7R, 17R, 20, 27R 32, 44R, 47R, 48R, 55) and General Functioning (12 items: 1R, 6, 11R, 16, 21R, 26, 31R, 36, 41R, 46, 51R, 56). These dimensions are consistently found by researchers, clinicians, and family life educators to be associated with healthy or unhealthy family relationships (Arcus, 1987; Beavers et al., 1985; Connan & Treasure, 2000; Cunha et al, 2009; Epstein et al., 1993; Erol et al., 2007; Garnfinkel et al., 1983; Gottman, 1994; Lattimore et al., 2000; Lerner, 1986; Olson et al., 1989; Uenhara et al., 2001; Vidović et al., 2004; Wisotsky et al., 2003).

The questionnaire is based on the McMaster Model of Family Functioning (MMFF), which assumes that family health is related to the accomplishment of essential tasks and provides a clinically oriented conceptualisation of families (Epstein et al., 1978). The scale measures structural, organisational, and transactional characteristics of families. The first six scales of the FAD assess the six dimensions of MMFF (previously described in section 3.2 of Chapter 3) and the seventh scale, General Functioning, assesses the overall health or pathology of the family (Epstein et al., 1983). This scale can be used as a single index representing the overall family functioning (Byles et al., 1988; Kabacoff et al., 1990). FAD's seven scales are intercorrelated but sufficiently independent to be distinguishable, reflecting the notion that aspects of a family's life are not always completely independent (Epstein et al., 1983).

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Psychometric evidence suggests that the FAD is internally consistent (Cronbach's α ranging from .72 to .92), reliable over short periods, unrelated to measures of social desirability and successful in discriminating clinical from non-clinical groups (Friedman et al., 1997; Kabacoff et al., 1990; Waller et al., 1989). It has been used in a Greek population displaying good reliability and validity (Francis & Papageorgiou, 2004; Kostakou, 2014; Statharou et al., 2011, Theodoropoulou, 2014), as well as in many studies worldwide with ED related studies among them (e.g. Lewandowski et al., 2010; McDermott & Cobham, 2012; Staccini et al., 2015).

4) Eating Disorder Examination Questionnaire (EDE-Q 6)

The Eating Disorder Examination Questionnaire 6.0 (EDE-Q) (Fairburn & Beglin, 2008) is derived from the Eating Disorder Examination (EDE) which is a semi-structured interview (Fairburn & Cooper, 1993) used worldwide for diagnostic purposes to assess the core psychopathology of eating disorders. The EDE is widely regarded as the instrument of choice for the assessment and diagnosis of eating disorders according to the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1994; Garner, 2002) and is the gold standard to assess eating pathology (Aardoom et al., 2012).

The EDE-Q has been used both in clinical and non-clinical populations (Lavender et al., 2010; Rø et al., 2010; Rose et al., 2013; Welch et al., 2011; Yucel et al., 2011). It is a popular alternative to the EDE because it is less time consuming and the interviewer does not need to be trained in order to administer it (Aardoom et al., 2012; Lavender et al., 2010). Studies of the validity of the EDE-Q have demonstrated a significant correlation between the EDE-Q and EDE in assessing the major characteristics of eating disorder psychopathology in nonclinical populations (Fairburn & Beglin, 1994; Mond et al., 2004c). Acceptable internal

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consistency, test–retest reliability and temporal stability have also been demonstrated (Berg et al., 2011; Luce & Crowther, 1999; Mond et al., 2004) therefore the EDE-Q has been excessively used as a self-report measure in numerous epidemiologic and clinical studies of eating disorders (Pike et al., 2001; Wilfley et al., 1997).

The EDE-Q is composed of 28 items and three types of data: a) four subscale scores (Restraint, Eating Concern, Shape Concern and Weight Concern), b) a global score which is the average of the four subscale scores and c) frequency data on key eating and compensatory behaviours (objective binge-eating episodes, self-induced vomiting, laxative misuse, excessive exercise) measured by 6 items (13-18). All items refer to the preceding 28 days, and frequency or intensity are rated on six-point Likert scales (0 = *feature was absent* to 6 = *feature was present every day or to an extreme degree*). Restraint is composed of 5 items (1: restraint over eating, 2: avoidance of eating, 3: food avoidance, 4: dietary rules, 5: empty stomach). Eating Concern is composed of 5 items (7: preoccupation with food, eating or calories, 9: fear of losing control over eating, 19: eating in secret, 20: guilt about eating, 21: social eating). Shape Concern is composed of 8 items (6: flat stomach, 8: preoccupation with shape or weight, 23: importance of shape, 10: fear of weight gain, 26: dissatisfaction with shape, 27: discomfort seeing body, 28: avoidance of exposure, 11: feelings of fatness). Weight Concern is composed of 5 items (8: preoccupation with shape or weight, 24: reaction to prescribed weighing, 22: importance of weight, 25: dissatisfaction with weight, 12: desire to lose weight). The subscales of weight and shape concern share item 8.

Subscale scores are calculated by averaging the available item responses, when less than half of the relevant items are missing (Fairburn & Cooper, 1993; Mond et al., 2006). Failure to respond to a behavioural item (13-18) is interpreted as non-endorsement of the particular behaviour (Mond et al., 2006) and is scored as zero. The subscale scores are obtained by calculating the average of the items forming each subscale, and the global score is the

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average of the four subscale scores. The scores are reported as means and standard deviations ranging from 0 to 6.

A cut-off point of four points or more (≥ 4) indicates clinical significance for each subscale and for the global score (Carter et al., 2001; Luce et al., 2008; Mond et al., 2006; Rø et al., 2010). An empirically derived threshold of ≥ 2.30 (versus < 2.30) is used for the Global score to indicate eating disturbances but not an eating disorder (Arigo et al., 2014; Berger et al., 2014; Hilbert et al., 2012; Mond et al., 2004; Penelo et al., 2013). This empirically derived threshold has been used to differentiate clinical from non-clinical groups (Arigo et al., 2014; Heron, 2011) or high vs low eating pathology (Anokhina, 2015). Cooper explained that a score less than one standard deviation above the community mean on the global EDEQ score is typically used to create the threshold and it is well accepted as a good outcome (Z. Cooper, personal communication October 17, 2014).

The EDE-Q 6 has been standardized and used in Greek population providing adequate test-retest reliability and internal consistency both for adults (Giovazolias et al., 2013) and adolescents (Pliatskidou et al., 2012).

5) Sociodemographic and Relevant Clinical Information

Included in the demographic sheet are questions about personal and contextual factors, such as sociodemographic characteristics, psychological and physical complaints at present (that could possibly affect their QoL), body satisfaction, BMI (kg/m^2), duration of illness and age of illness onset, times hospitalized. BMI was calculated from self-reported height and weight. Body satisfaction was measured through the use of whole body silhouette pictures of varying size (Ogden, 2004a) and participants were asked to state which figure is close to how they look now (perceived current shape) and which figure best illustrates how they would like to look (own ideal shape). Body dissatisfaction (BD) was calculated by subtracting the ideal body shape from the current one to obtain the discrepancy index. In this

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way, negative BD scores indicate a desire to be bigger and positive scores indicate a desire to be thinner. A score of zero indicates that there is no BD (Rekha & Maran, 2012; Wade et al., 2001). Table 1 presents Cronbach's α for the measures used in Study 1 both for ED patients and controls.

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Table 1.

Cronbach's α for the measures used in Study 1 both for ED patients and controls.

	Cronbach's alpha	
	ED patients N = 154	Controls N = 154
McMaster Family Assessment Device (family functioning)		
<i>Problem Solving</i>	.80	.72
<i>Communication</i>	.88	.76
<i>Roles</i>	.72	.75
<i>Affective Responsiveness</i>	.85	.81
<i>Affective Involvement</i>	.77	.69
<i>Behaviour Control</i>	.70	.51
<i>General Functioning</i>	.92	.87
WHOQOL-BREF (quality of life)		
<i>Psychological Domain</i>	.86	.77
<i>Social Relationships</i>	.72	.70
Rosenberg Self-Esteem Scale (self-esteem)	.90	.82
Eating Disorders Examination Questionnaire-EDE-Q 6 (eating pathology)		
<i>Restraint</i>	.81	.79
<i>Eating Concern</i>	.78	.77
<i>Shape Concern</i>	.88	.88
<i>Weight Concern</i>	.75	.80
<i>Global EDE-Q</i>	.93	.93

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Study 2a

The following questionnaires of Study 2a were used in Study 1 as well: 1) World Health Organisation Brief Quality of Life Assessment Scale (WHOQOL-BREF), 2) Rosenberg Self-esteem scale (RSES), 3) Eating Disorder Examination Questionnaire (EDE-Q 6), 4) Sociodemographic and Relevant Clinical Information. In addition to this, three measures were also used:

5) The Childhood Trauma Questionnaire - Short Form (CTQ-SF)

The Childhood Trauma Questionnaire (CTQ)-Short Form is a 28-item retrospective self-report screening tool that aims to detect experiences of childhood abuse and neglect in five domains with strong psychometric properties (Bernstein & Fink, 1998; Bernstein et al., 2003; Grassi-Oliveira et al., 2014). It is the most widely used instrument to screen for emotional (EA), physical (PA), sexual (SA) abuse, and emotional (EN) and physical neglect (PN) occurred during childhood. It has been used in more than 339 studies (Grassi-Oliveira et al., 2014) and has been found to be more reliable compared to other methods assessing childhood abuse (Polanczyk et al., 2009). The CTQ is internationally accepted as a key tool for the assessment of traumatic childhood experiences in different countries (Daalder & Bogaerts, 2011; Grassi-Oliveira et al., 2014; Grassi-Oliveira et al., 2006; Garrusi & Nakhaee, 2009; Gerdner & Allgulander, 2009; Hernandez et al., 2013; Kim et al., 2013; Kim et al., 2011; Klinitzke et al., 2012; Paquette et al., 2004; Thombs et al., 2009; Wingenfeld et al., 2010).

The original CTQ was developed from a 70-item retrospective questionnaire in which participants were required to rate the frequency on a 6-point scale (0-never true to 5-very often true) of abuse and neglect events that took place while they were growing up (Bernstein et al., 1994). In further studies, the length of the scale was reduced to 28 items based on

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exploratory and confirmatory factor analyses (Bernstein et al., 2003). Convergent and discriminant validity was demonstrated as CTQ was significantly correlated to Childhood Trauma Interview (Bernstein et al., 1994; Bernstein et al., 2003).

Each scale consists of five items with a total score ranging from 5 to 25. Seven items are reverse scored (items: 2, 5, 7, 13, 19, 26, 28). Three additional items compose the Minimization/Denial subscale (MD) for detecting socially desirable responses or false-negative trauma reports (Kim et al., 2011) but this scale was not used in the analyses. The total CTQ score takes into account the severity of multiple forms of abuse and neglect. The internal consistency reliability coefficients of the original version ranged from .60 (physical neglect) to .95 (sexual abuse) (Bernstein et al., 2003). Alphas from the current sample are reported below.

The CTQ scales definition was developed on the basis of the childhood trauma literature (Crouch & Milner, 1993; Finkelhor, 1994). *Emotional abuse* refers to verbal assaults on a child's sense of worth or well-being, or any humiliating, demeaning, or threatening behaviour directed toward a child by an older person. *Physical abuse* refers to bodily assaults on a child by an older person that pose a risk of, or result in, injury. *Sexual abuse* refers to sexual contact or conduct between a child and an older person, including explicit coercion. *Emotional neglect* refers to the failure of caretakers to provide basic psychological and emotional needs, such as love, encouragement, belonging and support. *Physical neglect* refers to failure to provide basic physical needs including food, shelter, and safety.

Translation of the CTQ into Greek

The CTQ-SF was first translated into Greek (forward-translation) by the author, with the approval of NCS Pearson, Inc., after signing a research translation licence agreement, and

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then back-translated three times by bilingual Greek–English native speakers. A panel of PhD- and master’s-level psychologists checked the translation and back-translation until no major discrepancies were noted, and the final Greek version was agreed. Pilot studies were also conducted to check the instrument’s performance in the general population prior to the main study. The WHO guidelines for the translation of instruments, along with guidelines for the questionnaire’s translation in cross-cultural health care research, were followed (Sousa & Rojjanasrirat, 2011). The pilot study Cronbach’s alpha coefficients are displayed in Table 3.

The third back-translation was necessary due to the incorrect back-translation of the word ‘molestation’ in item 24. The word “molestation” in Greek does not imply sexual abuse but in English it does. As a result, the word *sexual* had to be added in order to retain the meaning of “molestation”. The same problem appeared in the Dutch version, but the authors decided to remove the item (Spinhoven et al., 2014). The back-translated version was compared with the original version by a panel of psychologists who have translated and standardised other mental health instruments for the Greek population. There were two changes that did not alter the meaning of the items: a) several items begin with ‘people in my family’, but this is not a common expression in Greek, so the word ‘people’ was replaced with ‘members’; b) an exact translation of item 1 (‘I didn’t have enough to eat’) was not possible, as this is not an expression used in Greek, so it was replaced with ‘I didn’t have enough food’.

A pilot study, pre-testing the Greek version of the CTQ, was conducted in April 2015, employing 31 female participants from the community in Athens, Greece to represent 10% of the main study’s sample (Connelly, 2008; Hertzog, 2008; Lackey & Wingate, 1998). The clinical population was not a part of the pilot study for feasibility reasons, as there was no reason to expect the clinical population to interpret the meaning of these words any

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differently. The mean age of the pilot study population was 32.29 ($SD = 9.34$), and they had an average educational level of 16 years (29% high school; 32.3% college; 38.7% postgraduate studies). Twenty-one of the participants ($n = 21$) scored zero on the CTQ minimisation/denial scale. Ten participants ($n = 10$) had a score ranging from 1 to 3 in the minimisation/denial scale. Of these ten participants, six ($n = 6$) had a score of 1, two ($n = 2$) had a score of 2 and two ($n = 2$) had a score of 3. Any score ranging from 1 to 3 in this scale suggests possible under-reporting of maltreatment (false negatives) according to the instrument's manual (Bernstein & Fink, 1998).

Participants were asked to comment on the clarity of the items and instructions and the overall presentation of the scales. Table 2 presents the items for which participants had comments and the changes made.

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Table 2.

Participants' comments on the CTQ during the 1st pilot study and changes made

Item	Problem	Change made
Instructions	The CTQ instructions read “ <i>These questions ask about....</i> ” but they noticed that there are no question marks and the items are actually statements (n = 31)	None
Instructions	They were confused with the statement “ <i>for each question, circle the number that best describes how you feel</i> ” because the actual items require for responses based not on how they <i>feel</i> about the statement but how often each statement applied to them (frequency). The guidelines of the questionnaire actually verify that it measures frequency (Bernstein & Fink, 1998) (n = 31)	“ <i>For every question circle the number that best describes the frequency of each statement</i> ”
Scoring	They reported being confused from the wording of the range of the scoring options (<i>never true to very often true</i>). In particular, they reported being confused by the word “true” saying they would prefer this word to be omitted because they have not used seeing questionnaires measuring frequency with <i>never true</i> expression and it is not a common use in Greek language. (n = 31)	Explained in the instructions
Scoring	They observed that there should be an option for “always true” at the end of the range of the responses since the first option is “never true” instead of having a <i>never to very often</i> option. It would make better sense to them an option of “ <i>very rarely to very often</i> ” or “ <i>never to always</i> ” rather than “ <i>never to very often</i> ” as they felt something was missing from their options (n = 31)	None

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Item	Problem	Change made
Items that did not ask something related to people in their family.	Most of the questions are related to people in their family making then conditioned to think about parents or caregivers and when questions like item 15 appear (<i>I believe that I was physically abused</i>) came up they didn't know if it was related to people in their family or they had to answer if they had been physically abused by anyone ($n = 31$)	Clarified in the instructions: <i>Some statements particularly refer to the familial environment while some others are general (e.g. I believe I was emotionally abused). When the statement does not mention the family it means that what the statement describes could have happened by anybody.</i>
Item 23 <i>someone tried to make me do sexual things or watch sexual things</i>	They asked what <i>sexual things</i> include ($n = 17$)	“things” was explained in a parenthesis writing (<i>anything of sexual nature</i>)
Item 4 (<i>my parents were too drunk or high to take care of the family</i>)	In Greek language the word <i>high</i> is used to indicate both that someone is high on substances and that someone is very angry and people understand the meaning from the rest of the sentence's context ($n = 22$)	The word <i>high</i> was explained in a parenthesis with the indication “on substances”.
Item 3 (<i>people in my family called me things like “stupid”, “lazy” or “ugly”</i>) and item 11 (<i>people in my family hit me so hard that it left me bruises or marks</i>).	They wanted to know if they could count for this answer an older sibling and if this behaviour from an older sibling actually accounts for abuse since they have a very good relationship with him/her and since “ <i>this is common when underage siblings have a fight or a heated argument</i> ” ($n = 20$)	It was decided to make a clarification in a parenthesis (<i>anyone in the family</i>).
Item 1 (<i>I didn't have enough food</i>)	Not correct expression in Greek ($n = 12$)	clarification in parenthesis (<i>none had cooked or the food was less than it was supposed to be</i>)
Item 6 (<i>I had to wear dirty clothes</i>).	Not correct expression in Greek ($n = 12$)	Clarification in parenthesis (<i>I could not find clean clothes ready to wear</i>).
Item 12 (<i>I was punished with a belt, a board, a cord, or some other hard object</i>).	The word <i>board</i> ($n = 15$). Six out of the fifteen who made that comment wrote “ <i>do you mean a twig?</i> ” The Greek word for “twig”, a thin piece of wood coming from a plant, makes sense as a punishment tool in Greece instead of a board.	The word <i>board</i> was replaced by <i>twig</i> .

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Item	Problem	Change made
Instructions	Unclear (n = 31)	<p><i>The questions ask about some of the experiences you may have had growing up as a child and adolescent. For each question circle the number that best describes the frequency of each statement. "Never true" means that what the statement says never happened to "very often true" which means that the statement happened very often. Some statements particularly refer to the familial environment while some others are general (e.g. I believe I was emotionally abused). When the statement does not mention the family it means that what the statement describes could have happened by anybody. Even though some of the questions are of personal nature, please try to respond as honestly as you can. Your answers will be confidential".</i></p> <p><i>Before the changes the instructions were: "The questions ask about some of the experiences you may have had growing up as a child and adolescent. For each question please circle the number that describes best how you feel. Even though some of the questions are of personal nature, please try to respond as honestly as you can. Your answers will be confidential".</i></p>

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A second pilot study was conducted to test the improved, clarified version. The second pilot study was conducted on 30 adult women from the community ($n = 30$) with a mean age of 27.57 years ($SD = 8.11$) and a mean educational level of 16 years (23.3% high school; 53.3% college; 23.3% postgraduate studies). Seventeen of the participants ($n = 17$) scored zero on the minimisation/denial scale. Thirteen participants ($n = 13$) had a score ranging from 1 to 3 on the CTQ minimisation/denial scale. Of these thirteen participants, seven ($n = 7$) had a score of 1, five ($n = 5$) had a score of 2 and one ($n = 1$) had a score of 3. Any score ranging from 1 to 3 on this scale suggests possible under-reporting of maltreatment (false negatives) according to the instrument's manual (Bernstein & Fink, 1998). No issues came up in this final Greek version of the CTQ (Appendix 35).

Cronbach's alpha values were produced for the CTQ scales for the two pilot studies (see Table 3). Regarding the three scales of interest, emotional abuse was improved (probably due to the clarifications regarding older siblings' inclusion in certain items) and emotional and physical neglect produced a lower value compared to the first pilot study. Removing items did not improve the scale's alpha value. To overcome the performance problems of the CTQ's non-physical abuse scales, especially the neglect scales, the study incorporated an additional measure for neglect.

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Table 3.

Cronbach's a results of the two pilot studies along with a comparison of those reported in other studies both in English- and non-English-speaking populations.

Scale	Pilot Study 1	Pilot Study 2	Bernstein & Fink (1998)	CTQ Spain (Hernandez et al., 2013)	CTQ Korea (Kong & Bernstein, 2009)	CTQ Korea (Kim et al., 2011)	US 2001 (Scher et al., 2001)
CTQ Emotional Abuse	.61	.85	.89	.87	.81	.82	.83
CTQ Physical Abuse	.81	.79	.78	.88	.87	.88	.69
CTQ Sexual Abuse	.93	.89	.72	.94	.88	.87	.94
CTQ Emotional Neglect	.88	.76	.92	.83	.87	.86	.85
CTQ Physical Neglect	.38	.24	.60	.66	.68	.68	.58
CTQ Total	.81	.85	-	-	.91	.92	.91

Note. The scales of interest for this study are in bold. Bernstein's results refer to his non-clinical sample (students) because it was the closest to the population of the pilot studies. The mean age for pilot study 1 was 32.29 ($SD = 9.34$) and for pilot study 2 it was 27.57 years ($SD = 8.11$). Both samples had a mean educational level of 16 years. The low physical neglect reliability value is reported in other studies ranging from .58 to .79 both in English and non-English versions of the CTQ (Bernstein & Fink, 1998; Dovran et al., 2013; Gerdner & Allgulander, 2009; Grassi-Oliveira et al., 2014; Hernandez et al., 2013; Kim et al., 2011; Kong & Bernstein, 2009; Scher et al., 2001).

6) The Multidimensional Neglectful Behaviour Scale Adult Recall Version Short Form-MNBS

AS

This scale was designed as a brief measure of neglectful behaviour either for adolescents (currently living with parent or caregiver) or for adults who are asked to recall past abuse (Straus, 2006; Straus et al., 1995). The MNBS-AS is composed of 8 items that

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measure cognitive, emotional, physical and supervisory neglect by using two items for each domain. The authors selected the four dimensions based on review of previous neglect measures (Straus, 2006). These eight questions of MNBS AS come from the full version (MNBS A) which has 20 items, five for each of the previously mentioned domains (Straus et al., 1995). In detail, the eight items for the short form were selected from the full MNBS by choosing two items from each of the four subscales/domains and they were the ones with the highest item-total correlation maintaining the representation of the four domains in the short form (Straus et al., 1995). The MNBS AS is highly correlated with MNBS A ($\alpha = .95$).

The MNBS AS is measured on a 4-Likert scale (1 = strongly disagree to 4 = strongly agree) and four items are reverse scored (items: 1, 3, 4, 6) in order for higher scores to indicate neglect. The alpha value was .89 for this questionnaire (Straus et al., 1995). It was also developed to be included as the Neglect History scale of the Personal and Relationships Profile (PRP) (Straus et al., 1999; Straus & Mouradian, 1999). Straus et al., (1995) suggest that the score can be computed as a sum, mean or dichotomized variable that measures the number of neglectful behaviours experienced.

The authors of the MNBS (Straus et al., 1995) tried to overcome the difficulties of measuring parents' neglectful behaviour by handling the difficulty of the vagueness of the operational definition of neglect, a well-known problem in the relevant literature, which is also considered responsible for the small body of empirical knowledge on neglect (Besharov, 1981; Dubowitz, 1994; Dubowitz et al., 1993; Glaser, 2002; Harrington et al., 2002; Wolock & Horowitz, 1984; Zuravin, 1991). Unlike the physical forms of childhood abuse, neglect is multidimensional making difficult to be measured as there is lack of consensus of the aspects that constitute neglect (Harrington et al., 2002).

The definition used to build the MNBS questionnaire is: "*Behaviour by a caregiver which is characterised by failing to engage in behaviour which is needed to meet the*

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developmental needs of a child *and* which is the *responsibility of the caregiver* to provide” (Straus et al., 1995). Previous efforts to operationalise neglect have led to the importance of identifying specific subtypes of neglect in order to produce a comprehensive measure (Magura & Moses, 1986; Zuravin, 1991). Zuravin (1991) differentiated 14 subtypes of neglect and Magura and Moses (1986) 18 neglect areas. The development of the MNBS began by reviewing these measures and led to the identification of four general but basic needs of children (Straus et al., 1995): Physical needs (food, clothes, shelter, medical care), Emotional needs (affection, companionship, support), Cognitive needs (being played with, read to, help with school homework), Supervision needs (limit setting, attending to misbehaviour, knowing child’s whereabouts and friends). Straus et al., (1995) believe that by representing each of the basic neglect domains in the scale, the content validity is increased.

The MNBS AS has been translated into Greek language as part of the PRP translation (Kalaitzaki et al., 2010) displaying a coefficient alpha of .65 (A. Kalaitzaki, personal communication, May 25, 2015). The Greek translation includes the eight items of the original MNBS AS version before the change of 2 items for cultural adaptation (Straus, 2006). The unchanged version fits better the Greek cultural context as school is highly valued. Permission from the authors was obtained to use the MNBS AS in this study (G. Kantor, personal communication, 27 May 2015).

7) *The Experiences in Close Relationships Structures (ECR-RS) Questionnaire*

The Experiences in Close Relationships-Relationship Structures (ECR-RS) questionnaire (Fraley et al., 2011; Fraley et al., 2006) is a self-report measure developed to assess attachment patterns/styles in various close relationships using the same 9 items for each attachment target (mother, father, partner, best friend). For each attachment target two scores are computed: Attachment-related avoidance and attachment-related anxiety. The avoidance score is computed by averaging items 1-6 after the reverse scoring of items 1, 2, 3,

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4. The anxiety score is computed by averaging items 7-9. The two scores are computed separately for each relationship target. There is also the option to create a global attachment score by averaging the scores for mother, father, partner and best friend and computing their mean, which was the practice selected for this study. High scores indicate insecure attachment and low scores indicate secure attachment. A person with secure attachment would score low to both scores (Fraley et al., 2011). The instrument has displayed adequate psychometric properties with Cronbach's α ranging from .85 to .92. The ECR-RS 9 items derive from the Experiences on Close Relationships-Revised (ECR-R) inventory which also reports good psychometric properties (Fraley et al., 2011).

The Experiences in Close Relationships-Revised (ECR-R) measure (Fraley, Waller, & Brennan, 2000), is a 36-item dimensional adult attachment measure, coming from the revised version of Brennan and associates (1998) Experiences in Close Relationships (ECR) questionnaire. The ECR-R is based on the assumptions of the attachment model developed by Bartholomew and Horowitz (1991), in which the internal operational models of attachment are characterised in two types of relationships with other persons: fearful and avoidant thus the ECR-R measures individuals on two subscales of attachment: Avoidance and Anxiety. Anxiety refers to the fear of rejection and abandonment and avoidance refers to the experience of discomfort triggered by closeness and addiction to others (Kamenov & Zelić, 2005). In general, avoidant individuals find discomfort with intimacy and seek independence, whereas anxious individuals tend to fear rejection and abandonment.

There is a Greek version of the Experiences in Close Relationships-Revised (G-ECR-R). The instrument has been standardized for the Greek population displaying good psychometric properties in terms of internal consistency, test-retest reliability, convergent and criterion validity (Tsagarakis et al., 2007) and has been used in Greek studies (Kormas et al., 2014). For the purposes of the current study, the 9 ECR-RS items were selected from the

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Greek ECR-R questionnaire (G-ECR-R) which asked for mother, father, partner and best friend. The G-ECR-R has reported good psychometric properties (avoidance scale $\alpha = .91$; anxiety scale $\alpha = .91$) (Tsagarakis et al., 2007)

8) *Difficulties in Emotion Regulation Scale (DERS)*

According to Gratz and Roemer (2004), who drew from all related theoretical models on the field to provide a comprehensive and integrative conceptualisation, emotion regulation is the ability to experience a full range of emotions, modulate emotional experience, appropriately display emotion, as well as the ability to respond effectively to others' emotions; failure to one of the above results in emotion dysregulation. In that sense, the DERS was developed to assess emotion dysregulation in a multi-dimensional way by extending related clinical and social personality theories to assess adaptive emotion regulation using an integrated conceptualisation (Gratz & Roemer, 2004). Bardeen and colleagues (2012) maintained that a self-report measure that would provide a comprehensive assessment of the emotion regulation had been lacking in the relevant literature, until Gratz and Roemer (2004) developed the Difficulties in Emotion Regulation Scale (DERS).

The DERS is a 36-item self-report questionnaire designed to assess multiple aspects of emotion dysregulation (Gratz and Roemer, 2004). The measure yields a total score, as well as scores on the following six scales derived through factor analysis: 1) Nonacceptance of emotional responses, (NONACCEPT: 11, 12, 21, 23, 25, 29); 2) Difficulties engaging in goal directed behaviour (GOALS: 13, 18, 20R, 26, 33); 3) Impulse control difficulties (IMPULSE: 3, 14, 19, 24R, 27, 32); 4) Lack of emotional awareness (AWARENESS: 2R, 6R, 8R, 10R, 17R, 34R); 5) Limited access to emotion regulation strategies (STRATEGIES: 15, 16, 22R, 28, 30, 31, 35, 36) and 6) Lack of emotional clarity (CLARITY: 1R, 4, 5, 7R, 9). *Nonacceptance* subscale refers to the tendency of experiencing a non-accepting reaction to

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one's own distress. *Goals* subscale is the difficulty in concentrating and completing tasks when experiencing negative emotions. *Impulse* subscale is the difficulty in controlling one's own behaviour under distress. *Awareness* subscale reflects a lack of awareness / lack of attention to emotional responses. The *Awareness* subscale measures an individual's trend to acknowledge emotional states rather than the ability to recognise and describe internal emotional experiences and it has been used to measure an individual's tendency to allow an emotion experience to run its course instead of suppressing or avoiding it (Robertson et al., 2014). *Strategies* subscale represents the notion of inability to regulate oneself when upset. *Clarity* subscale reflects the extent to which a person's understanding and clarity regarding his/her emotions.

Each item is rated on a 5-point scale based on how often participants believe each item relates to them (1 = almost never to 5 = almost always) and some items are reverse scored (R) in order for higher scores to indicate greater emotion regulation difficulty. Subscale scores are obtained by summing the corresponding items. The DERS total score, which comes from the sum of all subscales, ranges from 36 to 180 and there are no cut-offs, meaning that the higher the score the greater the emotion dysregulation. However, researchers can acquire a general sense of their results' meaning by comparing them to the average scores coming from the non-clinical sample used by Gratz and Roemer (2004) during the development of the questionnaire: Overall score: women = 78; men = 80, Nonacceptance: women = 12; men = 12, Goals: women = 14; men = 14, Impulse: women = 11; men = 12, Awareness: women = 14; men = 16, Strategies: women = 16; men = 16, Clarity: women = 11; men = 11. Regarding the clinical population the mean score was 120-128 for outpatients with BPD (Gratz & Gunderson, 2006). Bardeen and associates (2012) computed a total DERS mean score of 76.70 for female students. A DERS subscale is considered invalid if

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more than one item is left unanswered. More than seven missing items invalidates the DERS total score.

In the measure's initial publication, the DERS displayed good internal consistency ($\alpha = .93$), construct and predictive validity, and test-retest reliability across 4–8 weeks ($p < .01$) (Gratz & Roemer, 2004). Both the overall DERS score and subscale scores have displayed high internal consistency within both clinical (Fox et al., 2007; Gratz et al., 2008) and nonclinical populations (Gratz & Roemer, 2004; Robertson et al., 2014). The Cronbach's α for the DERS subscales was $> .80$ (Gratz & Roemer, 2004). There is also evidence for the construct and predictive validity of DERS scores in clinical and nonclinical populations (Fox et al., 2007; Gratz & Roemer, 2004, 2008; Gratz et al., 2006). Gratz and Roemer (2004) have also established the predictive validity of the DERS by exploring correlations between the DERS scores and two clinically important behavioural outcomes (frequency of deliberate self-harm and frequency of intimate partner abuse) thought to be associated with emotion dysregulation. Moreover the DERS has been found to predict deliberate self-harm with an optimal cut-off score of 14 (Venta et al., 2011) and its total score has successfully discriminated high/average/low BPD features in adolescents (Fossati et al., 2014).

The DERS has been initially examined in a sample of students aged 18 and above and in a clinical sample of women with BPD (Gratz & Roemer, 2004; Gratz et al., 2006) with empirical results displaying that it could be also used in adolescents (Weinberg & Klonsky, 2009). It is considered a promising psychometric measure that reaches an important domain for the field of childhood trauma, and even though almost all empirically accepted treatments for child trauma address the important domain of emotion regulation, few measures actually assess it (Bardeen et al., 2012).

The instrument has also been utilized in many ED studies along with EDE-Q and CTQ (Burns et al., 2012; Harrison et al., 2009; Lafrance-Robinson et al., 2013; Reiser, 2013;

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Thompson et al., 2014) and in studies investigating childhood traumatic experiences (Selwood, 2013). The DERS has been widely used to assess emotion regulation in both clinical and nonclinical population (Fowler et al., 2014; Fox et al., 2007; Kökönyei et al., 2014) including ED patients (Cooper et al., 2014), as well as in studies assessing emotion regulation with respect to childhood abuse (Stevens et al., 2013); in studies assessing emotion regulation and childhood trauma both in ED patients (Vajda & Láng, 2014) and in BPD patients (Carvalho-Fernando et al., 2014); and in studies testing Linehan's biosocial theory (Reeves et al., 2010).

Translation of the DERS into Greek language

The DERS was translated by the author into Greek (forward-translation) with the permission of K. Gratz (personal communication, 23 February 2015). Then, it was back-translated twice by bilingual Greek–English native speakers. A panel of PhD- and master's-level psychologists checked the translation and back-translation and compared it with the English version to detect any discrepancies or inadequate expressions of the translation before proceeding to back-translate it, which did not result in any suggestions for changes. The WHO guidelines for translating instruments were followed (Sousa & Rojjanasrirat, 2011).

Gratz reviewed the back-translations and commented on them (K. Gratz, personal communication, 5 May 2015). This process revealed the need to find better synonyms in Greek for items 3, 36 and 10 that would not change the original meaning and would make sense in Greek. In items 3 and 36, 'overpowering' replaced 'overwhelming' and 'recognise' replaced 'acknowledge' in item 10. For item 31, Gratz proposed another expression ('staying stuck') that has the same meaning as 'wallowing in it', in order to be compatible with a Greek expression. A pilot study was conducted to pre-test the reliability of the subscales, the clarity

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of the items and instructions, as well as the overall presentation of the questionnaires, before the main study.

In the pilot study, the DERS was administered to 31 women from the community in order to represent 10% of Study 2's participants. The pilot study took place in April 2015. The administration of the pilot study questionnaires did not involve the ED population for feasibility reasons, as it would not be expected that a clinical population would understand the items' meanings differently. The pilot population's mean age was 32.29 ($SD = 9.34$), and they had a mean educational level of 16 years (29% high school; 32.3% college; 38.7% postgraduate studies). The participants were able to understand the instructions, and they all ($n = 31$) commented that they felt that some questions were repeated or looked similar, which was sometimes tiring ($n = 31$). For this type of comment, it was decided that changing the translation was not necessary because repeated or similar questions serve the purposes of the original DERS, and it was not a matter of translation. Table 4 presents the comments and the changes.

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Table 4.

Participants' comments on the DERS during the 1st pilot study and the changes made

Item	Problem	Change made
Items 3 & 36	They needed a periphrastic explanation of <i>overwhelming</i> (n = 20)	A periphrastic explanation was made in a parenthesis (they overwhelm me, I can't manage them)
Item 2 (<i>I pay attention to how I feel</i>)	Confused some participants as to whether they should pay attention regarding the expression of their feelings or pay attention to their feelings, meaning if they should "listen" to their feelings (n = 8)	A clarification in a parenthesis (<i>I carefully observe how I feel</i>)
Item 17	Clarification for the word <i>valid</i> (n = 9)	A clarification in a parenthesis (<i>it's understandable that I feel the way I do</i>)

A second pilot study was conducted for the improved final Greek version of the DERS with the clarifications (Appendix 36). This was conducted on 30 adult women from the community ($n = 30$) with a mean age of 27.57 years ($SD = 8.11$) and a mean educational level of 16 years of completed education (23.3% high school; 53.3% college; 23.3% postgraduate studies). Two participants commented that some items seemed to be repeated. Ten participants said that for certain items, the parenthetical clarification was helpful. Removing an item did not improve the scale's alpha value. Certain scales had an improved α value (nonaccept, impulse, awareness, clarity and total DERS), and others slightly decreased (goals and strategies). The awareness scale displayed the lowest alpha value in both pilot studies. This scale has displayed modest intercorrelations with the rest of the scales (Gratz & Roemer, 2004; Neumann et al., 2010). The awareness scale is the only DERS scale where the items are reverse scored, which could reduce the scale's validity (Hinkin, 1995). Table 5 presents the assessment of the DERS scales' internal consistency in both pilot studies, followed by Gratz's and Roemer's values (2004).

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Table 5.

Cronbach's α results of the two pilot studies along with a comparison of those reported by Gratz and Roemer (2004)

Scale	Pilot Study 1	Pilot Study 2	Gratz & Roemer (2004)
DERS Nonaccept	.83	.85	.84
DERS Goals	.89	.82	.89
DERS Impulse	.78	.85	.86
DERS Awareness	.68	.72	.80
DERS Strategies	.90	.85	.88
DERS Clarity	.81	.93	.84
Totals DERS	.93	.94	.93

Note: The mean age for Pilot Study 1 was 32.29 ($SD = 9.34$), and for Pilot Study 2 it was 27.57 years ($SD = 8.11$). Both samples had a mean educational level of 16 years.

Study 2b

The following measures are described earlier in this chapter: 1) World Health Organisation Brief Quality of Life Assessment Scale (WHOQOL-BREF), 2) The Childhood Trauma Questionnaire (CTQ), 3) The Experiences in Close Relationships-Revised (ECR-R), 4) Difficulties in Emotion Regulation Scale (DERS), 5) Rosenberg's Self-Esteem Scale (RSES), 6) The Multidimensional Neglectful Behaviour Scale Adult Recall Version Short Form-MNBS AS, 7) Sociodemographic and Relevant Clinical Information.

8) Adult Temperament Questionnaire-Short Form (ATQ-SF)

The ATQ-SF (Evans & Rothbart, 2007a) is a self-report questionnaire and it accounts for the short version of ATQ (177 items) which was adapted from the Physiological Reactions Questionnaire (Derryberry & Rothbart, 1988). It includes the same general

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constructs and sub-constructs as the long form and the correlations of the short form scales with the long form ones range from .85 to .96.

The ATQ-SF measures 4 dimensions (negative affect, effortful control, surgency/extraversion, orienting sensitivity) and 13 sub-dimensions (fear, sadness, discomfort, frustration, inhibitory control, activation control, effortful control, sociability, high pleasure, positive affect, general perceptual sensitivity, affective perceptual sensitivity, associative sensitivity) of the Rothbart temperament model (Rothbart et al., 2000). This means that there are motivational-emotional-attentional constructs identified at a general level and then more specific temperamental constructs are defined to be explored at lower levels (Evans & Rothbart, 2007a). In particular, the four general constructs are referred to by the authors as *factor scales* and the thirteen more specific sub-constructs as *scales* (Evans & Rothbart, 2007b). Regarding the four general constructs (factor scales), orienting sensitivity and effortful control are attentional constructs, negative affect is an emotional construct and extraversion/surgency is a motivational construct (Evans & Rothbart, 2009). Orienting sensitivity involves perception and peripheral thinking in current tasks while effortful control involves the ability of planning, detecting errors and controlling attention under conflict situations (Evans & Rothbart, 2009). Negative affect is the tendency to express feelings of anxiety, irritability, sadness fear and anger (Gouze et al., 2012) and extraversion/surgency is a characteristic of high-intensity pleasure seeking, high activity level, low shyness and impulsivity (Berdan et al., 2008).

Factor Scale 1: Negative Affect (26 items): Composed of 4 scales: *Fear* which the negative affect related to anticipation of stress and has 7 items. *Sadness* which is the negative affect and lowered mood and energy related to exposure to suffering, disappointment and job loss and has 7 items. *Discomfort* which is the negative affect related to sensory qualities of

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stimulation such as intensity, rate or complexity of visual, auditory, smell/taste and tactile stimulation. It has 6 items. Frustration which is the negative affect related to interruption of ongoing tasks or goal blocking. It has 6 items.

Factor Scale 2: Extraversion/Surgency (17 items): Composed of 3 scales: *Sociability* which is the enjoyment derived from social interaction and being in the presence of others. It has 5 items. *Positive Affect* which has to do with latency, threshold, intensity, duration and frequency of experiencing pleasure. It has 5 items. *High Intensity Pleasure* which is pleasure related to situations involving high stimulus intensity, rate, complexity, novelty and incongruity. It has 7 items.

Factor Scale 3: Effortful Control (19 items): Composed of 3 scales: *Attentional Control* which is the capacity to focus attention as well as to shift attention when desired. It has 5 items. *Inhibitory Control* which is the capacity to suppress inappropriate approach behaviour and it has 7 items. *Activation Control* which refers to the capacity to perform an action when there is strong tendency to avoid it. It has 7 items.

Factor Scale 4: Orienting Sensitivity (15 items): Composed of 3 scales: *Neutral Perceptual Sensitivity* which is the detection of slight, low intensity stimuli from both within the body and the external environment. It has 5 items. *Affective Perceptual Sensitivity* which is the spontaneous emotional valence, conscious cognition associated with low intensity stimuli. It has 5 items. *Associative Sensitivity* which refers to the spontaneous cognitive content that is not related to standard associations with the environment. It has 5 items.

The ATQ-SF is composed of 77 items in total rated on a 7-response option Likert scale (1 = extremely untrue to 7 = extremely true). There is also a response option x = not applicable which if answered accounts for a missing item. The mean item responses from the whole sample are used to replace the missing values. Twenty nine questions are reverse

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scored and there are no cut-off scores (Evans & Rothbart, 2007b). The score of the 13 scales is computed by adding the scores and then dividing by the number of valid item responses. The scores of the 4 factor scales is computed by adding the scores of all scales belonging to that factor scale and then dividing by the number of items that belong to that factor scale.

Coefficient alphas for the scales range from .62 to .85 (Evans & Rothbart, 2007b) and from .53 to .84 for the non-English versions (Laverdière et al., 2010; Wiltink et al., 2006). The ATQ has been translated in many languages (Laverdière et al., 2010).

Translation of the ATQ into Greek language

The ATQ was translated by the author into Greek (forward-translation) after obtaining permission from the Marry Rothbart Temperament Lab (Putnam, personal communication, 14 May 2015). Guidance was provided during the translation process regarding certain items that troubled the researcher (Putnam, personal communication, 18 May 2015), namely, for item 8, an explanation for ‘flow through with’ was provided. For item 10, a confirmation of the researcher’s perception of ‘barely noticeable’ was provided. For item 13, the researcher proposed that a parenthetical clarification would be appropriate, and it was suggested that the clarification should read something similar or identical to (*I tend to notice aspects of musical pieces that convey minor or small changes in emotion*). Due to how it would read in Greek, the word *minor* was omitted. For item 39, the initial response was that if an example were provided and the participants did not have that particular experience, the item could be under-reported. The researcher then explained that she requested an example because when people close their eyes in brightly lit environments, they may see abstract shapes and colours while the eye adjusts to the new conditions, and this has nothing to do with temperament. There was no suggestion by Putnam to clarify the item. For item 76, the researcher requested an example and *laughing at church or library* was suggested, with the option to change it if this

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was incompatible with Greek culture. The example remained the same as it is compatible with Greek culture.

I also made a proposal to the authors regarding the responses for the ATQ, which range from extremely untrue to extremely true because it was a known problem from the CTQ questionnaire. The proposal was to use the range of responses from another of Rothbart's questionnaires, the IBQ-R, which has 7 responses as well, ranging from never to always. The response was that changing the scale could lead to comparison problems with other studies, adding though that this change would be essentially fine if the researcher uses the scale to study relations between temperament and other variables in Study 2. It was decided that the response range would not change in the instructions but the table with the responses on every page would only explain numbers 1 (extremely false), 4 (neither true nor false) and 7 (extremely true), omitting the in-between explanations as they seem troubling in Greek. Additionally, the word 'untrue' was replaced with 'false'. By doing this, there were no essential changes to the questionnaire, allowing comparability with other studies because, in Putnam's opinion, respondents should not have any problems inferring the intent of the intermediate scores (i.e. 2, 3, 5, 6), and 'false' is a good synonym to use in place of 'untrue.' After the issues during the translation process were resolved, the questionnaire was back-translated by bilingual Greek-English native speakers. A panel of PhD- and master's-level psychologists checked the translation and back-translation and compared it with the English version to detect any discrepancies or inadequate expressions of the translation before proceeding to back-translate it, which did not result in any changes. The WHO guidelines for translating instruments were followed (Sousa & Rojjanasrirat, 2011).

Putnam (personal communication, 18 May 2015) also commented on the first back-translation, and this resulted in a few changes to the Greek version. For item 5, the translation

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changed to clarify that the item means that someone can switch focus between two tasks. For item 14, ‘serving the public’ was chosen instead of ‘socialising’, so that the item’s meaning was clearer in both the translation and the back-translation. For item 31, the back-translation of ‘agitated’ was ‘nervous’, and this resulted in a change to the translation. For item 41, the back-translation resulted in another way of saying ‘loosely connected’. For item 44, the researcher added ‘rollercoaster’ as an example to ensure that the item kept its original meaning in the translation. For items 48 and 58, Putnam proposed a synonym (annoyed) for the word ‘frustrated’, as the translation produced ‘disappointment’ in the back-translation. After making changes to the forward-translation resulting from the first back-translation, the Greek version was back-translated again, and Putnam decided that the instrument was ready for use.

A pilot study was conducted to check how the translated questionnaire (Appendix 37) performs in a Greek population. The pilot study took place in May 2015. The ATQ-SF was administered to thirty ($n = 30$) female participants from the community in Athens, Greece to represent 10% of Study 2’s overall participants, as this was the lowest sufficient number for a pilot study (Connelly, 2008; Hertzog, 2008; Lackey & Wingate, 1998). A clinical population was not included in the pilot study for feasibility reasons, as there was no reason to expect that a clinical population would interpret the meaning of these words any differently. Their mean age was 33.43 ($SD = 8.41$), and their mean educational level was 16 years of education.

Participants were asked to comment on the items’ and instructions’ clarity and the overall presentation of the scales. Ten ($n = 10$) people asked for clarification regarding item 54. It was decided to add an explanation in parentheses (advertising signs, flashing lights in clubs). For item 77, ‘to me’ was added after ‘comes’ in order to be more specific (*I would not enjoy the feeling that comes to me from yelling as loud as I can*). To avoid the answer ‘x =

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not applicable' being erroneously used to indicate that something never happens to them, a remark was added in the box with the answers on every page reading *check instructions for this answer*. In the instruction right after the sentence, as it is in the English version, an extra clarification was added: The x answer does not mean that what you read in the sentence does not happen to you. In the case that what you read in the statement never happens to you, answer 'I', according to the above instructions. Table 6 presents Cronbach's α results of the pilot study along with the results reported by the ATQ authors. Table 7 presents Cronbach's α for the measures used in Study 2.

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Table 6.

Cronbach's α results of the pilot study along with those reported by the ATQ authors and a study in a non-English-speaking population

ATQ 4 factor scales & 13 scales	Pilot Study Cronbach's α of the Greek version N = 30 females	Original version by the test authors (Evans & Rothbart, 2007b) N = 258 undergraduates	Finnish version (Kiuru et al., 2019) N= 685 parents
NEGATIVE AFFECT	.83	.81	.65
<i>Fear</i>	.57	.64	
<i>Sadness</i>	.67	.62	
<i>Discomfort</i>	.69	.69	
<i>Frustration</i>	.68	.72	
EFFORTFUL CONTROL	.70	.78	.69
<i>Inhibitory Control</i>	.36	.60	
<i>Activation Control</i>	.64	.69	
<i>Attentional Control</i>	.67	.73	
EXTRAVERSION/SURGENCY	.66	.75	.53
<i>Sociability</i>	.38	.71	
<i>High Intensity Pleasure</i>	.48	.68	
<i>Positive affect</i>	.51	.62	
ORIENTING SENSITIVITY	.68	.85	.69
<i>Neutral Perceptual Sensitivity</i>	.32	.64	
<i>Affective Perceptual Sensitivity</i>	.68	.79	
<i>Associative Sensitivity</i>	.46	.67	

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Note: Factor scales listed in capitals. Scales (i.e. sub-constructs) for factor scales listed in non-capitals below their factor scale. The mean age of participants was 33.43 ($SD = 8.41$) years and they had a mean educational level of 16 years of education. The subscales alpha values in the non-English version were not available. Study 2b used the factor scales.

Table 7.

Cronbach's α for the measures used in Study 2 both for ED patients and controls

	Cronbach's alpha	
	ED patients N = 80	Controls N = 188
Childhood Trauma Questionnaire (non-physical abuse)		
<i>Emotional Abuse</i>	.87	.82
<i>Emotional Neglect</i>	.87	.89
<i>Physical Neglect</i>	.64	.53
Multidimensional Neglect (non-physical neglect)	.82	.69
Adult Temperament Questionnaire (temperament)		
<i>Negative Affect</i>	.81	.74
<i>Orienting Sensitivity</i>	.76	.71
<i>Extraversion Surgency</i>	.65	.59
<i>Effortful Control</i>	.74	.71
WHOQOL-BREF (quality of life)		
<i>Psychological Domain</i>	.88	.76
<i>Social Relationships (without national items)</i>	.64	.66
Rosenberg Self-Esteem Scale (self-esteem)	.88	.88
Difficulties in Emotion Regulation Scale (emotion regulation)		
<i>DERS total score</i>	.94	.93
Relationship Structure Questionnaire (attachment)		
<i>Global avoidance</i>	.84	.88
<i>Global Anxiety</i>	.89	.87
Eating Disorders Examination Questionnaire-EDE-Q 6 (eating disturbances)		
<i>Global EDE-Q score</i>	.90	.92

Note: The CTQ physical neglect scale and the ATQ extraversion surgency scale were not used in Study 2 as explained in Chapter 6.

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Questionnaires' performance. An α value above .70 is considered acceptable, but above .80 is preferred (Cortina, 1993). Authors suggest an acceptable α value is above .60 but below .95 (Hulin et al., 2001), as a high α value could be an indication of items' redundancy. As can be seen in Tables 1 and 7, certain scales do not satisfy this requirement of an α value of at least .70, mainly for the control group. In Study 1, the α for patients ranges from .70 to .93 and for controls from .51 to .93. In Study 2, α for patients ranges from .64 to .94 and for controls from .59 to .93. Having a low Cronbach's alpha makes it difficult to find statistically significant results, if they exist, due to a higher percentage of random error variance (Tavakol & Dennick, 2011). For instance, an alpha value of .59 (the lowest value in Table 7), suggests that there is .65 random error variance ($.59 \times .59 = .35$; $1 - .35 = .65$). When the reliability increases, the random error variance decreases (Tavakol & Dennick, 2011). Classical test theory (Novick, 1966) proposes that each person has a true score that could be obtained if there were no measurement error. A participant's true score is the expected score over numerous independent administrations of the questionnaire. Researchers obtain the observed score rather than the true score, so it is hypothesised that the true score is the score observed plus some error (Tavakol & Dennick, 2011). In other words, a low Cronbach's alpha will result in a lower true score and higher error. However, it is still possible to identify meaningful and statistically significant effects with scales with moderate alphas (Kline, 2011), and both studies used SEM models, which measure random error variance, resulting in unbiased estimates of the relationships between latent constructs (Kline, 2011). Moreover, some scales performed better in the second pilot study regarding the translated ATQ (temperament) and CTQ (childhood non-physical abuse) questionnaires, with alphas ranging from .66 to .83 and from .76 to .85, respectively (see Tables 6 and 3). These values are similar to the original authors' values (e.g. .89 to .92 for the CTQ) and to other studies that have used the measures in non-English-speaking populations (see Tables 6 and 3).

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Specifically, for the ATQ, coefficient alphas for the scales range from .62 to .85 (Evans & Rothbart, 2007b) and from .53 to .84 for the non-English versions (Laverdière et al., 2010; Wiltink et al., 2006), indicating that the ATQ's mediocre alpha values are not unusual, especially in non-English-speaking populations. The ATQ's moderate reliabilities were improved in Study 2 by using explanatory parentheses to clarify certain questions, which improved the reliability values for the scales of orienting sensitivity and effortful control (see Tables 6 and 7). As the ATQ was the only measure available for temperament, along with the consideration of its α values in the non-English versions, moderate reliability was deemed to be acceptable. The extraversion surgency scale, which had the lowest alpha value, was not used in the SEM models, as explained in Chapter 6, Section 6.4.3.

Selection of the questionnaires. All measures were chosen for their ability to comprehensively assess the psychological construct of interest and for their robust psychometric properties, as described above. Given the robustness of SEM and the potential for scales with moderate alphas to still produce meaningful effects (Kline, 2011), all scales were used in the PhD studies.

In sum, much consideration was given prior to the main studies regarding selection of reliable and valid questionnaires. As described above, some of these decisions were made with the aim of identifying a scale that had an appropriately high alpha. The additional measure of the MNBS was employed in case the CTQ's neglect scales displayed low internal consistency reliability. The MNBS was ultimately used in Study 2, as the CTQ physical neglect scale did not perform well and was, therefore, excluded from the analyses. The same measures were taken regarding the CTQ emotional abuse scale, as approval was given (A. Kent, personal communication, 18 May 2015) to use the emotional abuse scale from the Child Abuse Trauma Scale (CATS) (Kent & Waller, 1998) to supplement the CTQ. The same

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process of translation and back-translation was followed, and the author approved the final version (Kent, personal communication, 26 May 2015). However, the CATS emotional abuse scale was not needed, as the CTQ emotional abuse scale proved to be internally consistent. Regarding the ATQ temperament questionnaire, there are no theoretically comparable and psychometrically robust alternative measures in Greek. Cloninger's Temperament and Character Inventory (TCI) (Cloninger et al., 1993), for example, is fraught with technical and theoretical issues. The ATQ was the only choice as it is based on a contemporary theory that better conceptualises temperament. Additionally, the authors were available to support the translation (see Section 4.3.3 in this chapter). The DERS was selected for emotion regulation as the conceptualisation captures multiple aspects of emotion regulation (Gratz & Roemer, 2004; Hallion et al., 2018), and the literature review indicated that it is psychometrically robust (Hallion et al., 2018). Furthermore, the DERS proved reliable in the pilot study.

In addition to selecting supplementary scales, this thesis's author included explanatory parentheses to clarify several items and instructions within the three translated questionnaires, which did in fact improve their performances in the last pilot studies (see Section 4.3.3 in this chapter). The rest of the questionnaires used in Studies 1 and 2 have already been used in Greek studies, and their alpha levels do not really differ from those reported in other countries. Moreover, Study 1 had to use the same measures as the author's master's study, as it was an expanded design. These measures performed well in the master's study.

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Study 3

For this study, a semi-structured interview schedule was used. At first, there were questions regarding age, education, working status, duration of illness and age of ED onset, followed by the interview questions:

- 1) Could you tell me about your experience of non-physical abuse in your adult life?
- 2) Is there something similar or different regarding your adult experience of non-physical abuse compared with your relevant experiences as a child/teenager?
- 3) Why do you believe this abuse happened when you were a child/teenager?
- 4) Why do you think it is still happening?
 - What factors, in your opinion, maintain this behaviour towards you?
 - How do you feel about the abuse?
- 5) How do you cope?
 - Is there something that makes it difficult/easy for you to cope?
 - Has therapy changed/not changed the way you see it?
- 6) A) Do you think the abuse is linked with your eating disorder symptoms now in anyway?
 - *In case they answer yes*: How do you think is it linked?
 - *In case they answer no*: What factors are linked with your eating disorder symptoms?B) Do you think the abuse as a child is linked with your experience of eating disorder development?
 - *In case they answer yes*: How do you think it is linked?
 - *In case they answer no*: What factors are linked to the development of your eating disorder?
- 7) Do you think this situation will change?
 - *In case they answer yes*: How do you think it will change?
 - *In case they answer no*: What factors do you think will prevent this change?

4.3.4. Procedure

Study 1 and Study 2. The studies received ethics approval from the PSYSOC ethics committee at the University of Central Lancashire (UCLan) (Appendix 1). Questionnaires for patients were placed at the outpatients units' reception desks, and patients were informed about the study by their therapists. Those who agreed to participate took a copy, which was returned in a sealed envelope within one week. Controls were recruited from colleges in Athens, Greece. The questionnaires were placed at the college reception desks with instructions that the questionnaires could also be given to family and friends and should be returned to the college reception desk in sealed envelopes. Participants' information packs included information (Appendix 3 for Study 1 and Appendix 5 for Study 2) about the study, as well as a debriefing form (Appendix 4 for Study 1 and Appendix 6 for Study 2). Codes were used instead of names for anonymity. Participants could withdraw from the study by not returning the questionnaires or by emailing the author with their code one month after completion. Hard copies of the questionnaire were kept in a locked room in the author's office. The data was stored coded and password-protected, accessible only to the author.

Study 3. The study received ethics approval from UCLan University's PSYSOC ethics committee (Appendix 2). The patients' therapists informed them about the nature of the study prior to recruitment, and an appointment for the interview was booked in the outpatient unit immediately before their regular therapy session. Patients were informed in writing and orally about withdrawal, anonymity, the use of pseudonyms in the analysis, data storage and that members of their clinical team would not be aware of the interview content (Appendix 7). The first two interviews took place in a quiet room in the outpatients unit. The last four interviews were conducted online through Skype. The author conducted all of the interviews. The mean length of the interviews was 56.5 minutes. There were a few minutes of

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small talk to establish rapport before the interviews commenced. I introduced myself, and there was a general discussion about the importance of psychologists' work led by participants upon hearing that I am a professional psychologist. To close the interviews, I gave a positive statement about how things that make people feel uncomfortable can be improved through discussion in psychotherapy. Participants also received a debriefing form via email at the end of the interview (Appendix 8).

4.3.5. Ethical Considerations

All three studies received ethics approval from UCLan's PSYSOC committee (Appendices 1 and 2). Anonymity was preserved for Studies 1 and 2. Full anonymity was not possible in Study 3, but the data were made anonymous through the use of pseudonyms. The patients recruited for the three studies were already under psychotherapy or had been referred to begin psychotherapy by a psychologist or psychiatrist. This means that they had a therapist to refer to if certain interview topics (i.e. childhood abuse, family relationships) were difficult for them to process. The participants in the control group were able to book an appointment for free in a psychiatric clinic two days after their request if they felt overwhelmed by the study. The British Psychological Society guidelines for research and conduct were followed (BPS, 2009, 2010).

Chapter 5: Study 1 – Effects of Family Functioning on Eating Pathology and Psychosocial Quality of Life: The Mediating Role of Self-esteem

5.1. Abstract

Background: Eating disorders (EDs) are complex regarding aetiology and symptom maintenance, and the psychosocial quality of life (QoL) of ED patients adds to this complexity, remaining unchanged even after ED symptoms have reduced. Family dysfunction and self-esteem play an important role in ED development, but this role has not been sufficiently examined regarding eating pathology and psychosocial QoL in a clinical population with the use of a parsimonious yet comprehensive theoretical model. The purpose of this study was to therefore assess the mediating role of self-esteem between family dysfunction and both eating pathology and psychosocial QoL in ED patients and healthy controls.

Method: 154 female adult ED patients and 154 female healthy adult controls were recruited from Athens, Greece, and self-reported measures were used to assess family dysfunction, eating pathology, self-esteem and psychosocial QoL. Structural equation modelling (SEM) was employed to test the mediation hypotheses.

Results: The mediation hypotheses were confirmed, as self-esteem mediated the relationship between family dysfunction and both ED and psychosocial QoL in both groups. Family dysfunction did not affect ED directly in any group, but it did have an indirect effect through self-esteem as the mediator. Family dysfunction, self-esteem and eating pathology had a direct effect on both groups' psychosocial QoL.

Conclusion: Self-esteem's important role in ED was confirmed along with its sensitivity to family dysfunction. Both the clinical and non-clinical sample had the same direct and indirect

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effects, highlighting the importance of family functioning and self-esteem beyond the presence of EDs. It is time to move on from simple direct-effects models of family functioning in order to better understand the psychological mechanisms involved in this complex relationship. Future studies should further investigate the way self-esteem is disturbed, leading to lifelong consequences for ED patients.

5.2. Introduction

Eating disorders continue to puzzle researchers and clinicians due to their complexity regarding aetiology and symptom maintenance, as well as their resistance to treatment and high mortality rates (Abbate-Daga et al., 2013; Eshkevvari et al., 2014; Gonidakis et al., 2015; Halmi, 2013; Hay et al., 2012; Kroplewski et al., 2019; Le Grange et al., 2014; Touyz et al., 2013; Walsh, 2013). Moreover, EDs appear to be highly prevalent in high school and academic grounds (Aparicio-Llopis et al., 2014; Hilbert & Czaja, 2009; Khodabakhsh et al., 2015; Taylor et al., 2006), meaning that they are disorders that can directly disrupt normal growth and physical health; thus, early intervention is important (Ozier & Henry, 2011).

Empirical literature also demonstrates that ED patients report significantly lower QoL compared to healthy controls, mood disorder patients and patients with a chronic physical illness (Bamford & Sly, 2010; Hay & Mond, 2005; Jenkins et al., 2011; de la Rie et al., 2005; de la Rie et al., 2007). Moreover, no significant differences on reported QoL have been observed between the ED subtypes (González et al., 2001; de la Rie et al., 2005), suggesting that low QoL cannot be explained by ED symptoms. Furthermore, longitudinal studies demonstrate that ED patients maintain low QoL even after years of treatment (Padierna et al., 2002). These findings suggest that reducing ED symptoms is not enough to improve QoL, and that illness chronicity cannot adequately explain low QoL. With the aetiology of EDs still an issue and their prevalence rising in Western societies, including the UK and Greece (Bilali et al., 2010; Fairburn & Harrison, 2003; Kaplan & Sadock, 2007; Keski-Rahkonen &

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Mustelin, 2016; Kyrgios et al., 2015; Micali et al., 2013; Rikani et al., 2013; Watson et al., 2016), it is important to identify new pathways that are potential risk factors for eating pathology. This is imperative to the development of effective prevention programmes and therapeutic interventions.

Family dysfunction

Family dysfunction is consistently associated with eating pathology (Cerniglia et al., 2017; Horesh et al., 2015; McDermott et al., 2002; Polivy & Herman, 2002; Tetzlaff et al., 2016). Moreover, it has been found in all ED patients, regardless of subtype, with significant difference when compared to healthy control families (Cerniglia et al., 2017; Holtom-Viesel & Allan, 2014; McDermott et al., 2002), indicating that it is a transdiagnostic factor in EDs. Leading theories of EDs have focused on unresolved familial conflicts to explain the origins of ED symptoms (Bruch, 1973, 1982; Minuchin et al, 1975). Despite criticism that these theories lack empirical support and the suggestion that problematic dynamics may be the result of an ED and not necessarily the cause (Eisler, 2005), the literature indicates that dysfunction in the family pre-exists ED onset, and the diagnosis adds to the dysfunction (Allen et al., 2014; Holtom-Viesel & Allan, 2014; Karwautz et al., 2003; Wade et al., 2007). In addition, reviews indicate that family areas of functioning, as identified by the McMaster model of family functioning (Epstein et al., 1978), have value for ED families (Holtom-Viesel & Allan, 2014). According to this model, family functioning is a complex phenomenon involving many aspects, such as managing problems, enhancing personal development, effective communication, and expressing and managing emotions.

Self-esteem

Trait self-esteem is a global and unidimensional construct of one's personal judgment regarding one's worth (Rosenberg, 1965). Low trait self-esteem has been associated with

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eating pathology in a range of studies and samples, both clinical and non-clinical, including all ED subtypes (e.g. Button & Warren, 2002; Cervera et al., 2003; Dunkley & Grilo, 2007; Fisher et al., 1994; Gual et al., 2002; Holston & Cashwell, 2000; Katsourani, 2009; Leon et al., 1999; Mintz & Betz, 1988; Newns et al., 2003; Peck & Lightsev Jr, 2008; Stice, 2002; Woodward et al., 2019). Low self-esteem's presence in all ED subtypes makes it a transdiagnostic factor. It has also been associated with low QoL in ED patients (de la Rie et al., 2005), along with family dysfunction (Ha et al., 2006; Lau & Kwok, 2000; Smets & Hartup, 1988) and poor psychosocial functioning (Ciao et al., 2015). Self-esteem is considered a major risk factor for onset, maintenance and relapse in EDs (Biney et al., 2019). Despite the empirical support between self-esteem and disordered eating, the direction of causality in the relationship between self-esteem and EDs is unclear (Adamson et al., 2019).

Family dysfunction and self-esteem

The familial environment is crucial for the development of the self (Harter, 2012). Longitudinal studies indicate its enduring effects on trait self-esteem, which can be observed in adulthood (Orth, 2018). A review of the available literature suggests that family dysfunction may lead to the development of an ED through lowering self-esteem, leading to body dissatisfaction/drive for thinness, and dieting and associated behaviours (use of diuretics, laxatives, self-induced vomiting). For example, studies indicate that people with dysfunctional families report higher levels of body dissatisfaction and a higher drive for thinness, which are both risk factors for ED development (Knobloch-Westerwick & Crane, 2012; Leung et al., 1996; Ordman & Kirschenbaum, 1986). Moreover, body dissatisfaction is associated with low self-esteem (Balcetis et al., 2013; Tiggemann et al., 2009). Body image and appearance, along with self-esteem, belong to the psychological domain of QoL, indicating that family dysfunction could have a negative effect on that domain via low self-

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esteem. This suggests an association between family dysfunction and low self-esteem with respect to both ED development and psychosocial QoL, given that family dysfunction and low self-esteem have emerged as risk factors for eating pathology (Anokhina, 2015; Wisotsky et al., 2003).

In sum, the empirical literature provides ample support for relationships between family dysfunction, trait self-esteem, eating pathology and QoL. Trait self-esteem is estimated to be a major risk factor, as well as an aetiological factor for EDs (Adamson et al., 2019; Biney et al., 2019; Holston & Cashwell, 2000; Stice, 2002). Furthermore, it has previously emerged as a mediator in the relationship between family functioning and increased risk for eating pathology in a non-clinical population (Kroplewski et al., 2019). However, research is lacking regarding self-esteem's mediating role between family dysfunction and eating pathology, as well as psychosocial QoL in a clinical population. No study has collectively explored these psychological constructs together in a combined theoretical model in ED patients. This includes considering the impact of these constructs on ED patients' psychosocial QoL as an important therapeutic outcome.

In light of this, the purpose of this study was to examine the mediating role of self-esteem between family functioning and ED, and family functioning and psychosocial QoL in ED patients, comparing the results to a non-clinical healthy control group. This thesis hypothesised that self-esteem mediates the relationship between family dysfunction and ED/psychosocial QoL in both groups.

5.3. Method

5.3.1. Study Design

Structural equation modeling was used to assess associations between the study variables in which the dependent variables (endogenous) were: a) two dimensions of psychosocial QoL (psychological health, social relationships), and b) four dimensions of ED psychopathology (restraint, eating concern, shape concern, weight concern). The independent variables (exogenous) were the dimensions of family functioning that measured: problem solving, communication, roles, general functioning, affective responsiveness, affective involvement, behaviour control. The mediator was global self-esteem.

5.3.2. Participants

A total of 308 Greek adult female participants (154 ED patients and 154 controls) completed the survey over two different periods (February 2013–June 2013 and March 2015–June 2015) and were eligible for inclusion in the analyses. The first 88 ED patients and 44 controls had already been drawn as participants for the author's master's research study. The 154 ED outpatients had a mean age of 31.12 years ($SD = 10.82$) and the 154 controls had a mean age of 32.80 years ($SD = 10.33$) and were drawn from the community (students, employees). Of the total number of patients, 45 (14.6%) had been diagnosed with anorexia nervosa (AN) (18 with the restricting type, 18 with the purging type, 9 with other specified AN); 60 (19.5%) had been diagnosed with the bulimia nervosa (BN) (58 with BN, 2 with other specified BN) and 49 (15.9%) had been diagnosed with binge eating disorder (BED). The controls did not have a score in the eating disorders examination questionnaire (EDE-Q) indicating an ED, nor did they have a score over 2.30 indicating a subclinical ED, meaning it was indeed a non-clinical sample (Thurston et al., 2008).

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Participation was voluntary. The inclusion criteria for the ED group were being an adult female of Greek nationality with an ED diagnosis. The inclusion criteria for the control group were the same, minus the ED diagnosis, and there was also an instruction in their envelopes not to fill in the questionnaires if they have eating disturbances. The exclusion criteria for both groups were participant's subjective judgement of severe chronic physical illness that could affect their QoL (asked in the questionnaires), developmental disorders, current pregnancy, psychosis, current major depression, addictions and bipolar disorder. The exclusion criteria for the ED patients were also available from their therapists, who were briefed about the participation criteria.

ED outpatients were drawn from the psychiatric department's outpatient unit at Eginition Hospital, National and Kapodistrian University of Athens; 18 ANO outpatient ED department at Dafni Psychiatric Hospital; the ANASA Day Care Centre for ED outpatients and from Binge Eaters Anonymous. All are located in Athens, Greece, and the author received written permission to recruit the clinical population from these units. ED patients met the criteria for ED diagnosis according to the Diagnostic and Statistical Manual for Mental Disorders (DSM-V) (APA, 2013) after being evaluated by clinicians via the standard clinical interview, the eating disorders inventory and the EDE-Q prior to their assignment for treatment in the outpatient units. The diagnoses of the first 88 ED patients, who were part of the author's master's study, were reanalysed post-hoc according to the DSM-V criteria, as they first met the DSM-IV-TR (APA, 2000) criteria. This practice is also used by other authors (Wolz et al., 2015).

For a full description of the population's socio-demographic and clinical characteristics see Tables 8 and 9, respectively.

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Table 8.

Descriptive statistics in means (standard deviation) and frequencies (%) of the ED patients' (both by diagnostic group and as a whole) and the control group's socio-demographics – Study 1.

	AN PATIENTS n = 45 (14.6%)	BN PATIENTS n = 60 (19.5%)	BED PATIENTS n = 49 (15.9%)	EATING DISORDER PATIENTS n = 154 (50%)	CONTROLS n = 154 (50%)
EDUCATIONAL					
LEVEL (total years of study)					
<i>Primary school (6 years)</i>	0(0%)	0(0%)	0(0%)	0(0%)	1 (.6%)
<i>High school gymnasium (9 years)</i>	0(0%)	0(0%)	1 (2%)	1 (.6%)	1 (.6%)
<i>High school lyceum (12 years)</i>	10 (22.2%)	11 (18.3%)	11 (22.4%)	32 (20.8%)	43 (27.9%)
<i>After school (14 years)</i>	5 (11.1%)	4 (6.7%)	10 (20.4%)	19 (12.3%)	11 (7.1%)
<i>College (16 years)</i>	28 (62.2%)	34 (56.7)	20 (40.8%)	82 (53.2%)	62 (40.3%)
<i>Post graduate studies (17–19 years)</i>	2 (4.4%)	11 (18.3%)	7 (14.3%)	20 (13%)	36 (23.4%)
WORKING STATUS					
<i>Full time</i>	10 (22.2%)	18 (30%)	22 (44.9%)	50 (32.5%)	93 (60.4%)
<i>Part time</i>	4 (8.9%)	11 (18.3%)	8 (16.3%)	23 (14.9%)	14 (9.1%)
<i>Not working (retired (or householder)</i>	1 (2.2%)	3 (5%)	10 (20.5%)	14 (9.1%)	7 (4.5%)
<i>Not working (unemployed)</i>	4 (8.9%)	10 (16.7%)	6 (12.2%)	20 (13%)	7 (4.5%)
<i>Not working (college student)</i>	26 (57.8%)	18 (30%)	3 (6.1%)	47 (30.5%)	33 (21.4%)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
MARITAL STATUS					
<i>Single</i>	41 (91.1%)	52 (86.7%)	19 (38.8%)	112 (72.7%)	97 (63%)
<i>Married</i>	4 (8.9%)	5 (8.3%)	21 (42.9%)	30 (19.5%)	50 (32.5%)
<i>Separated/divorced</i>	0(0%)	3 (5%)	9 (18.3%)	12 (7.8%)	7 (4.5%)
PARENT					
<i>Yes</i>	3 (6.7%)	5 (8.3%)	28 (57.1%)	36 (23.4%)	48 (31.2%)
<i>No</i>	32 (93.3%)	55 (91.7%)	21 (42.9%)	118 (76.6%)	106 (68.8%)
NUMBER OF CHILDREN	.09 (.36)	.18 (.68)	1.00 (1.10)	.42 (.87)	.55 (.86)
SIBLINGS					
<i>Yes</i>	42 (93.3%)	53 (88.3%)	42 (85.7%)	137 (89%)	138 (89.6%)
<i>No</i>	3 (6.7%)	7 (11.7%)	7 (14.3%)	17 (11%)	16 (10.4%)
NUMBER OF SIBLINGS	1.24 (.77)	1.28 (.87)	1.35 (.97)	1.29 (.87)	1.45 (1.10)
SIBLING GENDER					
<i>Female</i>	10 (22.2%)	12 (20%)	11 (22.4%)	33 (21.4%)	32 (20.8%)
<i>Male</i>	20 (44.4%)	17 (28.3%)	9 (18.4%)	46 (29.9%)	22 (14.3%)
<i>Both genders</i>	1 (2.2%)	5 (8.3%)	3 (6.1%)	9 (5.8%)	3 (1.9%)
<i>Not mentioned</i>	11 (24.4%)	19 (31.7%)	19 (38.8%)	49 (31.8%)	81 (52.6%)
<i>None (no sibling)</i>	3 (6.7%)	7 (11.7%)	7 (14.3%)	17 (11%)	16 (10.4%)
BIRTH ORDER					
<i>1st born</i>	11 (24.4%)	20 (33.3%)	25 (51%)	56 (36.4%)	59 (38.3%)
<i>2nd born</i>	26 (57.8%)	27 (45%)	13 (26.5%)	66 (42.9%)	54 (35.1%)
<i>3rd born</i>	2 (4.4%)	5 (8.3%)	3 (6.1%)	10 (6.5%)	18 (11.7%)
<i>4th born</i>	0(0%)	0(0%)	0(0%)	0 (0%)	3 (1.9%)
<i>5th born</i>	1 (2.2%)	0(0%)	1 (2%)	2 (1.3%)	1 (.6%)
<i>Twin</i>	2 (4.4%)	1 (1.7%)	0(0%)	3 (1.9%)	3 (1.9%)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
<i>Only child</i>	3 (6.7%)	7 (14.3%)	7 (14.3%)	17 (11%)	16 (10.4%)
LIVING STATUS					
<i>Alone</i>	8 (17.8%)	17 (28.3%)	5 (10.2%)	30 (19.5%)	31 (20.1%)
<i>Not alone</i>	37 (82.2%)	43 (71.7%)	44 (89.8%)	124 (80.5%)	123 (79.9%)
PEOPLE LIVING WITH					
<i>Family members (e.g. parents, siblings)</i>	31 (68.9%)	32 (53.3%)	18 (36.6%)	81 (52.7%)	62 (40.4%)
<i>Boyfriend/spouse, kids</i>	6 (13.3%)	10 (16.7%)	26 (53.2%)	42 (27.2%)	59 (38.2%)
<i>Roommate/friend</i>	0 (0%)	0 (0%)	0 (0%)	0 (0%)	4 (2.6%)
<i>None</i>	8 (17.8%)	18 (30%)	5 (10.2%)	31 (20.1%)	29 (18.8%)
RESIDENCE					
<i>Urban Athens</i>	37 (82.2%)	48 (80%)	39 (79.6%)	124 (80.5%)	107 (69.5%)
<i>Urban other</i>	2 (4.4%)	7 (11.7%)	8 (16.3%)	17 (11%)	21 (13.6%)
<i>Rural (village, island)</i>	6 (13.3%)	5 (8.3%)	2 (4.1%)	13 (8.4%)	26 (16.9%)
LONGER STAY RESIDENCE					
<i>Urban Athens</i>	26 (57.8%)	42 (70%)	37 (75.5%)	105 (68.2%)	97 (63%)
<i>Urban other</i>	2 (4.4%)	8 (13.3%)	7 (14.3%)	17 (11%)	20 (13%)
<i>Rural (village, island)</i>	17 (37.8%)	10 (16.7%)	5 (10.2%)	32 (20.8%)	37 (24%)

Note: The ED subgroups are mentioned for informative reasons, and they were not used in the analyses.

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Table 9.

Descriptive statistics in means (standard deviation) and frequencies (%) of the ED patients' (both by diagnostic group and as a whole) and the control group's clinical data – Study 1.

	AN PATIENTS n = 45 (14.6%)	BN PATIENTS n = 60 (19.5%)	BED PATIENTS n = 49 (15.9%)	EATING DISORDER PATIENTS n = 154 (50%)	CONTROLS n = 154 (50%)
	Mean (SD) or frequency %	Mean (SD) or frequency %	Mean (SD) or frequency %	Mean (SD) or frequency %	Mean (SD) or frequency %
Current age (years)	24.60 (5.66)	27.88 (7.30)	41.06 (11.16)	31.12 (10.82)	32.80 (10.33)
Age of ED onset (years)	17.48 (3.37)	16.88 (4.24)	18.18 (8.15)	17.47 (5.60)	0 (0)
Duration of illness (years)	7.12 (6.00)	11.00 (8.22)	22.88 (12.89)	13.65 (.92)	0 (0)
Hospitalised for ED					
<i>Yes</i>	8 (17.8%)	1 (1.7%)	3 (6.1%)	12 (7.8%)	0 (0%)
<i>No</i>	37 (82.2%)	59 (98.3%)	46 (93.9%)	142 (92.2%)	154 (100%)
Times hospitalised for ED	.31 (.73)	.02 (.13)	.12 (.53)	.14 (.51)	0 (0)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
Duration of hospitalisation (days)	11.47 (48.13)	.03 (.26)	1.14 (5.52)	3.73 (26.47)	0 (0)
Perceived health status					
<i>Very bad</i>	3 (6.7%)	2 (3.3%)	1 (2%)	6 (3.9%)	1(.6%)
<i>Bad</i>	2 (4.4%)	3 (5%)	3 (6.1%)	8 (5.2%)	0(0%)
<i>Neither good nor bad</i>	13 (28.9%)	9 (15%)	10 (20.4)	32 (20.8%)	11 (7.1%)
<i>Good</i>	19 (42.2%)	32 (53.3%)	22 (44.9%)	73 (47.4%)	67 (43.5%)
<i>Very good</i>	8 (17.8%)	14 (23.3%)	13 (26.5%)	35 (22.7%)	75 (48.7%)
Facing any health problem					
<i>Yes</i>	20 (44.4%)	20 (33.3%)	21 (42.9%)	61 (39.6%)	25 (16.2%)
<i>No</i>	25 (55.6%)	40 (66.7%)	28 (57.1%)	93 (60.4%)	129 (83.8%)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
Type of Problem					
<i>None</i>	25 (55.6%)	41 (68.3%)	28 (57.1%)	94 (61%)	136 (88.3%)
<i>Mental health</i>	9 (20%)	5 (8.3%)	2 (4.1%)	16 (10.4%)	0(0%)
<i>Cardiological</i>	2 (4.4%)	0(0%)	6 (12.2%)	8 (5.2%)	3 (1.9%)
<i>Ophthalmological</i>	0(0%)	0(0%)	1 (2%)	1 (.6%)	0(0%)
<i>Endocrinological</i>	2 (4.4%)	4 (6.7%)	1 (2%)	7 (4.5%)	2 (1.3%)
<i>Orthopaedic</i>	1 (2.2%)	3 (5%)	3 (6.1%)	7 (4.5%)	9 (5.8%)
<i>Gastrointestinal</i>	2 (4.4%)	2 (3.3%)	2 (4.1%)	6 (3.9%)	0(0%)
<i>Respiratory (asthma included)</i>	0(0%)	1 (1.7%)	0(0%)	1 (.6%)	0(0%)
<i>Autoimmune</i>	1 (2.2%)	1 (1.7%)	5 (10.2%)	7 (4.5%)	4 (2.6%)
<i>Gynaecological</i>	1 (2.2%)	1 (1.7%)	0(0%)	2 (1.3%)	0(0%)
<i>Dermatological</i>	2 (4.4%)	2 (3.3%)	1 (2%)	5 (3.2%)	0(0%)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
<hr/>					
Perceived seriousness and attribution of any health problem (physical or mental) affecting their life					
<i>ED-related</i>	33 (73.3%)	35 (58.3%)	31 (63.3%)	99 (64.3%)	2 (1.3)
<i>Non-ED- related</i>	2 (4.4%)	1 (1.7%)	4 (8.2%)	7 (4.5%)	24 (15.6%)
<i>None</i>	10 (22.2%)	24 (40%)	14 (28.6%)	48 (31.2%)	128 (83.1%)
Mental health pro visit					
<i>Yes</i>	44 (97.8%)	54 (90%)	47 (95.9%)	145 (94.2%)	47 (30.5%)
<i>No</i>	1 (2.2%)	6 (10%)	2 (4.1%)	9 (5.8%)	107 (69.5%)
Psychotherapy (at any point in their life)					
<i>Yes</i>	33 (73.3%)	45 (75%)	40 (81.6%)	119 (77.3%)	18 (11.7%)
<i>No</i>	12 (26.7%)	12 (25%)	9 (18.4%)	35 (22.7%)	136 (88.3%)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
Currently in psychotherapy					
<i>Yes</i>	36 (80%)	38 (63.3%)	36 (73.5%)	111 (72.1%)	9 (5.8%)
<i>No</i>	9 (20%)	22 (36.7%)	13 (26.5%)	43 (27.9%)	145 (94.2%)
Total Duration of reported Psychotherapy (months)	26.44 (41.47)	16.35 (20.21)	44.31 (108.11)	27.68 (5.40)	4.08 (1.74)
Body weight (kg)	47.40 (7.93)	61.76 (12.95)	91.08 (25.82)	66.89 (24.52)	61.20 (9.53)
BMI	17.54 (2.42)	22.16 (4.10)	33.47 (9.53)	24.41 (8.88)	22.19 (3.18)
Body Discrepancy Index	.69 (1.91)	2.20 (1.55)	2.65 (1.25)	1.90 (1.76)	.97 (1.19)

Note: The duration of illness is calculated by subtracting the age of ED onset from the current age. The body discrepancy index indicated body dissatisfaction by subtracting the ideal body shape from the perceived body shape. The negative scores indicate a desire to be bigger, and positive scores indicate a desire to be thinner. A score of zero indicated no body dissatisfaction. In this table, only positive scores are observed. Patients who answered that they have never undergone psychotherapy were on a waiting list to start. The ED subgroups are mentioned for informative reasons, and they were not used in the SEM analyses.

5.3.3. Measures

The discussion of the following measures used in this study can be found in Chapter 4 (4.3.3.): 1) World Health Organisation Brief Quality of Life Assessment Scale (WHOQOL-BREF), 2) Rosenberg Self-esteem scale (RSES), 3) McMaster Family Assessment Device (FAD), 4) Eating Disorder Examination Questionnaire (EDE-Q 6), 5) Sociodemographic and Relevant Clinical Information.

5.3.4. Procedure

As described in 4.3.4. of Chapter 4.

5.3.5. Data Analytic Strategy

One structural equation model for each of the two participants groups was performed to explore the hypothesised mediational models, using AMOS 22 (Arbuckle, 2013). The maximum likelihood chi-square (χ^2) statistic was used to evaluate both the measurement and structural models. This statistic is routinely reported, but even minor deviations from multivariate normality may lead to model rejection, even when the model is properly specified (Hooper et al., 2008). The χ^2 statistic is also sensitive to large samples and lacks the power to discriminate between well- and poor-fitting models (Iacobucci, 2010; Kenny & McCoach, 2003). In light of this, several fit indices were used: the comparative fit index (CFI), the Tucker–Lewis index (TLI) and the root mean square of approximation (RMSEA). These measures of fit are the most frequently recommended (Jackson et al., 2009). RMSEA has the ability to calculate a confidence interval around its value, allowing poor fit to be assessed more precisely (MacCallum et al., 1996; McQuitty, 2004). It is generally considered an informative index due to its sensitivity to the number of estimated parameters in the model, favouring this way parsimony by choosing the model with the least number of

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parameters (Hooper et al., 2008). A good and adequate fit can be indicated by cut-off levels $\leq .06$ and $\leq .08$, respectively (Browne & Cudeck, 1993; Schreiber, 2008). The CFI considers sample size and can perform well even when the sample is small (<100) (Cangur & Ercan, 2015; Kline, 2005; Tabachnick & Fidell, 2007). TLI is also not significantly affected by sample size (Cangur & Ercan, 2015). The CFI and TLI values should be greater than .90 and .95 to indicate an acceptable and excellent fit, respectively (Browne & Cudeck, 1993; Hu & Bentler, 1999).

The hypothesised structural models for patients and controls are shown in Figures 6 and 7, respectively, displaying the path coefficients for the standardised direct and indirect effects. Examination of the standardised residuals and modifications indices revealed no high values, and the standardised residuals were random, without systematic patterns, indicating that both conceptual models are viable. Standardised regression weights were used to interpret the direct effects, and the significance of the indirect effects was based on bias-corrected bootstrap method confidence intervals (Cheung, 2009; MacKinnon et al., 2004). Regarding the bootstrap, 1,000 bootstrap samples were created by random sampling and replacement of the original dataset. If the values of a 95% CI for mean indirect effect do not include/cross a zero value, it is an indication that the indirect effect is significant at a $p < .05$ level. In small sample sizes, BC bootstrap confidence intervals are recommended to generate a minimum of 500 bootstrap samples to avoid a Type I error (Cheung & Lau, 2008). The R mediation program (Tofighi & MacKinnon, 2011) was also used to further test the mediational effects of the significant indirect paths to minimise the possibility that the BC confidence intervals were a Type I error (Fritz et al., 2012). Post-hoc power is reported, as it can be helpful—especially with non-statistically significant results—to demonstrate the study's contribution to the literature, offering evidence that the effect in question does not exist (Derr & Goldsmith, 2003).

5.4. Results

5.4.1. Data Screening and Preliminary Analyses

Missing values and data entry errors. Data were analysed with the use of IBM SPSS Statistics 21.0 (IBM Corporation, 2012). Before proceeding to the statistical analyses, data were screened for missing values, data entry errors, univariate and multivariate outliers and for distribution's normality (Tabachnick & Fidell, 2007). Following correction of data entry errors, the Missing Value Analysis (MVA) revealed that there were no missing items.

Outliers and Normality. Univariate and multivariate outliers were checked and corrected for each statistical analysis. Normality was then explored with respect to the variables entered in each statistical model. Skewness, kurtosis and histograms were preferred rather than global significance tests (e.g. Shapiro-Wilk test) as the latter are considered overly sensitive in large sample sizes (Field, 2009; Hae-Young, 2012). For medium sample sizes (50 to 300) it is recommended to reject the null hypothesis at absolute z value over 3.29 which corresponds at alpha level.05 (Hae-Young, 2013).

Prior to SEM, univariate outliers were checked for all variables separately for both the ED ($n = 154$) and control ($n = 154$) group. There were 13 univariate outliers for the ED group and 46 univariate outliers for the control group. After the univariate outliers were dealt with, a check for multivariate outliers was performed for each group. For the 14 variables in the ED group (first path analysis model), there were no multivariate outliers. For the 14 variables used in the control group (second path analysis model), one multivariate outlier was deleted. The deleted participant was a 27-year-old public servant with 12 years of education, single, with no children, second-born with one sister, living alone, in good health, who had historically undergone three months of psychotherapy, a subnormal BMI and no reported

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body dissatisfaction. The revised sample size was 307 participants (154 ED patients and 153 controls).

Normality was checked for the 14 variables used in the SEM analysis, both for the patient and control groups (see Table 10). There were no z-values over 3.29 for the patient group, thus, no significant deviance from normality. Regarding the control group, all 14 variables, except for the four EDE-Q scales, did not have z-values over 3.29. The four EDE-Q scales measuring eating pathology (EDE-Q restraint, EDE-Q eating concern, EDE-Q shape concern, EDE-Q weight concern) had z-skewness values higher than 3.29, ranging from 3.85 to 5.79, even though their actual skewness and kurtosis scores were not above 2. The histograms and boxplots of these variables revealed a moderate positive skewness, which is expected in a sample of non-ED participants recruited from the general population, as the EDE-Q measures eating pathology, and high scores have been reported in the literature (Hilbert et al., 2012). The proposed transformation for this type of skewness is square root (Tabachnick & Fidell, 2007), but it did not resolve the issue for the eating concern scale, and neither did logarithm transformation. Thus, inverse transformation was employed, adding 1 to the variables due to the presence of zero in their scores $[-1 / (\text{variable} + 1)]$, and the eating concern z-value was improved to fall within the 3.29 limitation (Kim, 2013). Table 10 presents the means and standard deviations of the scales used. A comparison of these means to those of other published studies can be found in Appendix 38.

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Table 10.

Means (standard deviation) of questionnaires' results for ED patients (both by diagnostic group and as a whole) and the control group – Study 1.

	AN PATIENTS n = 45 (14.6%)	BN PATIENTS n = 60 (19.5%)	BED PATIENTS n = 49 (15.9%)	EATING DISORDER PATIENTS n = 154 (50%)	CONTROLS n = 154 (50%)
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Rosenberg's SELF-ESTEEM (range 0-30)	14.6 (4.93)	14.18 (5.77)	16.37 (5.67)	15 (5.55)	20.68 (4.05)
FAD PROBLEM SOLVING (range 1-4)	2.38 (.45)	2.45 (.56)	2.26 (.57)	2.37 (.54)	2.06 (.36)
FAD COMMUNICATION (range 1-4)	2.49 (.61)	2.55 (.58)	2.33 (.62)	2.46 (.61)	2.09 (.37)
FAD ROLES (range 1-4)	2.53 (.43)	2.41 (.38)	2.57 (.37)	2.49 (.40)	2.33 (.38)
FAD AFFECTIVE RESPONSIVENESS (range 1-4)	2.52 (.69)	2.64 (.60)	2.36 (.73)	2.52 (.68)	2.17 (.53)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
FAD AFFECTIVE INVOLVEMENT	2.21	2.35	2.19	2.26	2.03
(range 1-4)	(.55)	(.48)	(.53)	(.52)	(.38)
FAD BEHAVIOUR CONTROL	2.21	2.18	2.25	2.21	2.00
(range 1-4)	(.43)	(.40)	(.41)	(.41)	(.30)
FAD GENERAL FUNCTIONING	2.41	2.52	2.28	2.41	1.92
(range 1-4)	(.65)	(.63)	(.61)	(.64)	(.38)
EDE-Q RESTRAINT	3.17	3.20	2.52	2.98	1.28
(range 0-6)	(1.99)	(1.56)	(1.44)	(1.68)	(1.16)
EDE-Q EATING CONCERN	2.53	2.99	2.36	2.66	.64
(range 0-6)	(1.48)	(1.56)	(1.42)	(1.51)	(.71)
EDE-Q SHAPE CONCERN	3.60	4.20	3.60	3.84	1.61
(range 0-6)	(1.71)	(1.59)	(1.40)	(1.59)	(1.29)
EDE-Q WEIGHT CONCERN	3.25	3.91	3.12	3.46	1.24
(range 0-6)	(1.62)	(1.62)	(1.30)	(1.56)	(1.20)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
EDE-Q GLOBAL SCORE	3.14	3.57	2.89	3.23	1.20
(range 0-6)	(1.53)	(1.35)	(1.07)	(1.35)	(.95)
BREF PSYCHOLOGICAL QoL	11.20	10.88	11.73	11.24	14.58
(range 4-20)	(3.06)	(3.21)	(2.88)	(3.06)	(2.00)
BREF SOCIAL RELATIONSHIPS QoL	11.91	11.64	11.78	11.76	14.92
(range 4-20)	(3.97)	(3.56)	(3.80)	(3.74)	(2.90)

Note: These means are derived from the scales with corrected univariate outliers prior to any transformation. The diagnostic groups' M(SD) are reported for informative reasons and were not used in the analysis.

5.4.2. The impact of family functioning on ED and QoL: The mediational effects of self-esteem

Measurement Models

In the structural model the latent variable of family functioning is the exogenous variable, the observed variable of self-esteem is the mediator, and the two latent variables of psychosocial QoL and eating pathology are the endogenous variables. Prior to analysis of the structural models both for patients and controls, the measurement models were assessed in order to evaluate the appropriateness of the latent variables i.e., the relations between the measured variables and their proposed latent constructs (Kline, 2011). Both models (patients and controls) have three latent variables (family functioning, eating pathology, psychosocial

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QoL) and one observed variable, self-esteem. The latent variable of family functioning has seven indicators (Problem Solving, Communication, Roles, Affective Responsiveness, Affective Involvement, Behaviour Control, General Functioning), psychosocial QoL has two indicators (Psychological Health, Social Relationships), and eating pathology four indicators (Restraint, Eating Concern, Shape Concern, Weight Concern) The examination of the factor loadings in both groups' measurement models showed that the indicators loaded significantly in the predicted directions of their respective latent factors as described below.

ED Patients: the model fit the data well: $\chi^2(62, N = 154) = 107.823, p < .001$, CFI = .97, TLI = .96, RMSEA = .07. All standardized factor loadings between the measured variables and their respective latent variables were significant ($p < .001$) and ranged from .48 to .97. The correlations between the latent variables were as follows: family functioning and psychosocial QoL was $r = -.44, p < .001$; psychosocial QoL and eating pathology was $r = -.60, p < .001$; family functioning and eating pathology was $r = .23, p = .012$.

Controls: the model was an adequate fit to the data: $\chi^2(62, N = 153) = 120.532, p < .001$, CFI = .95, TLI = .94, RMSEA = .08. All standardized factor loadings between the measured variables and their respective latent variables were significant ($p < .001$) and ranged from .52 to .98. The correlation between family functioning and psychosocial QoL was $r = -.61, p < .001$; psychosocial QoL and eating pathology was $r = -.42, p < .001$ and the correlation between family functioning and eating pathology was not significant ($p > .05$).

Structural Model Analyses

The structural model examines the relationships between the theoretically proposed latent constructs. Prior to the SEM analysis examination of the two models, relationships between all variables were calculated using Pearson Product moment coefficients (two tailed), as shown in Tables 11 (patients) and 12 (controls) respectively. All significant

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correlations regarding patients were in the expected direction. That is, increased family dysfunction was related to greater ED and lower QoL, and low self-esteem was related to greater family dysfunction, greater ED and lower QoL. Regarding controls, prior to transformation, all significant correlations were in the expected direction. That is, increased family dysfunction was related to greater ED and lower QoL, and low self-esteem was related to greater family dysfunction, greater ED and lower QoL. However, after transformation this changed for: restraint with social relationships, eating concern with both QoL scales. The decision regarding the direct and indirect paths was driven by sample limitations along with the number of variables used and background literature. Thus, emphasis was given to the issue of statistical power and model fit preserving the simplicity of the models to answer the primary mediation hypotheses. When the model fit allowed it, all possible relationships were explored.

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Table 11.

Bivariate correlations (two-tailed), means and standard deviations for observed variables with the structural equation model for the patient group (n = 154) – Study 1.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Problem solving	-													
2. Communication	.76 ^{***}	-												
3. Roles	.53 ^{***}	.56 ^{***}	-											
4. Affective Responsiveness	.61 ^{***}	.79 ^{***}	.44 ^{***}	-										
5. Affective Involvement	.59 ^{***}	.72 ^{***}	.56 ^{***}	.64 ^{***}	-									
6. Behaviour Control	.51 ^{***}	.41 ^{***}	.48 ^{***}	.29 ^{***}	.43 ^{***}	-								
7. General Functioning	.80 ^{***}	.89 ^{***}	.59 ^{***}	.79 ^{***}	.77 ^{***}	.46 ^{***}	-							
8. Self-esteem	-.31 ^{***}	-.29 ^{***}	-.19 [*]	-.33 ^{***}	-.23 ^{**}	-.29 ^{***}	-.31 ^{***}	-						
9. Psychological Health	-.38 ^{***}	-.36 ^{***}	-.23 ^{**}	-.35 ^{***}	-.30 ^{***}	-.30 ^{***}	-.41 ^{***}	.74 ^{***}	-					
10. Social Relationships	-.22 ^{**}	-.23 ^{**}	-.25 ^{**}	-.23 ^{**}	-.25 ^{**}	-.26 ^{**}	-.30 ^{***}	.51 ^{***}	.65 ^{***}	-				
11. Restraint	.13	.10	.07	.12	.16	.00	.18 [*]	-.25 ^{**}	-.26 ^{**}	-.16	-			
12. Eating Concern	.22 ^{**}	.23 ^{**}	.14	.21 ^{**}	.23 ^{**}	.12	.25 ^{**}	-.43 ^{***}	-.54 ^{***}	-.40 ^{***}	.53 ^{***}	-		
13. Shape Concern	.21 ^{**}	.16 [*]	.13	.12	.20 [*]	.09	.24 ^{**}	-.48 ^{***}	-.54 ^{***}	-.40 ^{***}	.52 ^{***}	.72 ^{***}	-	
14. Weight Concern	.18 [*]	.13	.05	.08	.17 [*]	.02	.20 [*]	-.47 ^{***}	-.51 ^{***}	-.38 ^{***}	.53 ^{***}	.69 ^{***}	.90 ^{***}	-
Mean	2.37	2.46	2.49	2.52	2.26	2.21	2.41	15.00	11.24	11.76	2.98	2.66	3.84	3.46
SD	.54	.61	.40	.68	.52	.41	.64	5.55	3.06	3.74	1.68	1.51	1.59	1.56
Range	1-4	1-4	1-4	1-4	1-4	1-4	1-4	0-30	4-20	4-20	0-6	0-6	0-6	0-6

* $p < .05$, ** $p < .01$, *** $p < .001$

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Table 12.

Bivariate correlations (two-tailed), means and standard deviations for observed variables with the structural equation model for the control group (n = 153) – Study 1.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Problem Solving	-													
2. Communication	.59 ^{***}	-												
3. Roles	.48 ^{***}	.47 ^{***}	-											
4. Affective Responsiveness	.44 ^{***}	.70 ^{***}	.41 ^{***}	-										
5. Affective Involvement	.40 ^{***}	.56 ^{***}	.42 ^{***}	.40 ^{***}	-									
6. Behaviour Control	.33 ^{***}	.46 ^{***}	.37 ^{***}	.38 ^{***}	.36 ^{***}	-								
7. General Functioning	.66 ^{***}	.80 ^{***}	.56 ^{***}	.72 ^{***}	.63 ^{***}	.48 ^{***}	-							
8. Self-esteem	-.27 ^{**}	-.35 ^{***}	-.28 ^{***}	-.28 ^{***}	-.35 ^{***}	-.29 ^{***}	-.41 ^{***}	-						
9. Psychological Health	-.36 ^{***}	-.39 ^{***}	-.42 ^{***}	-.30 ^{***}	-.34 ^{***}	-.30 ^{***}	-.46 ^{***}	.63 ^{***}	-					
10. Social Relationships	-.26 ^{**}	-.39 ^{***}	-.34 ^{***}	-.39 ^{***}	-.32 ^{***}	-.29 ^{***}	-.49 ^{***}	.41 ^{***}	.59 ^{***}	-				
11. Restraint	-.02	.04	.09	.14	.14	.01	.03	-.11	-.08	.09	-			
12. Eating Concern	-.00	-.10	-.11	-.13	-.22 ^{**}	-.06	-.16 [*]	-.36 ^{***}	.34 ^{***}	.08	-.45 ^{***}	-		
13. Shape Concern	-.07	.06	.13	.14	.16 [*]	.03	.11	-.35 ^{***}	-.41 ^{***}	-.19 [*]	.55 ^{***}	-.80 ^{***}	-	
14. Weight Concern	-.13	-.01	.13	.11	.18 [*]	-.02	.06	-.31 ^{***}	-.35 ^{***}	-.15	.59 ^{***}	-.74 ^{***}	.91 ^{***}	-
Mean	2.05	2.08	2.34	2.16	2.03	2.00	1.92	20.73	14.61	14.95	1.28	.64	1.60	1.24
SD	.36	.37	.37	.53	.38	.29	.38	4.03	1.98	2.89	1.16	.71	1.28	1.20
Range	1-4	1-4	1-4	1-4	1-4	1-4	1-4	0-30	4-20	4-20	0-6	0-6	0-6	0-6

* $p < .05$, ** $p < .01$, *** $p < .001$

Note: Means (SD) reported for the four eating pathology variables are prior to transformation but their correlation values are reported post-inverse transformation as they were entered transformed in the SEM analysis.

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ED Patients: Model fit indices indicated that the hypothesised model (Figure 6) fit the data well: $\chi^2(72, N = 154) = 121.257, p < .001, CFI = .97, TLI = .96, RMSEA = .07$. The direct path from family functioning to self-esteem was significant ($\beta = -.33, p < .001$), meaning that when family functioning increased by 1 standard deviation then self-esteem decreased by .33 standard deviations. The direct paths from self-esteem to eating pathology ($\beta = -.48, p < .001$), and from self-esteem to psychosocial QoL ($\beta = .58, p < .001$) were also significant, meaning that when self-esteem increased by 1 standard deviation then psychosocial QoL increased by .58 standard deviations. The other significant direct paths were from family functioning to psychosocial QoL ($\beta = -.18, p = .001$) and from eating pathology to psychosocial QoL ($\beta = -.25, p < .001$). All the direct paths have a post hoc power 1. The two latter direct paths were not part of the mediation hypothesis but all possible paths were examined in the model. It is interesting to note that there was no significant direct effect from family functioning to eating pathology and the post hoc power of this path is 1. In summary, the direct paths in the clinical sample revealed that: i) high levels of family dysfunction leads to low self-esteem and poor psychosocial QoL, ii) that low levels of self-esteem lead to higher levels of ED and poor QoL and iii) high eating pathology results in poor QoL.

The indirect path from family functioning to eating pathology with self-esteem as a mediator was significant (standardized indirect effect = .16, bias-corrected bootstrap lower CI = .068 and upper CI = .253, $p = .002$). In addition, the indirect effect of family functioning to psychosocial QoL with self-esteem as a mediator was significantly different from zero (standardized indirect effect = -.25, bias-corrected bootstrap lower CI = -.360 and upper CI = -.129, $p = .002$). The RMediation program was used (Tofighi & MacKinnon, 2011) to further test the mediational effects using the distribution-of-product method. The unstandardised coefficients and standard errors of the a and b paths between family functioning and self-

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esteem and between self-esteem and eating pathology were entered into the program and yielded lower and upper 95% confidence limits of .155 and .575 providing a significant indirect effect. Next, the unstandardized coefficients and standard errors of the a and b paths between family functioning and self-esteem and between self-esteem and psychosocial QoL were entered into RMediation resulting in lower and upper confidence limits of -2.006 and -0.658, thus displaying a significant indirect effect. The results are consistent with those of a partial mediation effect given a direct path from family functioning to self-esteem in the presence of significant indirect effects both for eating pathology and psychosocial QoL. This means that self-esteem partially mediated the relationship between family dysfunction and ED/psychosocial QoL.

Controls: Model fit indices indicated that the hypothesised model (Figure 7) fit the data adequately: $\chi^2(72, N = 153) = 128.054, p < .001, CFI = .96, TLI = .95, RMSEA = .07$. The direct path from family functioning to self-esteem was significant ($\beta = -.43, p < .001$) and so were the direct paths from self-esteem to eating pathology ($\beta = .38, p < .001$) and from self-esteem to psychosocial QoL ($\beta = .46, p < .001$). The other direct paths that were significant were from eating pathology to psychosocial QoL ($\beta = .22, p = .003$) and from family functioning to psychosocial QoL ($\beta = -.35, p < .001$). As with the patients model, there was no direct effect of family functioning on eating pathology even though the post hoc power was 1 in all direct paths. Summarising, the direct paths revealed that high levels of family dysfunction leads to low self-esteem and poor psychosocial QoL, and low self-esteem leads to poor QoL. However, high levels of ED seem here to lead to higher levels of QoL and high self-esteem to high eating pathology, something inconsistent not only to literature but to psychological mechanisms. It could be explained as a suppressor variable effect for the self-esteem - ED path, a common problem reported in literature with multiple regression and latent variables of SEM models (Maassen & Bakker, 2001), like the ED variable in this case.

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The answer to ED - QoL issue may be the transformation of ED scales for controls. The correlations of ED scales, prior to transformation, with QoL were all negative as they should be. Post transformation correlation of these variables led to mixed results with QoL (some negative and some positive within the same scales).

The indirect path from family functioning to eating pathology with self-esteem as a mediator was significant (standardized indirect effect = $-.17$, bias-corrected bootstrap lower CI = $-.267$ and upper CI = $-.082$, $p = .001$). The indirect path from family functioning to psychosocial QoL with self-esteem as a mediator was also significant (standardized indirect effect = $-.22$, bias-corrected bootstrap lower CI = $-.330$ and upper CI = $-.133$, $p = .001$). The unstandardized coefficients and standard errors of the a and b paths between family functioning and self-esteem and between self-esteem and eating pathology were entered into RMediation and yielded lower and upper 95% confidence limits of -0.651 and -0.178 providing a significant indirect effect. Next, the unstandardized coefficients and standard errors of the a and b paths between family functioning and self-esteem and between self-esteem and psychosocial QoL were entered resulting in lower and upper confidence limits of -2.455 and -0.785 , producing a significant indirect effect. The results are consistent with those of a partial mediation effect given a direct path from family functioning to self-esteem in the presence of significant indirect effects both for eating pathology and psychosocial QoL. In other words, self-esteem partially mediated the relationship between family dysfunction and ED/psychosocial QoL.

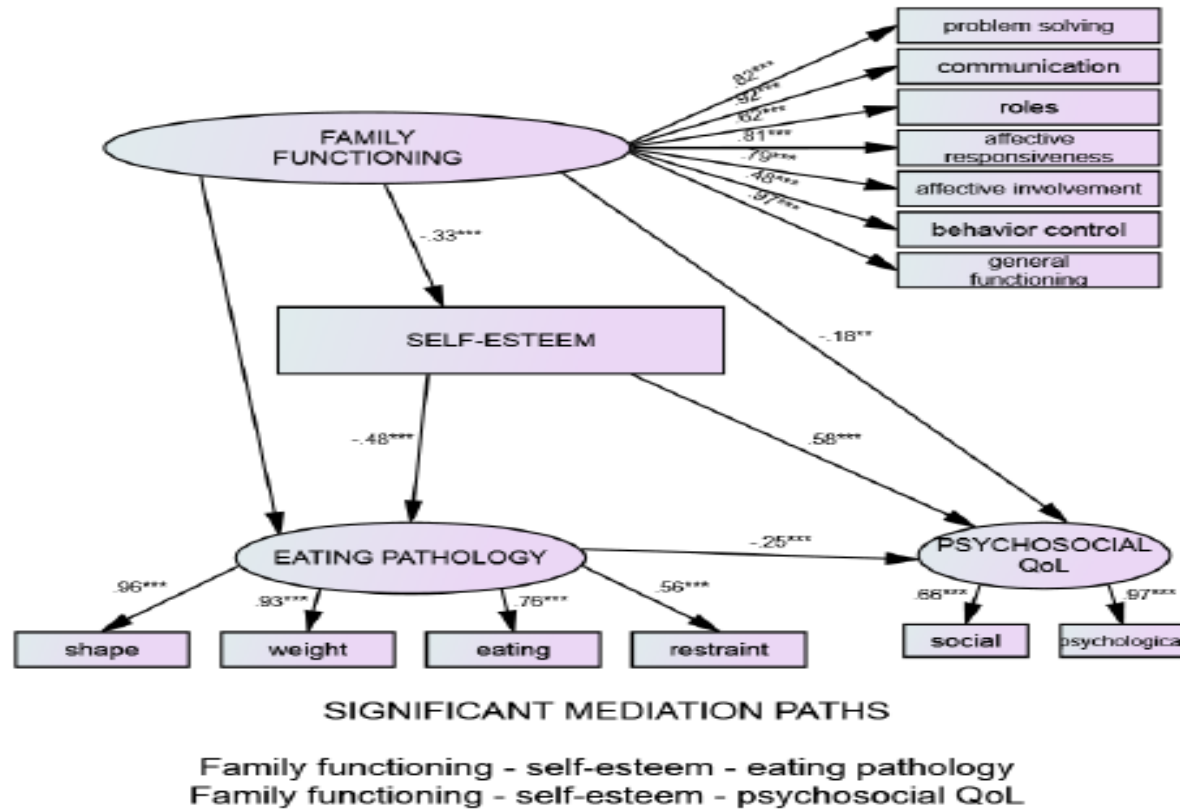
Comparing the beta values between ED patients and controls it is evident that greater family dysfunction had a stronger effect on lowering self-esteem [$(\beta = -.43, p < .001)$ vs $(\beta = -.33, p < .001)$] and QoL [$(\beta = -.35, p < .001)$ vs $(\beta = -.18, p = .001)$] for controls, and low self-

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esteem had a stronger effect on lowering QoL for patients [$(\beta = .58, p < .001)$ vs $(\beta = .46, p < .001)$].

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Figure 6. Structural equation model testing the mediational effect of self-esteem on the relationships between family functioning and eating pathology, and family functioning and psychosocial QoL in ED patients.

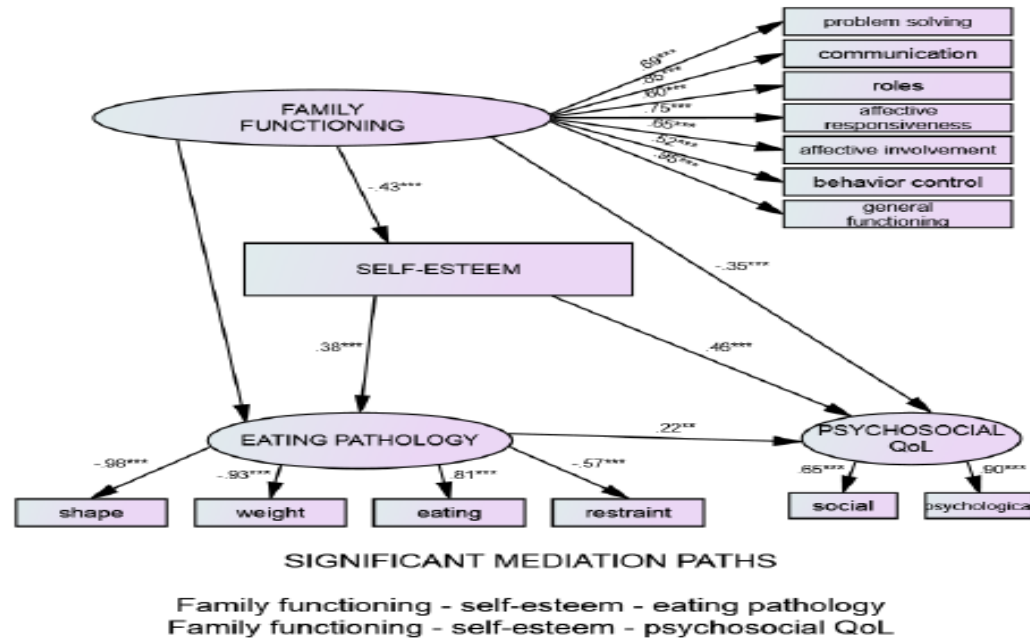


* $p < .05$, ** $p < .01$, *** $p < .001$

Note: Standardised paths shown are significant.

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Figure 7. Structural equation model testing the mediational effect of self-esteem on the relationships between family functioning and eating pathology, and family functioning and psychosocial QoL in the control group.



* $p < .05$, ** $p < .01$, *** $p < .001$

Note: Standardised paths shown are significant.

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Further exploratory analyses: Further exploratory analyses were conducted to check for differences in the mean scores of the variables used in this study both between the ED subtypes and between the ED patients and controls. This is important in order to identify how distinct the groups are and whether any similarities in the SEM models might be due to overlap in the control and ED groups. Moreover, the ED subtypes were not analysed in this thesis's studies, adopting a transdiagnostic perspective regarding the factors related to onset and maintenance of eating pathology. However, it is important to check for differences in the study variables among the ED subtypes in order to clarify if a specific diagnostic type was responsible for the SEM results. In addition to this, and based on the study's demographic information, illness and psychotherapy duration were also considered among the ED subtypes to see if the results were affected both by chronicity and treatment resistance, since this is a chronic, treatment-resistant group, as shown in the demographics (see Table 9). Prior to the analyses, the data for each group were checked for normality, outliers and the assumptions of the tests. For economy of space, the results are presented in tables. The alpha level for all the analyses was set at .05.

ED subtypes: One-way analysis of variance (ANOVA) was used to determine the differences between the ED subtypes for self-esteem, family functioning and psychosocial QoL. The assumptions for this analysis were met (continuous dependent variable, independence of observations in the three categorical groups, no outliers, dependent variables approximately normally distributed, homogeneity of variance). Omega squared (ω^2) was used to calculate the effect sizes of one-way ANOVA, as it is considered to be a more accurate estimate of the true population value of strength of association, and it is less biased than eta squared, which is a descriptive statistic of the sample (Albers & Lakens, 2018; Hinkle et al., 1998; Olejnik & Algina, 2003). Omega squared indicates the proportion of the variance in the dependent variable that is accounted for by the levels of the independent variable (Hinkle et

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al, 1998), in this case the ED subgroups. Because it is an unbiased estimate of population variances, it is always computed as lower than the value produced for eta squared (Meyers et al., 2006).

Table 13.

Means, standard deviations and one-way analyses of variance (ANOVA) in self-esteem, family functioning and psychosocial QoL among the ED Subtypes – Study 1.

Measure	AN		BN		BED		<i>F</i> (2,151)	<i>p</i>	ω^2
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>			
Self-esteem	14.60	4.93	14.18	5.77	16.37	5.67	2.29	.105	.02
General family functioning	2.41	.65	2.52	.63	2.28	.61	1.97	.144	.01
Problem solving	2.38	.45	2.45	.56	2.26	.57	1.63	.199	.01
Communication	2.49	.61	2.55	.58	2.33	.62	1.90	.154	.01
Roles	2.53	.43	2.41	.38	2.57	.37	2.25	.108	.02
Affective responsiveness	2.52	.69	2.64	.60	2.36	.73	2.43	.092	.02
Affective involvement	2.21	.55	2.35	.48	2.19	.53	1.54	.217	.01
Behaviour control	2.21	.43	2.18	.40	2.25	.40	.44	.648	.01
Psychological health (QoL)	11.20	3.06	10.88	3.21	11.73	2.88	1.05	.354	.00
Social relationships (QoL)	11.91	3.97	11.64	3.56	11.78	3.80	.07	.934	.01

*** $p < .001$; ** $p < .01$; * $p < .05$

Note: AN: $n = 45$, BN: $n = 60$, BED: $n = 49$

As shown in Table 13, there were no significant differences among the ED subtypes regarding self-esteem, family functioning and psychosocial QoL, suggesting that it was

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unlikely that the mean score of a specific ED subtype affected the direct and indirect effects in the SEM model for the ED patient group.

ED patients and controls: One-way ANOVA was used to determine the differences between the ED patients and the controls with respect to their mean scores for self-esteem, family functioning and psychosocial QoL. The assumptions for this analysis were met in all cases except for homogeneity of variance, as Levene's test was statistically significant for all of the dependent variables. When the equal variance assumption has been violated, the Type I error rate can be affected, and the Welch test is recommended as the most powerful (Field, 2009; Gastwirth et al., 2009). Moreover, the adjusted F statistic was preferred over the non-parametric *Kruskal–Wallis* test, both because it is considered more robust (Chen et al., 2005) and because there was only one assumption violation (there was no violation on both normality and homogeneity of variance). Furthermore, the larger group variance was not 4 or 5 times larger than the smaller group variance, which would have been a bigger problem meaning one-way ANOVA could not have been used (Field, 2009). Adjusted omega squared formula (est. ω^2) was calculated except for the only scale that met the homogeneity of variance assumption (roles of family functioning). For this scale, the standard F statistic is reported.

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Table 14.

Means, standard deviations and one-way analyses of variance (ANOVA) in self-esteem, family functioning and psychosocial QoL between ED patients and controls – Study 1.

Measure	ED patients		Controls		Welch <i>F</i> (df)	<i>F</i> (1,305)	<i>p</i>	est. ω^2	ω^2
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>					
Self-esteem	15.00	5.55	20.73	4.03	107.14*** (1, 279.198)		.000	.26	
General family functioning	2.42	.64	1.92	.38	69.68*** (1, 249.544)		.000	.18	
Problem solving	2.37	.54	2.05	.36	37.82*** (1, 265.987)		.000	.11	
Communication	2.46	.61	2.08	.37	44.53*** (1, 252.361)		.000	.12	
Roles	2.49	.40	2.34	.37		12.93***	.000		.04
Affective responsiveness	2.52	.68	2.16	.53	26.37*** (1, 288.047)		.000	.08	
Affective involvement	2.26	.52	2.03	.38	19.85*** (1, 281.480)		.000	.06	
Behaviour control	2.21	.41	2.00	.29	25.85*** (1, 278.199)		.000	.07	
Psychological health (QoL)	11.24	3.06	14.61	1.98	131.17*** (1, 261.805)		.000	.30	
Social relationships (QoL)	11.76	3.74	14.95	2.89	69.57*** (1, 287.715)		.000	.18	

*** $p < .001$; ** $p < .01$; * $p < .05$

Note: ED patients: $n = 154$, controls: $n = 153$

The results indicate a statistically significant difference in the mean scores of all the dependent variables between ED patients and controls. In other words, having an ED diagnosis resulted in lower self-esteem, higher family dysfunction and lower psychosocial QoL compared to healthy controls. For example, there was a statistically significant difference in self-esteem: $F(1, 279.198) = 107.14, p < .001, \omega^2 = .26$. Approximately 26% of the total variance of self-esteem is due to the groups (having an ED diagnosis resulted in

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lower self-esteem compared to healthy controls). Along with the SEM results, these findings suggest that the ED and control group differ in levels of severity across the measured constructs, but differ little in terms of the nature of theoretical model that can explain relationships between these constructs.

ED subtypes, controlling for illness duration: One-way analysis of covariance (ANCOVA) between subjects was conducted to determine statistically significant differences between AN, BN and BED patients regarding self-esteem, family functioning and psychosocial QoL (psychological health and social relationships), controlling for the effect of illness duration. The assumptions were met, and the conservative Bonferroni correction was used, as recommended (Field, 2009).

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Table 15.

Observed and Adjusted Means and Standard Deviations of Self-Esteem, Family Functioning and Psychosocial QoL among the ED Subtypes after Controlling for Duration of Illness – Study 1.

Measure	AN		BN		BED		AN		BN		BED	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Self-esteem	14.60	4.93	14.18	5.77	16.37	5.67	14.87	.88	14.29	.72	15.98	.90
General family functioning	2.41	.65	2.52	.63	2.28	.61	2.42	.10	2.53	.08	2.27	.10
Problem solving	2.38	.45	2.45	.56	2.26	.54	2.38	.09	2.45	.07	2.26	.09
Communication	2.49	.61	2.55	.58	2.33	.62	2.50	.10	2.56	.08	2.31	.10
Roles	2.52	.43	2.41	.38	2.57	.37	2.54	.06	2.42	.05	2.54	.06
Affective responsiveness	2.52	.69	2.64	.60	2.36	.73	2.51	.11	2.64	.09	2.37	.11
Affective involvement	2.21	.55	2.35	.48	2.19	.53	2.24	.08	2.36	.07	2.16	.09
Behaviour control	2.21	.43	2.18	.40	2.25	.40	2.21	.07	2.18	.05	2.25	.07
Psychological health (QoL)	11.20	3.06	10.88	3.21	11.73	2.88	11.40	.49	10.96	.40	11.44	.50
Social relationships (QoL)	11.91	3.97	11.64	3.56	11.78	3.80	12.02	.60	11.68	.49	11.63	.62

Note: AN: n = 45, BN: n = 60, BED: n = 49; illness duration considered in the ANCOVA was 13.65 years.

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Table 16.

ANCOVA results after controlling for duration of illness – Study 1.

Measure	<i>F</i> (2,150)	<i>p</i>	<i>partial η</i> ²
Self-esteem	1.00	.371	.01
General family functioning	1.72	.182	.02
Problem solving	1.26	.288	.02
Communication	1.83	.164	.02
Roles	1.73	.181	.02
Affective responsiveness	1.69	.189	.02
Affective involvement	1.91	.152	.03
Behaviour control	.32	.727	.00
Psychological health (QoL)	.40	.669	.01
Social relationships (QoL)	.12	.885	.00

*** *p* <.001; ** *p* <.01; * *p* <.05

The covariate, illness duration, was not significantly related to self-esteem $F(1,150) = .77, p = .382, partial \eta^2 = .00$, as it was not significantly related to any other dependent variable. There was no significant difference between the ED subtypes regarding self-esteem $F(2,150) = 1.00, p = .371, partial \eta^2 = .01$ nor for any of the other dependent variables, after controlling for illness duration.

High and low chronicity: One-way ANOVA between subjects was conducted to determine whether there is a statistically significant difference between the high and low

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chronicity groups regarding self-esteem, family functioning and psychosocial QoL (psychological health and social relationships). Duration of illness was conceptualised as high and low chronicity in the patient group using the median. This further analysis was conducted in order to overcome the ANCOVA limitations when using it for non-experimental research (Miller & Chapman, 2001). The assumptions for the analysis were met.

Table 17.

Means, Standard Deviations and One-Way ANOVA in Self-Esteem, Family Functioning and Psychosocial QoL between ED patients with high and low chronicity – Study 1.

Measure	High chronicity		Low chronicity		<i>F</i> (1,152)	<i>p</i>	ω^2
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>			
Self-esteem	15.81	5.70	14.19	5.31	3.29	.072	.01
General family functioning	2.38	.62	2.45	.66	.55	.462	.00
Problem solving	2.34	.59	2.39	.49	.33	.568	.00
Communication	2.45	.61	2.47	.61	.03	.860	.01
Roles	2.49	.37	2.50	.42	.01	.926	.01
Affective responsiveness	2.48	.72	2.56	.63	.54	.465	.00
Affective involvement	2.29	.50	2.23	.63	.60	.440	.00
Behaviour control	2.22	.41	2.20	.41	.15	.695	.00
Psychological health (QoL)	11.54	3.12	10.94	2.99	1.47	.227	.00
Social relationships (QoL)	12.03	3.64	11.49	3.84	.81	.371	.00

*** $p < .001$; ** $p < .01$; * $p < .05$

Note: high chronicity: $n = 77$, low chronicity: $n = 77$; total $n = 154$ ED patients

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The results indicate that there is no significant difference in self-esteem depending on whether the patients have high or low eating pathology chronicity $F(1,152) = 3.29, p = .072, \omega^2 = .01$, and there is no significant difference in family functioning and psychosocial QoL. This further indicates that illness chronicity is not likely to have played a role in the outcome of the structural models in the clinical sample.

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ED subtypes, controlling for psychotherapy duration: One-way between subjects ANCOVA was conducted to determine a statistically significant difference between AN, BN and BED on self-esteem, family functioning and psychosocial QoL (psychological health and social relationships) controlling for the effect of psychotherapy duration. The assumptions for the analysis were met.

Table 18.

Observed and Adjusted Means and Standard Deviations for Self-Esteem, Family Functioning and Psychosocial QoL among the ED Subtypes after Controlling for Duration of Psychotherapy – Study 1.

Measure	AN		BN		BED		AN		BN		BED	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Self-esteem	14.60	4.93	14.18	5.77	16.37	5.67	14.59	.82	14.06	.71	16.51	.79
General family functioning	2.41	.65	2.52	.63	2.28	.61	2.41	.09	2.53	.08	2.27	.09
Problem solving	2.38	.45	2.45	.56	2.26	.57	2.38	.08	2.46	.07	2.25	.08
Communication	2.49	.61	2.55	.58	2.33	.62	2.49	.09	2.56	.08	2.32	.09
Roles	2.52	.43	2.41	.38	2.57	.37	2.53	.06	2.41	.05	2.57	.06
Affective responsiveness	2.52	.69	2.64	.60	2.36	.73	2.52	.10	2.65	.09	2.35	.10
Affective involvement	2.21	.55	2.35	.48	2.19	.53	2.21	.08	2.35	.07	2.19	.08
Behaviour control	2.21	.43	2.18	.40	2.25	.40	2.21	.06	2.18	.05	2.25	.06
Psychological health (QoL)	11.20	3.06	10.88	3.21	11.73	2.88	11.19	.46	10.83	.40	11.79	.44
Social relationships (QoL)	11.91	3.97	11.64	3.56	11.78	3.80	11.91	.56	11.66	.49	11.75	.54

Note: AN: n = 45, BN: n = 60, BED: n = 49; psychotherapy duration considered in the ANCOVA was 27.68 months.

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Table 19.

ANCOVA results after controlling for duration of psychotherapy – Study 1.

Measure	<i>F</i> (2,150)	<i>p</i>	<i>Partial η</i> ²
Self-esteem	2.76	.067	.04
General family functioning	2.26	.108	.03
Problem solving	2.13	.123	.03
Communication	2.21	.113	.03
Roles	2.27	.107	.03
Affective responsiveness	2.54	.083	.03
Affective involvement	1.59	.208	.02
Behaviour control	.37	.695	.01
Psychological health (QoL)	1.29	.280	.02
Social relationships (QoL)	.06	.944	.00

*** *p* <.001; ** *p* <.01; * *p* <.05

Psychotherapy duration was not significantly related to self-esteem ($F[1,150] = 1.88$, $p = .172$ *partial η*² = .01), as it was not significantly related to any other dependent variable. There was no significant difference between the ED subtypes for self-esteem ($F[2,150] = 2.76$, $p = .067$, *partial η*² = .04), nor for any of the other dependent variables, after controlling for psychotherapy duration.

Short-term and long-term psychotherapy: One-way ANOVA between subjects was conducted to determine whether duration of psychotherapy resulted in a statistical difference for self-esteem, family functioning and psychosocial QoL. The categorical variable was

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created based on the literature (Knekt et al., 2016) defining long-term as over six months and short-term as less than six months. This further analysis was undertaken to overcome the ANCOVA limitations (Miller & Chapman, 2001). The assumptions for the analysis were met.

Table 20.

Means, Standard Deviations and One-Way ANOVA in Self-Esteem, Family Functioning and Psychosocial QoL between short and long-term psychotherapy groups of ED patients – Study 1.

Measure	Long-term therapy		Short-term therapy		<i>F</i> (1,152)	<i>p</i>	ω^2
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>			
Self-esteem	14.15	5.40	16.48	5.55	6.50*	.012	.03
General family functioning	2.47	.65	2.33	.61	1.71	.192	.00
Problem solving	2.41	.54	2.30	.53	1.31	.255	.00
Communication	2.52	.63	2.36	.56	2.44	.120	.01
Roles	2.51	.41	2.47	.38	.44	.507	.00
Affective responsiveness	2.55	.67	2.45	.69	.86	.356	.00
Affective involvement	2.28	.56	2.22	.45	.39	.536	.00
Behaviour control	2.23	.41	2.18	.41	.54	.464	.00
Psychological health (QoL)	10.87	3.10	11.89	2.91	4.05*	.046	.02
Social relationships (QoL)	11.71	3.69	11.85	3.85	.05	.828	.01

*** $p < .001$; ** $p < .01$; * $p < .05$

Note: long-term therapy: $n = 98$, short-term therapy: $n = 56$; total $n = 154$ ED patients.

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There was a significant difference between the two groups regarding self-esteem ($F[1,152] = 6.50, p = .012, \omega^2 = .03$) and psychological health QoL ($F[1,152] = 4.04, p = .046, \omega^2 = .01$), indicating that patients in short-term psychotherapy report better self-esteem and psychological health QoL compared to patients in long-term psychotherapy. There was no significant difference between the two groups regarding family functioning and social relationship QoL. In sum, the results indicate some association between self-esteem and psychotherapy duration, and between psychological health QoL and psychotherapy duration.

5.5. Discussion

Even though an empirically established association between family dysfunction and eating pathology exists, the specific pathways of the mechanisms involved have not been adequately established (Friedman et al., 1997). The results of this study provide evidence to suggest that self-esteem is an important mediator between family dysfunction and eating pathology, as well as between family dysfunction and psychosocial QoL, in both ED patients and the general population. These results extend the limited studies in the field (Kroplewski et al., 2019) by testing an original model that includes these key theoretically pertinent factors. Therefore, this study provides insight into the possible mechanism that explains the link between the family and EDs.

Both low self-esteem and family dysfunction are considered risk factors for ED development, and they are reported characteristics of people diagnosed with EDs (Adamson et al., 2019; Cerniglia et al., 2017). Self-esteem appears to play a more direct role in EDs than family dysfunction, supporting theories of self-esteem's unique contribution to ED development (Amianto et al., 2016; Silverstone, 1992). Self-esteem's importance is unchanged regardless of the ED subtype: in this study, there was no difference in the self-esteem levels of AN, BN or BED patients, even when illness duration was considered. This finding also indicates that self-esteem is affected by family functioning and might, therefore,

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become a risk factor for poor psychosocial QoL, as well as ED development, further highlighting the potential lifelong significance of self-esteem. The fact that self-esteem had a direct effect on EDs in both populations also supports this argument. Yet, longitudinal studies will need to confirm causality.

Family functioning had a direct effect on self-esteem and psychosocial QoL, and an indirect effect on eating pathology and psychosocial QoL via self-esteem, both in ED patients and controls. Theoretically, this suggests that trait self-esteem is an important factor, sensitive to family dysfunction regardless of psychopathology. This result was strengthened by the group comparison tests, which resulted in significant differences between ED patients and controls regarding self-esteem, psychosocial QoL and family dysfunction, indicating that the similarity in the theoretical models' outcomes were not the result of similar mean scores in the questionnaires. It also indicates that these psychological constructs are important at different levels of severity, but fundamentally they operate in the same manner when it comes to theoretical pathways/relationships. These findings also validate other studies in which family dysfunction has been associated with low self-esteem (Ha et al., 2006; Lau & Kwok, 2000; Rezaei-Dehaghani et al., 2015; Smets & Hartup, 1988) and low psychosocial QoL (Ciao et al., 2015). Additionally, this study confirms that the effect of the familial environment is enduring and observed in adulthood (Orth, 2018). Family functioning did not have a direct effect on eating pathology in either population, supporting Le Grange and colleagues' position (2010) that family cannot be considered the sole or primary risk factor for ED development. Yet, family functioning did have a small direct effect on both populations' psychosocial QoL, suggesting that family dysfunction has an important effect on psychological health and social relationships in adult life, independent of a clinical diagnosis.

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The direct effect of EDs on psychosocial QoL is in accordance with the relevant literature (Ura & Preston, 2015). These findings, along with other results that indicate psychosocial functioning impairment in EDs (Bentley et al., 2015), highlight the overlooked psychosocial aspects of QoL. Given that QoL is not improved even after ED remission (Bamford and Sly, 2010; de la Rie et al., 2005; Padierna et al., 2002; Pohjolaine et al., 2016), it can be hypothesised that factors besides ED symptoms are more responsible for poor QoL, and these factors should be addressed in treatment. One such factor could be self-esteem, as the results indicate that it had a stronger direct effect on QoL than eating pathology and family dysfunction in both groups. The ED subgroups did not differ in their perceived QoL, in line with other studies (de la Rie et al., 2005; González et al., 2001). Furthermore, as other studies found, the duration of eating pathology did not affect patients' psychosocial QoL (Bamford & Sly, 2010). The duration of psychotherapy as a covariate also did not affect QoL, in line with the empirical finding that ED patients report poor QoL even while they are undergoing psychotherapy (Mond et al., 2005). However, there was a significant difference in psychological health QoL when the patients were divided into long- and short-term therapy groups: the short-term group reported better psychological QoL. This result could possibly be explained through the result of self-esteem, which was significantly lower in the long-term therapy group, as the psychological health QoL scale contains a question regarding self-esteem. The discovery that the long-term therapy group has worse self-esteem and psychological health than the short-term therapy group suggests that impaired self-esteem delays the therapeutic outcome.

The fact that there was no significant difference in family functioning and self-esteem between the ED subtypes, despite the differences in illness duration among the subtypes (see Table 9), supports the transdiagnostic nature of these factors. Overall, the findings further

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suggest that both self-esteem and family dysfunction are important for people without an ED as well, emphasising self-esteem in relation to ED prevention.

5.5.1. Clinical and Theoretical Implications

The finding that self-esteem emerged as a mediator between family dysfunction and ED and QoL and also having a direct relationship with these dependent variables suggests that self-esteem enhancement sessions should be designed as an addition to standard ED therapy. There is already evidence that group self-esteem enhancement sessions can reduce symptoms in ED patients (Newns et al., 2003). The fact that family functioning had an indirect effect on ED via self-esteem means that dysfunction in the family can have a long-lasting effect by affecting the formation of important psychological variables that have been proven to be risk factors for eating pathology, such as self-esteem. The fact that the duration of psychotherapy as a covariate did not affect self-esteem and psychosocial QoL in the ED subgroups further indicates the need to incorporate self-esteem into ED patients' psychotherapy. The difference in self-esteem and psychological health between long- and short-term therapy groups may further indicate this need, as incorporating self-esteem in standard therapy could accelerate therapeutic outcomes and lower the duration of psychotherapy.

These findings also demonstrate the need to include a psychosocial QoL assessment as part of ED treatment, instead of the sole assessment of symptomatology reduction, which may not result in better QoL. By addressing the underlying factors in treatment that seem to affect psychological and social QoL, such as family functioning and self-esteem, the impairment in various life domains may be reduced, resulting in an improvement of the ED pathology and better adjustment outside of clinical settings. This way, treatment will focus more on patients rather than the disorder itself, improving patient-centred care. Focusing on

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self-esteem and improving psychosocial QoL might also improve patients' overall functionality, thereby reducing the risk of relapse, as social relationships are consistently considered important for mental and physical health (Theodoropoulou et al., 2011; Umberson et al., 2010; Umberson & Montez, 2010). Furthermore, the bidirectional relationship between EDs and QoL observed in Mitchison and colleagues' study (2015) could be revisited by considering the mediating role of low self-esteem (see Section 2.4.2, Chapter 2).

Moreover, family dysfunction's effect on self-esteem highlights the need for the inclusion of family therapy in adults' ED treatment. Family therapy is a necessary component of any biopsychosocial approach to ED treatment, and the literature suggests that adults with an ED report more pathological family functioning than adolescents (Ciao et al., 2015). Family dysfunction has been observed in ED patients (Holtom-Viesel & Allan, 2014); therefore, individual, dyadic and family sessions could be arranged in order to examine and resolve the dysfunction of roles and communication in these families. This approach could be implemented as soon as ED patients seek treatment, so that all family members are part of the psychotherapeutic process. As revealed by this study's population's socio-demographic information, the majority of ED patients live with at least one family member, implying that family interactions and communication are part of their everyday life, which consequently can magnify and maintain the family dynamics throughout an individual's life.

Effective psychosocial prevention depends on the identification and targeting of specific risk and protective factors that influence the onset and course of a disorder (Phelps et al., 2000). Since eating pathology had a direct effect on the controls' psychosocial QoL, the need for ED prevention programmes in the community is also evident. A review of ED prevention programmes (Stice et al., 2013) revealed that the majority target body dissatisfaction and the thin ideal internalisation, with the authors suggesting the inclusion of

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other risk factors in order to enhance the programmes' effectiveness. The internalisation process in psychology is psychodynamic in nature and involves the developmental formation of a super ego which is related with the sense of self (Corsini, 1999). Moreover, EDs are considered to be the result of the dysfunctional internalisation of stressful situations that the individual cannot otherwise emotionally handle (Tandon et al., 2009). Considering that programmes that include the thin ideal as a target have been effective (Stice et al., 2013), it would be of additional help to include the possible causes of the thin ideal internalisation and body dissatisfaction, as both are consequences of other psychological variables, such as low self-esteem. The inclusion of self-esteem strengthening in ED prevention programmes might increase their efficacy (O'Dea & Abraham, 2000) by acting as a mediating factor between family functioning and ED pathology, as well as between family functioning and psychosocial QoL. Both factors had an impact on the controls' psychosocial QoL and eating pathology in this study, a finding that indicates their importance as psychological factors, regardless of the involvement of a clinical diagnosis.

Theoretically, the results indicate that existing theories of EDs should be revisited and adapted in order to form more comprehensive models that could sufficiently explain the role of family functioning and self-esteem regarding the formation and maintenance of eating pathology, as well as the ways family dysfunction affects self-esteem.

5.5.2. Limitations and Future Directions

The known limitations of cross-sectional designs apply to this study in that causality cannot be implied because the nature of the data does not permit testing of the temporal relationships between the constructs (Burns et al., 2012). However, Rindfleisch and colleagues (2007) argue that a thoroughly developed theoretical background strengthens causal inference and that under certain conditions (strong correlations among constructs and a

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combination of strong theory and statistical tools), the results from cross-sectional studies can be comparably valid to those from longitudinal ones. These conditions were satisfied in this study. Nevertheless, the use of experimental data to estimate the strength of the paths would strengthen these results (Warner, 2013). Additionally, both methods used in this study to explore the effects of illness chronicity and psychotherapy duration have limitations, as ANCOVA may produce biased results in non-experimental studies (Miller & Chapman, 2001). Furthermore, turning a continuous variable to a categorical one (one-way ANOVA) may attenuate statistical relationships because raw data are removed and replaced with categories instead, which could lower the power (Aiken & West, 1991; Altman & Royston, 2006; Cohen, 1983). However, using both methods offered a more thorough exploration of the factors of interest. It should be also noted that while psychotherapy duration was categorised as either short- or long-term based on the literature (Knekt et al., 2016), chronic illness was categorised based on a median-split analysis since there are no official cut-off scores to define chronic illness and it is usually described as a condition that lasts more than three months (Bernell & Howard, 2016). The clinical sample in this study did not have participants with low chronicity numbers, hence the median was appropriate. Yet, the median may have acted as an arbitrary cut-off point (McClelland et al., 2015).

In addition, only White women were sampled; thus, the results cannot be applied to other racial/ethnic groups or men. This is important because it has been found that there are variations in EDs, both between and within diverse groups (Assari & DeFreitas, 2018; Franco, 2007; Lydecker & Grilo, 2016). However, previous studies have found no gender differences in the self-esteem levels of ED patients (Kinasz et al., 2016), further supporting the importance of this psychological factor with respect to EDs, regardless of gender. Also, most ED studies rely on college populations or athletes, a biased sample when it comes to eating pathology, as the literature indicates (Currie, 2010; Striegel-Moore et al., 1989;

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Sundgot-Borgen & Torstveit, 2004). The inclusion of a non-student/non-athlete sample only as the control group in this study overcame this limitation. Additionally, having officially clinically assessed and diagnosed participants in the ED group increased reliability in terms of diagnosis, as the diagnosis procedure is more objective and thorough than self-reporting (Aboraya et al., 2016) and does not rely on the use of one specific instrument at one given point in time, which could lead to over- or misdiagnosis.

Family functioning was assessed by asking the participants only, instead of administering the questionnaire to their entire family, which would have provided a complementary perspective of family functioning. Yet, this study was interested in participants' evaluation of their family rather than the discrepancy of this evaluation among family members (Waller et al., 1990), since this is what matters most to these individuals. It is worth noting that while some scales had low reliability (which can reduce the likelihood of detecting significant effects), these all produced significant paths in the SEM model.

Furthermore, as explicit and not both implicit and explicit self-esteem was measured, the results can be generalised only to the former (Buhrmester et al., 2011). Yet, studies using both types of measurement did not find that the inclusion of implicit self-esteem explained eating pathology to a greater degree than explicit self-esteem alone (Anokhina, 2015). These findings, therefore, highlight that explicit self-esteem is sufficient to explain complex psychological relationships.

In addition to addressing the aforementioned limitations, this study should be extended in the following two ways. First, a reconceptualisation of family dysfunction is needed since this study conceptualised dysfunction in broad terms. In particular, conceptualising family dysfunction more specifically as childhood non-physical abuse is important for contextualising the effects of self-esteem. Second, exploring additional

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mediator variables as potential risk factors will clarify whether family dysfunction exerts its influence on EDs and QoL only through self-esteem or through other theoretically associated mechanisms as well. Both attachment and emotion regulation are affected by invalidating familial environments and are, therefore, important targets for study.

5.6. Conclusion

The association between family functioning and EDs has not been empirically clarified yet, and this study found that self-esteem plays a greater role than family functioning since it has a mediating effect between family functioning and ED pathology, rather than family functioning being influential by itself.

It is time to move on from investigations into family functioning alone in order to better understand the psychological mechanisms involved in the complex relationship with eating behaviour. This could be achieved by conceptualising family dysfunction in terms of non-physical childhood abuse in order to identify its consequences in adult life and how they may relate to ED pathology. This way dysfunction in the family becomes more specific in order to understand how it affects self-esteem. The addition of attachment and emotion regulation will further clarify the range of mediational mechanisms regarding family dysfunction and its developmental consequences on EDs.

Chapter 6 – Study 2: Childhood Non-physical Abuse in Adult Eating Disorder

Patients: A Transdiagnostic Developmental Model of Eating Pathology

6.1. Abstract

Background: Childhood non-physical abuse and its associated mechanisms remain under-researched despite their significant role in eating disorder (ED) development. The purpose of this study was to a) examine the relationship between childhood non-physical abuse with eating pathology and psychosocial quality of life (QoL) to determine whether self-esteem, attachment and emotion dysregulation act as mediators; and b) to explore attachment and temperament as mediators between childhood non-physical abuse and self-esteem, emotion regulation, EDs and psychosocial QoL. Kent and Waller's ED theory and Linehan's biosocial theory were extended and tested in Studies 2a and 2b, respectively.

Method: 80 female adult ED patients and 188 female adult healthy controls were recruited from Athens, Greece, and self-reported measures were used for childhood non-physical abuse, eating pathology, self-esteem, emotion regulation, temperament, attachment and psychosocial QoL. For the mediation hypotheses, structural equation modelling (SEM) was employed.

Results: Regarding Study 2a, only self-esteem mediated childhood non-physical abuse and eating pathology in patients. The relationship between childhood non-physical abuse and psychosocial QoL was mediated by self-esteem and emotion regulation, with self-esteem being the stronger mediator. In the controls, only emotion regulation mediated childhood non-physical abuse and EDs, and all three mediators were significant for psychosocial QoL, with self-esteem being the strongest. Regarding Study 2b, in patients, attachment mediated all the relationships except for EDs, while temperament mediated all the proposed relationships. In the controls, both attachment and temperament mediated all the proposed relationships.

Conclusion: Self-esteem is the most important risk factor for eating pathology in patients, while emotion regulation is a risk factor for the controls. Additionally, psychosocial QoL is affected more by psychological factors than ED symptoms, explaining why it remains low after symptoms' reduction. These results can enable targeted treatment and prevention designs, and the biosocial theory could benefit from the inclusion of attachment and self-esteem. Overall, these modifications can benefit the expansion of the biosocial theory regarding EDs. The proposed transdiagnostic developmental model could possibly offer insight regarding the origins and maintenance of EDs.

6.2. Introduction

The mechanisms involved in the development and maintenance of EDs is still unclear (Monell et al., 2020), though some literature suggests that childhood non-physical abuse is an important mechanism (Rai et al., 2019). Additionally, the literature has focused on the physical forms of childhood abuse (sexual and physical) and EDs, overlooking non-physical abuse (Burns et al., 2012; Kimber et al., 2017; Vajda & Láng, 2014), even though findings show that emotional abuse is a stronger predictor of eating pathology than physical forms (Burns et al., 2012; Fischer et al., 2010). Authors have maintained that childhood emotional abuse (CEA) is the most damaging form of childhood abuse (Kent & Waller, 2000; Rorty et al., 1994), with empirical evidence confirming that repeated emotional abuse affects brain development in children, despite the absence of physical abuse (van Harmelen et al., 2010). Systematic literature reviews on EDs and childhood abuse conclude the need for studies on causation regarding non-physical abuse forms and ED development (Rai et al., 2019).

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Non-physical childhood abuse in ED literature

In the limited studies on CEA—sometimes termed as psychological abuse, emotional maltreatment or psychological maltreatment (Baker & Maoiorino, 2010; Witkiewitz & Dodge-Reyome, 2000)—there is no sufficient understanding of the psychological factors that could explain the relationship with ED onset (Kent & Waller, 2000; Waller et al., 2007). There are even fewer studies on childhood neglect and EDs even though both types of neglect have been found in ED patients (Pignatelli et al., 2017). Furthermore, reviews indicate that family dysfunction has been broadly used in research to assume emotional abuse (Kent & Waller, 2000) and has been associated with the invalidating environment and childhood abuse (Crowell et al., 2009; Gonçalves et al., 2019; Mullen et al., 1996). The invalidating environment (Linehan, 1993) has been further used in the abuse literature regarding EDs to imply emotional abuse (Haslam et al., 2012) and to better explain family dysfunction (Gonçalves et al., 2019).

ED patients frequently report family dysfunction, which does not directly cause disordered eating (as supported by Study 1's results) but, rather, severely affects emotion regulation and core beliefs about the self (self-esteem), which in turn influence ED development (Wade et al., 2000). Reviews highlight the association of family factors both with the onset and maintenance of EDs, suggesting that family dysfunction should be better examined in terms of developmental psychopathology (Erriu et al., 2020). Therefore, this thesis proposes that the role of family dysfunction in EDs could be better understood if conceptualised as childhood non-physical abuse. Further expanding Study 1's results, family dysfunction/invalidating environment/childhood non-physical abuse can be better explained as an attachment disturbance that might lead to ED development (Hewett, 2014; Tasca et al., 2013), through low self-esteem and emotion dysregulation.

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Developmental consequences of childhood non-physical abuse

Attachment, self-esteem and emotion regulation are developmental factors formed in the early years of life through interaction with primary caregivers (Bowlby, 1973, 1979). Attachment is associated with EDs, affected by childhood maltreatment and responsible for shaping emotion regulation and self-esteem (Bowlby, 1973; Pearlman, 2005). An insecure attachment style could also have a negative effect on the individual's psychological health and later social relationships (Mikulincer & Shaver, 2012; Pearlman & Courtois, 2005), meaning that a disrupted attachment could also affect psychosocial QoL. Disrupted attachment due to childhood abuse is explained by the trauma betrayal theory, which proposes that betrayal due to bad attachment in early years of life can lead to the suppression of painful emotions (emotion dysregulation) and memories to preserve attachment (Freyd, 1996). Summarising the above, non-physical abuse disrupts attachment, and attachment affects the development of the self and emotion regulation.

Emotion regulation is a developmentally significant accomplishment that is essential for positive social adjustment (Stifter et al., 2011), indicating that it could affect individuals' social QoL. Emotion regulation has been found to be affected by non-physical childhood abuse (Vajda & Láng, 2014) and invalidating environments (Corstrophine, 2006; Fox, 2009; Fox & Power, 2009; Linehan, 1993). Moreover, non-physical forms of abuse are found to affect emotion regulation aspects more than physical forms of abuse (Zlotnic et al., 2001). Furthermore, trait self-esteem has a significant impact on the brain's response and severity of response to positive and negative stimuli (Wang & Wu, 2019), indicating that trait self-esteem affects emotion regulation.

Low trait self-esteem is a major risk factor for ED development (Adamson et al., 2019; Biney et al., 2019). Even though scholars have either implied or empirically supported

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self-esteem's important role in both ED development and non-physical childhood abuse, self-esteem has not been used as a mediator between non-physical abuse forms and EDs in clinical populations to date.

Study 2a. Given the psychological connection of the aforementioned factors and extending Study 1's research outcomes, Study 2a aimed to further examine the pathway through which family dysfunction, now conceptualised as non-physical abuse, affects eating pathology and psychosocial QoL. The term childhood non-physical abuse used in this study includes emotional abuse, emotional neglect and physical neglect. This study also expanded Kent's and Waller's model (2000), which proposes that emotional abuse plays a direct causal role in ED development, but this relationship could also be mediated by other factors, such as low self-esteem. In view of this, the first hypothesis explored in Study 2a was that self-esteem, emotion regulation and attachment mediate the relationship between childhood non-physical abuse and EDs/QoL in ED patients and healthy controls.

The invalidating environment as childhood non-physical abuse

Linehan (1993) theorised that an invalidating environment (non-physical childhood abuse) and a genetic tendency to be over-emotional (temperament) constitute the two major causes of borderline personality disorder (BPD) by affecting emotion regulation. Individuals coming from invalidating familial environments are likely to avoid emotional triggers or use impulsive (binge eating, purging, self-injury) and compulsive (food restriction, compulsive exercise, obsessive-compulsive features) behaviours to cope with negative affect (Haslam et al., 2008). The invalidating environment (Linehan, 1993) has been considered very useful in modelling the impact of CEA and in understanding the psychological interpretations of ED patients' individual experiences (Waller et al., 2007). Neglect could be also included, since Linehan's chaotic environment involves physically and emotionally unavailable parents

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(Linehan, 1993), and it has been related to negative core beliefs in ED patients (Ford et al., 2011). Temperament cannot cause an ED but can increase vulnerability to eating pathology in the presence of other risk factors (Martin et al., 2000). The concept of the invalidating environment has been extended to EDs (e.g. Gonçalves et al., 2019), but it has not been sufficiently researched (Reeves et al., 2010). Authors suggest that it is unclear how the invalidating environment may influence ED development (Haslam et al., 2012). Additionally, the SPAARS-ED model (Fox & Power, 2009) theoretically connects the invalidating environment with attachment and self-esteem to explain the origins of emotion dysregulation in eating pathology. It is the only model that considers schemas for the self and attachment, as self-esteem and attachment have been overlooked by affect regulation theories, even though attachment is responsible for the formation of self-esteem and emotion regulation. BPD and ED patients share psychological features, such as being raised in an invalidating environment, low self-esteem, poor attachment and emotion dysregulation. This indicates that there could be a link in the aetiology of the two disorders or at least common influential factors. Farber (2008) maintains that the manifestation of self-damaging behaviour, such as self-injury behaviour and/or EDs, is the result of disorganised attachment to a caregiver that has caused pain, with the person maintaining the pain in order to preserve the attachment. In that sense, it could be theoretically beneficial to include attachment in the biosocial theory model (Linehan, 1993) since attachment dysfunction is linked to emotion regulation.

Study 2b. Building upon Studies 1 and 2a, the purpose of Study 2b was to test and adapt Linehan's biosocial theory (1993) with respect to EDs. This study extends the biosocial model by incorporating attachment as a mediator, and self-esteem and psychosocial QoL as outcome variables. Non-physical childhood abuse (emotional abuse, emotional neglect, physical neglect) was used to represent the effects of the invalidating environment and family dysfunction, expanding Studies 1 and 2a. As a result, the association of non-physical

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childhood abuse, attachment and temperament with emotion regulation, self-esteem and the psychosocial aspects of QoL in ED patients was explored and compared with a healthy control group.

6.3. Method

6.3.1. Study Design

Study 2a is a structural equation modeling (SEM) analysis in which the endogenous variables are a) psychosocial QoL assessed on two domains through the World Health Organisation Brief Quality of Life Assessment Scale (WHOQOL-BREF) (psychological health, social relationships) and b) ED psychopathology assessed on the global score of the Eating Disorders Examination Questionnaire (EDEQ). The exogenous variable is non-physical abuse assessed using two domains of the Childhood Trauma Questionnaire (CTQ) (emotional abuse, emotional neglect) and the multidimensional neglect measure by the Multidimensional Neglectful Behaviour Scale (MNBS). The mediators are: a) global self-esteem measured through Rosenberg's SES, b) attachment measured by the two global scales of the Experiences in Close Relationships-Revised (ECR-RS) Questionnaire (global attachment-related anxiety, global attachment-related avoidance), c) emotion regulation measured by the global score of Difficulties in Emotion Regulation Scale (DERS). Study 2b is a SEM analysis in which the endogenous variables are: a) psychosocial QoL assessed on two domains through WHOQOL-BREF (psychological health, social relationships) b) emotion regulation assessed by the global DERS score and c) self-esteem assessed by Rosenberg's Self-Esteem Scale, d) eating pathology assessed by the global score of EDEQ. The exogenous variable is non-physical abuse assessed on two domains by the CTQ (emotional abuse, emotional neglect), and multidimensional neglect measured by MNBS. The mediators are a) the four temperament scales of Adult Temperament Questionnaire (ATQ) (negative affect, extraversion/surgency, effortful control, orienting sensitivity) and b)

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attachment measured by the two global scales of the ECR-RS (global attachment-related anxiety, global attachment-related avoidance). The two mediators were explored in different models each to preserve simplicity of the models and given the small sample size with respect to the number of variables.

6.3.2. Participants

A total of 268 Greek adult female participants completed the survey (between September 2015 and December 2016) and were eligible for inclusion in this study's analyses. Of the 268 participants, 80 (29.9%) were ED outpatients with a mean age of 29.10 years ($SD = 11.02$), and 188 (70.1%) were controls with a mean age of 25.54 years ($SD = 9.31$) drawn from the community (mostly college students), as shown in Table 21. Participation was voluntary, and the inclusion and exclusion criteria were the same as in Study 1 (see Section 5.3.2, Chapter 5). Of the 80 ED outpatients, 40 were drawn from the psychiatric department's EDs outpatient unit at Eginition Hospital, National and Kapodistrian University of Athens; 20 from the 18 ANO outpatient ED department at Dafni Psychiatric Hospital and 20 from the ANASA Day Care Centre for ED outpatients. All are located in Athens, Greece, and the author had written permission to recruit the clinical population from these units. The ED patients met the criteria for diagnosis according to the Diagnostic and Statistical Manual for Mental Disorders (DSM-V) (APA, 2013) after being evaluated by clinicians via the standard clinical interview, the eating disorders inventory and the eating disorders examination questionnaire (EDE-Q) prior to their assignment for treatment in the outpatient units. Of the total number of ED patients, 18 (22.5%) had been diagnosed with anorexia nervosa (AN) (5 with the restricting type, 9 with the bingeing/purging type, 4 other specified AN), 32 (40%) had been diagnosed with bulimia nervosa (BN) (31 BN, 1 other specified BN) and 30 (37.5%) had been diagnosed with binge eating disorder (BED) (29 BED, 1 other specified BED). The controls did not have an EDE-Q score indicating an ED, nor did they have a score

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over 2.30 indicating subclinical ED, meaning it was indeed a non-clinical sample (Thurston et al., 2008).

For a full description of the socio-demographic and clinical characteristics of the population see Tables 21 and 22, respectively.

Table 21.

Descriptive statistics in means (standard deviation) and frequencies (%) of the ED patients' (both by diagnostic group and as a whole) and the control group's socio-demographics – Study 2.

	AN PATIENTS n = 18 (6.7%)	BN PATIENTS n = 32 (11.9%)	BED PATIENTS n = 30 (11.2%)	EATING DISORDER PATIENTS n = 80 (29.9%)	CONTROLS n = 188 (70.1%)
EDUCATIONAL					
LEVEL					
<i>Primary school (6 years)</i>	0(0%)	0(0%)	0(0%)	0(0%)	1 (.5%)
<i>High school gymnasium (9 years)</i>	0(0%)	0(0%)	0(0%)	0 (0%)	0 (0%)
<i>High school lyceum (12 years)</i>	5 (27.8%)	7 (21.9%)	7 (23.3%)	19 (23.8%)	85 (45.2%)
<i>After school (14 years)</i>	5 (5.6%)	3 (9.4%)	3 (10%)	7 (8.8%)	10 (5.3%)
<i>College (16years)</i>	8 (44.4%)	21 (65.6%)	17 (56.7%)	46 (57.5%)	67 (35.6%)
<i>Post graduate studies (17-19 years)</i>	1 (5.6%)	1 (3.1%)	3 (10%)	8 (10%)	25 (13.3%)
WORKING STATUS					
<i>Full time</i>	6 (33.3%)	7 (21.9%)	10 (33.3%)	23 (28.8%)	40 (21.3%)
<i>Part time</i>	4 (22.2%)	8 (25%)	4 (13.3%)	16 (20%)	19 (10.1%)
<i>Not working (retired or householder)</i>	0(0%)	0(0%)	3 (10%)	3 (3.8%)	0(0%)
<i>Not working (unemployed)</i>	2 (11.1%)	2 (6.3%)	3 (10%)	7 (8.8%)	6 (3.2%)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
BIRTH ORDER					
<i>1st born</i>	8 (44.4%)	13 (40.6%)	14 (46.7%)	35 (43.8%)	68 (36.2%)
<i>2nd born</i>	5 (27.8%)	15 (46.9%)	13 (43.3%)	33 (41.3%)	65 (34.6%)
<i>3^d born</i>	1 (5.6%)	2 (6.3%)	1 (3.3%)	4 (5%)	13 (6.9%)
<i>4th born</i>	0(0%)	0(0%)	1 (3.3%)	1 (1.3%)	4 (2.1%)
<i>5th born</i>	0 (0%)	0(0%)	0(0%)	0(0%)	0(0%)
<i>Twin</i>	0 (0%)	0 (0%)	0(0%)	0(0%)	1 (.5%)
<i>Only child</i>	4 (22.2%)	2 (6.3%)	1 (3.3%)	8 (10%)	38 (20.2%)
LIVING STATUS					
<i>Alone</i>	4 (22.2%)	9 (28.1%)	9 (30%)	22 (27.5%)	31 (16.5%)
<i>Not alone</i>	14 (77.8%)	23 (71.9%)	21 (70%)	58 (72.5%)	157 (83.5%)
PEOPLE LIVING WITH					
<i>Family members(e.g. parents, siblings)</i>	11 (61.1%)	18 (56.3%)	14 (46.7%)	43 (53.8%)	122 (64.9%)
<i>Boyfriend/spouse, kids</i>	1 (5.6%)	3 (9.4%)	6 (20%)	10 (12.5%)	27 (14.4%)
<i>Roommate/friend</i>	2 (11.1%)	2 (6.3%)	1 (3.3%)	5 (6.3%)	8 (4.3%)
<i>None</i>	4 (22.2%)	9 (28.1%)	9 (30%)	22 (27.5%)	31 (16.5%)
RESIDENCE					
<i>Urban Athens</i>	15 (83.3%)	26 (81.3%)	27 (90%)	68 (85%)	149 (79.3%)
<i>Urban other</i>	3 (16.7%)	6 (18.8%)	2 (6.7%)	11 (13.8%)	30 (16%)
<i>Rural (village, island)</i>	0(0%)	0(0%)	1 (3.3%)	1 (1.3%)	8 (4.3%)
LONGER STAY RESIDENCE					
<i>Urban Athens</i>	11 (61.1%)	18 (56.3%)	23 (76.7%)	52 (65%)	119 (63.3%)
<i>Urban other</i>	3 (16.7%)	6 (18.8%)	2 (6.7%)	11 (13.8%)	30 (16%)
<i>Rural (village, island)</i>	4 (22.2%)	8 (25%)	5 (16.7%)	17 (21.3%)	39 (20.7%)

Note: The ED subgroups are mentioned for informative reasons, and they were not used in the analyses.

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Table 22.

Descriptive statistics in means (standard deviation) and frequencies (%) of the ED patients' (both by diagnostic group and as a whole) and the control group's clinical data – Study 2.

	AN PATIENTS n = 18 (6.7%)	BN PATIENTS n = 32 (11.9%)	BED PATIENTS n = 30 (11.2%)	EATING DISORDER PATIENTS n = 80 (29.9%)	CONTROLS n = 188 (70.1%)
	Mean (SD) or frequency %	Mean (SD) or frequency %	Mean (SD) or frequency %	Mean (SD) or frequency %	Mean (SD) or frequency %
Current Age (years)	25.83 (7.34)	26.72 (8.41)	33.60 (13.78)	29.10 (11.02)	25.54 (9.31)
Age of ED onset (years)	19.61 (6.34)	17.09 (3.46)	15.27 (7.28)	16.98 (5.96)	0 (0)
Duration of illness (years)	6.22 (5.84)	9.63 (8.33)	18.33 (12.35)	12.13 (10.76)	0 (0)
Hospitalised for ED					
<i>Yes</i>	80 (0%)	7 (21.9%)	4 (13.3%)	11 (13.8%)	0 (0%)
<i>No</i>	18 (100%)	25 (78.1%)	26 (86.7%)	69 (86.3%)	188 (100%)
Times hospitalised for ED	0 (0)	.38 (.98)	.30 (.92)	.26 (.84)	0 (0)
Duration of hospitalization (days)	0 (0)	18.88 (70.81)	16.40 (68.03)	13.70 (61.02)	0 (0)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
Perceived health status					
<i>Very bad</i>	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0(0%)
<i>Bad</i>	1 (5.6%)	0 (0%)	3 (10%)	4 (5%)	2(1.1%)
<i>Neither good nor bad</i>	10 (55.6%)	10 (31.3%)	6 (20%)	26 (32,5%)	12 (6.4%)
<i>Good</i>	6 (33.3%)	10 (3.3%)	11 (36.7%)	27 (33.8%)	83 (44.1%)
<i>Very good</i>	1 (5.6%)	12 (37.5%)	10 (33.3%)	23 (28.8%)	91 (48.4%)
Facing any health problem					
<i>Yes</i>	8 (44.4%)	8 (25%)	13 (43.3%)	29 (36.3%)	11 (5.9%)
<i>No</i>	10 (55.6%)	24 (75%)	17 (56.7%)	51 (63.8%)	176 (93.6 %)
Type of problem					
<i>None</i>	10 (55.6%)	24 (75%)	17 (56.7%)	51 (63.8%)	176 (93.6%)
<i>Mental health</i>	2 (11.1%)	2 (6.3%)	2 (6.7%)	6 (7.5%)	0(0%)
<i>Cardiological</i>	1 (5.6%)	0(0%)	2 (6.7%)	3 (3.8%)	1 (.5%)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
<i>Ophthalmological</i>	0(0%)	0(0%)	0(0%)	0 (0%)	0(0%)
<i>Endocrinological</i>	0 (0%)	4 (6.7%)	3 (10%)	2 (2.5%)	4 (2.1%)
<i>Orthopaedic</i>	1 (5.6%)	1 (3.1%)	2 (6.7%)	4 (5%)	4 (2.1%)
<i>Gastrointestinal</i>	2 (11.1%)	4 (12.5%)	0(0%)	5 (6.3%)	1(.5%)
<i>Respiratory (asthma included)</i>	0(0%)	0(0%)	1 (3.3%)	1 (1.3%)	1(.5%)
<i>Autoimmune</i>	0(0%)	0(0%)	1 (3.3%)	1 (1.3%)	1(.5%)
<i>Gynaecological</i>	1 (5.6%)	1 (3.1%)	2 (6.7%)	4 (5%)	0(0%)
<i>Dermatological</i>	1 (5.6%)	0(0%)	0(0%)	1 (1.3%)	0(0%)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
<hr/>					
Perceived seriousness and attribution of any health problem (physical or mental) affecting their life					
<i>ED related</i>	11 (61.1%)	18 (56.3%)	19 (63.3%)	48 (60%)	0 (0%)
<i>Non ED related</i>	0 (0%)	2 (6.3%)	2 (6.7%)	4 (5%)	10 (5.3%)
<i>None</i>	7 (38.9%)	12 (37.5%)	9 (30%)	28 (35%)	178 (94.7%)
Mental health pro visit					
<i>Yes</i>	14 (77.8%)	27 (84.4%)	27 (90%)	68 (85%)	96 (51.1%)
<i>No</i>	4 (22.2%)	5 (15.6%)	3 (10%)	12 (15%)	92 (48.9%)
Psychotherapy (at any point in their life)					
<i>Yes</i>	12 (66.7%)	19 (59.4%)	24 (80%)	55 (68.8%)	55 (29.3%)
<i>No</i>	6 (33.3%)	13 (40.6%)	6 (20%)	25 (31.3%)	133 (70.7%)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
Currently in psychotherapy					
<i>Yes</i>	8 (44.4%)	16 (50%)	16 (53.3%)	40 (50%)	21 (11.2%)
<i>No</i>	10 (55.6%)	16 (50%)	14 (46.7%)	40 (50%)	167 (88.8%)
Total Duration of reported Psychotherapy (months)	11.56 (23.22)	31.50 (20.21)	28.87 (34.28)	21.28 (3.50)	7.65 (18.86)
Body weight (kg)	43.94 (9.05)	61.16 (11.79)	84.43 (21.17)	66.01 (22.05)	60.97 (10.93)
BMI	17.83 (7.01)	22.28 (4.35)	30.50 (7.64)	24.36 (8.07)	21.94 (3.62)
Body Discrepancy Index	.72 (1.99)	2.25 (1.50)	2.70 (1.15)	2.08 (1.67)	.89 (1.16)

Note: The duration of illness is calculated by subtracting the age of ED onset from the current age. The body discrepancy index indicated body dissatisfaction by subtracting the ideal body shape from the perceived body shape. The negative scores indicate a desire to be bigger, and positive scores indicate a desire to be thinner. A score of zero indicated no body dissatisfaction. In this table, only positive scores are observed. Patients who answered that they have never undergone psychotherapy were on a waiting list to start. The ED subgroups are mentioned for informative reasons, and they were not used in the analyses.

6.3.3. Measures

The discussion of the following measures used in this study can be found in Chapter 4 (4.3.3.): 1) World Health Organisation Brief Quality of Life Assessment Scale (WHOQOL-BREF), 2) Rosenberg Self-esteem scale (RSES), 3) Eating Disorder Examination Questionnaire (EDE-Q 6), 4) The Childhood Trauma Questionnaire - Short Form (CTQ-SF) 5) The Multidimensional Neglectful Behaviour Scale Adult Recall Version Short Form-MNBS AS, 6) The Experiences in Close Relationships-Relationship Structures (ECR-RS) Questionnaire, 7) Difficulties in Emotion Regulation Scale (DERS), 8) Adult Temperament

Questionnaire-Short Form (ATQ-SF), 9) Sociodemographic and Relevant Clinical Information.

6.3.4. Procedure

As described in 4.3.4 of Chapter 4.

6.3.5. Data Analytic Strategy

One structural equation model for each of the two participants groups was performed to explore the hypothesised mediational models, using AMOS 22 (Arbuckle, 2013). The hypothesised structural models for patients and controls are shown in Figures 8 and 9, respectively, displaying the path coefficients for the standardised direct effects. The examination of the standardised residuals and modifications indices revealed no high values, and the standardised residuals were not systematic, indicating that both conceptual models are viable. Standardised direct and indirect effects are reported for both models. Standardised regression weights were used to interpret the direct effects, and the significance of the indirect effects was based on the bias-corrected bootstrap method confidence intervals (Cheung, 2009; MacKinnon et al., 2004), which are reported below, along with the direct paths. Regarding the bootstrap, 500 bootstrap samples were created by random sampling and replacement of the original dataset for the patients and 1,000 for controls. This difference in the bootstrap sample number for the two groups is because AMOS could not achieve a solution when the number of ED patients' samples was greater than 500. The rest of the strategy is the same as in Study 1 (see Section 5.3.5 in Chapter 5).

6.4. Results

6.4.1. Data Screening and Preliminary Analyses

Before proceeding to the statistical analyses, the data were screened for missing values, data entry errors, univariate and multivariate outliers, and for the distribution's normality (Field, 2009; Tabachnick & Fidell, 2007).

Outliers and Normality. Univariate outliers were checked and corrected with respect to each statistical analysis separately. Multivariate outliers were checked after the univariate ones have been corrected and after the transformation of variables to succeed normality when needed. Normality was dealt with using the same procedures as in Study 1 (see Section 5.4.1 in Chapter 5).

The univariate outliers were checked for all of Study 2a's and 2b's variables separately for the ED group ($n = 80$) and the control ($n = 188$) group. There were 4 univariate outliers for the ED group and 53 univariate outliers for the controls. After the univariate outliers had been addressed, the check for multivariate outliers was performed for each group using Mahalanobis distance, identifying no multivariate outliers for the patient group in either Study 2a or 2b, nor for the controls in Study 2a. There was one multivariate outlier in the control group in Study 2b, participant 69. This participant was a 21-year-old college graduate with no reported health problems or psychotherapy for any reason and a BMI of 24. This participant was deleted and the check for Mahalanobis distance revealed no other masked multivariate outliers in Study 2b's control group.

For the ED patients group ($n = 80$), only the physical neglect scale had z-value over 3.29 ($p < .05$), being positively skewed, (z -skewness = 4.18, z -kurtosis = .75) and was corrected with square root (z -skewness = 3.27, z -kurtosis = -.52). For the controls ($n = 188$), three variables had z-values over 3.29. Emotional abuse was positively skewed (z -skewness =

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5.65, z-kurtosis = $-.20$) and was corrected with logarithm (z-skewness = 2.69, z-kurtosis = $-.35$). Emotional neglect was positively skewed (z-skewness = 4.83, z-kurtosis = $-.44$) and was corrected with square root (z-skewness = 3.11, z-kurtosis = 1.92). Physical neglect was positively skewed (z-skewness = 6.65, z-kurtosis = $.42$), and it was not corrected, even when transformed with inverse transformation. It was decided not to use this scale in the analysis as abuse was also assessed by the scale of multidimensional neglect that represents the physical neglect part of non-physical abuse. For comparability reasons, it was not included in the patients' analysis either. Table 23 presents the means and standard deviations for the scales used in the analyses for both Studies 2a and 2b as they examine the same population. A comparison of these means to those of other published studies can be found in Appendix 38.

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Table 23.

Means (standard deviation) of questionnaires' results for ED patients (both by diagnostic group and as a whole) and the control group – Study 2.

	AN PATIENTS n = 18 (6.7%)	BN PATIENTS n = 32 (11.9%)	BED PATIENTS n = 30 (11.2%)	EATING DISORDER PATIENTS n = 80 (29.9%)	CONTROLS n = 188 (70.1%)
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Rosenberg's SELF-ESTEEM (range 0–30)	12.78 (5.55)	13.84 (6.60)	14.47 (5.01)	13.84 (5.77)	20.06 (5.23)
CTQ Emotional Abuse (range 5-25)	12.78 (5.78)	13.31 (5.55)	13.07 (5.78)	13.10 (5.62)	8.81 (3.87)
CTQ Emotional Neglect (range 5-25)	11.06 (4.60)	12.44 (5.66)	12.37 (5.06)	12.10 (5.18)	9.11 (3.84)
CTQ Physical Neglect (range 5-25)	6.78 (2.88)	7.53 (2.78)	7.57 (2.80)	7.38 (2.79)	6.16 (1.58)
Multidimensional Neglect (range 8-32)	14.67 (4.35)	14.25 (4.15)	15.23 (4.86)	14.71 (4.44)	11.99 (3.17)
ATQ Extraversion Synergy (range 1-7)	4.08 (.94)	3.97 (.66)	4.06 (.68)	4.03 (.73)	4.56 (.73)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
ATQ Negative Affect (range 1-7)	4.36 (.82)	4.64 (.78)	4.71 (.77)	4.60 (.79)	4.12 (.65)
ATQ Orienting Sensitivity (range 1-7)	4.81 (1.04)	4.76 (.95)	4.94 (.80)	4.84 (.91)	4.61 (.74)
ATQ Effortful Control (range 1-7)	4.42 (.83)	4.06 (.83)	3.83 (.69)	4.06 (.80)	4.38 (.74)
ECRS Global Anxiety (range 1-7)	3.34 (1.74)	3.28 (1.71)	3.03 (1.63)	3.20 (1.67)	2.03 (.96)
ECRS Global Avoidance (range 1-7)	3.46 (.97)	3.28 (.88)	3.27 (.98)	3.32 (.93)	2.82 (.92)
DERS total score (range 36-180)	105.00 (27.32)	105.09 (25.04)	114.00 (25.58)	108.41 (25.80)	81.16 (20.33)
EDE-Q GLOBAL SCORE (range 0-6)	3.62 (1.20)	4.15 (1.01)	3.36 (1.20)	3.73 (1.17)	1.24 (.99)
BREF PSYCHOLOGICAL QoL (range 4-20)	10.22 (3.19)	10.54 (3.20)	11.11 (2.63)	10.68 (2.98)	14.24 (2.04)

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	AN PATIENTS	BN PATIENTS	BED PATIENTS	EATING DISORDER PATIENTS	CONTROLS
BREF SOCIAL RELATIONSHIPS QoL	11.93	11.83	11.42	11.70	14.39
(range 4-20)	(3.87)	(4.05)	(3.26)	(3.69)	(3.15)

Note: These means are derived from the scales with corrected univariate outliers prior to any transformation.

6.4.2. Study 2a: Non-physical abuse effects on eating pathology and psychosocial QoL:

The mediating role of attachment, self-esteem and emotion regulation

Measurement Models

Regarding fit indices used to evaluate models see section 5.4. of Chapter 5. Prior to analysis of the structural models both for patients and controls, the measurement models were assessed in order to evaluate the appropriateness of the latent variables as the measurement model stage of SEM examines the relations between the measured variables and their proposed latent constructs (Kline, 2011). Both initial models (patients and controls) had three latent variables (non-physical abuse, attachment, psychosocial QoL) and three observed variables (self-esteem, emotion regulation, eating pathology). The latent variable of non-physical abuse has three indicators (emotional abuse, emotional neglect, multidimensional neglect) as measured by the CTQ and MNBS. The latent variable of psychosocial QoL has two indicators (Psychological Health, Social Relationships) as measured by WHOQoL BREF and the latent variable of attachment has two indicators (global avoidance, global anxiety) as measured by the ECR-RS.

ED Patients: The model had a poor fit; $\chi^2(11, N = 80) = 22.300, p = .022, CFI = .96, TLI = .91, RMSEA = .11$. According to the modification indices, the deletion of CTQ

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emotional neglect and avoidance attachment would improve the fit. As this change does not affect the main theoretical model it was implemented. Following this amendment, the new measurement model had a good fit: $\chi^2(1, N = 80) = .705, p = .401, CFI = 1, TLI = 1.02, RMSEA = .00$. The latent variables of non-physical abuse and psychosocial QoL were correlated ($r = -.54, p = .014$) and the factors loaded significantly ($p < .001$) with their latents ranging from .65 to .95. Thus, the adjusted model has two latent variables and four observed variables.

Controls: The model had a poor fit: $\chi^2(11, N = 188) = 28.088, p = .003, CFI = .96, TLI = .92, RMSEA = .09$. According to the modification indices, the deletion of anxiety (attachment) and CTQ emotional neglect would improve the fit and as this would not affect the main theoretical model, the change was implemented. As a result, the new measurement model had a good fit: $\chi^2(1, N = 188) = .002, p = .965, CFI = 1, TLI = 1.04, RMSEA = .00$. The latent variables of non-physical abuse and psychosocial QoL were correlated ($r = -.63, p < .001$) and the factors loaded significantly ($p < .001$) with their latents ranging from .64 to .78. Thus, the adjusted model has two latent variables and four observed variables.

After the changes in the two models to improve the fit, the following differences are noticed between patients and controls: 1) global anxiety was used for patients and global avoidance for controls, 2) a direct path was drawn from ED to psychosocial QoL for controls, 3) a direct path was drawn from emotion regulation to self-esteem for controls. For both group models non-physical abuse and psychosocial QoL were latent variables and self-esteem, attachment, emotion regulation and eating pathology were observed variables.

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Structural Model Analyses

The preliminary correlations between all variables within the hypothesised models for patients and controls are shown in Tables 24 and 25 respectively. The proposed mediational paths in these theoretical models for patients and controls are: The exogenous variable is non-physical abuse, the mediators are emotion regulation, attachment and self-esteem and ED and psychosocial QoL is the endogenous variable. The paths are as follows: Path 1 = non-physical abuse to ED through attachment, path 2 = non-physical abuse to psychosocial QoL through attachment, path 3 = non-physical abuse to ED through self-esteem, path 4 = non-physical abuse to psychosocial QoL through self-esteem, path 5 = non-physical abuse to ED through emotion regulation, path 6 = non-physical abuse to psychosocial QoL through emotion regulation. All significant correlations were in the expected direction in patients. That is, increased non-physical abuse was related to greater ED, emotion dysregulation, bad attachment and temperamental difficulties and lower QoL and self-esteem. Higher attachment difficulties and emotion dysregulation were related to lower QoL and higher ED. Low self-esteem was related to higher ED and lower QoL. High temperament was related to lower QoL and higher ED. Regarding controls, all significant correlations were in the expected direction as well. That is, increased non-physical abuse was related to greater ED, emotion dysregulation, bad attachment and temperamental difficulties and lower QoL and self-esteem. Higher attachment difficulties and emotion dysregulation were related to lower QoL and higher ED. Low self-esteem was related to higher ED and lower QoL. High temperament was related to lower QoL and higher ED. The decision regarding the direct and indirect paths is the same as in Study 1 (see 5.4.2. Chapter 5).

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Table 24.

Bivariate Correlations (two-tailed), Means and Standard Deviations for Observed Variables with the Structural Equation Model for the Patient Group (n = 80) – Studies 2a & 2b.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Negative affect	-													
2. Effortful control	-.53 ^{***}	-												
3. Extraversion Surgency	-.27 [*]	.11	-											
4. Orienting Sensitivity	.24 [*]	-.09	.10	-										
5. Emotional Abuse	.32 ^{**}	-.36 ^{**}	-.08	.34 ^{**}	-									
6. DERS Total	.58 ^{***}	-.65 ^{***}	-.16	.23 [*]	.47 ^{***}	-								
7. EDEQ Global	.45 ^{***}	-.34 ^{**}	-.13	.14	.24 [*]	.43 ^{***}	-							
8. Multi-Dimensional Neglect	-.00	-.15	-.03	.27 [*]	.62 ^{***}	.26 [*]	.16	-						
9. Self-esteem	-.38 ^{***}	.39 ^{***}	.32 ^{**}	-.06	-.48 ^{***}	-.55 ^{***}	-.56 ^{***}	-.35 ^{**}	-					
10. Psychological Health	-.46 ^{***}	.43 ^{***}	.38 ^{***}	-.19	-.40 ^{***}	-.64 ^{***}	-.58 ^{***}	-.24 [*]	.77 ^{***}	-				
11. Social Relationships	-.40 ^{***}	.21	.37 ^{**}	-.20	-.40 ^{***}	-.45 ^{***}	-.23 [*]	-.30 ^{**}	.51 ^{***}	.60 ^{***}	-			
12. Global Anxiety	.23 [*]	-.20	.00	.33 ^{**}	.63 ^{***}	.25 [*]	.38 ^{**}	.51 ^{***}	-.41 ^{***}	-.26 [*]	-.25 [*]	-		
13. Global Avoidance	-.00	-.03	-.13	.09	.40 ^{***}	.18	.08	.50 ^{***}	-.19	-.27 [*]	-.47 ^{***}	.46 ^{***}	-	
14. Emotional Neglect	.10	-.19	-.17	.24 [*]	.74 ^{***}	.27 [*]	.12	.73 ^{***}	-.42 ^{***}	-.31 ^{**}	-.34 ^{**}	.61 ^{***}	.47 ^{***}	-
Mean	4.60	4.06	4.03	4.84	13.10	108.41	3.73	14.71	13.84	10.68	11.70	3.20	3.32	12.10
SD	.79	.80	.73	.91	5.62	25.80	1.17	4.44	5.77	2.98	3.69	1.67	.93	5.18
Range	1-7	1-7	1-7	1-7	5-25	36-180	0-6	4-32	0-30	4-20	4-20	1-7	1-7	5-25

* $p < .05$, ** $p < .01$, *** $p < .001$

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Table 25.

Bivariate Correlations (two-tailed), Means and Standard Deviations for Observed Variables with the Structural Equation Model of the Controls Group (n = 188) – Study 2a.

	1	2	3	4	5	6	7	8	9	10
1. DERS Total	-									
2. EDEQ Global	.25***	-								
3. Multi-Dimensional Neglect	.26***	.04	-							
4. Self-esteem	-.60***	-.23**	-.33***	-						
5. Psychological Health	-.59***	-.34***	-.31***	.68***	-					
6. Social Relationships	-.39***	-.19**	-.29***	.46***	.56***	-				
7. Global Anxiety	.33***	.23**	.15*	-.30***	-.32***	-.41***	-			
8. Global Avoidance	.23**	.08	.39***	-.28***	-.41***	-.35***	.41***	-		
9. Emotional Abuse	.23**	.09	.47***	-.36***	-.36***	-.33***	.33***	.41***	-	
10. Emotional Neglect	.19**	.02	.61***	-.35***	-.35***	-.23**	.26***	.51***	.64***	-
Mean	81.16	1.24	11.98	20.06	14.24	14.39	2.03	2.82	8.81	9.11
SD	20.33	.99	3.12	5.23	2.03	3.15	.96	.92	3.87	3.84
Range	36-180	0-6	8-32	0-30	4-20	4-20	1-7	1-7	5-25	5-25

* $p < .05$, ** $p < .01$, *** $p < .001$

Note: Means and standard deviations reported for the CTQ emotional abuse and CTQ emotional neglect variables are prior to transformation. The correlation numbers reported for the same variables are after transformation as they were entered transformed in the SEM analysis.

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ED Patients: Model fit indices indicated that the hypothesised model fit the data well: $\chi^2(10, N = 80) = 10.893, p = .366, CFI = 1, TLI = .99, RMSEA = .03$. The direct path from non-physical abuse to anxiety attachment was significant ($\beta = .88, p < .001$) and so were the direct paths from: non-physical abuse to self-esteem ($\beta = -1.17, p = .001$), non-physical abuse to emotion regulation ($\beta = 1.67, p = .003$), self-esteem to psychosocial QoL ($\beta = .73, p < .001$), emotion regulation to psychosocial QoL ($\beta = -.51, p = .004$), self-esteem to eating pathology ($\beta = -.88, p = .002$) and attachment anxiety to emotion regulation ($\beta = -1.22, p = .019$). The last direct path was not part of the mediation hypothesis but all possible paths that have a theoretical meaning without causing fit issues were examined in the model. These results indicate that high levels of childhood non-physical abuse lead to worse attachment, low self-esteem and emotion dysregulation, low self-esteem raises eating pathology diminishing QoL and emotion dysregulation lowers QoL. However, in contrast to the Pearson's correlation coefficient which was positive, the negative path coefficient suggests that high levels of anxiety attachment seem to lead to low levels of emotion dysregulation. This indicates the presence of a suppressor variable effect i.e., another variable in the model changed the direction of this relationship by suppressing irrelevant variance (Maassen & Bakker, 2001).

The indirect path from non-physical abuse to eating pathology with attachment as a mediator (path 1) was significant (standardized indirect effect = .68, bias-corrected bootstrap lower CI = .133 and upper CI = 4.510, $p = .002$) and so were the indirect paths from: non-physical abuse to eating pathology with self-esteem as a mediator (path 3) (standardized indirect effect = .30, bias-corrected bootstrap lower CI = .073 and upper CI = 2.561, $p = .003$), non-physical abuse to psychosocial QoL with self-esteem as a mediator (path 4) (standardized indirect effect = -.47, bias-corrected bootstrap lower CI = -1.290 and upper CI = -.205, $p = .002$), non-physical abuse to eating pathology with emotion regulation as

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mediator (path 5) (standardized indirect effect = .65, bias-corrected bootstrap lower CI = .130 and upper CI = 4.049, $p = .002$) and non-physical abuse to psychosocial QoL with emotion regulation as mediator (path 6) (standardized indirect effect = -.47, bias-corrected bootstrap lower CI = -1.568 and upper CI = -.178, $p = .004$).

It is interesting to note that non-physical abuse had a direct effect on all mediators but no direct effect on the dependent variables (ED and QoL), affecting them only through mediation. Regarding the mediators, only self-esteem and emotion regulation had an effect on psychosocial QoL and only self-esteem had an effect on eating pathology. The RMediation program was used (Tofighi & MacKinnon, 2011) to further test these mediational effects using the distribution-of-product method. The unstandardized coefficients and standard errors of the a and b paths for each of the five significant indirect effects yielded lower and upper 95% confidence limits of .072 and .598 for path 3, -.866 and -.156 for path 4 and -.972 and -.095 for path 6 providing significant indirect effects, thus confirming mediation for the three out of five significant indirect paths: for non-physical abuse to ED with self-esteem as mediator, from non-physical abuse to psychosocial QoL with self-esteem as mediator, and from non-physical abuse to psychosocial QoL with emotion regulation as mediator.

The results are consistent with those of a partial mediation effect given a direct path from non-physical abuse to self-esteem and emotion regulation in the presence of significant indirect effects both for eating pathology and psychosocial QoL when self-esteem was the mediator, and significant indirect effect for psychosocial QoL when emotion regulation was the mediator. All the explored paths in the model have a post hoc power 1 including the ones that were not significant. In other words, self-esteem partially mediated the relationship between non-physical childhood abuse and ED and psychosocial QoL, and emotion

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regulation partially mediated the relationship between non-physical childhood abuse and psychosocial QoL.

Controls: Model fit indices indicated that the hypothesised model fit the data well: $\chi^2(9, N = 188) = 5.089, p = .826, CFI = 1.03, TLI = 1, RMSEA = .00$. The direct path from non-physical abuse to avoidance attachment was significant ($\beta = .59, p < .001$) and so were the direct paths from: non-physical abuse to emotion regulation ($\beta = .33, p = .009$), non-physical abuse to self-esteem ($\beta = -.36, p = .002$), emotion regulation to eating pathology ($\beta = .19, p = .037$), self-esteem to psychosocial QoL ($\beta = .45, p < .001$), emotion regulation to psychosocial QoL ($\beta = -.25, p < .001$), attachment to psychosocial QoL ($\beta = -.20, p = .007$), emotion regulation to self-esteem ($\beta = -.49, p < .001$) and eating pathology to psychosocial QoL ($\beta = -.17, p = .001$). The two latter direct paths were not part of the mediation hypothesis but all possible paths that would make a theoretical meaning without causing fit issues were examined in the model. According to the results, high levels of childhood non-physical abuse lead to worse attachment, low self-esteem and emotion dysregulation, low self-esteem leads to low QoL, emotion dysregulation leads to ED and low QoL, worse attachment lowers QoL, emotion dysregulation lowers self-esteem and ED makes QoL worse.

The indirect path from non-physical abuse to psychosocial QoL with attachment as mediator (path 2) was significant (standardized indirect effect = -1.68, bias-corrected bootstrap lower CI = -4.036 and upper CI = -.296, $p = .021$) and so were the indirect paths from: non-physical abuse to psychosocial QoL with self-esteem as mediator (path 4) (standardized indirect effect = -2.33, bias-corrected bootstrap lower CI = -4.613 and upper CI = -1.075, $p = .001$), non-physical abuse to eating pathology with emotion regulation as mediator (path 5) (standardized indirect effect = .49, bias-corrected bootstrap lower CI = .024 and upper CI = 1.444, $p = .032$) and non-physical abuse to psychosocial QoL with emotion

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regulation as mediator (path 6) (standardized indirect effect = -1.22, bias-corrected bootstrap lower CI = -2.775 and upper CI = -.355, $p = .004$).

It is interesting to note that non-physical abuse did not have a direct effect on eating pathology and psychosocial QoL, but there was an effect on these two dependent variables through mediation. Non-physical abuse did have a direct effect on all mediators and all the mediators had a direct effect on psychosocial QoL, but only emotion regulation had a direct effect on eating pathology.

The RMediation program was used (Tofighi & MacKinnon, 2011) to further test these mediational effects using the distribution-of-product method. The unstandardized coefficients and standard errors of the a and b paths for each of the four significant indirect effects yielded lower and upper 95% confidence limits of -3.174 and -.422 for path 2, -4.149 and -.822 for path 4, .025 and 1.171 for path 5 and -2.553 and -.255 for path 6, providing significant indirect effects, thus confirming mediation for these four paths.

The results are consistent with those of a partial mediation effect given a direct path from non-physical abuse to all the mediators in the presence of significant indirect effects both for eating pathology and psychosocial QoL when emotion regulation was the mediator and significant indirect effect for psychosocial QoL in the presence of all mediators. All the explored paths in the model have a post hoc power ranging from .86 to 1 including the ones that were not significant. In other words, emotion regulation mediated the relationship between childhood non-physical abuse and ED and QoL, and emotion regulation and self-esteem mediated the relationship between abuse and QoL.

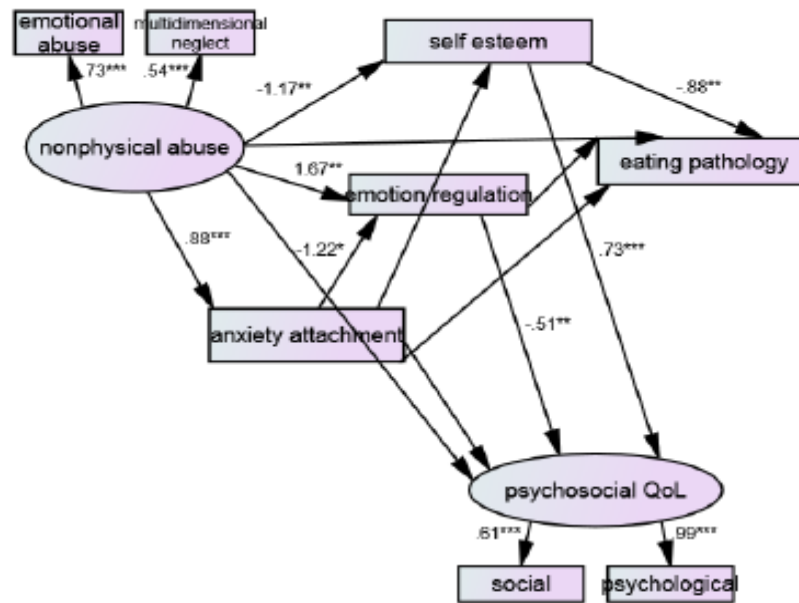
Summary: There were some differences between the two SEM models/samples. Regarding the beta values in the two groups in their common direct paths, non-physical abuse had a stronger effect on self-esteem [$(\beta = -1.17, p = .001)$ vs $(\beta = -.36, p = .002)$], attachment

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[($\beta = .88, p < .001$) vs ($\beta = .59, p < .001$)] and emotion regulation [($\beta = 1.67, p = .003$) vs ($\beta = .33, p = .009$)] for patients and self-esteem had a stronger effect on QoL for patients as well [($\beta = .73, p < .001$) vs ($\beta = .45, p < .001$)]. The indirect effects that were common for the two groups are childhood non-physical abuse - self-esteem – psychosocial QoL and childhood non-physical abuse – emotion regulation – psychosocial QoL.

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Figure 8. Structural equation model testing the mediational effect of self-esteem, emotion regulation and attachment on the relationships between non-physical abuse and eating pathology and non-physical abuse and psychosocial QoL in ED patients.



SIGNIFICANT MEDIATION PATHS

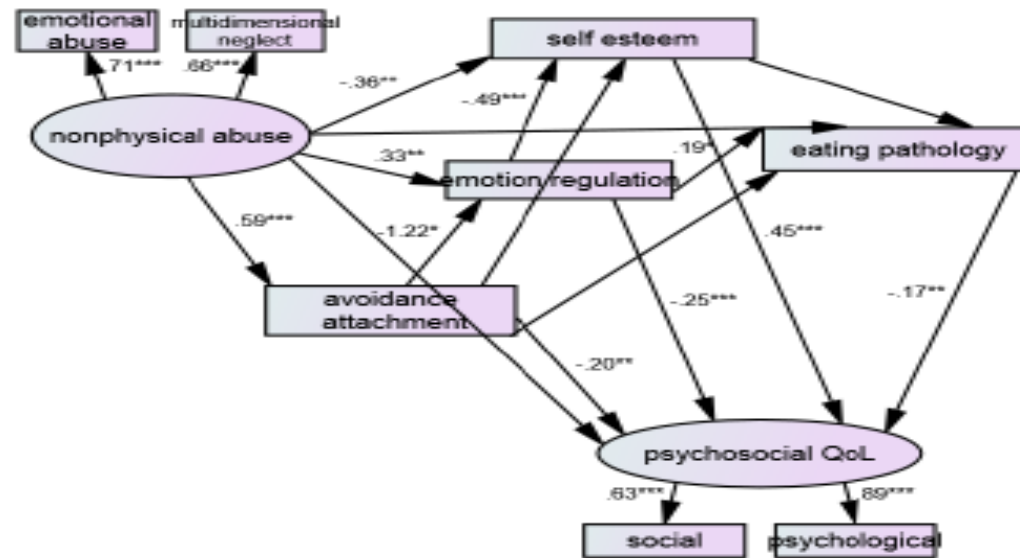
- Nonphysical abuse - self-esteem - eating pathology
- Nonphysical abuse - self-esteem - psychosocial QoL
- Nonphysical abuse - emotion regulation - psychosocial QoL

* $p < .05$, ** $p < .01$, *** $p < .001$

Note: Standardised paths shown are significant.

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Figure 9. Structural equation model testing the mediational effect of self-esteem, emotion regulation and attachment on the relationships between non-physical abuse and eating pathology and non-physical abuse and psychosocial QoL in the control group.



SIGNIFICANT MEDIATION PATHS

- Nonphysical abuse - attachment - psychosocial QoL
- Nonphysical abuse - self-esteem - psychosocial QoL
- Nonphysical abuse - emotion regulation - eating pathology
- Nonphysical abuse - emotion regulation - psychosocial QoL

* $p < .05$, ** $p < .01$, *** $p < .001$

Note: Standardised paths shown are significant.

6.4.3. Study 2b: Childhood non-physical abuse effects on eating pathology, self-esteem, emotion regulation and psychosocial QoL: The mediating role of attachment and temperament

Attachment as a mediator

Measurement Models

Both initial models (patients and controls) have three latent variables (non-physical abuse, attachment, psychosocial QoL) and three observed variables. The latent variable of non-physical abuse has three indicators (emotional abuse, emotional neglect, multidimensional neglect) as measured by the CTQ and MNBS. The latent variable of psychosocial QoL has two indicators (Psychological Health, Social Relationships) as measured by WHOQoL BREF and the latent variable of attachment has two indicators (global avoidance, global anxiety) as measured by the ECR-RS.

ED Patients: The model had a poor fit: $\chi^2(11, N = 80) = 22.300, p = .022, CFI = .96, TLI = .91, RMSEA = .11$. According to measurement model analysis, the deletion of avoidance (attachment) and CTQ emotional neglect as well as the correlation of CTQ emotional abuse with multidimensional neglect and emotional abuse with emotion regulation, would improve the model fit without changing the main theoretical model. As a result, the new measurement model had a good fit: $\chi^2(1, N = 80) = .705, p = .401, CFI = 1, TLI = 1.02, RMSEA = .00$. The latent variables of non-physical abuse and psychosocial QoL were correlated ($r = -.54, p = .014$) and the factors loaded significantly ($p < .001$) with their latents ranging from .65 to .95. The adjusted model includes two latent and four observed variables.

Controls: The model had a poor fit $\chi^2(11, N = 187) = 28.093, p = .003, CFI = .96, TLI = .92, RMSEA = .09$. Analysis of the measurement indicated that the deletion of CTQ

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emotional neglect and anxiety attachment improved fit without changing the theory of the model $\chi^2(1, N = 187) = .003, p = .956, CFI = 1, TLI = 1.04, RMSEA = .00$. The latent variables of non-physical abuse and psychosocial QoL were correlated ($r = -.64, p < .001$) and the factors loaded significantly ($p < .001$) with their latents ranging from .63 to .79. The adjusted model includes two latent and four observed variables.

After the changes in the two models to improve the fit the following difference is noticed between patients and controls: 1) global anxiety was used for patients and global avoidance for controls. This change resulted in two latent variables (non-physical abuse and psychosocial QoL) and four observed variables (attachment, self-esteem, eating pathology, and emotion regulation) for both groups.

Structural Model Analyses

Preliminary Pearson Product moment coefficients for patients and controls are shown in Tables 24 and 26 respectively. Regarding controls, all significant correlations were in the expected direction. That is, increased non-physical abuse was related to greater ED, emotion dysregulation, bad attachment and temperamental difficulties and lower QoL and self-esteem. Higher attachment difficulties and emotion dysregulation were related to lower QoL and higher ED. Low self-esteem was related to higher ED and lower QoL. High temperament was related to lower QoL and higher ED. The decision regarding the direct and indirect paths is the same as in Study 1 (see 5.4.2. Chapter 5). Study 2b had a different aim than Study 2a thus, different SEM models were used. Attachment and temperament were explored in different models instead of a single one in order a) to test Linehan's theory (1993) as a model per se (temperament as a mediator) and then modify it for EDs and b) preserve the models fit and statistical power given the small sample size in relation to the number of variables and the complexity of the models.

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Table 26.

Bivariate Correlations (two-tailed), Means and Standard Deviations for Observed Variables with the Structural Equation Model for the control group (n = 187) – Study 2b.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Negative Affect	-													
2. Effortful Control	-.34 ^{***}	-												
3. Extraversion Surgency	-.31 ^{***}	-.17 [*]	-											
4. Orienting Sensitivity	.15 [*]	-.01	.04	-										
5. DERS Total	.43 ^{***}	-.58 ^{***}	-.04	-.02	-									
6. EDEQ Global	.12	-.14	.03	.02	.27 ^{***}	-								
7. Multi-Dimensional Neglect	.13	-.14	-.28 ^{***}	-.02	.26 ^{***}	.04	-							
8. Self-esteem	-.33 ^{***}	.39 ^{***}	.19 [*]	.09	-.61 ^{***}	-.23 ^{**}	-.33 ^{***}	-						
9. Psychological Health	-.38 ^{***}	.42 ^{***}	.25 ^{***}	-.02	-.59 ^{***}	-.35 ^{***}	-.31 ^{***}	.68 ^{***}	-					
10. Social Relationships	-.32 ^{***}	.23 ^{**}	.25 ^{**}	-.06	-.39 ^{***}	-.20 ^{**}	-.29 ^{***}	.46 ^{***}	.56 ^{***}	-				
11. Global Anxiety	.22 ^{**}	-.20 ^{**}	-.08	-.04	.34 ^{***}	.20 ^{**}	.15 [*]	-.30 ^{***}	-.34 ^{***}	-.41 ^{***}	-			
12. Global Avoidance	.22 ^{**}	-.22 ^{**}	-.35 ^{***}	-.05	.23 ^{**}	.09	-.39 ^{***}	-.28 ^{***}	-.40 ^{***}	-.35 ^{***}	.42 ^{***}	-		
13. Emotional Abuse	.36 ^{***}	-.28 ^{***}	-.29 ^{***}	.19 [*]	.24 ^{**}	.07	.47 ^{***}	-.36 ^{***}	-.37 ^{***}	-.34 ^{***}	.31 ^{***}	.43 ^{***}	-	
14. Emotional Neglect	.14	-.18 [*]	-.33 ^{***}	-.06	.20 ^{**}	.01	.62 ^{***}	-.34 ^{***}	-.35 ^{***}	-.23 ^{**}	.25 ^{**}	.51 ^{***}	.64 ^{***}	-
Mean	4.13	4.37	4.55	4.61	81.26	1.23	11.98	20.06	14.23	14.39	2.02	2.82	8.76	9.09
SD	.65	.73	.72	.74	20.34	.98	3.13	5.24	2.04	3.15	.95	.92	3.82	3.84
Range	1-7	1-7	1-7	1-7	36-180	0-6	8-32	0-30	4-20	4-20	1-7	1-7	5-25	5-25

* $p < .05$, ** $p < .01$, *** $p < .001$

Note: Means and standard deviations reported for the emotional abuse and emotional neglect variables are prior to transformation. The correlation numbers reported for the same variables are after transformation as they were entered transformed in the SEM analysis.

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In the structural model the latent variable of non-physical abuse is the exogenous variable, the observed variable of attachment is the mediator, the latent variable of psychosocial QoL and the observed variables of eating pathology, emotion regulation and self-esteem are the endogenous variables. The hypothesised structural models for patients and controls are shown in Figures 10 and 11 respectively, displaying the path coefficients for the standardized direct effects. The examination of the standardized residuals and modifications indices revealed no high values and the standardized residuals were not systematic indicating that both conceptual models are viable. Standardized direct and indirect effects are reported for both models.

ED Patients: The model had a good fit: $\chi^2(13, N = 80) = 16.675, p = .215, CFI = .99, TLI = .97, RMSEA = .06$. The direct path from non-physical abuse to anxiety attachment was significant ($\beta = .93, p < .001$) and so were the direct paths from: non-physical abuse to psychosocial QoL ($\beta = -2.54, p = .003$), anxiety attachment to psychosocial QoL ($\beta = 2.11, p = .014$), non-physical abuse to eating pathology ($\beta = 1.46, p = .007$), non-physical abuse to self-esteem ($\beta = -2.03, p = .004$), non-physical abuse to emotion regulation ($\beta = 1.71, p = .003$), anxiety attachment to self-esteem ($\beta = 1.48, p = .033$) and anxiety attachment to emotion regulation ($\beta = -1.34, p = .017$). Non-physical abuse had a direct effect on the mediator (attachment) and on all the dependent variables (self-esteem, emotion regulation, QoL, ED). The mediator had a direct effect on all dependent variables except for ED. The results mean that high childhood non-physical abuse makes attachment worse, raises ED and emotion dysregulation, and lowers QoL and self-esteem. However, in contrast to the Pearson's correlation coefficient which was positive for emotion regulation and anxiety attachment and negative for anxiety attachment and self-esteem and QoL, the negative path coefficient suggests that high levels of anxiety attachment lead to low levels of emotion dysregulation and the positive path coefficient suggest that high levels of anxiety attachment

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lead to high levels of self-esteem and QoL. This indicates the presence of a suppressor variable effect i.e., another variable in the model changed the direction of this relationship by suppressing irrelevant variance (Maassen & Bakker, 2001).

The indirect path from non-physical abuse to psychosocial QoL with attachment as mediator (path 1) was significant (standardized indirect effect = 1.96, bias-corrected bootstrap lower CI = .795 and upper CI = 6.385, $p = .002$) and so were the indirect paths from: non-physical abuse to ED with attachment as mediator (path 2) (standardized indirect effect = -.92, bias-corrected bootstrap lower CI = -4.506 and upper CI = -.100, $p = .019$), non-physical abuse to self-esteem with attachment as mediator (path 3) (standardized indirect effect = 1.38, bias-corrected bootstrap lower CI = .518 and upper CI = 5.268, $p = .002$) and non-physical abuse to emotion regulation with attachment as mediator (path 4) (standardized indirect effect = -1.25, bias-corrected bootstrap lower CI = -4.232 and upper CI = -.487, $p = .002$).

The RMediation program was used (Tofighi & MacKinnon, 2011) to further test these mediational effects using the distribution-of-product method. The unstandardized coefficients and standard errors of the a and b paths for each of the four significant indirect effects yielded lower and upper 95% confidence limits of .294 and 2.839 for path 1, .167 and 4.153 for path 3, -.15.994 and -.1.424 for path 4, confirming mediation effects for the three out of four significant indirect paths.

The results are consistent with those of a partial mediation effect given a direct path from non-physical abuse to attachment in the presence of significant indirect effects for emotion regulation, self-esteem and psychosocial QoL. All the explored paths in the model have a post hoc power 1 including the ones that were not significant. This means that attachment partially mediated the relationship between non-physical childhood abuse and self-esteem/emotion regulation/psychosocial QoL but not non-physical abuse with ED.

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Controls: The model had a good fit: $\chi^2(14, N = 187) = 13.510, p = .487, CFI = 1, TLI = 1, RMSEA = .00$. The direct path from non-physical abuse to avoidance attachment was significant ($\beta = .84, p < .001$) and so were the direct paths from: non-physical abuse to psychosocial QoL ($\beta = -1.50, p < .001$), avoidance attachment to psychosocial QoL ($\beta = .79, p = .019$), non-physical abuse to ED ($\beta = .62, p = .002$), non-physical abuse to self-esteem ($\beta = -1.42, p < .001$), non-physical abuse to emotion regulation ($\beta = 1.25, p < .001$), avoidance attachment to ED ($\beta = -.42, p = .023$), avoidance attachment to self-esteem ($\beta = .91, p = .003$) and avoidance attachment to emotion regulation ($\beta = -.82, p < .003$). In other words, high levels of non-physical abuse make attachment worse, they lower QoL and self-esteem and they raise eating pathology and emotion dysregulation. However, in contrast to the Pearson's correlation coefficient which was positive for emotion regulation and eating pathology and negative for self-esteem, the negative path coefficient suggests that high levels of avoidance attachment lead to low levels of emotion dysregulation and eating pathology and the positive path coefficient suggest that high levels of avoidance attachment lead to high levels of self-esteem. This indicates the presence of a suppressor variable effect i.e., another variable in the model changed the direction of this relationship by suppressing irrelevant variance (Maassen & Bakker, 2001).

Notably, non-physical abuse had a direct effect on all dependent variables and so did the mediator. All the indirect effects proposed in the theoretical model were significant: non-physical abuse to QoL with attachment as mediator (path 1) (standardized indirect effect = .66, bias-corrected bootstrap lower CI = .163 and upper CI = 2.242, $p = .001$) also confirmed by RMediation program (Tofighi & MacKinnon, 2011) yielding lower and upper 95% confidence limits of .141 and 1.796, non-physical abuse to emotion regulation with attachment as mediator (path 2) (standardized indirect effect = -.69, bias-corrected bootstrap lower CI = -2.069 and upper CI = -.237, $p = .001$) with lower and upper 95% confidence

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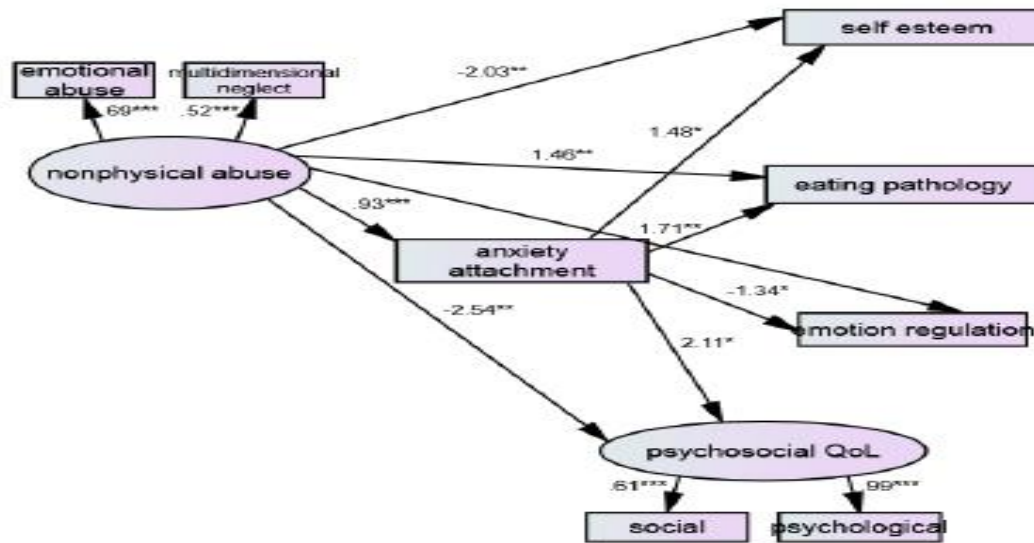
limits of -17.634 and -2.97 in RMediation, non-physical abuse to self-esteem with attachment as a mediator (path 3) (standardized indirect effect = .77, bias-corrected bootstrap lower CI = .283 and upper CI = 2.150, $p = .001$) with lower and upper 95% confidence limits of .865 and 5.045 in RMediation and non-physical abuse to ED (path 4) (standardized indirect effect = -.36, bias-corrected bootstrap lower CI = -1.222 and upper CI = -.026, $p = .029$) with lower and upper 95% confidence limits of -.482 and -.032 in RMediation.

The results are consistent with those of a partial mediation effect given a direct path from non-physical abuse to attachment in the presence of significant indirect effects for all the dependent variables. All the explored paths in the model have a post hoc power 1 including the ones that were not significant. This means that attachment partially mediated the relationship between childhood non-physical abuse and ED/QoL/self-esteem/emotion regulation.

Summary: Regarding the beta values in the common direct relationships between the two groups, higher levels of childhood non-physical abuse had the stronger effect on ED [$(\beta = 1.46, p = .007)$ vs $(\beta = .62, p = .002)$], QoL [$(\beta = -2.54, p = .003)$ vs $(\beta = -1.50, p < .001)$], self-esteem [$(\beta = -2.03, p = .004)$ vs $(\beta = -1.42, p < .001)$] and emotion regulation [$(\beta = 1.71, p = .003)$ vs $(\beta = 1.25, p < .001)$] for ED patients. The indirect effects indicated that attachment was a mediator for all outcome variables except for ED in patients and a mediator for all outcome variables for controls.

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Figure 10. Structural equation model testing the mediational effect of attachment on the relationships between non-physical abuse and eating pathology and non-physical abuse, ED, self-esteem, emotion regulation and psychosocial QoL in ED patients.



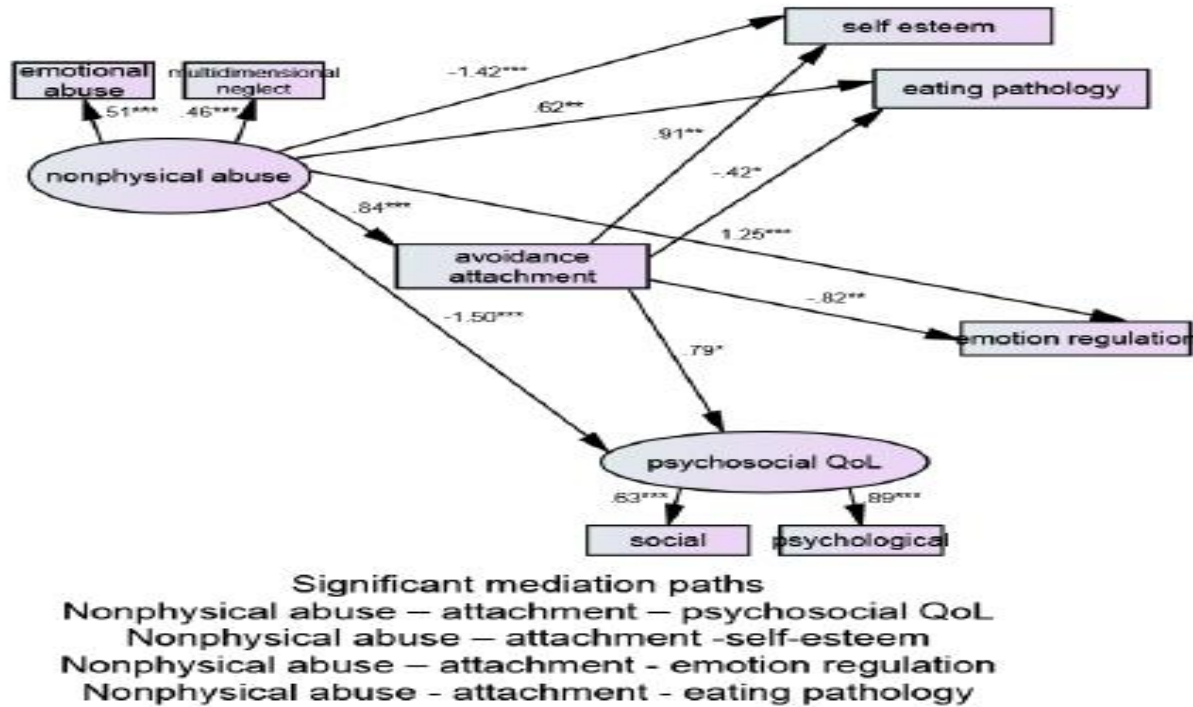
Significant mediation paths
 Nonphysical abuse – attachment – psychosocial QoL
 Nonphysical abuse – attachment – self-esteem
 Nonphysical abuse – attachment – emotion regulation

* $p < .05$, ** $p < .01$, *** $p < .001$

Note: Standardised paths shown are significant.

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Figure 11. Structural equation model testing the mediational effect of attachment on the relationships between non-physical abuse and eating pathology and non-physical abuse, ED, self-esteem, emotion regulation and psychosocial QoL in the control group.



* $p < .05$, ** $p < .01$, *** $p < .001$

Note: Standardised paths shown are significant.

Temperament as a mediator

Measurement models

Both initial models (patients and controls) have three latent variables (non-physical abuse, temperament, psychosocial QoL) and three observed variables (emotion regulation, eating pathology, self-esteem). The latent variable of non-physical abuse has three indicators (emotional abuse, emotional neglect, multidimensional neglect) as measured by the CTQ and MNBS. The latent variable of psychosocial QoL has two indicators (Psychological Health, Social Relationships) as measured by WHOQoL BREF and the latent variable of temperament has four indicators (orienting sensitivity, effortful control, extraversion surgency, negative affect) as measured by the ATQ.

ED Patients: The model had a poor fit $\chi^2(24, N = 80) = 52.570, p = .001, CFI = .89, TLI = .83, RMSEA = .12$. Measurement model analysis indicated that the deletion of CTQ emotional neglect, ATQ extraversion surgency and ATQ effortful control along with the correlation of multidimensional neglect and ATQ negative affect error variances would improve the fit without changing the main theory behind the model $\chi^2(5, N = 80) = 4.586, p = .468, CFI = 1, TLI = 1.01, RMSEA = .00$. The latent variables were correlated: non-physical abuse with psychosocial QoL ($r = -.57, p < .001$), non-physical abuse with temperament ($r = .65, p = .001$) and temperament with psychosocial QoL ($r = -.81, p < .001$). All factors loaded significantly with their latent variables ($p < .001$ to $p = .007$) ranging from .41 to .90. The adjusted model has three latent variables and three observed ones.

Controls: The model had a poor fit $\chi^2(24, N = 187) = 112.079, p < .001, CFI = .81, TLI = .71, RMSEA = .14$. Measurement model analysis indicated that the deletion of CTQ emotional neglect and ATQ extraversion surgency along with the correlation of CTQ emotional abuse and ATQ orienting sensitivity error variances would improve the fit without

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changing the main theoretical model $\chi^2(10, N = 187) = 15.724, p = .108, CFI = .98, TLI = .95, RMSEA = .06$. The latent variables were correlated: non-physical abuse to psychosocial QoL ($r = -.56, p < .001$), non-physical abuse to temperament ($r = .63, p < .001$) and temperament to psychosocial QoL ($r = .77, p < .001$). All factors loaded significantly ($p < .001$) with their latent variables ranging from .14 to .86. The adjusted model has three latent variables and three observed ones.

After the changes in the two models to improve the fit the following differences are noticed between patients and controls: 1) the latent variable of temperament includes orienting sensitivity and negative affect for patients and for the controls it includes the same plus effortful control, 2) there is a direct path drawn from ED to psychosocial QoL and from self-esteem to psychosocial QoL for the controls.

Structural Model Analyses

ED Patients: The model (Figure 12) had a good fit to the data $\chi^2(17, N = 80) = 22.810, p = .156, CFI = .98, TLI = .96, RMSEA = .07$. The direct path from non-physical abuse to temperament was significant ($\beta = .75, p = .003$) and so were the direct paths from: temperament to psychosocial QoL ($\beta = -1.34, p = .001$), temperament to ED ($\beta = .86, p = .001$), temperament to self-esteem ($\beta = -1.01, p = .001$) and temperament to emotion regulation ($\beta = .76, p < .001$). In other words, high levels of abuse raise temperament and then high temperament lowers QoL and self-esteem and raises emotion dysregulation and eating pathology.

It is interesting to note that non-physical abuse (independent variable) had a direct effect on temperament (mediator) and temperament had a direct effect on all dependent variables. The independent variable had no direct effect on the dependent variables and it affected them only through mediation. All the indirect effects proposed in the theoretical

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model were significant: non-physical abuse to psychosocial QoL with temperament as a mediator (path 1) was significant (standardized indirect effect = -.1, bias-corrected bootstrap lower CI = -7.331 and upper CI = -.275, $p = .021$) also confirmed by RMediation program (Tofighi & MacKinnon, 2011) yielding lower and upper 95% confidence limits of -.98 and -.113, non-physical abuse to ED with temperament as mediator (path 2) (standardized indirect effect = .64, bias-corrected bootstrap lower CI = .282 and upper CI = 4.147, $p = .009$) with lower and upper 95% confidence limits of .031 and .264 in RMediation, non-physical abuse to self-esteem with temperament as mediator (path 3) (standardized indirect effect = -.75, bias-corrected bootstrap lower CI = -5.747 and upper CI = -.243, $p = .014$) with lower and upper 95% confidence limits of -1.541 and -.175 in RMediation and non-physical abuse to emotion regulation with temperament as mediator (path 4) (standardized indirect effect = .57, bias-corrected bootstrap lower CI = .193 and upper CI = 3.323, $p = .016$) with lower and upper 95% confidence limits of .669 and 5.059 in RMediation.

The results are consistent with those of a partial mediation effect given a direct path from non-physical abuse to temperament in the presence of significant indirect effects for all the dependent variables. All the explored paths in the model have a post hoc power 1. This means that temperament mediated the relationship between childhood non-physical abuse and ED/self-esteem/QoL/emotion regulation.

Controls: The model (Figure 13) had a good fit to the data $\chi^2(26, N = 187) = 38.904$, $p = .050$, CFI = .98, TLI = .96, RMSEA = .05. The direct path from non-physical abuse to temperament was significant ($\beta = .61$, $p < .001$) and so were the direct paths from: temperament to ED ($\beta = .35$, $p = .002$), temperament to self-esteem ($\beta = -.65$, $p < .001$), temperament to psychosocial QoL ($\beta = -.36$, $p < .001$), temperament to emotion regulation ($\beta = 1.11$, $p < .001$), non-physical abuse to emotion regulation ($\beta = -.35$, $p = .034$), self-esteem to

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psychosocial QoL ($\beta = .41, p < .001$) and ED to psychosocial QoL ($\beta = -.18, p = .001$). The two last direct paths were not part of the theoretical model but all possible relationships were explored with respect to the model data fit. The independent variable had a direct effect on the mediator and the mediator had a direct effect on all dependent variables. The independent variable had a direct effect only on emotion regulation affecting the other three dependents only through mediation. In other words, high levels of abuse raise temperament and then high temperament lowers QoL and self-esteem and raises emotion dysregulation and eating pathology. In addition, low self-esteem diminishes QoL and high levels of ED lower QoL.

However, in contrast to the Pearson's correlation coefficient which was positive for emotion regulation and non-physical abuse, the negative path coefficient suggests that high levels of non-physical abuse lead to low levels of emotion dysregulation. This indicates the presence of a suppressor variable effect i.e., another variable in the model changed the direction of this relationship by suppressing irrelevant variance (Maassen & Bakker, 2001).

All the indirect effects proposed in the theoretical model were significant: non-physical abuse to ED with temperament as mediator (path 1) was significant (standardized indirect effect = .21, bias-corrected bootstrap lower CI = .095 and upper CI = .551, $p = .001$) also confirmed by RMediation program (Tofighi & MacKinnon, 2011) yielding lower and upper 95% confidence limits of .036 and .22, non-physical abuse to self-esteem with temperament as mediator (path 2) (standardized indirect effect = -.40, bias-corrected bootstrap lower CI = -.786 and upper CI = -.228, $p = .001$) with lower and upper 95% confidence limits of -1.946 and -.527 in RMediation, non-physical abuse to psychosocial QoL with temperament as mediator (path 3) (standardized indirect effect = -.42, bias-corrected bootstrap lower CI = -.683 and upper CI = -.290, $p < .001$) with lower and upper 95% confidence limits of -.415 and -.073 in RMediation and non-physical abuse to emotion

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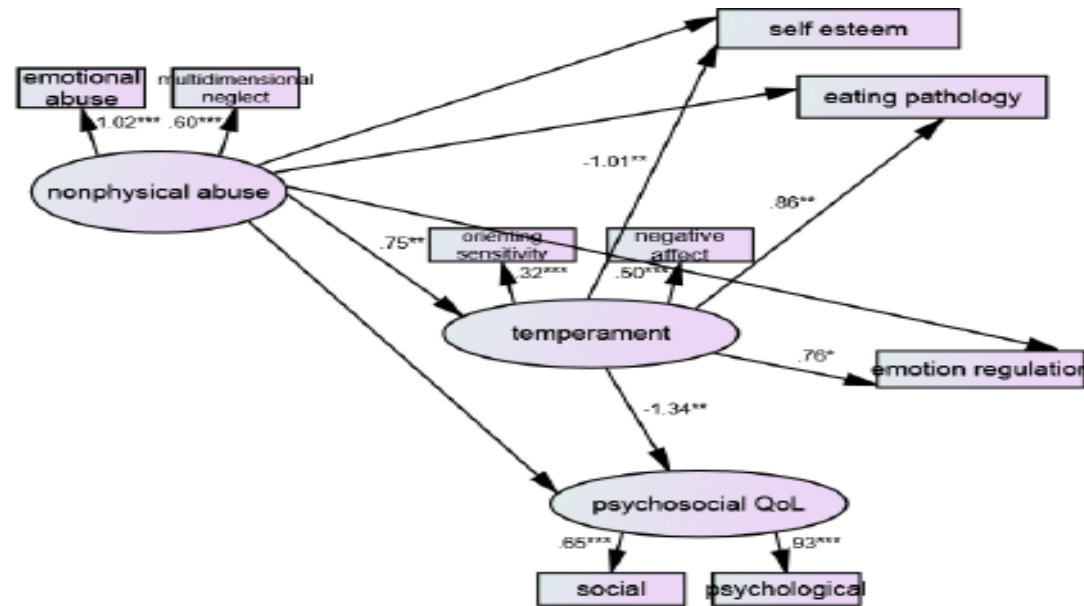
regulation with temperament as mediator (path 4) (standardized indirect effect =.68, bias-corrected bootstrap lower CI =.391 and upper CI = 1.625, $p < .001$) with lower and upper 95% confidence limits of 3.464 and 12.945 in RMediation.

The results are consistent with those of a partial mediation effect given a direct path from non-physical abuse to temperament in the presence of significant indirect effects for all the dependent variables. All the explored paths in the model have a post hoc power ranging from .96 to 1. This means that temperament mediated the relationship between childhood non-physical abuse and ED/self-esteem/QoL/emotion regulation.

Summary: Regarding the beta values in the common direct relationships between the two groups, childhood non-physical abuse had a stronger effect on temperament for the patients [$(\beta = .75, p = .003)$ vs $(\beta = .61, p < .001)$], temperament had a stronger effect on QoL [$(\beta = -1.34, p = .001)$ vs $(\beta = -.36, p < .001)$], ED [$(\beta = .86, p = .001)$ vs $(\beta = .35, p = .002)$] and self-esteem [$(\beta = -1.01, p = .001)$ vs $(\beta = -.65, p < .001)$] for the patients but temperament's effect on emotion regulation was stronger for the controls [$(\beta = 1.11, p < .001)$ vs $(\beta = .76, p < .001)$]. The indirect effects suggest that temperament mediated all proposed relationships in both groups.

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Figure 12. Structural equation model testing the mediational effect of temperament on the relationships between non-physical abuse and eating pathology and non-physical abuse, ED, self-esteem, emotion regulation and psychosocial QoL in ED patients.



Significant mediation paths

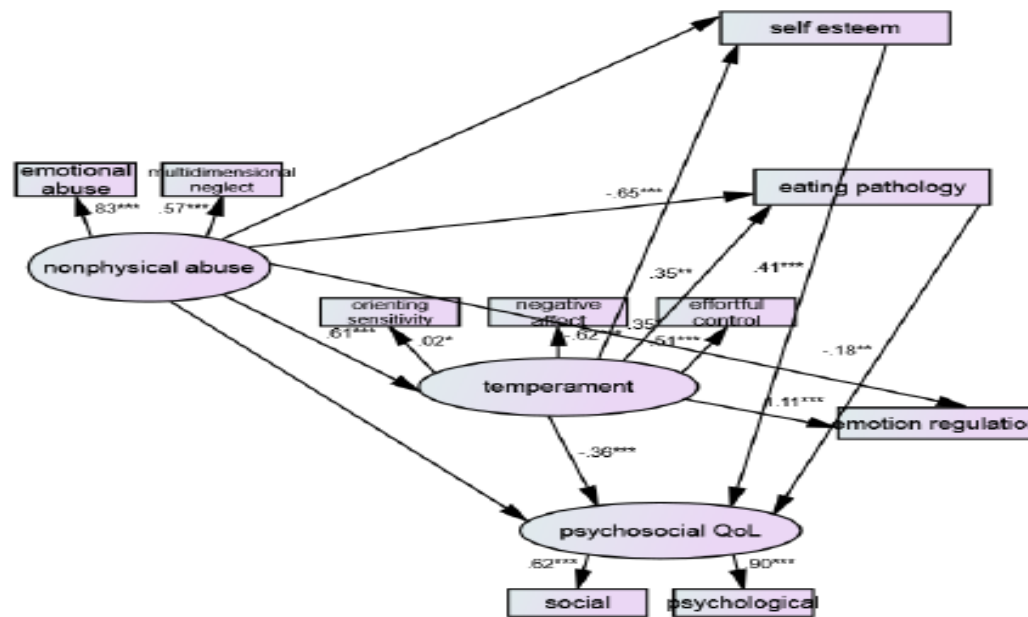
- Nonphysical abuse – temperament – psychosocial QoL
- Nonphysical abuse – temperament – self-esteem
- Nonphysical abuse – temperament – emotion regulation
- Nonphysical abuse – temperament – eating pathology

* $p < .05$, ** $p < .01$, *** $p < .001$

Note: Standardised paths shown are significant.

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Figure 13. Structural equation model testing the mediational effect of temperament on the relationships between non-physical abuse and eating pathology and non-physical abuse, ED, self-esteem, emotion regulation and psychosocial QoL in the control group.



Significant mediation paths
 Nonphysical abuse – temperament – psychosocial QoL
 Nonphysical abuse – temperament – self-esteem
 Nonphysical abuse – temperament – emotion regulation
 Nonphysical abuse – temperament – eating pathology

* $p < .05$, ** $p < .01$, *** $p < .001$

Note: Standardised paths shown are significant.

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Further exploratory analyses: Further exploratory analyses were employed to check for differences in the mean scores of the core variables used in this study both between the ED subtypes, and between the ED patients and the controls, following the reasoning of Study 1 (see Section 5.4 in Chapter 5). For all the analyses, the alpha level was set at .05.

ED subtypes: One-way analysis of variance (ANOVA) was used to determine differences between the ED subtypes with respect to their mean scores for self-esteem, temperament, emotion regulation, attachment, non-physical childhood abuse and psychosocial QoL. The assumptions for this analysis were met. Additionally, and based on this study's demographic information, illness and psychotherapy duration were also considered among the ED subtypes to see if the results were affected by chronicity and treatment resistance, as these numbers in the demographics were high. Prior to the analyses, the data for each group were checked for normality, outliers and the assumptions of the following tests. For economy of space, the results are presented in tables.

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Table 27.

Means, Standard Deviations and One-Way Analyses of Variance (ANOVA) in Self-Esteem, Emotion Regulation, Psychosocial QoL, Attachment, Childhood Non-physical Abuse and Temperament among the ED Subtypes – Study 2.

Measure	AN		BN		BED		<i>F</i> (2,77)	<i>p</i>	ω^2
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>			
Self-esteem	12.78	5.55	13.84	6.60	14.47	5.01	.48	.624	.01
Emotion regulation	105.00	27.32	105.09	25.04	114.00	25.58	1.13	.329	.00
Psychological health (QoL)	10.22	3.19	10.54	3.20	11.11	2.63	.55	.577	.01
Social relationships (QoL)	11.93	3.87	11.83	4.05	11.42	3.26	.14	.872	.02
Avoidance attachment	3.46	.97	3.28	.88	3.27	.98	.28	.755	.02
Anxiety attachment	3.34	1.74	3.28	1.71	3.03	1.63	.26	.774	.02
Emotional abuse	12.78	5.78	13.31	5.55	13.07	5.78	.05	.950	.02
Emotional neglect	11.06	4.60	12.44	5.66	12.37	5.08	.47	.629	.01
Multidimensional neglect	14.67	4.35	14.25	4.15	15.23	4.86	.38	.688	.02
Negative affect	4.36	.82	4.64	.78	4.71	.77	1.15	.322	.00
Orienting sensitivity	4.81	1.04	4.76	.95	4.94	.78	.30	.739	.02
Effortful control	4.42	.83	4.06	.83	3.83	.67	3.20*	.046	.05
Extraversion surgency	4.08	.94	3.97	.66	4.06	.68	.16	.852	.02

*** $p < .001$; ** $p < .01$; * $p < .05$

Note: AN: $n = 18$, BN: $n = 32$, BED: $n = 30$

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The results indicated that there was only a statistically significant difference in the temperamental characteristic of effortful control: $F(2, 77) = 3.20, p = .046, \omega^2 = .05$. Approximately 5% of the total variance of effortful control is due to the ED subtypes. Tukey's honest significant difference (HSD) post-hoc test revealed that AN ($M = 4.42, SD = .83$) and BED ($M = 3.83, SD = .67$) differed, with a mean difference of .59, $p = .035$. This means that in this sample, AN patients, who scored higher than BED patients for effortful control, can control their emotions and behaviour better than BED patients. There was no significant difference on any other variable between the ED subtypes.

ED patients and Controls: One-way ANOVA was used to determine differences between the ED patients and the controls with respect to their mean scores for self-esteem, temperament, emotion regulation, attachment, non-physical childhood abuse and psychosocial QoL. The assumptions for this analysis were met for certain dependent variables, but for others, there was violation of the homogeneity of variance assumption. For these cases, the Welch F is reported.

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Table 28.

Means, Standard Deviations and One-Way Analyses of Variance (ANOVA) in Self-Esteem, Emotion Regulation, Psychosocial QoL, Attachment, Childhood Non-physical Abuse and Temperament between ED patients and the Controls – Study 2.

Measure	ED patients		Controls		$F(1,266)$	ω^2	Welch $F(df)$	est. ω^2	p
	M	SD	M	SD					
Self-esteem	13.84	5.77	20.06	5.23	74.61***	.22			.000
Emotion regulation	108.41	25.80	81.16	20.33			70.59*** (1,122.618)	.21	.000
Psychological health (QoL)	10.68	2.98	14.24	2.03			95.07*** (1,111.569)	.26	.000
Social relationships (QoL)	11.70	3.69	14.39	3.15			32.52*** (1,130.254)	.11	.000
Avoidance attachment	3.32	.93	2.82	.92	16.21***	.05			.000
Anxiety attachment	3.20	1.67	2.03	.96			34.25*** (1,101.74)	.11	.000
Emotional abuse	13.10	5.62	8.81	3.87			38.85*** (1,112.176)	.12	.000
Emotional neglect	12.10	5.18	9.11	3.84			21.63*** (1,117.467)	.07	.000
Multidimensional neglect	14.71	4.44	11.98	3.12			25.07*** (1,113.622)	.08	.000
Negative affect	4.60	.79	4.12	.65			22.96*** (1,126.964)	.08	.000
Orienting sensitivity	4.84	.91	4.61	.74			4.02** (1,125.846)	.01	.000
Effortful control	4.06	.80	4.38	.74	10.23**	.03			.002
Extraversion surgency	4.03	.73	4.56	.73	29.51***	.10			.000

*** $p < .001$; ** $p < .01$; * $p < .05$

Note: ED patients: $n = 80$, controls: $n = 188$

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There were statistically significant differences in all the dependent variables' mean scores between ED patients and the controls. In other words, having an ED diagnosis of any subtype resulted in lower self-esteem and psychosocial QoL, higher childhood non-physical abuse and emotion dysregulation, worse attachment and more difficulties in temperament than healthy controls. For example, there was a statistically significant difference in self-esteem: $F(1, 266) = 74.61, p < .001, \omega^2 = .22$. Approximately 22% of the total variance for self-esteem is due to the groups: having an ED diagnosis of any subtype resulted in lower self-esteem than healthy controls. This means that the similarity of ED patients and controls in the SEM results is not due to their means being similar, as they are not.

ED subtypes after controlling for duration of illness: One-way ANCOVA between subjects was conducted to determine statistically significant differences between AN, BN and BED patients regarding self-esteem, childhood non-physical abuse, emotion regulation, attachment, temperament and psychosocial QoL (psychological health, social relationships), controlling for the effect of illness duration. The assumptions for this analysis were met.

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Table 29.

Observed and Adjusted Means and Standard Deviations of Self-Esteem, Psychosocial QoL, Emotion Regulation, Temperament, Attachment and Non-physical Abuse among the ED Subtypes after Controlling for Illness Duration – Study 2.

Measure	AN		BN		BED		AN		BN		BED	
	<i>M</i>	<i>SD</i>	Observed means		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	Adjusted means		<i>M</i>	<i>SD</i>
Self-esteem	12.78	5.55	13.84	6.60	14.47	5.01	12.76	1.44	13.83	1.05	14.49	1.15
Emotion regulation	105.00	27.32	105.09	25.04	114.00	25.58	103.93	6.36	104.64	4.64	115.13	5.09
Psychological health (QoL)	10.22	3.19	10.54	3.20	11.11	2.63	10.32	.74	10.58	.54	11.01	.59
Social relationships (QoL)	11.93	3.87	11.83	4.05	11.42	3.26	11.78	.92	11.77	.67	11.58	.74
Avoidance Attachment	3.46	.97	3.28	.88	3.27	.98	3.46	.23	3.28	.17	3.27	.19
Anxiety Attachment	3.34	1.74	3.28	1.71	3.03	1.63	3.31	.42	3.27	.31	3.06	.34
Emotional Abuse	12.78	5.78	13.31	5.55	13.07	5.78	13.12	1.40	13.46	1.02	12.71	1.12
Emotional Neglect	11.06	4.60	12.44	5.66	12.37	5.08	11.60	1.27	12.67	.93	11.79	1.02
Multidimensional neglect	14.67	4.35	14.25	4.15	15.23	4.86	15.05	1.10	14.41	.80	14.83	.88
Negative affect	4.36	.82	4.64	.78	4.71	.77	4.40	.19	4.65	.14	4.67	.16
Orienting sensitivity	4.81	1.04	4.76	.95	4.94	.80	4.85	.23	4.78	.17	4.90	.18
Effortful control	4.42	.83	4.06	.83	3.83	.67	4.38	.19	4.05	.14	3.87	.15
Extraversion surgency	4.08	.94	3.97	.66	4.06	.68	3.93	.17	3.91	.13	4.22	.14

Note: AN: n = 18, BN: n = 32, BED: n =30; illness duration considered in the ANCOVA was 12.13 years.

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Table 30.

ANCOVA Results Controlling for Illness Duration – Study 2.

Measure	<i>F</i> (2,76)	<i>p</i>	<i>partial</i> η^2
Self-esteem	.41	.668	.01
Emotion regulation	1.29	.282	.03
Psychological health (QoL)	.26	.772	.01
Social relationships (QoL)	.02	.980	.00
Avoidance attachment	.24	.785	.01
Anxiety attachment	.13	.883	.00
Emotional abuse	.12	.891	.00
Emotional neglect	.33	.724	.01
Multidimensional neglect	.13	.875	.00
Negative affect	.69	.507	.02
Orienting sensitivity	.10	.901	.00
Effortful control	1.98	.145	.05
Extraversion surgency	1.38	.257	.04

*** $p < .001$; ** $p < .01$; * $p < .05$

There was no significant difference between the ED subtypes regarding any of the dependent variables after controlling for illness duration. For example, there was no significant difference between the ED subtypes for self-esteem after controlling for illness duration: $F(2,76) = .41$, $p = .668$, *partial* $\eta^2 = .01$. Illness duration was not significantly related to self-esteem: $F(1,76) = .00$, $p = .955$, *partial* $\eta^2 = .00$, as it was not significantly related to any other dependent variable.

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High and low chronicity: One-way ANOVA between subjects was conducted to determine whether there was a statistically significant difference between the high and low chronicity groups regarding self-esteem, non-physical abuse, emotion regulation, attachment, temperament and psychosocial QoL (psychological health, social relationships). Duration of illness was conceptualised as high and low chronicity in the patient group using the median. This further analysis was conducted in order to overcome the limitations of ANCOVA (Miller & Chapman, 2001). Assumptions for the analysis were met, except for negative affect, which did not display homogeneity of variance; thus, Welch F is reported for this variable.

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Table 31.

Means, Standard Deviations and One-Way Analyses of Variance (ANOVA) for Self-Esteem, Emotion Regulation, Psychosocial QoL, Attachment, Childhood Non-physical Abuse and Temperament between high and low chronicity groups – Study 2.

Measure	High chronicity		Low chronicity		F(1,78)	p	Welch F(df)	est. ω^2	ω^2
	M	SD	M	SD					
Self-esteem	13.83	5.91	13.84	5.70	.00	.990			.01
Emotion regulation	109.78	24.30	106.97	27.54	.23	.630			.01
Psychological health (QoL)	10.63	3.01	10.74	2.99	.02	.881			.01
Social relationships (QoL)	11.25	3.74	12.17	3.62	1.25	.268			.00
Avoidance attachment	3.29	.96	3.34	.91	.07	.789			.01
Anxiety attachment	2.97	1.76	3.44	1.57	1.59	.211			.01
Emotional abuse	13.63	5.87	12.54	5.36	.76	.387			.00
Emotional neglect	12.98	4.79	11.18	5.48	2.44	.122			.02
Multidimensional neglect	15.22	4.48	14.18	4.39	1.10	.298			.00
Negative affect	4.69	.87	4.51	.69		.301	1.08 (1, 75.352)	.00	
Orienting sensitivity	4.90	.83	4.77	.99	.39	.534			.01
Effortful control	3.92	.85	4.20	.73	2.42	.124			.02
Extraversion surgency	3.88	.65	4.18	.79	3.48	.066			.03

*** $p < .001$; ** $p < .01$; * $p < .05$

Note: high chronicity: n = 41, low chronicity: n = 39; total n = 80 ED patients

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The results indicate that high and low chronicity groups did not display significant difference in self-esteem $F(1,78) = .00, p = .990, \omega^2 = .01$ and they did not have a significant difference in any other variable.

ED subtypes after controlling for psychotherapy duration: One-way ANCOVA between subjects was conducted to determine whether there was a statistically significant difference in the scores for self-esteem, childhood non-physical abuse, emotion regulation, attachment, temperament and psychosocial QoL (psychological health, social relationships) between AN, BN and BED patients, controlling for the effect of psychotherapy duration. The assumptions for this analysis were met.

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Table 32.

Observed and Adjusted Means and Standard Deviations for Self-Esteem, Psychosocial QoL, Emotion Regulation, Temperament, Attachment and Non-physical Abuse among the ED Subtypes after Controlling for Psychotherapy Duration – Study 2.

Measure	AN		BN		BED		AN		BN		BED	
	<i>M</i>	<i>SD</i>	Observed means		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	Adjusted means		<i>M</i>	<i>SD</i>
Self-esteem	12.78	5.55	13.84	6.60	14.47	5.01	12.49	1.38	13.80	1.02	14.69	1.07
Emotion regulation	105.00	27.32	105.09	25.04	114.00	25.58	105.83	6.15	105.23	4.56	113.35	4.76
Psychological health (QoL)	10.22	3.19	10.54	3.20	11.11	2.63	10.17	.72	10.53	.53	11.15	.56
Social relationships (QoL)	11.93	3.87	11.83	4.05	11.42	3.26	11.75	.88	11.80	.66	11.56	.69
Avoidance Attachment	3.46	.97	3.28	.88	3.27	.98	3.51	.22	3.29	.16	3.23	.17
Anxiety Attachment	3.34	1.74	3.28	1.71	3.03	1.63	3.42	.40	3.29	.30	2.97	.31
Emotional Abuse	12.78	5.78	13.31	5.55	13.07	5.78	13.26	1.31	13.40	.97	12.67	1.02
Emotional Neglect	11.06	4.60	12.44	5.66	12.37	5.08	11.49	1.21	12.51	.90	12.03	.94
Multidimensional neglect	14.67	4.35	14.25	4.15	15.23	4.86	15.06	1.03	14.32	.77	14.93	.80
Negative affect	4.36	.82	4.64	.78	4.71	.77	4.37	.19	4.64	.14	4.70	.15
Orienting sensitivity	4.81	1.04	4.76	.95	4.94	.80	4.83	.22	4.76	.16	4.92	.17
Effortful control	4.42	.83	4.06	.83	3.83	.67	4.36	.18	4.06	.14	3.87	.14
Extraversion surgency	4.08	.94	3.97	.66	4.06	.68	4.03	.17	3.96	.13	4.09	.13

Note: AN: n = 18, BN: n = 32, BED: n =30; psychotherapy duration considered in ANCOVA was 21.28 months.

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Table 33.

ANCOVA Results Controlling for Psychotherapy Duration – Study 2.

Measure	<i>F</i> (2,76)	<i>p</i>	<i>Partial η</i> ²
Self-esteem	.78	.460	.02
Emotion regulation	.85	.430	.02
Psychological health (QoL)	.63	.538	.02
Social relationships (QoL)	.03	.967	.00
Avoidance attachment	.53	.589	.01
Anxiety attachment	.46	.631	.01
Emotional abuse	.13	.875	.00
Emotional neglect	.24	.789	.01
Multidimensional neglect	.23	.796	.01
Negative affect	.98	.380	.03
Orienting sensitivity	.22	.800	.01
Effortful control	2.20	.118	.06
Extraversion surgency	.24	.784	.01

*** *p* <.001; ** *p* <.01; * *p* <.05

There was no significant difference between the ED subtypes regarding any of the dependent variables after controlling for psychotherapy duration. For example, there was no significant difference between the ED subtypes regarding self-esteem: $F(2,76) = .78$, $p = .460$, $partial \eta^2 = .02$, after controlling for psychotherapy duration, as there was no significant difference regarding any other dependent variable. Psychotherapy duration was not significantly related to self-esteem: $F(1,76) = 1.96$, $p = .166$, $partial \eta^2 = .03$, as it was not significantly related to any other dependent variable.

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Short-term and long-term psychotherapy: One-way ANOVA between subjects was conducted to determine the presence of a statistically significant difference between short- and long-term psychotherapy groups regarding self-esteem, childhood non-physical abuse, emotion regulation, attachment, temperament and psychosocial QoL (psychological health, social relationships). The categorical variable was created based on the literature (Knekt et al., 2016), defining long-term as over six months and short-term as less than six months. This further analysis was performed in order to overcome the ANCOVA limitations (Miller & Chapman, 2001). The assumptions for the analysis were met.

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Table 34.

Means, Standard Deviations and One-Way Analyses of Variance (ANOVA) for Self-Esteem, Childhood non-physical Abuse, Emotion Regulation, Attachment, Temperament and Psychosocial QoL between Short and Long-term Psychotherapy groups of ED patients – Study 2.

Measure	Long-term therapy		Short-term therapy		$F(1,78)$	p	ω^2
	M	SD	M	SD			
Self-esteem	13.11	6.32	14.72	4.97	1.55	.217	.01
Emotion regulation	112.59	26.34	103.31	24.53	2.62	.110	.02
Emotional abuse	14.45	5.70	11.44	5.11	6.05*	.016	.06
Emotional neglect	13.93	4.70	9.86	4.92	14.26***	.000	.14
Multidimensional neglect	15.66	4.39	13.56	4.28	4.65*	.034	.04
Avoidance attachment	3.48	.85	3.11	.99	3.27	.075	.03
Anxiety attachment	3.44	1.72	3.02	1.62	.74	.392	.00
Psychological health (QoL)	10.29	2.92	11.17	3.03	1.74	.191	.01
Social relationships (QoL)	10.82	3.68	12.78	3.44	5.95*	.017	.06
Negative affect	4.70	.86	4.48	.69	1.46	.230	.01
Orienting sensitivity	5.01	.82	4.62	.97	3.82	.054	.03
Effortful control	3.91	.85	4.24	.70	3.58	.062	.03
Extraversion surgency	3.90	.68	4.19	.76	3.26	.075	.03

*** $p < .001$; ** $p < .01$; * $p < .05$

Note: long-term therapy: $n = 44$, short-term therapy: $n = 36$; total $n = 80$ ED patients

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There was a significant difference between the long-term therapy and short-term therapy groups regarding emotional abuse: $F(1,78) = 6.05, p = .016, \omega^2 = .06$; emotional neglect: $F(1,78) = 14.26, p < .001, \omega^2 = .14$; multidimensional neglect: $F(1,78) = 4.65, p = .034, \omega^2 = .04$ and in social relationships: QoL $F(1,78) = 5.94, p = .017, \omega^2 = .06$. This means that patients in the long-term psychotherapy group displayed higher levels of childhood non-physical abuse and lower QoL in the social relationships domain compared to patients in the short-term psychotherapy group. There was no significant difference between the two groups regarding self-esteem, temperament, emotion regulation, attachment and psychological QoL.

6.5. Discussion

Although there are other studies exploring the relationships between this current study's constructs, there is no study incorporating all these variables together in one theoretical model to explain the role of non-physical abuse in EDs (Kimber et al., 2017; Vajda & Láng, 2014; Waller et al., 2007).

In Study 2a, family dysfunction was conceptualised as childhood non-physical abuse to explore its effects on eating pathology and psychosocial QoL, with self-esteem, attachment and emotion regulation as mediators. The results showed that self-esteem is an important correlate of EDs in patients since it was the only variable that mediated the relationship between non-physical abuse and both EDs and psychosocial QoL. However, in the control group, self-esteem only mediated psychosocial QoL. It could be argued that self-esteem is most affected by non-physical abuse, since non-physical abuse did not directly affect EDs in the patient group, but only in the presence of self-esteem as mediator (but not in the presence of attachment and emotion regulation as mediators). In the theory that views EDs as self-damaging behaviour (Farber, 2008), the current findings support the notion that the pathway between childhood abuse and EDs is mediated by self-esteem. Therefore, EDs may reflect the physical manifestation of the person's negative self-representations (e.g. low self-esteem), which developed early in life because of childhood maltreatment (Claes & Vandereycken, 2007; Low et al., 2000; Yates, 2004). Additionally, this finding supports other studies' suggestions that low self-esteem is more responsible than emotion dysregulation regarding ED development (Monell et al., 2015). It also validates other studies regarding the long-lasting effects of the early childhood environment on self-esteem in adulthood (Orth, 2018). The fact that attachment was not a mediator supports the argument that it is not sufficient to

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explain EDs but, rather, is responsible for the impairment of ED risk factors, such as self-esteem and emotion regulation (Cortés-García et al., 2019). This may indicate that attachment's impact on disrupted self-development takes the form of low self-esteem, which appears to be the major risk factor for EDs. The importance of the self in AN has already been discussed in the literature (Amianto et al., 2016; Williams et al., 2016), but the current study presents its transdiagnostic importance in eating pathology through an empirically tested developmental model.

For ED patients, psychosocial QoL was also mediated by emotion regulation, indicating that both their social relationships and psychological well-being are affected by how they manage and express their emotions. Attachment did not play a mediating role in this sample. Self-esteem had a stronger direct effect on the patients' QoL than emotion regulation, which further elucidates this psychological construct as important for ED patients. This finding echoes other studies where low self-esteem negatively affected ED patients' QoL (de la Rie et al., 2005).

Childhood non-physical abuse had a direct effect on self-esteem, emotion regulation and attachment in the clinical group, confirming its reported impact on these important developmental factors (Briere et al., 2012; Corstrophine et al., 2007; Finzi-Dottan & Karu, 2006; Hund & Espelage, 2006; Waller, 1998, 2000). Interestingly, only self-esteem mediated the link between childhood non-physical abuse and eating pathology in patients, even though abuse had a bigger direct effect on emotion regulation than self-esteem. In addition, attachment's direct effect on emotion regulation highlights its importance in the development of the self-regulatory process (Ford, 2005). Overall, it could be stated that the proposed theory of non-physical abuse → self-esteem → ED paints a clear picture of the potential

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aetiological pathway to ED development that could be explored in longitudinal studies. This result is in line with Silverstone's (1992) hypothesis that chronic low self-esteem is a necessary prerequisite for ED development and that chronic low self-esteem is the final common pathway through which the multiple aetiological factors involved in the causation of EDs act.

Regarding the controls, only emotion regulation mediated non-physical abuse and EDs. It was the only mediator that had a direct effect on eating pathology, even though non-physical abuse's had a bigger direct effect on attachment and self-esteem than on emotion regulation. Following Cortés-García and colleagues' argument (2019), it could be proposed that attachment's impact on the controls manifested through emotion regulation. Emotional expression serves the purpose of preserving the relationship to the attached person, meaning that emotion regulation and attachment are psychologically connected (Scheidt & Waller, 2002). The model of non-physical abuse → emotion regulation → ED indicates that the way individuals handle their emotions is important for eating pathology, confirming related studies with non-patient samples (Burns et al., 2012; Moulton, 2013; Vajda & Láng, 2014). In line with this result, the literature supports the notion that emotional eating is used by the general population as a means to regulate negative emotions (Goossens et al., 2009; Nguyen-Rodriguez et al., 2009; Norwood et al., 2011). This finding further supports other studies that have established an association between disordered eating, emotional abuse, emotion dysregulation and being female in the general population (Mills et al., 2015). In addition, emotion regulation had a direct effect on self-esteem, whereas attachment did not. However, due to issues with the model's fit, it was not possible to explore self-esteem's direct effect on emotion regulation, which would have helped clarify their relationship. However, it can be

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concluded that self-esteem was not a mediator for the control group because this population had no EDs, not even at a subclinical level. This argument, combined with the finding that self-esteem is the only mediator for EDs in patients, concurs with other studies' findings that concluded that self-esteem is more important in EDs than emotion regulation (Monell et al., 2015; Monell et al., 2020). Chronic childhood maltreatment is an identified cause of emotion dysregulation (Ehring & Quack, 2010; Thompson et al., 2014). As a result, the proposed theory of non-physical abuse → emotion regulation → ED potentially explains eating disturbances in the general population.

All three mediators (self-esteem, emotion regulation, attachment) mediated the relationship between non-physical abuse and psychosocial QoL in the control group, with self-esteem having the strongest effect, thereby indicating its importance in psychosocial adjustment of non-clinical groups as well. Emotion regulation and attachment have been linked to social adjustment (Bowlby, 1969, 1973, 1979; Stifter et al., 2011), with the current results supporting their direct effect on psychosocial QoL. However, self-esteem had a bigger direct effect on QoL than attachment and emotion regulation, further indicating its importance regardless of the presence of psychopathology. The mediating role of attachment also indicates its significance regarding social relationships and psychological states in the general population, confirming Bowlby's (1969) theory that low attachment negatively shapes the child's psychological development, and severely disturbs social and emotional behaviour. In fact, in this sample, attachment was more affected by childhood non-physical abuse, as abuse had a bigger direct effect on attachment than on emotion regulation and self-esteem. Although eating pathology had a direct effect on psychosocial QoL, its effect was smaller than attachment's and self-esteem's. This finding could probably explain why former

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ED patients' report that their QoL is similar to that of current ED patients, despite the symptoms' remission (Bamford and Sly, 2010; Padierna et al., 2002; Pohjolaine et al., 2016; de la Rie et al., 2005). In other words, psychological factors, such as self-esteem, emotion regulation and attachment, when affected, may be more important than the ED symptoms themselves. It is known that these symptoms are the means to alleviate the pain from the impairment of these psychological factors. This is particularly evident from the control group's results, which indicate that it is not the presence of ED symptoms that impacts psychosocial QoL. This means that Study 2a's transdiagnostic theoretical model offers insight regarding the common psychological factors responsible for the onset and maintenance of EDs and how these factors become impaired (childhood non-physical abuse). Overall, the results from both groups could explain why the childhood non-physical abuse → self-esteem → ED pathway is relevant for ED patients, as the disorder has already developed, and their scores indicate low self-esteem and emotion dysregulation. On the contrary, the childhood non-physical abuse → emotion regulation → ED pathway is relevant for the controls in terms of potential eating disturbances, as the control group was ED-free with normal scores for self-esteem and emotion regulation.

Study 2b adapted Linehan's biosocial theory (1993), adding attachment as a mediator, and self-esteem, EDs and QoL as outcome variables, separate from emotion regulation, in order to expand and explore all the possible important theoretical mechanisms and risk factors of eating pathology. For the patient group, attachment mediated all the proposed relationships, except for eating pathology, indicating that the theory of childhood non-physical abuse → attachment → ED is not explanatory for the clinical group. The fact that the theory of childhood non-physical abuse → attachment → self-esteem was significant

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indicates that attachment plays a significant role in the formation of self-esteem, which is a highly important correlate of eating pathology, according to Studies 1 and 2a. Moreover, childhood non-physical abuse had the biggest direct effect on self-esteem, rather than on attachment or emotion regulation. It could be stated that the manifestations of self-damaging behaviour, such as EDs, results from dysfunctional attachment, causing psychological pain to the individual, and this pain is preserved through self-damaging behaviour, hiding the real self to maintain the attachment (Bruch, 1973; Farber, 2008). In accordance with this concept, attachment had a bigger direct effect on self-esteem than on emotion regulation. It could be hypothesised at this point, also given the results of Study 2a, that the path of non-physical abuse → attachment → ED was not significant because self-esteem was missing from the equation. Furthermore, childhood abuse may also generate trauma and lead to emotional reduction behaviour, such as an ED, in order to forget psychological pain (Claes & Vandereycken, 2007). This could explain the significant path of non-physical abuse → attachment → emotion regulation in ED patients. Attachment had a stronger direct effect on psychosocial QoL than on self-esteem, EDs and emotion regulation. Non-physical abuse had a stronger direct effect on QoL compared to attachment. Studies indicate that individuals with high attachment anxiety tend to perceive other people's emotions earlier, and as a result, they respond in a quick and defensive manner, affecting their interpersonal relationships (Fraley et al., 2006), thus, affecting their psychosocial QoL. Farber (2008) also noted that attachment impacts the child's perception on whether they are worthy of being cared for by others. Expanding this argument alongside the current findings, it could be argued that a disturbed attachment affects the child's self-esteem (model of self) and social relationships (model of others), impacting psychosocial QoL in adult life.

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In the control group, all paths were significant, indicating that the consequences of non-physical abuse on attachment have an impact on self-esteem, emotion regulation, eating pathology and psychosocial QoL in adult life. This finding supports the theory that attachment is linked to the formation of self-representations, emotions and social relationships (Mikulincer & Shaver, 2012; Pearlman & Courtois, 2005; Ross & Green, 2011; Thompson, 1998). Authors maintain that emotion regulation is not present at birth but develops throughout life (Dodge & Garber, 1991; Morris et al., 2013), and is mostly affected by attachment (Malekpour, 2007; Sroufe, 2005). Attachment theory claims that emotion regulation is learned through the individual's interaction with attachment figures, and this interaction defines how the individual will cope in the face of distress cues (Cole-Detke & Kobak, 1996). It is believed that CEA in particular is linked with ED development as it serves the function of managing strong emotions through suppression or avoidance (Fox & Power, 2009; Kent et al., 1999; Overton et al., 2005). Rorty and Yager (1996) suggested that the self-destructiveness and persistence of ED symptoms of abused women could be better explained as desperate efforts to regulate negative affect and compose self-coherence. They maintained that psychological trauma leads to the dysregulation of the arousal system to affect suppression. Consequently, self-coherence is damaged, and the world is perceived as threatening, just as in BPD (Jørgensen, 2006; Lafleur, 2013; Linehan, 1993; Nolen-Hoeksema, 2003). Moreover, attachment had a bigger direct effect on self-esteem than on emotion regulation in both the patient and control groups. These results indicate that the inclusion of attachment and self-esteem could benefit the biosocial theory (Linehan, 1993).

Temperament mediated all the proposed relationships both for patients and controls, indicating that the biosocial theory (Linehan, 1993) can be expanded to EDs. This is

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consistent with previous studies that revealed a relationship between the childhood invalidating environment and eating pathology (Haslam et al., 2008; Haslam et al., 2012). Eating pathology's direct effect on the controls' psychosocial QoL underlines the importance of ED prevention. As in Study 2a, EDs' direct effect on psychosocial QoL was smaller than the effects of self-esteem and temperament, with self-esteem having the biggest impact on QoL. This finding again evinces that factors other than ED symptoms are more important for QoL, most notably self-esteem. However, EDs' effect on QoL in comparison to the other factors could only be explored in the control group due to model fit issues in the patient group. Nevertheless, these results regarding the factors affecting QoL provide a plausible explanation as to why QoL is not improved after ED symptoms reduce. This could be explored in larger ED patient samples. The results also highlight the fact that non-physical abuse can affect temperament, which in turn, impacts self-esteem, emotion regulation, ED and psychosocial QoL, supporting the theory that temperament is subject to change affected by family dynamics (Butler, 2010; Fox, 1998; Hong & Park, 2012; Wilson et al., 2000) and can affect human socialisation (Rothbart et al., 1994; Rothbart & Bates, 1998). Study 2b's empirical findings also help us understand the proposed links between temperament and self-esteem (Robins et al., 2010), and temperament and emotion regulation (Posner & Rothbart, 2000; Stifter et al., 2011). Temperament appears to affect developmental factors that are important in human psychology. Simultaneously, temperament is affected by non-physical childhood abuse. Non-physical abuse had a stronger impact on temperament in patients compared to the controls, and temperament had a stronger impact on all the variables in the patient group than in the control group, except for emotion regulation (the effect was bigger in the control group). These findings suggest a link between non-physical abuse and temperament with respect to eating pathology, and they also echo Study 2a's findings in the

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sense that emotion regulation seems to be more important for controls than it is for patients. Attachment had a stronger effect on self-esteem than temperament did in both the patient and control group. This indicates that attachment is the most important factor in the creation of self-esteem, a process that occurs early in life and has strong effects in adulthood, supporting attachment theories. It is important to note that these models were valid for the controls, indicating that the proposed developmental relationships are not due to the presence of psychopathology.

A comparison of the two mediators indicates that childhood non-physical abuse had a stronger direct effect on attachment than on temperament in both groups, signifying abuse's strong impact on this important developmental process. Attachment had a stronger direct effect on self-esteem, emotion regulation and QoL in patients compared to temperament, suggesting that attachment is more important than temperament in the formation of core psychological variables that define human behaviour and subsequently affect their psychosocial QoL. Moreover, this finding further suggests that the inclusion of attachment and self-esteem could benefit the biosocial theory (Linehan, 1993) and that temperament's role could be revisited. In the control group, attachment had a stronger direct effect on QoL, self-esteem and ED compared to temperament, highlighting attachment's developmental role, regardless of the presence of psychopathology. Temperament had a bigger effect on emotion regulation compared to attachment in the controls, demonstrating that in the absence of highly disturbed attachment, emotion regulation is linked with temperamental traits.

In addition, the group comparison analyses indicate the transdiagnostic nature of these factors, as the ED subtypes did not differ in their mean scores for all the variables, except for the temperamental characteristic of effortful control, which varied between AN and BED

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patients. Effortful control has been characterised as a transdiagnostic characteristic of internalising (e.g. mood disorders) and externalising (e.g. substance abuse) psychopathology differentiation (Santens et al., 2020). This could offer some merit in the differentiation between bingeing/purging and restrictive EDs as there is a reported difference between their scores for effortful control (Claes et al., 2010). However, there was no difference between AN and BN patients, and the .046 significance between AN and BED was borderline. When considered alongside all the other non-significant findings, including the other three temperament scales, the results do not provide support for meaningful differences in the ED subgroups. The chronicity of the disorder, along with the duration of therapy, which are expected in EDs and still unexplained (Geller et al., 2001; Keller et al., 1992; Wonderlich et al., 2012), did not affect the results either. The possible impacts of chronicity and psychotherapy duration were checked using both ANCOVA and median split to overcome the limitations of each method (Aiken & West, 1991; Miller & Chapman, 2001). Patients with high and low illness chronicity did not significantly differ in their mean scores for the variables. However, patients who were undergoing or had undergone long-term psychotherapy had significantly higher childhood non-physical abuse scores and lower social relationships QoL compared to the patients who had had or were receiving short-term therapy. This indicates the need to identify and consider childhood non-physical abuse in treatment, addressing the consequences that these types of abuse create in order to avoid treatment resistance, and achieve a positive and quicker therapeutic outcome. Moreover, this finding could also indicate a dose–effect relationship between childhood non-physical abuse and treatment resistance. The fact that there is a difference in social relationships QoL between patients in long- or short-term therapy highlights the instability that non-physical

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abuse creates in their adult interpersonal relationships probably through the impairment of attachment.

The transdiagnostic nature of the adapted biosocial model is as well supported by the fact that the ED subtypes did not display differences across the studies' variables, as the two studies shared the same population. The significant differences among the variables between the patients and the controls indicate their importance regarding prevention and demonstrate that the proposed models have value regardless of the presence of psychopathology. ED chronicity and psychotherapy duration did not seem to play a significant role in the SEM results, but their presences indicates that something maintains the disorder, and this could probably be the non-addressed childhood non-physical abuse and its consequences in adult life.

6.5.1. Clinical, Theoretical and Research Implications

This study's results confirm the need to consider non-physical childhood traumatic experiences stemming from invalidating environments when designing therapy for ED patients (Haslam et al., 2008; Mountford et al., 2007) and prevention for the general population (Smyth et al., 2008). The results also provide a theoretical explanation of how family dysfunction and the invalidating environment (i.e. childhood non-physical abuse) might lead to the development of EDs, mainly through damaging self-esteem. Longitudinal studies would be needed to identify whether the presence of low self-esteem in the early years of life constitutes the sole risk/causal factor for adult EDs. There is also the need to conceptualise eating pathology as a result of non-physical abuse, self-esteem, emotion regulation, attachment and temperament by testing the proposed models longitudinally in order to clarify causality and the sequence of events to help prevention during the early years

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of life. For example, these studies hypothesise that childhood non-physical abuse affects attachment and temperament in the early years of life, which in turn, affect self-esteem and emotion regulation, leading to eating pathology. These factors are transdiagnostic in nature and responsible for both the onset and maintenance of EDs, regardless the subtype.

The clinical implications of Study 1 regarding self-esteem also apply here. Clinicians may find these results useful in addressing the reasons behind ED patients' difficulty with emotion regulation by focusing on self-esteem. Based on reviews of the development of the self, a basic suggestion for improving self-esteem and how to prevent its negative development and associated consequences is to reduce the discrepancy between the real and ideal selves (Harter, 1999). Therapeutic designs should include attachment, self-esteem and emotion-regulation-focused techniques to people reporting non-physical abuse, as these important psychological factors are affected by disturbed primary relationships with significant others, leading to the development of eating pathology and low psychosocial QoL. They should also look for non-physical abuse history as it is often under-reported. Enhancing the patient's understanding and awareness of this possible link could fortify the psychotherapy's outcome and prevent relapse. Prevention programmes in colleges focused on emotion regulation could offer significant value to the outcome, as Study 2's sample comprised mostly college students in whom a link between disordered eating and emotion dysregulation has previously been established (Messman-Moore & Garrigus, 2007). The results of these studies also indicate that including self-esteem in BPD models and treatment could offer therapeutic value, as these factors are common in both disorders; yet, further research is needed to compare the specificity of the models to EDs and BPD. The literature indicates that self-esteem is important for emotion regulation, and self-esteem was an

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outcome variable in Study 2b's revised model. In other words, there is enough evidence to suggest that self-esteem was the missing factor from ED patients' dialectical behaviour therapy (DBT) all along.

Theoretically, the results indicate that existing ED theories (as discussed in Section 2.3.1 in Chapter 2) could be integrated to form a complete and comprehensive model incorporating all the important psychological factors. Such a model could sufficiently explain the formation and maintenance of eating pathology. The findings also indicate that the factors responsible for ED onset and maintenance are transdiagnostic. The current study's results echo Linehan's (1993) concept of the invalidating environment, the SPAARS (Power & Dalgleish, 2008) and the SPAARS-ED (Fox & Power, 2009) models of emotions.

Regarding future research, the findings highlight the need to concentrate research on the role of non-physical abuse in EDs, reconceptualising family dysfunction/the invalidating environment and exploring the effects of self-esteem on the development of emotion regulation (see Section 3.5.3 in Chapter 3).

6.5.2. Limitations and Future Directions

The study is cross-sectional and although the findings support the existence of the proposed mediational relationships, they should be treated with caution because data from cross-sectional studies cannot be used to imply causality (as discussed in Section 5.5 in Chapter 5). However, cross-sectional studies are important for determining whether data are compatible with the proposed models and for strengthening the case for using the model in future prospective designs (Waller et al., 2001).

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Additionally, only White women were sampled; thus, the results cannot be generalised to other racial/ethnic groups or to men, as it has been suggested that there are variations in ED symptoms within and between diverse groups (Assari & DeFreitas, 2018; Franco, 2007; Lydecker & Grilo, 2016).

The childhood trauma questionnaire (CTQ) asks for information based on recalling past events, which can affect autobiographical memory (Hassan, 2005). However, the majority of the participants were in their early/mid 20s, meaning that the recalled events were not too distant (though this will vary based on the individual's age when the abuse occurred). Furthermore, evidence suggests that adults tend to accurately recall experiences that were unique and had a significant effect on them (Brewin et al., 1993). Authors suggest that the anonymity provided by self-report questionnaires encourages honest responses (Andrews, 1997; Hartt & Waller, 2002). Nevertheless, due to the nature of the CTQ questions, underreporting has been reported (Lochner et al., 2010) but is typically lower in patients with ED (MacDonald et al., 2015) possibly due to them being in psychotherapy thus having the chance to bring psychological issues to the surface. In this study the CTQ Minimisation/Denial scale had a mean of .89 ($SD = .08$) for the controls and .49 ($SD = .10$) for patients, suggesting low rates of under-reporting. Moreover, asking ED patients to reflect on and answer questions about their emotions, with respect to the emotion regulation variable, is empirically challenging as they cannot conceptualise their emotional experiences (Lane et al., 1997; Mauss & Robinson, 2009), which can lead to inaccurate responses.

Another limitation is that of the attachment measure. There are differences in attachment classification among studies, and these various attachment measures do not always correlate (Holmes & Johnson, 2009). However, dimensional measures of attachment

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have been found to be more accurate than categorical ones, and continuous attachment models better conceptualise attachment (Fraley et al., 2000; Fraley et al., 2015; Fraley & Spieker, 2003).

The complexity of the proposed theoretical models, along with the difficulties coming from certain questionnaire scales that did not perform well, led to the models' modification in order to achieve adequate fit, a not-uncommon procedure when using SEM (Kline, 2011). However, these modifications addressed the removal of certain scales of latent variables, which did not alter the hypotheses about mediation and the central theoretical principles behind the proposed models. In spite of this limitation, both studies produced significant results in both groups, and all the variables involved in the studies contributed to these significant results. Moreover, SEM considers the measurement of error variance in the models (Kline, 2011).

In addition, both methods used to explore the effects of illness chronicity and psychotherapy duration have limitations. ANCOVA may produce misleading results in non-experimental studies (Miller & Chapman, 2001), and the dichotomisation of a continuous variable could reduce statistical power (Cohen, 1983). However, using both methods offered a more thorough exploration of factors of interest, leading to similar results. There are no official cut-off scores to define chronic illness, and it is usually described as a condition that lasts more than three months (Bernell & Howard, 2016). The clinical sample in this study did not have participants with low chronicity numbers, so the median was used. Regarding psychotherapy duration, the created categorical variable for the analysis was based on the literature, according to which, short-term psychotherapy means six months and long-term psychotherapy refers to three years (Knekt et al., 2016).

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Despite the usual limitations of cross-sectional studies, these models together provide a theory-driven approach to empirically assess the key psychological factors regarding the onset and maintenance of EDs. Moreover, the majority of the relevant studies examined non-clinical populations, whereas this study used ED patients along with a community population, allowing for better understanding and interpretation of the proposed theoretical models. Given the similarity of the results in the ED and control groups in Study 2b, testing these models across severity of ED symptoms, as well as across restrictive and purging EDs, would benefit the literature and enhance understanding of the role of these psychological factors. Moreover, Study 2 should be replicated using a larger clinical population.

6.6. Conclusion

These results clarify the importance of childhood non-physical abuse in EDs, as well as its impact on core psychological factors that affect psychosocial QoL in adult life. They also imply that non-physical abuse could possibly be an aetiological correlate, since it impacts self-esteem, attachment, emotion regulation and temperament in controls as well. These factors are all related to the development of various types of psychopathology, also affecting QoL, offering grounds for replicating the studies. In sum, the results provide a clearer cross-sectional picture of the potential pathway leading to eating pathology, simultaneously clarifying that the most important factor for ED patients is self-esteem (based on the strongest effect size), which, following replication in longitudinal studies, might be identified as a necessary focus of prevention and treatment. Moreover, the findings suggest that the biosocial theory (Linehan, 1993) can be expanded for EDs, and prevention programmes could be advanced.

Chapter 7 – Study 3: The Presence of Non-physical Abuse in Adult Eating Disorder Patients: An Interpretative Phenomenological Analysis

7.1. Abstract

Background: There is no literature exploring whether adult eating disorder (ED) patients who have experienced childhood non-physical abuse continue to endure abuse as adults, and if they do, which psychological factors are affected. These factors could also contribute to the continuation of abuse. The purpose of this study was to explore the lived experiences of ED patients who have suffered non-physical childhood abuse.

Method: Six adult female ED outpatients were recruited, and each took part in a semi-structured interview. This study used the interpretative phenomenological analysis (IPA) framework.

Results: Three master themes and eleven superordinate themes were drawn from the analysis. The master themes that addressed the research question were continuation of non-physical abuse across a lifetime, developmental factors and non-physical abuse, and non-physical abuse and eating pathology.

Conclusion: The participants' accounts indicate that childhood non-physical abuse is related to ED onset, and the abuse's continuation in adulthood contributes to the disorder's maintenance. Moreover, the lifelong consequences of non-physical childhood abuse impact psychological factors, such as self-esteem, attachment and emotion regulation, affecting the individuals' adult lives and keeping participants inside a vicious cycle of trauma re-enactment.

7.2. Introduction

Statistics from the Crime Survey for England and Wales (CSEW) indicate that one in five adults have experienced a form of abuse before the age of 16 years, with more women than men affected (CSEW, 2020). Moreover, it is estimated that 51% of people who suffered childhood abuse will suffer abuse as adults, with abused girls four times more likely to be abused as adults than abused boys (CSEW, 2016). Evidence suggests that abused children experience multiple forms of victimisation during their lives, possibly because they display certain characteristics that make others view them easy targets. These characteristics include submission, in order to preserve safety when coming from a chaotic home environment, as well as feelings of helplessness and low self-confidence, because they have learned that their needs are ignored, so they are probably unimportant (Duncan, 1999, 2004; Finkelhor & Browne, 1985; Koenig et al., 2000). This could lead abused individuals to generalise their cognitions and behaviour in extra-familial relationships across their lifespans (Schwartz et al., 1993; Wolke & Samara, 2004), indicating the long-lasting consequences of an adverse home environment (Copeland et al., 2013). It has been proposed that people who have been psychologically maltreated internalise the expressions of maltreatment and, eventually, accept them as true (Wiehe, 1990). This further indicates that emotional abuse can affect self-esteem, leading to self-blame, perpetuating the feeling of victimisation. Moreover, high betrayal trauma from a caregiver in early life can damage the cognitive mechanisms that enable the acknowledgement of trauma, making people prone to abuse continuation by trying to establish the desired attachment at any cost (DePrince, 2005; Gobi & Freyd, 2009). Expanding this argument, it could be noted that a disrupted attachment and low self-esteem caused by abuse and neglect in childhood could lead to deficient effective coping, adaptation

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and socialising later in life due to vulnerability to harm. Vulnerability to harm is a maladaptive schema defined as an exaggerated fear that imminent catastrophe will strike at any time that one will be unable to prevent (Young et al., 2003).

Empirical evidence indicates that there is a link between non-physical childhood abuse and the vulnerability to harm schema (Hartt & Waller, 2002; O'Dougherty Wright et al., 2009). Studies indicate that students from violent and dysfunctional families are more vulnerable to school bullying victimisation (Craig et al., 1998; Lereya et al., 2013; Mohr, 2006). This could suggest a link between being bullied in childhood and having suffered non-physical childhood abuse, since childhood emotional abuse (CEA) and neglect are present in dysfunctional and hostile families. Maltreatment has also been found to place children at risk of victimisation by their peers, and bullying victims have displayed problems with emotion regulation as a consequence of abuse (Shields & Cicchetti, 2001). In the same study, the effect of maltreatment on children's risk of bullying and victimisation were mediated by emotion dysregulation, and in other studies, low self-esteem was associated with violence and bullying victimisation (Delfabbro et al., 2006; O'Moore & Kirkham, 2001; Rigby & Slee, 1991; Slee & Rigby, 1993). In addition, adult anorexia nervosa (AN) patients reported that being bullied by other children during their childhood added to their pre-existing anger (Fox, 2009). The abovementioned could indicate that childhood non-physical abuse that causes emotion dysregulation makes people susceptible to victimisation as adults due to the vulnerability to harm schema and low self-esteem, and this is an enduring life-long effect. Moreover, low self-esteem and experiences of childhood abuse have been associated with partner violence victimisation and perpetration (Papadakaki et al., 2009), suggesting a continuation link between childhood and adult abuse. In studies comparing the bullying and

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stigmatisation experiences of people with EDs to people with depression, the ED group reported higher levels of being ignored by their peers (Froom, 2007) and stigmatisation (Roehrig & McLean, 2010). Evidence also suggests that there is a correlation between being bullied about weight and appearance as a child and later developing an ED (Lie et al., 2019; Sweetingham & Waller, 2008). Bullying about physical appearance is a form of emotional abuse that could lead to low-self-esteem, body dissatisfaction, the drive for thinness and depression, escalating the chances of ED development (Eisenberg et al., 2003; Hill & Murphy, 2000; Jackson et al., 2000).

Additional empirical results indicate that ED sufferers are stigmatised (Bannatyne & Stapleton, 2018; Crisp, 2005; Froom, 2007; Mond et al., 2006; Roehrig & McLean, 2010; Zwickert & Rieger, 2013), which may indicate the presence of non-physical abuse in adult life. Studies indicate that people negatively differentiate EDs from other mental illnesses because they have the view that ED patients are responsible for the development of the disorder (Crisp, 2005; Mond et al., 2006; Roehrig & McLean, 2010). This could be associated both with the media presentation of the female body, along with the ego-syntonic nature of certain ED features (e.g. dietary restraint). In combination, they make the disordered behaviour look like a behaviour that does not cause discomfort to the patients (Mond et al., 2006; Peroutsi & Gonidakis, 2011) and that the ED patients have control of their illness compared to disorders, such as schizophrenia (Roehrig & McLean, 2010). In this respect, it has been suggested that people in the ED patient's familial and social environment may envy them in the early stages of the disorder for being able to lose weight and maintain a *socially desirable* figure, especially in AN (Mond et al., 2006).

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Waller and associates (2007) in their expansion of O'Hagan's (1995) definition of emotional abuse in order to include adult experiences argued that ED patients report emotional abuse in their adult years, either as primary experiences or as a revival of their childhood experiences. The latter echoes the psychoanalytical concept of *repetition compulsion* coined by Freud (1920, pp. 282–283) to explain that humans seek comfort in the familiar, which he famously defined as 'the desire to return to an earlier state of things'. Repetition compulsion posits that the individual who has experienced trauma seeks symbolic repetition in order to master the trauma (van der Kolk, 1989; Winnikott, 1965).

Existing qualitative studies have focused predominantly on ED patients' experiences regarding their symptoms and treatment (e.g. Fox & Diab, 2015; Fox et al., 2011; Fox & Whittlesea, 2017; Hoskins et al., 2019; Lazare et al., 2019; McKenna et al., 2014). In addition, reviews of the abuse literature indicate that the psychological effects of sexual abuse survivors are emphasised (MacGinley et al., 2019). The ED literature is missing evidence indicating whether ED patients who have suffered non-physical childhood abuse experience this kind of abuse as adults and, if so, explaining the psychological mechanism behind this phenomenon. Given the results from Studies 1 and 2, and the fact that adult ED patients regularly report non-physical abuse in their psychotherapy, the purpose of this study was to explore the lived experiences of ED patients who have experienced non-physical childhood abuse and who are still victims in adult life and identify the potential psychological factors behind this continuing phenomenon. Since this has not been researched before, it will be a pioneer study. The research question is therefore: what are the lived experiences of adult ED patients in relation to continuing non-physical abuse?

7.3. Method

7.3.1 Study Design

In order to address the gap in the current literature regarding ED patients' continuing non-physical abuse, this research focuses on identifying themes within the participants' own experiences and understanding of non-physical abuse through a qualitative study. IPA (Smith et al., 2009) was used to explore the patients' unique experiences of non-physical abuse and enhance the understanding of the results from Studies 1 and 2.

Interpretative Phenomenological Analysis. IPA was chosen as it combines phenomenology and hermeneutics to elucidate people's experiences of a phenomenon, along with the sense they make of this phenomenon, instead of focusing on the phenomenon itself (Smith et al., 2009). This was consistent with the purpose of this study. The objective of IPA is to thoroughly explore how participants make sense of their personal world, as well as the meaning they make of a particular experience, using a combination of hermeneutics (interpretation), phenomenology and idiography (Smith, 2004; Smith et al., 2009). Phenomenology (from the Greek word *phenomenon*, meaning 'something that appears', and *logos*, referring to speech, reasoning and rationale) deals with exploring and understanding human conscious subjective experience, a core practice in psychology (Leahey, 2001, p. 381). Hermeneutics (from the Greek word *hermēneuō*, meaning 'translate' or 'interpret') is the theory and practice of interpreting textual meanings. A thorough interpretation involves linguistic and psychological analysis (Smith et al., 2009). Idiography focuses on individuals' specific experiences that took place in a particular context (Eatough & Smith 2008), thus, offering an in-depth understanding of the phenomenon. These procedures, however, rely on participants' and researchers' communication skills. Furthermore, even though

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phenomenological research aims to elucidate people's experiences, it is limited in explaining why these experiences occur (Tuffour, 2017). Nevertheless, this study was interested in the participants' own explanations of the phenomenon's aetiology.

IPA is a popular qualitative method in the field of health psychology, as it helps clinicians understand patients' experiences in order to improve therapeutic practices (Smith, 1999), and it has become popular in other health disciplines over the years (Cassidy et al., 2011). IPA is considered particularly helpful when the research topic involves sensitive issues, such as mental illness (Tuffour, 2017). In IPA, the research process is dynamic as the researcher is actively involved by trying to understand and interpret the participant's world using their own personal conceptions. This two-stage interpretation process is referred to as the double hermeneutic (Smith & Osborn, 2003): the participants try to make sense of their experiences and elaborate on them by answering the researcher's questions, while the researcher tries to make sense of the participant's efforts to make sense of their experiences during the analysis and write-up. A basic limitation of this procedure is that two researchers analysing the same data may end up with different interpretations (Brocki & Wearden, 2006). However, this may occur in with any qualitative method. To overcome this limitation, I conducted the analysis as thoroughly as possible (see Section 7.3.6) and discussed the results with my supervisors.

7.3.2. Participants

Six Greek female adult ED outpatients, currently in treatment for EDs, were recruited for this study (see Section 4.3.2 in Chapter 4). Demographic and clinical information can be found in Table 35. The inclusion criteria, aiming for a homogenous sample as preferred in

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IPA, were that participants should have an ED diagnosis, be in psychotherapy and have reported non-physical abuse when underage, as well as in their adult life. While IPA guidelines favour a fairly homogenous sample (Smith et al., 2009), they also consider the potential problems in sample recruitment if exclusion/inclusion criteria are too restrictive (Eatough & Smith, 2008). Thus, the age and subdiagnosis of the participants were not part of the criteria. Participants were recruited from 18 ANΩ, an EDs outpatient unit in Athens, Greece. Recruitment took place during January and February 2019 and September to November 2020. Pseudonyms have been used to protect the participants' confidentiality. Apart from non-physical abuse in childhood, Participant 1 had endured physical abuse and Participant 2 sexual abuse.

Table 35.

Demographic and clinical information of Study 3's participants

Pseudonym	Age	Diagnosis	Psychotherapy (years)	Illness Duration (years)	Education	Working status
Laura	28	BED	4	10	College	Employed
Betty	39	BED	5	23	High school	Employed
Jenny	41	AN-B/P	5	23	High school	Employed
Carol	35	BN	2	17	College	Employed
Kate	28	AN-B/P (former AN-R)	2	10	College	Unemployed
Amanda	38	BED	4	24	College	Employed

Note: AN-R = anorexia nervosa restricting subtype; AN-B/P = anorexia nervosa binge-purge subtype; BN = bulimia nervosa; BED = binge eating disorder

7.3.3. Data Collection

I employed semi-structured interviewing with the use of follow-up questions (Smith & Osborn, 2003). The interviews were conducted in Greek, and the quotations were translated into English. A demographic questionnaire was also used (see Appendix 9). In IPA, the questions are exploratory, non-directive and semi-structured in an interactive process, as the researcher has the freedom and flexibility to ask further questions, according to the participants' speech flow, in order to help them understand and explain the phenomenon through elaboration (Cassidy, 2011). Semi-structured interviews are helpful to obtain a detailed account of participants' perceptions regarding their unique experiences, leaving room for novel concepts of interest, as the participants direct the interview and not the schedule (Alase, 2017; Smith, 1995; Smith & Osborn, 2003). The material obtained from the semi-structured interviews is consistent with phenomenology as it is of psychological importance to the participant when offering their explanation of the experienced phenomenon in question (Bevan, 2014).

Albeit the interview is co-determined, as previously stated, an interview schedule was developed in advance, allowing me to consider the general areas to be covered in the interviews, along with the wording of sensitive topics (Smith, 1995), in this case non-physical abuse. In line with Smith (1995), the sequence of questions was also decided in advance based on the most logical order. It is important to stress that the questions were general and not leading regarding references to self-esteem, attachment, emotion regulation or any other psychological construct.

7.3.4. Procedure

As described in Section 4.3.4 in Chapter 4.

7.3.5. Self-Reflexivity

I am a Greek female practitioner psychologist with 15 years of experience in almost all types of psychopathology, and further experienced and trained in ED treatment. I do not have personal experience with EDs. I was holding assumptions that there is probably a link between non-physical abuse and EDs through the impairment of core developmental factors. These assumptions resulted from my experience of working with ED patients. Therefore, I was extremely careful not to ask leading questions, and I was aware of these assumptions during the interview and analysis in order to minimise their influence on the way I interpreted the data. The research was supervised by my three supervisors to further ensure that my assumptions would not affect the analysis.

Although using qualitative research for the first time, conducting the interviews was not a difficult process, as talking to patients is familiar to me. The interview questions were based upon a research focus, and I was consulting my interview schedule during the process. The participants knew that I am a psychologist from the information they received from their therapist prior to participation. All the participants asked me prior to the interview if I have experience treating ED patients and, upon my positive answer, they looked relieved and comfortable. Somehow, I feel this helped them open up. Some of the interviews took place during the COVID-19 pandemic lockdown, meaning that people were under psychological distress, and I was no exception. After the interviews, I felt psychologically burdened, as hearing so many negative experiences and negative feelings was overwhelming. The

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participants' accounts did not include positive experiences, which could be the result of many factors, for example, the topic of the interview itself, their own psychological pressure resulting from the pandemic or the fact that they are used to focusing on negative experiences when talking to therapists.

I believe that the fact that my participants were in psychotherapy for quite some time enabled them to make these psychological connections. I did not feel that they withheld information, nor did I feel that they could have said something more but chose not to.

7.3.6. Data Analysis

The analysis followed the steps of Pietkiewicz and Smith (2014), Smith and colleagues (2009) and Smith and Osborn (2003). I transcribed the data collected from the participants (Appendices 10–15), and my initial thoughts and ideas were noted down. The transcribed data were read several times, and the recordings were listened to many times to check the accuracy of the transcription, as well as to ensure my closeness to the data. Initial comments were noted on the right side, making exploratory notes (see Appendix 16 for an example). The second stage involved the use of the left side margin to note emergent themes (codes) from the initial notes (Appendix 16), and they were listed chronologically (Appendices 17–22). Next, I clustered these emergent themes according to conceptual similarities and gave each cluster a descriptive label in order to form related superordinate themes for each participant (Appendices 23–28). Then, a table was produced with the superordinate themes and quotation references for each interview (Appendices 29–34). I reviewed the tables with the superordinate themes by re-reading the transcripts to ensure accurate representation. The superordinate list that resulted from the first participant's analysis was used to inform the next participants' analyses. By being aware of what had come

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before, it was possible to identify similarities and differences in the participants' accounts. Simultaneously, it enabled me to understand how the themes evolved, along with their repeated patterns. Starting each transcript analysis from a clean slate is also an option in IPA, but it is recommended for studies with a small number of participants, such as three (Smith & Osborn, 2003). The next stage involved looking for patterns across cases, and the superordinate themes were clustered together to form master themes (Table 36). This selection was made according to which superordinate themes best answered the research question, as well as those that offered novel areas of the patients' experience of non-physical abuse. Through this process, three master themes and eleven superordinate themes were produced. The table of master themes and superordinate themes is presented in the analysis that follows (see Table 36).

7.4. Analysis

IPA of the six semi-structured interviews resulted in the identification of three master themes. The exploration of these master themes and their constituent superordinate themes is presented below (see Table 36), with each superordinate theme illustrated by quotes from the interviews. These quotes can be found in the text. The analysis is presented following the quantitative studies' structure, meaning that the connection to the literature is highlighted in the discussion part.

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Table 36.

Master themes and related superordinate themes from Study 3's transcripts.

Master Themes	Superordinate Themes
Continuation of non-physical abuse across the lifespan	The experience of non-physical abuse in childhood
	The experience of non-physical abuse in adulthood
	Non-physical abuse consequences
	Feelings about non-physical abuse
	Dealing with non-physical abuse
	Searching for understanding in non-physical abuse's onset and continuation
Developmental factors and non-physical abuse	The development of self-esteem
	Disrupted attachment
	Emotion regulation difficulties
Non-physical abuse and eating pathology	Non-physical abuse and ED onset
	Non-physical abuse and ED maintenance

7.4.1. Continuation of non-physical abuse across a lifespan

The first master theme refers to the participants' experiences of continuing non-physical abuse both in childhood and adulthood. The common features of childhood and adult non-physical abuse for the majority of participants were negative comments on their physical appearance, as well as a general devaluation of their self-worth. In their effort to understand their experiences, participants attributed the abuse to abusers' psychological issues. Furthermore, they attributed its continuation to their own difficulties in standing up for themselves. They discussed how they felt about the abuse and how they dealt with it. The prominent feelings regarding their abusive experiences were anger coupled with sadness.

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The experience of non-physical abuse in childhood

Participants reflected on their experiences of non-physical abuse during childhood, which for the most part, involved derogatory comments regarding their physical appearance, most notably their weight, for example, '*[my father] called me stupid, fat and ugly*' (Jenny). The abuse also included general devaluation of the self, and in many cases, it exceeded the familial environment, like in Amanda's case: '*kids at school used to call me fat, barrel and my teachers told me that I had to lose weight*'. It is evident that the participants experienced attacks on the self from a very young age, shaping their perceptions of themselves: '*I spent my whole life being the fat one*' (Amanda). The experience of criticism in shaping self-perception was a key element of the participants' narratives. For example, Kate, who said that she was an overweight child, reflected on how everyone in her social environment criticised her body weight. She described her experiences in a way that shows how disappointed she was with her mother who did not help her maintain a physical appearance that would prevent her abuse. When Kate was asked to reflect on her experiences regarding her childhood non-physical abuse she said:

[I experienced] bullying, which was essentially criticism, and I was also strongly criticised by my social environment. Uncles, aunts, teachers, friends' parents—they were all trying to do the job that my mother did not do, but not in a very nice way. (Kate)

Participants were also neglected by their parents—'*I felt there was emotional neglect from both my parents*' (Betty)—emphasising the importance of a stable familial environment on personal development, as reflected in Laura's accounts: '*family is the first step for someone to move on in life, and this first step was trembling from the beginning in my case*'. The participants perceived as neglect the fact that their parents did not care about helping them improve their physical appearance. Their parents' advice regarding abuse was not to eat too

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much, which only fuelled the negative image the participants had already formed about themselves. Jenny and Kate emphasised the mother's role. The perception of a mother as a protective figure caused the participants to be disappointed when their mothers let them down. For example, Kate said:

When I complained to her that I was called fat and ugly...she would say to me 'ok it is bad that they call you ugly, but objectively, you are fat'...generally my mother is not good at supporting someone emotionally...She certainly should not have let me gain so much weight and become so ugly. That was neglect. (Kate)

The experience of non-physical abuse in adulthood

Participants experienced the continuation of non-physical abuse throughout their adult lives. The content of the abuse remained the same, but as the participants matured, apart from parents, as in Laura's case (*'as I still live with my parents, it [the abuse] is a continuation of my childhood. Whatever I experienced as a child, I now experience as an adult as well'*), other people became involved in the continuation of the phenomenon, for instance, partners: *'he may make comments regarding my appearance, and this hurts my feelings'* (Laura). For some participants, colleagues were also involved in the abusive behaviour against them: *'you are shy. You do not understand what we are saying. Do not be so weak'* (Jenny). For other participants, devaluing behaviour was displayed from health care professionals: *'a doctor once told me that if I lose weight I will become more elegant. The doctor himself was fat'* (Betty). Bullying regarding physical appearance within an educational environment did not end with high school graduation for the participants, like in Kate's case, who reflected on her college experience of participating in a theatrical play audition:

...[the director] had picked me up for an exercise, and he was shooting me from some angles, and he was saying to me 'what a nice face you have... if only you didn't have this body', and he said that to me in a room full of people...

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All the participants mentioned being emotionally abused or neglected by their partners, indicating the difficulty of abused individuals to avoid abusive relationships later in life. Carol and Amanda moved a step forward, noticing the resemblance between their parents and partners' abusive behaviour. This comparison was not the result of the interview questions. For instance, Carol said:

It's like leaving my parents and going to the same environment...There is a lot in common between my parents and my boyfriend. It's something I was shocked when I realised. (Carol)

Non-physical abuse consequences

The participants reflected on how the ongoing abuse affected them, explaining the impact on how they connect with others, on their life development, on shaping their characters and feelings, and on the fact that they cannot feel joy. Life satisfaction and personal development seem to have been influenced by the non-physical abuse:

I do so many things in my job, and yet, I don't enjoy it because there is a black part in my life...In my mind, I am that child who suffered violence...I have not grown up. (Laura)

Jenny likened the consequences of abuse to carrying a burden, indicating the long-term and 'heavy' nature of abuse. She reflected on how abuse had interfered with her personality development. When asked how abuse has affected her, she said:

[the abuse affected] how my character was shaped... how I feel...I cannot feel joy...It's like carrying a burden...I am always sad...I did not feel acceptance and love, so to be strong and show this to others...The way I grew up is responsible for who I am now. How I think, react and feel.

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Carol added that being abused from a young age leads to familiarity with abusive relationships:

Even though [the abuse] bothers me, it is a situation that I know and am used to. I know everything. I'm waiting for them. Sometimes I think that if I find someone who treats me well, I will not know what to do.

Kate highlighted the long-term effects of non-physical abuse, which has manifested as an ED, by detailing the time she has lost:

I have lost huge periods of time in my life while fighting with the ED...I had focused on the weight, and I lost time from my life, mainly in the professional part...I made myself not eat, but not eating has been imposed on me by others through all these years of bullying.

Feelings about non-physical abuse

Participants shared their feelings about their non-physical abuse, for example, '*I feel anger, rage, sadness, despair*' (Laura). The internalisation of abuse was evident: '*I feel like a victim, and I am angry that I am so weak and that I cannot defend myself*' (Jenny). These emotions also reflected how the participants feel about themselves: '*very low self-esteem and anger. I was angry at myself for being fat...I always felt neglected. I always felt alone*' (Amanda).

Laura questioned the validity of her feelings, further indicating the internalisation of abuse, resulting in cognitive conflict. On the one hand, she thought the abuse could be her fault, but on the other hand, she knew that the abuse should not have happened (two beliefs):

I wonder if I am crazy, if they are right, if I am doing something wrong, a victimisation so to speak...I wonder if I'm doing something wrong, and I'm doomed because two beliefs conflict within me.

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The participants not only named anger as the most prominent emotion, but they also laid out the origin of their anger, which is non-physical abuse. Explaining the direction of their anger, it is clear that it is twofold. For example, Jenny stated:

[I am angry] at my father, who was bad to me; at my mother, who did not protect me or teach me to be strong; at myself because I was not strong enough to protect myself. Often, I feel angry at the whole world. I also get angry when I see others who did not grow up that way. I envy them, and that makes me angry with them. Why should they be strong, and I am not? This makes me angry.

Kate's description shows both the intensity and the overwhelming nature of anger:

[anger] really is like holding a hand grenade and not knowing where I want to throw it. I mean, I blame myself. I blame my mother because she let me gain weight. I blame society for being so oppressive about women and weight, but most of all, I burst out my anger with bulimic episodes. I do not know who to blame first.

While being angry at abusive others is a common experience, being angry at themselves for their weakness in reacting to the abuse could explain the participants' self-damaging behaviour in the form of EDs. The participants turned to themselves in an effort to improve what they considered responsible for the abuse, which was their weak self.

Dealing with non-physical abuse

This sub-theme captures how the participants handled their abuse in childhood and as adults. Passively accepting the abuse was and remained their way of dealing with abuse, and this can be clearly seen in Jenny's quote:

When something bothers me, I do not react. I usually leave...My reaction is the same. I did not speak then, nor do I speak now...I learned not to speak, so that there would be no bigger reaction from others...That was my mother's advice regarding my father...I have learned not to speak and walk away....I close myself off.

Being used to the abuse also played a role in not reacting, indicating the long-term effects of abuse, as Laura explained: '*at first it [the abuse] felt annoying but now I have got used to it*'.

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The effort to avoid abuse through food reduction displays the internalisation of abuse, indicating a connection between being bullied about weight and diet onset. For example, Kate's experiences illustrate how emotional abuse is an attack directly at the self that can affect self-esteem. Kate did not understand that being bullied for her weight was wrong, and she thought that she should change her physical appearance:

I did not understand, and I thought that the only solution not to accept such behaviour would be to lose weight...and I said that everyone is right, I am a monster, and I must definitely lose weight, otherwise nothing good will ever happen to me in my life. Then, I started my first paranoid diet. I said I would continue not eating until the fat was completely gone.

Kate used AN-R to eliminate her low self-esteem, indicating how angry she was at herself. Her quote demonstrates how low self-esteem, coming from emotional abuse, can lead to body dissatisfaction and, consequently, to restrictive dieting:

...I decided to protect myself but in the wrong way. I protected my vulnerable self, my low self-esteem self. Anorexia 'helped' me lose weight, become pretty, feel better about myself. It helped me kill the ugly overweight girl that everyone mocked...

Kate's quote above, further interpreted from a psychodynamic perspective, indicates that AN is the result of the disruption of self-concept, as Kate thoroughly explained the creation of a false self (thin, beautiful, with high self-esteem) to hide the true self (overweight, ugly, with low self-esteem). Kate's air quote for 'helped' indicates that she now acknowledges that restrictive dieting was an unhealthy way to deal with the abuse. She also tried to draw attention away from her body image by changing the way she looked. This further indicates the efforts she made to hide her 'responsible for the abuse' true self as she had internalised others' opinions about her physical appearance:

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At the end of high school, I had decided to be gothic to get the attention off of my weight because everyone was concerned with the fact that I had green hair and wore black clothes...

Amanda's experiences further show the internalisation of childhood non-physical abuse and how she used food to regulate the negative emotions resulting from her abuse:

Anything I was thinking that made me sad, I kept inside...My company was food. When I ate something that I liked, I felt good...All I thought about was food...I found an escape through food, which did not help because I continued to be fat...

Searching for understanding of non-physical abuse's onset and continuation

The participants were trying to understand their experiences of non-physical abuse and its continuing character. They attributed what happened to them to the abusers' own issues: *'people who did this have also been abused because people have psychological issues'* (Betty). The participants also attributed the onset of their abuse to the fact that they were not strong enough to react and stop it, and as a result, others perceive them as victims.

Carol explained:

If I had reacted from a young age, this would not have happened. I am a weak character. I avoid conflict with them [my parents] because I cannot stand it at all. I cannot argue...I'm used to it, I think. That is how I am as a character. That's what I know how to do. Others see me as a victim. Weak, I mean. You attack someone who looks weak, not someone who looks strong [sighs]...

Kate and Amanda added that abuse is frequently aimed towards overweight children as weight is considered the individual's fault. This is exemplified by Amanda: *'I think this [non-physical abuse] happens to children who are obese. Others make fun of them. It's not right but it happens'*. This indicates the stigmatisation of overweight people, as explained by Kate:

I think this [non-physical abuse] happens to children who are overweight. People consider it normal to do so [abuse overweight children]. If you go to an adult to ask for help, he will tell you to lose weight to resolve this. But if someone has a big nose,

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an adult can say that it is not okay to make fun of it, but for the weight they believe that it is your fault...

Kate also added the thin ideal as a reason for the ongoing abuse regarding physical appearance, highlighting the role of societal messages in weight stigmatisation: *'because I was born a woman, it is common to be stressed about appearance even when you have a normal body'*. The continuation of abuse throughout adulthood is mainly attributed to the participants themselves for not being able to become stronger and react. Being victimised and used to the abuse is considered by the participants factors that perpetuate the abusive behaviour against them, indicating that the consequences of abuse are also abuse's maintaining factors. This is best reflected in Laura's account:

Because I cannot react, and it is my fault. I don't know how to set limits so as not to be insulted, as I never learned to do it...[it is] a habit. That I have learned this way and I believe that some things are not going to change, and they are what they are...it is a habit. We have all learned this way. They [parents] learned this way...I have learned this way.

The psychological mechanism behind the internalisation of childhood abuse that leads to victimisation is self-esteem. The participants' low self-esteem is an important reason for the continuation of abuse, as it prevents them reacting, thus, making them easy victims. Amanda explained: *'[the abuse continues] because I still do not have self-esteem. When you have self-esteem, you look strong, and when you look strong, others do not comment on you. They do not insult you'*. The abuse will end when their self-esteem is improved because only then they will be able to defend themselves without considering the consequences: *'I have to become stronger, to defend myself, to have self-esteem and to show it, to set limits, to speak without caring what will happen'* (Carol). It is evident that high self-esteem is important to the participants' ability to express emotions and defend themselves.

7.4.2. Developmental factors and non-physical abuse

The second master theme presents the participants' views of the core developmental factors that were impaired due to non-physical abuse, as these emerged from the participants' accounts. These factors are self-esteem, attachment and emotion regulation.

The development of self-esteem

Self-esteem was very important to the participants, affecting how they perceive themselves, as Kate said: *'in general, my self-esteem is not good. It created instability in how I see myself'*. Childhood abuse affected the participants' self-esteem: *'I never had self-esteem. Never. I lost years of my life not having self-esteem. Not only then but now as well'* (Amanda). As a result, they are weak and easy targets for abuse, as Carol explained: *'It is important to have self-esteem. Others do not treat people with self-esteem like rubbish. They can't'*. Kate further discussed the power of self-esteem and the dynamics it can create in people's relationships: *'you know...self-esteem is visible. Others can see if you have it or not. And they treat you accordingly'*.

Jenny explained how low self-esteem prevents her from defending herself—*'I do not feel that I have the confidence to defend myself. I cannot fight back when I have to...because I don't have self-esteem'*—and from trying for more in her life—*'I do not have self-esteem...I do not believe in myself. I only do what I can, and I do not try for more'*. Amanda added that low self-esteem also prevents her from living: *'it's like I'm afraid to live. I do not know how else to describe it. I do not believe that self-esteem ever changes.'*

Jenny perceived that her life would be different if she had good self-esteem, revealing the importance of this developmental factor: *'things would be different if I had self-esteem, if I believed in myself'* (Jenny). According to Amanda and Kate, low self-esteem makes people

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weak: *'low self-esteem equals an easy target, an easy victim, weakness'* (Kate), defining them throughout their lives: *'it is very bad not to have self-esteem. It determines all your life. What will you do in your life...Who will you be?'* (Amanda). The participants perceived self-esteem as stable: *'this image for yourself and your weight follows you forever. To put it correctly, your self-esteem follows you forever'* (Kate) and difficult to change: *'if you didn't have self-esteem as a child, you will never have [it]. You will carry this burden throughout your life'* (Amanda).

The consequences of low self-esteem are strongly reflected in the participants' accounts, as they caused the participants to be angry with themselves to the point of engaging in self-destructive behaviour to change the self. For example, Kate described how she punished her low self-esteem through dieting: *'[I had] very low self-esteem...I was self-whipping endlessly, that is, I started dieting'*. Kate linked her self-esteem with weight: *'I have compulsions with weighing myself, and I think my self-esteem is related to the outcome'*. The participants tried to improve their self-esteem through dieting, highlighting self-esteem as a risk factor for ED onset, which is best reflected in Kate's quote:

Anorexia was the easy way towards a better self-esteem. It's not that I woke up one day and thought to myself 'let's have anorexia'...no. I started dieting like crazy, and at some point I lost control.

According to the participants, high self-esteem is a protective factor against unhealthy coping methods to regulate emotions. This indicates a connection between low self-esteem and emotion dysregulation, which is evident from Amanda's quote:

When you feel good about yourself, you will not be affected by others' behaviour, and you will not be hurt...If I were strong and [if] I had self-esteem, would I need to eat to feel relieved?

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Rhetorical questions, such as the one in Amanda's quote, are used in Greek culture to indicate strong beliefs.

The participants believed that self-esteem develops in the early stages of life and is affected by the relationship with significant others. Amanda highlighted that '*parents should make sure their children will have self-esteem*'. According to Kate, the development of self-esteem, in terms of physical appearance in women, can be affected by the mother's image and its reflection on her daughter:

I did not have self-esteem from my home. My mother did not have self-esteem, and so, I probably learned not to have it either. She did not care if she looked beautiful. She was very withdrawn and closed off. I had no idea what a beautiful woman looks like. I did not have an image as a guide to create my own.

Kate's quote might be interpreted as if she is looking for someone to blame for her low self-esteem, as her self-esteem is linked with her physical appearance. Kate's anger at her mother is evident here, and this quote was given in further explanation of her earlier account about feelings of anger (Section 7.4.1).

Disrupted attachment

Another important developmental factor affected by non-physical abuse is attachment. The participants either avoid attachment to avoid emotional hurt or they find it difficult to detach from abusive relationships so as not to feel abandoned. Relationships with significant others can be problematic, as Betty explained: '*some relationships can be addictive or primitive like mother-child*', indicating the constant need for love, acceptance and safety as described by Laura: '*I am still waiting for the love and support that I feel I should have received. But I know that won't happen*'. Amanda linked attachment with self-esteem: '*If I had good self-esteem, I wouldn't be afraid to attach*', indicating that she feels that low self-esteem prevents her from safe attachment. The participants' quotes suggested a bidirectional

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relationship between attachment and self-esteem, perceiving safe attachment as necessary for the development of good self-esteem and, once self-esteem is formed, defining how and if people will attach to others. The need to fill the emotional gap from a disrupted parental attachment is reflected by their difficulty in avoiding abusive relationships, such as in Carol's case:

I have thought about [leaving my boyfriend], and I have to do it. I do not know why I cannot. Maybe because I have nowhere to go. All my friends are in a relationship. I will be the only one alone. Who will I go out with? Will someone else be better than him? What if I do not find someone else? What if I am alone forever?

On the other hand, avoiding close relationships equals safety from painful feelings, and this is the only way to emotionally survive in an unsafe world, as Jenny explains:

I do not trust others. I do not have close relationships. I like distance. I feel safer when I keep my distance...I cannot attach. I'm afraid of rejection...so as not to get hurt, of course. When you get close to someone, you become vulnerable. Then if something goes wrong, you will be very hurt.

Further to this, Amanda described close attachment as being shot at from close range. This metaphor indicated the extent to which significant others have let her down early in her life. Consequently, distancing herself from others is a self-protective measure against emotional pain. The long-lasting effects of a disrupted attachment are illustrated in Amanda's quote:

When someone says something bad to me, it's like eating a bullet. I hurt. If they shoot you from a close distance, you will hurt more, while from a long distance it will not hurt so much.

Emotion regulation difficulties

Emotion regulation is another developmental factor affected by childhood non-physical abuse in the participants' accounts. The participants explained their difficulty in managing and tolerating negative emotions. They also reflected on their difficulty in

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expressing these emotions, and how, as a result, they could not defend themselves against non-physical abuse. For instance, Jenny explained how the perception of anger as a threat leads to emotional inhibition:

I cannot express my feelings...I have got used to not speaking up, and I only speak up to the point where I feel safe because I do not know what to do if the other person starts to get angry and shout. I do not know how to deal with the other person's anger.

In their accounts, the participants indicated that anger is an intense emotion that causes sadness and tiredness. Sadness is caused by the inability to confront the source of the anger, explaining the coupling of anger with sadness in almost all of the study's participants' accounts. Tiredness is the result of the intensity of emotions. Jenny's quote best explains these emotions:

After having thoughts that make me angry, I feel sad, sad and tired at the same time. Maybe [I feel] sad because I know I'm not capable of dealing with what makes me angry, those who make me angry. When you feel something intensely you get very tired, don't you?

Expressed emotions can destroy relationships; thus, they are suppressed. Jenny experienced the cost of unexpressed emotions:

[emotions] are stronger than me. This is why I am afraid to speak. I do not know what will happen if I express them. Instead of taking them out and destroying someone else, I leave them inside, and they destroy me. They eat me from the inside.

Jenny, in the above quote, vividly presented the destructive nature of unexpressed emotions. Being eaten from emotions is a common expression in Greek culture and is used to indicate the psychological cost of dealing with negative life events, negative emotions and bad personal/social relationships.

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Suppressed anger can also lead to negative ways of coping, as is reflected in Kate's accounts.

In her case, extreme negative affect interacted with social influences:

I had gotten into such cliques that everyone was angry and delinquent...I felt like we had something in common, so I vented it [my anger] a bit like that...A couple of times I put out cigarettes on myself...Another time, I took a key and damaged all the cars in the neighbourhood. I had suppressed anger, and so I took it out like that.

The participants explained how they used food to alleviate negative emotions. Eating is a way to cover unexpressed anger: *'I swallow emotions with food'* (Amanda). Food also helped them dissociate from painful emotions: *'I relieved my anger through eating. That's all I remember to tell you. It's like so much food has blocked my memories!'* (Amanda joked). These negative emotions are created from ongoing abuse, indicating trauma-induced dissociation: *'I continue to find an escape through food and eat to avoid listening to them (her parents)'* (Laura). Carol explained that not being able to express negative emotions preserves the need to rely on her relationship with food in order to dissociate from painful emotions:

It is easier for me to eat than speak up...This relieves me at that time, but then I feel bad...It's like I want to do something intense at that time, but I can't. Eating is like forgetting anger or drowning it out. {[It's] as if the food covers anger, and I forget [the anger]}.

Kate graphically explained how changing the direction of her anger changed her ED symptoms:

When I started with anorexia, I blamed myself for everything. I believed that in order to deserve better treatment, I had to be thin. The whole phase of anorexia was as if I had turned my anger on myself. I did not feel angry with others then...Now, with bulimia, I understand how angry I am with others. I understood that with psychotherapy...In bulimia, I started to think that I was not to blame for the bullying, and I started to take out my anger [through vomiting].

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Kate's account offers insight into the diagnostic migration observed in EDs. Being angry at herself for being overweight, thus, responsible for the non-physical abuse, led to AN-R. The realisation that it was not her fault, which led to anger at others, changed her symptoms to AN-B/P. She vomited her anger, as it was no longer her fault. The shifting of the ED symptoms because of the shifting in the direction of anger shows the importance of the unexpressed anger in EDs and the importance of recognising the source of this intense emotion, which is somatised. Kate referred to her latest diagnosis as bulimia; however, according to her therapist, her current diagnosis is AN-B/P.

7.4.3. Non-physical abuse and eating pathology

Regarding the last master theme, the participants discussed how their experiences of non-physical abuse related to the onset of their EDs and how the continuation of the abuse relates to the maintenance of ED symptoms. According to the participants' accounts, the three developmental factors described in the second master theme play an important role in this relationship.

Non-physical abuse and ED onset

Kate discussed that childhood non-physical abuse played a role in the development of the disorder: *'I think if I did not have these experiences, it [the ED] might not have happened. I wouldn't need to lose weight in order to be accepted and become obsessed with it'*. Negative childhood experiences disturbed Laura's relationship with food: *'I was criticised [by my parents] as kid for being overweight and being fat so eating was a reaction to that'*. Food is a form of addiction, resulting from traumatic experiences, according to Betty: *'anything traumatic can create a form of addiction'*. Emotion dysregulation is important in

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ED onset. Unexpressed emotions created by non-physical abuse ‘eat’ people, leading to unhealthy coping methods, as Jenny explained:

When you are not well and you cannot speak, you probably find another way to show it. You turn into yourself, and you shut yourself in there. But this way is destructive in the end...If I could get all these [unexpressed negative emotions] out of me, they would not eat me...Anger. The sadness about what is happening, but I cannot speak. The sorrow. Aren't these [feelings] eating people? This is why people get sick.

The need to restore the disrupted attachment with significant others is a contributing factor to the onset of EDs. In childhood, Amanda used food to attach to her emotionally distant mother.

I started eating so [my mother] wouldn't be upset. I had heard her tell my aunt that I was a difficult child. When I started eating, I could not stop...I think I started eating so my mother would be happy and satisfied with me. I thought that if I pleased her, she would [be] more tender with me, that I would gain her acceptance, that she would show me that she loves me.

Low self-esteem is an important risk factor for ED onset, as shown from Kate's experiences:

I thought that by losing weight my self-esteem would dramatically improve, and people do not bully people with high self-esteem. That was the solution in my mind. It was an unhealthy way to deal with it of course... In order to have self-esteem, I had to lose weight.

Non-physical abuse and ED maintenance

The participants reflected on how the continuation of non-physical abuse is associated with the maintenance of their ED symptoms. The continuation of non-physical abuse preserves the ED symptoms, creating a vicious cycle, as illustrated by Carol:

When [my parents and partner] see me eating, they tell me that I will become fat and my clothes will not fit. I continue eating! This calms me down, and I already know

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that it irritates them when do it...I feel relieved when I eat, but then I become disgusted with myself, and I want to vomit.

Carol's account explains the feeling of disgust, which has been observed in eating pathology, indicating that vomiting is linked with disgust, and disgust is directed at the vulnerable self rather than the food. Carol's disgust seems to be an automatic response to the fact that she cannot effectively react to the abuse by expressing her emotions.

Kate thoroughly explained how the ED symptoms are used to avoid rejection and deal with the painful feelings rejection may cause:

When I was not accepted onto a postgraduate programme, I had a bulimic episode. When I had to go to a job interview, two days before, I did not eat anything in preparation for success. When I feel rejected, I have a bulimic episode. To avoid rejection, I do not eat.

Kate explained how attaching to the disorder protects the self from painful emotions that could result from attaching to other people: *'I use the disorder as a buffer in my relationships with others...When I am closed inside the disorder, I do not care much what others say and how bad my relationships will be'*. As noted in the second master theme (see Section 7.4.2.), Kate said that her self-esteem is linked with her weight. The fact that she attaches to the disorder to avoid rejection and protect her self-esteem (which she tried hard to elevate through anorexia), reveals a link between the impairment of developmental factors and ED symptoms' maintenance.

Summary of the results. Non-physical abuse in childhood disturbed the participants' attachment and lowered their self-esteem. Thus, the development of a strong self that could effectively deal with abuse and avoid internalising the abuse was inhibited. The abuse created intense negative feelings, most notably anger for the weak self and sadness because of the

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inability to react and stop the abuse. The 'weak' self (low self-esteem) was not confident enough to express these feelings because the relationships with significant others would be destroyed. 'Eaten' by their strong emotions, they turned into themselves, using food as a coping mechanism. Due to the internalisation of abuse and victimisation, the participants' self-esteem remained low, making them vulnerable to continuing their non-physical abuse throughout adulthood. Childhood non-physical abuse triggered the ED onset, and its continuation into adulthood maintains the ED symptoms.

The mechanism behind this relationship, as revealed through the participants' accounts, is that the impairment of attachment, self-esteem and emotion regulation due to childhood non-physical abuse opened the door for EDs to act as coping mechanisms. These developmental factors never improved, and as a result, the ED symptoms are maintained as a coping mechanism. The participants still need the disorder to protect the vulnerable self and survive. The participants are afraid of rejection and abandonment, and the negative feelings these emotions will create about the self, so they either avoid attachment or preserve abusive relationships. They are afraid of their negative emotions, and they do not express them, as they cannot handle other people's reaction. The connecting factor is self-esteem. A strong self can survive rejection and is not afraid of the consequences of emotional expression. In these participants' cases, the self is by their own admission weak; thus, they are still attached to the disorder.

7.5. Discussion

This study aimed to gain an in-depth understanding of adult female ED patients' lived experiences of continuing non-physical abuse from childhood to adulthood. This was achieved through semi-structured interviews employing IPA. To the best of my knowledge,

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there are currently no qualitative or quantitative studies that have examined the construct of continuous non-physical abuse in EDs. It was, therefore, hoped that the current study would add to existing knowledge in this area, which to date, has primarily come from clinical observations. This study, through ED patients' lived experiences, expanded and elucidated the quantitative results of Studies 1 and 2, which identified self-esteem as the most important mediator between an invalidating environment in childhood and adult eating pathology.

The research question was answered in a unique way, by the participants offering coherent explanations regarding the sequence and interaction of factors related to eating pathology onset and maintenance: the non-physical abuse they endured during their childhoods, most notably emotional abuse, resulted in their ED onset by affecting self-esteem and emotion regulation through a disrupted attachment with significant others. These three impaired psychological factors made it difficult for the interviewees to stop the continuation of the non-physical abuse in adulthood, and as a result, they maintained their ED symptoms to alleviate negative feelings. This vicious cycle has affected their development. The themes derived from the analysis are connected in such a way that demonstrates that the consequences of non-physical childhood abuse persist into adulthood. Consequently, the discussion of the findings that follows is not divided into subheadings.

This study's results indicate that childhood non-physical abuse is related to both ED onset and maintenance in all the participants. The literature supports the association of a dysfunctional familial environment with ED development (e.g. Cerniglia et al., 2017; Tetzlaff et al., 2016), suggesting that non-physical forms of childhood abuse could have a significant effect on the development of EDs, which is supported by this study's participants' account. Many authors (Fosse & Holen, 2006; Groleau et al., 2012; Kent et al., 1999; Kong &

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Bernstein, 2009; Vajda & Láng, 2014; Waller et al., 2007; Witkiewitz & Dodge-Reyome, 2000) consider CEA to be the most significantly influential factor that deserves further empirical exploration with respect to eating pathology. Additionally, in studies exploring all forms of childhood abuse and eating pathology, CEA emerged as the strongest predictor of EDs (Burns et al., 2012; Kent et al., 1999; Witkiewitz & Dodge-Reyome, 2000). It has also been underlined that in instances where other forms of abuse appear to predict ED, they have a greater impact when an emotional abuse component is also involved (Kent & Waller, 2000; Kent et al., 1999), supporting the notion that emotional abuse unifies all forms of childhood abuse (Hart & Brassard, 1987). Laura and Betty experienced physical abuse as well, but throughout their interviews, they emphasised the emotional abuse they endured, along with neglect. This was not in response to my questioning, as I avoided leading them in that direction.

Authors maintain that CEA contributes to ED development by impacting self-esteem and emotion regulation (Groleau et al., 2012; Hund & Espelage, 2006; Waller et al., 2007). The symptoms of EDs serve as a means to block emotions (Corstorphine et al., 2007; Fox & Power, 2009; Meyer et al., 1998), and this study's participants stated that they managed their negative and intolerable feelings through 'swallowing emotions with food'. For instance, Laura, who suffers from binge eating disorder (BED), said that when she eats, she stops hearing her parents and so detaches from their abusive behaviour. In other words, binge eating is used as a way to escape self-awareness (Heatherton & Baumeister, 1991), indicating the trauma-induced dissociation observed in eating pathology (Vanderlinden et al., 1993). Janet (1889, 1907, 1919, 1925) was the first to study the relationship between traumatic experiences and dissociation in order to explain the manifestation of several psychiatric

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conditions, including EDs. He conceptualised dissociation as an important psychological mechanism used by the individual in the face of overwhelming trauma. He referred to the traumatic experiences as *idées fixes* (fixed ideas) and proposed that they split off from conscious awareness, resulting in several dissociative (in other terms, hysterical) symptoms (Janet, 1889, 1907, 1919, 1925; van der Kolk & van der Hart, 1989). For Janet, trauma-induced dissociation was an escape/avoidance mechanism, and severe pathological dissociation involved amnesia in relation to the traumatic event, something supported by the literature as well (Radulovic et al., 2018). Amanda, for example, joked about how too much food has blocked her memories. Dissociation is present in the participants' accounts and is ongoing in adulthood, indicating that in lieu of a strong self (self-esteem) that could help them tolerate and manage emotions effectively (emotion regulation), including detaching from abusive relationships (attachment), the maintenance of ED symptoms is the only way to find instant relief.

Studies indicate that childhood emotional maltreatment is associated with low self-esteem (Shaffer et al., 2009) as it can lead to negative core beliefs about the self (Frankel, 2002; Waller et al., 2007), severely affecting self-esteem (Finzi-Dottan & Karu, 2006; Kent & Waller, 1998, 2000). Self-esteem is an important trait of eating pathology (Fisher et al., 1991; Newns et al., 2003; Sassaroli & Ruggiero, 2005) and has been identified as a risk factor for ED development (Cervera et al., 2003; Gual et al., 2002; Holston & Cashwell, 2000; Katsourani, 2009; Leon et al., 1999; Stice, 2002). It is possible that being repeatedly insulted and criticised in childhood leads to ED symptoms because of the impact on the individual's self-concept (Groleau et al., 2012), which seems to be the case with these participants. Waller (1998, 2000) hypothesised that self-esteem could be a mediator specifically for the

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relationship between CEA and ED pathology, stating that CEA could lead to low self-esteem more than any other form of child abuse as it may be interpreted as a personal attack on the self, which is evident from the participants' experiences. Yates (2004) also suggested that self-damaging behaviours may reflect a physical manifestation of the individual's negative self-representations, which may originate in childhood through maltreatment. Elaborating on these lines, it could be proposed that emotional abuse leads to low self-esteem, and these negative representations of oneself physically manifest as ED symptoms by somatising anger towards the self. This study's participants used dieting to improve their self-esteem as their abuse involved bullying regarding their physical appearance. Kate, who suffers from AN, explained that she started her 'paranoid diet' in order to improve her self-esteem, supporting studies in which low self-esteem was revealed to be a stronger risk factor than restrictive dieting for AN (Schmidt, 2001). In other words, restrictive dieting would not occur without low self-esteem. Kate's experience is in line with the psychodynamic view that considers EDs to be the result of a disrupted self-concept (Bruch, 1973), which creates the need to develop a false self to hide the unaccepted true self (Winnicott, 1965). One could argue that this process is applicable to other physical manifestations of self-damaging behaviours as well, such as drug and/or alcohol abuse. Betty placed the relationship with food among addictive behaviours, as has been suggested in the literature (Curtis & Davis, 2014). The answer to this argument probably relies on the specifics of emotional abuse. In other words, when the abuse involves negative comments regarding food consumption and physical appearance, and when the parents emphasise dietary behaviours, it is more likely that the consequences of non-physical abuse will take the form of an ED (Copeland et al., 2015; Jones, 1985; Lie et al., 2019). The participants have experienced negative feedback regarding their body image and food consumption since childhood, and some participants have

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witnessed their mothers' disorganised eating behaviour. The participants' eating problems began early in childhood as a reaction to these physical appearance comments (e.g. eating secretly, dieting, eating a lot), and longitudinal studies indicate that eating problems in childhood are a risk factor for ED development in adulthood (Kotler et al., 2001). Being bullied about weight by one's peers and the associated negative emotions echo other qualitative studies that employed ED patients (Fox, 2009).

According to Wiehe (1990), people who have suffered psychological maltreatment internalise the expressions of maltreatment and accept them as true, which further indicates that emotional abuse can affect self-esteem when applied to the participants. For example, Kate had internalised the thin ideal and thought being bullied for her weight was normal. Studies indicate that instability of self-concept increases vulnerability to the thin ideal internalisation (Vartanian & Dey, 2013). The thin ideal is linked with body dissatisfaction (Stice, 2001), and body dissatisfaction is predicted by low self-esteem (Shahyad et al., 2015). Kate stated that her low self-esteem created instability regarding how she views herself, suggesting that low self-esteem due to childhood non-physical abuse created an unstable image of herself that made her vulnerable to the thin ideal internalisation, thus, making it difficult for her to recognise abuse. The unstable sense of self in AN is in line with other studies (Williams et al., 2016). Studies also confirm the significance of self-esteem on body image perception (Skorek et al., 2014), explaining why Kate had linked her self-esteem with her weight. Kate's experience could add to the dual pathway model of eating pathology (Stice, 2001) by offering the concept of childhood non-physical abuse to better explain family and peer pressure for thinness and by offering the concept of low self-esteem to explain the internalisation both of the thin ideal and the non-physical abuse that led to body dissatisfaction.

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This study's participants felt victimised. Furthermore, they were so used to being emotionally abused that it had turned into a habit, which further indicates the internalisation of early abuse. Many participants experienced second thoughts regarding their abuse, thinking that since they are overweight, negative comments are to be expected. The literature suggests that ED patients are stigmatised because they are perceived to be responsible for the disorder (Crisp, 2005; Mond et al., 2006; Roehrig & McLean, 2010), something echoed by Kate's and Amanda's experiences, who were advised to lose weight to stop the abuse because their weight was their fault. The participants' second thoughts about their abuse could be also explained by Briere's (1992) abuse dichotomy concept in which abused people face a cognitive conflict in their effort to explain the abuse. This means that they either attribute the abusive behaviour to others' interpersonal issues or they justify it, believing they deserve it. The dichotomy concept is reflected in the participants' accounts as they attributed the abuse to the abusers' psychological issues but simultaneously consider themselves responsible for being overweight or for not being good enough. Cognitive conflict is also observed in borderline personality disorder (BPD) and is associated with self-injury behaviours due to psychological distress (Suarez & Feixas, 2020) resulting from an invalidating environment (Linehan, 1993). EDs are considered a form of self-injury (Farber, 2008), and this thesis conceptualised childhood non-physical abuse incorporating the invalidating environment concept. The presence of cognitive conflict in this study's participants (angry at themselves for the abuse/ angry at others for the abuse), along with the self-punishing behaviour (dieting, self-harm), could indicate a common characteristic between EDs and BPD.

Moreover, it is believed that the impact of emotional abuse is stronger during childhood because of children's egocentric way of processing information and their simple

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situation classification (Waller et al., 2007). This immature cognitive style in childhood may lead to attributing negative experiences mostly to oneself (e.g. I am a bad person, I did something wrong, I probably deserve this), thus, directly impacting the development of self-esteem. During childhood, the development of core beliefs about the self and others is easily influenced by external experiences due to the inflexible cognitive style through which information is processed in children (Waller et al., 2007). Consequently, negative self-beliefs formed in childhood tend to be robust and difficult to change (Young, 1994). For instance, the participants perceived their self-esteem as a stable trait characteristic, holding the position that low self-esteem never changes, and it will define them forever, a concept in line with the relevant literature (Trzesniewski et al., 2006). These core beliefs in childhood are perceived as intolerable from the child. As a result, the need to develop protective coping strategies is created in order to survive in the invalidating environment and in an unsafe world, as has been proposed in the SPAARS-ED model (Fox & Power, 2009). Since the child has limited cognitive resources to adapt to their situation, the coping strategies are more likely to be of a detaching and avoiding nature, rather than focusing on thoughts and emotions that allow proper elaboration and resolution. In this study, this process is evident as the participants turned inward, finding comfort in food to alleviate negative emotions, and at the same time, they used dieting to stop the abuse.

The emotions reported as a result of non-physical abuse in the participants' accounts, are in line with the basic emotion perspective of the schematic, propositional, analogical and associative representational systems (SPAARS) model (Power & Dalgleish, 1997, 2008) and its extension to EDs (Fox & Power, 2009) (see Section 3.5.3 in Chapter 3). Anger is an important feeling in EDs (Fox, 2009; Fox & Power, 2009), and its presence and suppression

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is evident in the participants' accounts and in other qualitative studies (Fox, 2009). The intensity and dangerousness of anger is best reflected by Kate's comparison to a hand grenade. Unlike the AN participants in the study by Fox (2009), who appeared confused regarding who to blame for the overwhelming feeling of anger, the current study's participants reported anger aimed at their abusive caregivers and towards themselves for being unable to react, with the latter also creating sadness. The majority of this study's participants coupled anger with sadness, supporting other studies in which anger and sadness predicted eating pathology (Fox & Froom, 2009). Some of the current participants reported being disgusted with themselves for still behaving like victims and eating rather than reacting. Moreover, the perception of anger as threatening led participants to emotional inhibition, which agrees with other studies (Ioannou & Fox, 2009), as well as with the SPAARS-ED model (Fox & Power, 2009). Power and Dalglish (1999) proposed that sadness and disgust directed at the self create the depression observed in ED patients. It can be added here that it is the anger towards the low self-esteem self that causes these depressive feelings (see Section 3.5.4 in Chapter 3). Carol, who suffers from bulimia nervosa (BN), explained that she feels disgusted with herself for trying to find relief in food, and this causes her to vomit. Carol's experience is in line with the SPAARS-ED model (Fox & Power, 2009), which proposes that disgust in people with bulimic symptomatology is an automatic emotion. In the current study, it is also evident that anger is perceived as dangerous for interpersonal relationships. Therefore, it needs to be inhibited through the ED symptoms, which is in line with the relevant literature (Corstorphine, 2006; Fox, 2009; Fox & Power, 2009). The importance of anger in EDs is reflected in Kate's experiences, and in her case, the change in the target of her anger changed her ED symptoms. She explained that when she was angry at herself, feeling responsible for being abused, AN-R developed. When she realised during

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therapy that it was not her fault, and she started being angry at others, AN-B/P developed. This participant's experience and explanation could shed light on the shifting diagnosis seen in some ED patients. It could be hypothesised that the direction of anger alone or in combination with the nature of the abusive behaviour plays a role in this phenomenon. For instance, being angry at oneself leads to restriction, while being angry at others leads to bulimic symptomatology. However, only studies focused on the shifting of ED diagnosis could possibly offer an explanation. The participants also reported being unable to enjoy life and their accomplishments as a result of what they have been through and what they still endure. This experience could be related to their low self-esteem, resulting from their non-physical abuse, as studies indicate that self-esteem predicted life satisfaction over and above personality traits (Joshani & Afshari, 2011).

Bowlby (1988) stated that in a disrupted attachment in which parents do not allow negative emotional expression, children will learn to suppress their feelings in order to maintain attachment and survive. The participants of this study reported difficulty in managing and expressing negative emotions, turning to food for relief. They were 'eaten' by these emotions and were 'drowning' them with food. The link between attachment, emotion regulation and EDs is supported by the literature (Fox, 2009; Fox & Froom, 2009; Fox & Power, 2009). This study's participants experienced difficulties in forming and maintaining healthy relationships plus avoiding abusive ones. They all mentioned abusive partners whose behaviour was similar to that of their parents. Attachment is associated with childhood abuse and family dysfunction, and people who have experienced childhood abuse find it difficult to establish and maintain healthy intimate relationships in adulthood (Colman & Widom, 2004). Being let down by a significant other can cause betrayal trauma (Freyd, 1996), and people who have experienced this cannot easily detect signs of abuse later in life; thus, it is difficult

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for them to leave an abusive relationship (DePrince, 2005; Gobin & Freyd, 2009) as experienced by this study's participants, who had partners that negatively commented on their body image, and made them feel neglected and disliked. Moreover, Farber (2008) connected traumatic attachment with self-harm behaviour based on the notion that traumatic attachment to people that caused pain to the child leads to dissociation, and dissociation leads to self-harm. He proposed that self-harm manifestations, such as self-injury behaviour (SIB) and ED, are dissociated attempts to regulate affect and, ultimately, master the trauma through re-enactment. This vicious cycle could also explain why the participants find it difficult to detach themselves from their abusive relationships. For instance, Laura still wanted to be loved by her parents, and Betty described relationships with significant others as addictive. Farber's approach (2008) can also explain why the participants were still attached to the disorder, leading to ED maintenance. Kate explained that being detached from others and remaining attached to AN protects her from painful feelings. This is in line with other qualitative findings, according to which, AN protects the self (Olofsson et al., 2020; Williams et al., 2015). Kate's experience is also in line with the cognitive–interpersonal maintenance model of AN (Schmidt & Treasure, 2006), which maintains that AN is used to avoid close relationships that could cause painful emotions. Being attached to the disorder has been also associated with treatment resistance in eating pathology (Forsén Mantilla et al., 2019).

Attachment and emotion regulation are essentially connected, as attachment is a psychological factor responsible for the individual's self-regulatory process (Ford, 2005), and realisation and expression of emotions serve the purpose of preserving the relationship to the attached object (Scheidt & Waller, 2002). ED patients report insecure attachment to their parents, particularly to their mothers (Ward et al., 2000), and it is believed that the intense

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focus on food replaces the inadequate regulatory functions of early attachment (Farber, 2008; Pearlman, 2005). This is in line with the current study, as the participants emphasised their emotionally inadequate relationships with their mothers. For example, Amanda started eating a lot to gain her mother's love and acceptance, and Kate was disappointed with her mother for not helping Kate with her self-esteem. The literature indicates that a woman's trait self-esteem is affected by their relationship with their mother (Onayli & Erdur-Baker, 2013; Sholomskas & Axelrod, 1986). Studies also indicate a relationship between mother–daughter dynamics and positive body image development in adolescent girls (Usmiani & Daniluk, 1997), as well as a strong relationship between maternal warmth and female children's self-esteem (Ling et al., 2020).

The results from Studies 1 and 2 highlighted the importance of self-esteem in EDs, above any other factor (see Sections 5.4 and 6.4 of Chapters 5 and 6, respectively). Study 3's participants attributed their ED onset to the non-physical abuse they endured in childhood that caused their low self-esteem, and thus, their inability to defend themselves by expressing their emotions. This is in line with the literature, which suggests that people with high self-esteem can defend themselves (Greenberg et al., 1992). The participants attributed the continuation of non-physical abuse to their low self-esteem, which is 'visible' to others, who then treat them as victims. The 'visible' perception of low self-esteem is not a new concept in ED literature. When people without EDs were requested to identify the main problem of women with EDs, they reported low self-esteem instead of a mental health disorder (Mond et al., 2006; Mond et al., 2004a; Mond et al., 2004b). This indicates a match between ED patients' perception of their self-esteem and how people think about ED patients' self-esteem. The participants also defined themselves as weak and vulnerable because of their low self-esteem, and this is the reason they are angry at themselves. Kate discussed how her low self-

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esteem made her so angry at herself that she felt that she had to restrict her diet to improve her self-esteem, which led to loss of control and AN development. The non-improvement of their self-esteem maintains the disorder, as the disorder is still needed to protect the vulnerable self. The participants believed that self-esteem is stable and defines people's lives forever. Low self-esteem seems to be a key factor in both the onset and maintenance of EDs in this study's sample. In line with the participants' accounts, empirical studies have indicated that self-esteem is more important than emotion regulation in EDs (Monell et al., 2015; Monell et al., 2020). This is also reflected in Study 2's results, where self-esteem was the only mediator between childhood non-physical abuse and EDs in the clinical sample. While emotion dysregulation is considered responsible for ED symptoms, serving as a means to manage suppressed and intolerable affect, most notably anger (Fox & Power, 2009), it is important for therapy and research to clarify the origins of this anger. According to this study's participants, the anger stems from their childhood non-physical abuse, when they internalised the emotional abuse and neglect, thus, leading to low self-esteem. This intense anger is directed at the weak self, who is afraid to speak up and express emotions and is afraid to leave an abusive relationship. As Amanda put it: 'if I was strong and I had self-esteem would I need to eat to feel relief?'

The three psychological factors affected by childhood non-physical abuse, as demonstrated in this study, are the milestones of developmental psychology, and they are transdiagnostic in nature. These factors are related to each other; their impairment can lead to psychopathology onset, in this case EDs; they can affect psychological health and social relationships over the course of a lifetime and the continuation of their impairment maintains psychopathology.

7.5.1. Clinical, Theoretical and Research Implications

This study's results indicate which psychological factors could be addressed as standard in ED patients' psychotherapy. The interviewees described how self-esteem, attachment and emotion regulation are affected by childhood non-physical abuse, and therapists should, therefore, consider trying to identify and target these factors in their interventions at the beginning of the therapeutic procedure. Self-esteem's improvement should be a standard priority in ED patients' psychotherapy. Psychoeducating the patients regarding trauma connection to these psychological variables and how these variables affect their lives prior to individual psychotherapy onset would be helpful as it would increase patients' awareness and help them understand how a disturbed relationship with food works. This could be achieved in group sessions as a preliminary process before individual psychotherapy, preparing the patients for the psychological mechanism that will be addressed in their individual therapy. As a preventive measure, this procedure could be expanded to children and adolescents who have experienced such traumatic events, since we empirically know that disturbance in self-esteem, attachment and emotion regulation will lead, at the very least, to poor psychosocial quality of life (QoL) (poor psychological health and poor social relationships), and, in severe cases, will lead to psychopathology. If an individual has the time to develop positive self-perceptions, the impact of CEA, or even another type of abuse of one's self-perceptions, will be felt, but there is a great likelihood of recovery without destroying the self.

The literature indicates that the non-physical forms of childhood abuse are still overlooked in research and, consequently, in practice. For example, systematic reviews regarding childhood abuse and non-suicidal self-injury (NSSI) suggest that the non-physical

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forms are less considered (Liu et al., 2018). However, the findings of the current study reveal NSSI behaviour in Kate's case as a result of her anger at her low self-esteem self, and this anger was the result of the internalisation of her childhood non-physical abuse. Moreover, EDs could be perceived as a form of NSSI (Farber, 2008). The findings also indicate that childhood non-physical abuse could stand on its own in the psychology literature, as it seems able to affect important developmental factors. The literature on abuse focuses mostly on physical forms, probably because these types can be seen contrary to non-physical abuse, which is only visible in the psychology of the individual. Additionally, non-physical abuse is viewed as complementary to physical forms, which in a sense is correct, as it is difficult to have physical abuse without the non-physical forms being present. However, it is possible for someone to endure non-physical abuse without the presence of physical abuse. For example, AN patients reported neglect during childhood with minimal reports of physical abuse, and it was difficult for them to blame someone for their anger (Fox, 2009). This could be attributed to the fact that non-physical abuse is under-reported, probably because it is not well understood by the victim, as discussed earlier. Moreover, physical forms of abuse are more likely to occur over a distinct period of time and be perpetrated by specific people in the individual's life. In contrast, non-physical abuse is more likely to occur within a broader range of the person's social context. This is especially true when the abused individual displays impaired psychological factors that allow others to think they can psychologically maltreat the individual because they seem vulnerable, as was reported from these patients' accounts. This reveals a vicious cycle, meaning that the impaired self-esteem, attachment and emotion regulation resulting from abuse can actually enhance and maintain the abuse throughout the person's life. Thus, the literature might benefit from focusing separately on

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this psychological mechanism by treating these variables and their interplay not only as consequences of abuse but as the reason the abuse continues.

Due to limited research regarding non-physical abuse continuation in ED patients' adult lives, it is important to continue exploring this phenomenon, focusing on the consequences of abuse on self-esteem, attachment and emotion regulation via longitudinal studies. These studies could be expanded to populations who have suffered childhood non-physical abuse regardless of a diagnosis, as the abovementioned psychological factors are important for psychological development and are empirically linked to various psychopathology types.

The main theoretical implication involves the continuation of non-physical abuse into adult life due to the impairment of certain psychological factors. Moreover, this continuation relates to the maintenance of ED symptoms because of these impaired factors. Regarding the research implications, the present connection between childhood and adult non-physical abuse allows research to concentrate on the specific transdiagnostic psychological factors that are common to both ED onset and maintenance. These factors could also serve as protective factors when designing preventive programmes for EDs . The transdiagnostic nature of these risk and maintenance psychological factors support the argument of shifting attention towards the underlying transdiagnostic biopsychosocial processes in mental health (Dalglish et al., 2020). The current study's results also expand and offer empirical support to Linehan's (1993) concept of the invalidating environment, the SPAARS (Power & Dalglish, 1997, 2008) and SPAARS-ED (Fox & Power, 2009) models of emotions, and Corstorphine's (2006) emotional inhibition in EDs.

7.5.2. Limitations and Future Directions

The study used a small and fairly homogenous sample because idiography deals with elucidating individuals' lived experiences rather than generalisations of these experiences (Pringle et al., 2011; Smith & Osborn, 2003). The qualitative results cannot be statistically representative; rather, they contribute to existing empirical findings by further explaining them and setting the grounds for expanded research regarding the topic in question. Despite the fact that the population and context cannot be generalised, the concept and theory behind this study have grounds for analytical generalisation (Smith, 2018). In the current study, the theoretical assumption is that childhood non-physical abuse continues into ED patients' adulthoods, and this continuation may be caused by the impairment of self-esteem, emotion regulation and attachment during childhood. Furthermore, half of the study's participants had the same ED diagnosis rather than having one participant for each ED subtype, but there is no indication in the literature that there is any difference in the experience of non-physical abuse with respect to ED subtypes. Two participants reported experiencing a form of physical abuse, but the continuation of abuse from childhood through to adulthood involved only the non-physical abuse, which they emphasised throughout their interviews. It should be acknowledged that having experienced continuing non-physical abuse was part of the inclusion criteria for this study, as IPA's idiographic nature favours a purposive and homogeneous sample, seeking to form a group of people for whom the research question will be significant (Smith & Osborn, 2003). This means that while the results add detail to our overall understanding of the wider group, and will resonate with therapists dealing with similar clients, they do not describe the lives of all ED patients.

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It could be also argued that the researcher's clinical experience may have affected the data analysis and interpretation. However, the analysis was grounded in the data, and many quotations are almost self-explanatory. In agreement with other authors (Fox, 2009), it was the clinical experience that allowed deeper analysis in this study. It is also acknowledged that all the participants were undergoing therapy, meaning that psychotherapy could have facilitated the psychological connections they made during the interviews. However, being able to make psychological connections and understand oneself is the whole essence of psychotherapeutic treatment. If this study had employed participants who were not undergoing therapy, they might have answered differently. Therefore, Study 3 could be replicated employing ED patients who have been referred to but not yet begun psychotherapy. This way, we could explore how ED patients with no prior experience of psychotherapy would address the study's research questions, and how they comprehend their lived experiences with non-physical abuse and eating pathology. Such a study could provide valuable information regarding patients' insights at the beginning of psychotherapy, enabling clinicians' work. Moreover, research replication through quantitative studies would offer a more robust picture of these results. The inclusion of clinical populations other than ED patients, as well as the inclusion of victims of non-physical abuse without a diagnosis, could strengthen the generalisability of the psychological mechanism involved regarding the consequences of non-physical abuse.

In sum, this study contributes to the revelation and understanding of the phenomenon of non-physical abuse continuation in ED patients and highlights the ongoing consequences of non-physical childhood abuse on core psychological factors, such as self-esteem, attachment and emotion regulation. To the best of my knowledge, this study is the first to

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have conducted a qualitative exploration of this phenomenon, which has been neglected not only in ED studies, but in abuse-related studies as well. This in-depth exploration of patients' non-physical abuse experiences also adds to Studies 1 and 2, explaining and expanding their results, as the psychological factors used as mediators between childhood non-physical abuse, eating pathology and psychosocial QoL emerged qualitatively as factors affected by childhood non-physical abuse. Additionally, childhood non-physical abuse was further connected to ED onset and maintenance through the participants' experiences. Thus, the psychological mechanism that was theoretically proposed in this thesis's quantitative studies was supported by the patients' accounts, even though the current study was open to any issue that could be important to the patients. Therefore, even though the results cannot be statistically generalised, they may offer theoretical and conceptual generalisation (Smith, 2018) regarding the psychological mechanism involved in ED onset and maintenance. It is important to note that the transdiagnostic character of this thesis's variables was continued in this study as the participants belonged to all diagnostic categories.

7.6. Conclusion

This study captured the whole essence of this thesis, offering a phenomenological insight into ED patients' experiences of continuous non-physical abuse from childhood through to adulthood; non-physical abuse's consequences on self-esteem, attachment and emotion regulation; and the link to ED onset and maintenance. These results offer a clearer picture to clinicians who work with ED patients, as well as with non-physical abuse survivors, by highlighting the possible psychological mechanism regarding how this type of abuse may lead to psychopathology. Moreover, these results offer understanding regarding how the psychological factors affected by non-physical abuse preserve both the abusive

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behaviour towards the victims and the psychopathology symptoms. Since these risk factors are of a developmental nature and the developmental process is the same for all humans, a basic common underlying mechanism in mental health disorders could be further considered.

Chapter 8: General Discussion and Overall Conclusion

This thesis advances understanding of the onset and maintenance risk factors for eating disorders (EDs), adopting a transdiagnostic perspective of the underlying psychological mechanisms. A mixed-methods approach was employed—three structural equation modelling (SEM) studies and one interpretative phenomenological analysis (IPA) study—in order to enhance our understanding of non-physical abuse in eating pathology and psychosocial quality of life (QoL). The mediational effects of self-esteem, attachment, emotion regulation and temperament were explored, combining and expanding existing theories of EDs. This series of studies' results significantly contribute knowledge to both the fields of EDs and childhood abuse by providing empirical support for the existence of a transdiagnostic underlying mechanism of ED onset and maintenance, and for the pivotal role of childhood non-physical abuse in EDs and its continuation into adulthood. The current thesis filled a gap in the literature (Kimber, 2017). Furthermore, the transdiagnostic nature of the studies' design and their results advance the scientific literature regarding transdiagnostic underlying mechanisms, which is a contemporary need in mental health (Dalglish et al., 2020). Additionally, the literature about childhood abuse benefits from this thesis's results highlighting the importance of the overlooked non-physical forms of abuse, along with the core developmental factors affected by childhood non-physical abuse and their role in abuse's continuation in adulthood.

Theoretically pertinent correlates were tested in structural models to assess the fit of these models, which assume that certain factors cause EDs, most notably self-esteem, thus, identifying the significance of an important developmental factor, which is relevant to both EDs and the abuse literature. The fact that family dysfunction did not affect EDs unless self-

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esteem was present as a mediator in both groups (Study 1), along with the fact that self-esteem was the only mediator of eating pathology and the strongest mediator of psychosocial QoL in ED patients (Study 2a) supports Silverstone's (1992) suggestion that EDs are a symptom of low self-esteem. This concurs with this thesis's assertion that an individual with good self-esteem is unlikely to develop an eating disorder. Despite the belief that childhood emotional abuse (CEA) is involved in ED development through its impact on emotion regulation and self-esteem (Groleau et al., 2012), only self-esteem had this significant role in Studies 2a's and 3's clinical sample. Therefore, based on the combined results of its studies, this thesis argues that non-physical abuse, which is a direct attack of the self, lowers self-esteem through disrupted attachment with significant others, and the low self-esteem affects proper emotion regulation processes. Low self-esteem creates a weak and vulnerable self, prohibiting emotional expression and regulation; thus, maladaptive behaviours for emotional relief are employed to tolerate negative affect and elevate self-esteem. Yet, this thesis does not imply that all people with low self-esteem will develop an ED. Other factors are needed in order to clarify the potential consequences and manifestation of low self-esteem on mental health. In the case of EDs, one such factor could be repeated comments about physical appearance and an emphasis on food conveyed at a young age (Jones, 1985), as was revealed in the ED patients' experiences in Study 3. This could probably explain self-esteem's association with weight and body image that leads to unhealthy dieting efforts (see Section 3.5.4 in Chapter 3).

In Study 2b, in both the patient and control groups, childhood non-physical abuse had the strongest impact on attachment rather than on temperament, and attachment had a bigger direct effect on self-esteem, eating pathology, psychosocial QoL and emotion regulation

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compared to temperament. This indicates that non-physical abuse plays an important role in attachment; thus, attachment's inclusion in the adapted biosocial theory model is beneficial, informing the relevant literature. Previous theorists have argued that various factors, including the individual's early environmental experiences and their temperament (Rorty & Yager, 1996; Waller et al., 2007), play an important role in explaining how the relationship between non-physical childhood abuse and EDs impacts the individual. Studies 2a and 2b empirically tested alternative theoretical models that incorporated various important psychological factors and potential psychosocial consequences in an effort to address this relationship and simultaneously extend Linehan's (1993) biosocial theory. Study 3 explained and expanded these findings by analysing in-depth the ED patients' lived experiences of non-physical abuse. Study 3 highlighted the continuation of non-physical abuse in adult life of ED patients who experienced a connection between non-physical abuse, attachment, self-esteem, emotion regulation and ED maintenance. According to their experiences, these three developmental factors affected by childhood non-physical abuse are related to ED onset and maintenance, as well as to the continuation of abuse. The ongoing non-physical abuse preserves the ED symptoms, creating a vicious cycle from which patients cannot easily escape, delaying a positive therapeutic outcome. This could explain chronicity and treatment resistance in EDs, or else maintenance. Together, this thesis's results provide a comprehensive explanation of a complex disorder.

The reasoning behind the order of the studies was explained earlier in the thesis (see section 4.3.1 of Chapter 4). Explaining the logic that underpins the design of mixed methodology is important for quality and transparency of mixed methods in research (Wisdom et al., 2012). It should be acknowledged that while the depth and richness of

information could not have been possible without the qualitative study at the end of the thesis, one limitation of including a qualitative study after a quantitative one is the potential for the results of the quantitative study to influence the researcher's subjective interpretation of qualitative data. This limitation was carefully considered during all stages of Study 3 (see sections 7.3.1; 7.3.3; 7.3.5; 7.5.2 of Chapter 7). Undoubtedly, IPA's double hermeneutic procedure involves subjectivity as the researcher by definition has to interact with the participants, who also influence the meaning and interpretation of data (Brocki & Wearden, 2006). However, subjectivity is central to qualitative studies as it is considered the strength of the interpretive process and researchers constitute a structural part of the research process and outcome (Galdas, 2017). Therefore, their detachment (as opposed to quantitative studies) is neither possible nor compatible with the nature of the qualitative methodology (Galdas, 2017). What is feasible though is the implementation of multiple techniques to avoid research bias such as, careful interview ensuring the absence of any leading questions, peer review of the results, transparency and reflexivity (Galdas, 2017). These techniques were employed in Study 3 and are addressed in Chapter 7. In sum, even though both quantitative and qualitative methods have limitations and the order of the studies is important to consider, they each have strengths and can be effective in explaining complex psychological phenomena when combined. Qualitative methods are important in validating and contextualising data collected via quantitative methods especially in psychologically sensitive topics (Ruark, & Fielding-Miller, 2016) and the qualitative exploration that expands quantitative results can be found in published literature (e.g. Deasy et al., 2014; Lindsay-Smith et al., 2018; Odom et al., 2006; Salvo et al., 2018). Therefore, mixed methods were utilised in this thesis to elucidate qualitatively how the quantitatively derived mechanism of childhood non-physical abuse might operate with respect to eating pathology.

Theoretical Contributions

This thesis's proposed theoretical model is empirically valid, it can be re-tested, and is comprehensive and parsimonious, offering applied and heuristic value (Cramer, 2013). The theoretical psychological mechanism identified through this series of studies is that non-physical childhood abuse has an effect on eating pathology and psychosocial QoL in adult life through self-esteem, emotion regulation, attachment and temperament, with self-esteem standing out as the most important factor for ED patients. These variables combined also capture the essence of the traumatic consequences stemming from childhood abuse. In other words, persistent and recurrent child abuse leads to the violation of boundaries, trust issues, feelings of betrayal and a diminished sense of self, which then leads to difficulties in managing strong emotions and, consequently, to the adaptation of maladaptive coping strategies (Follette et al., 1998; Freyd, 1996; Goldsmith et al., 2004). Moreover, these mediators are important psychological factors linked to other psychopathology types, indicating that this proposed theoretical mechanism model for EDs (non-physical abuse → poor attachment, low self-esteem, emotion dysregulation → psychopathology) could be tested for other mental disorders too. The similarities between the patients and the controls regarding the findings indicate that these models could work independently of diagnostic levels of EDs. It is also evident that in cases of childhood non-physical abuse, the lifelong psychological consequences should be targeted in treatment.

In sum, this thesis offers a plausible and coherent explanation regarding possible ED onset and maintenance factors and proposes the underlying psychological mechanism, which appears to be the same in both cases. There are not many studies examining ED maintenance (Wonderlich et al., 2012) that also identify the correlated psychological factors. This research

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advances the collective empirical understanding of EDs and informs recommendations for the prevention and treatment of complicated and resistant disorders, such as EDs. Additionally, the reconceptualisation of the broadly defined family dysfunction and invalidating environment as recurrent childhood non-physical abuse is a conceptually important shift and answers concerns regarding how a specific feature of the invalidating environment is linked with EDs (Haslam, 2012). This reconceptualisation also resolves the arbitrary research assumption of family dysfunction as emotional abuse (Kent & Waller, 2000). Dysfunctional family dynamics do not necessarily lead to psychopathology, as more psychological factors are needed for this to occur, and focusing on specific and recurrent non-physical abusive patterns can offer a better and more coherent explanation to the much-discussed role of family in EDs. Furthermore, the thesis's findings support the SPAARS (Power & Dalgleish, 1997, 2008) and SPAARS-ED (Fox & Power, 2009) models of emotion, conceptualising them in a developmental model. It is important to note that even though Linehan's (1993) influential biosocial theory has been tested in terms of the effectiveness of dialectical behaviour therapy (DBT) in ED and borderline personality disorder (BPD) patients, it has not been tested in a mediational model as a theory *per se* for ED populations. This gap was filled by Study 2b, which also empirically highlights the transdiagnostic nature of biosocial theory. Finally, Study 3 adds to the literature by elucidating for the first time the continuation of childhood non-physical abuse in adult ED patients, simultaneously offering valuable insight into the phenomenon and its consequences on ED maintenance.

Clinical Implications

This series of studies' results inform clinical practice because they identify the key correlates that can exacerbate the risk of developing and maintain EDs, which is important to

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effectively design prevention and treatment programmes. Educating ED patients about the link between negative past experiences and their current difficulties with EDs will enable them to understand the mechanism of the disorder and how it works as a maladaptive coping strategy. It is essential to first understand why a problem is occurring before trying to resolve it. This way, treatment resistance could be diminished as the patient will be able to consciously join the therapist in the therapeutic effort (Plutchik, 2000). Self-esteem enhancement and emotion regulation skills should be part of standard treatment, not only to accelerate therapy, but also because the patient needs to understand these factors' connection with the ED, as well as the underlying psychological mechanism. This way, patients will more readily understand where their feelings are coming from, properly identify them and then handle them (Plutchik, 2000). In other words, a developmental model of emotion regulation (Cicchetti et al., 1995) is not only essential for scholars but for patients as well. Patients also need to understand why it is difficult for them to trust and attach to other people and why they experience certain feelings with intensity (e.g. anger), which they are afraid to express for fear of rejection. Corrective experiences formed during psychotherapy can offer valuable help (Castonguay & Hill, 2012), as these patients, for the most part, have experienced abandonment and rejection. An elevated self-esteem, and better emotion identification, acceptance and regulation along with transference and counter-transference in therapy (Westerling et al., 2019) will also encourage a better attachment pattern. Therapists should address the true *imperfect* self in a holding environment created in therapy to help patients understand that the maladaptive coping strategies of the false self are no longer needed (Bion, 1962; Winnicott, 1965). Given the developmental nature of the factors involved in EDs, cognitive analytic therapy (CAT) (Ryle, 1991) might be more beneficial for ED patients than cognitive-behavioural therapy (CBT) (e.g. Treasure & Ward, 1997), as it

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combines cognitive and psychodynamic psychotherapies (Wicksteed, 2016). CAT is informed by object relations theory (Ryle, 1991). Based on this thesis's results, psychological elaboration and links between core developmental factors could offer more consistent therapeutic outcomes.

This thesis also proposes that the negative, omnipotent and bullying 'ED voice' (Aya et al., 2019; Pugh & Waller, 2016; Tierney & Fox, 2010) is actually the pathogenic critic (Sagan, 1967) of low self-esteem, which focuses on success and failure related to body control since there an ED has developed. By improving self-esteem, this voice will not have grounds to exist, and the pride resulting from food restriction (Faija et al., 2017) will no longer be necessary to elevate self-esteem.

This thesis proposes that treatment's primary focus should be placed on self-esteem and then proceed to improving emotion regulation skills. Cognitive-behavioural approaches, along with mindfulness and compassion-focused therapies (Maric et al., 2019), could be used to design an intervention focused on self-esteem. DBT skills (Linehan, 1993) could be used for emotion regulation in combination with self-esteem's improvement. The absence of self-esteem's improvement as a consideration in DBT skills could be the reason this intervention offers little benefit to improving emotion regulation (Harvey et al., 2019). Elevated self-esteem is needed first in order for patients to be able to express their feelings without fear of the consequences (e.g. the other person's reaction, rejection, abandonment, the ruin of the relationship), as a high self-esteem can defend itself (Greenberg et al., 1992). Then, every small or big emotion regulation accomplishment should be checked and linked with how this made the patient feel about themselves (self-esteem). A strong self-esteem that can express emotions and handle adverse situations does not need to control the body in order to be

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maintained and experience pride. Moreover, a good self-esteem will not be entrapped in abusive relationships, including the relationship with the disorder (Tierney & Fox, 2011). If we accept that the ED voice is the pathogenic critic, it is this voice (the voice of low self-esteem) that maintains the abusive relationship. This voice was created early in life and was incorporated into the self as a result of recurrent non-physical abuse. In other words, emotionally abusive comments and neglectful experiences coming from significant others (poor attachment) developmentally manifested as internal intrusive negative thoughts (low self-esteem's pathological critic) that left the individual vulnerable to any kind of abusive relationship. In the case of EDs, and in any other self-harm-related disorder, this abusive relationship is with the self as a result of a tremendous, unexplained, unresolved and unexpressed anger. This anger is safely expressed at the self so that relationships with significant others are not jeopardised. The anger is also addressed at the vulnerable self for not being able to react when needed. Understanding the anger's origins and its link with self-esteem will allow its regulation, tolerance and expression through therapy. Anger is the most important emotion in EDs, coupled with sadness, with empirical findings supporting this concept (Fox & Froom, 2009). It has been suggested that depression is the result of anger and sadness due to the loss of a significant other (Bowlby, 1980; Freud, 1917), and studies confirm that depression can result from unexpressed anger (Mook et al., 1990). Freud (1930) also added that depression is aggression directed at the self. Empirical findings support this theory (Haddad et al., 2008). Using a psychodynamic interpretation, it could be proposed that the loss in the case of EDs is both the loss of the true self because of the betrayal of a significant other, and the loss of the significant other's love and approval. This loss creates anger both towards the significant other and towards the vulnerable self, who is unable to react and unworthy of love. Sadness caused by unexpressed anger due to low self-esteem

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could be the factor responsible for the negative mood states observed in ED patients (e.g. depression), as has already been proposed (Power & Dalgleish, 1999). Personality characteristics, such as self-esteem, become more stable and difficult to change as we mature (Donnellan et al., 2012; Kuster & Orth, 2013; Trzesniewski et al., 2003). This means that efforts to improve self-esteem would be easier and more successful in childhood and adolescence (Krauss et al., 2020). Thus, in terms of prevention, self-esteem enhancement programmes, combined with emotion regulation skills, should be implemented in schools and linked with emotional expression. This thesis holds the position that many disorders could possibly be prevented this way, not just EDs.

This thesis's results are also important in alerting therapists' to the importance of overlooked non-physical abuse, both in childhood and adulthood, which should be part of therapists' clinical evaluation instead of looking for single-case traumatic incidents (Goldsmith et al., 2004). Therapists should also be cautious of the fact that even though the physical forms of abuse are more easily communicated by the patient as conscious and physically painful events, this is not usually the case with non-physical abuse. The fact that Study 3's participants said that they are used to non-physical abuse, referring to it as a familiar situation (meaning they know how to live with it), explains its continuation in adult life through repetition compulsion (Freud, 1920). It may explain why they stayed with abusive partners, highlighting another difficulty, traumatic attachment continuance. Attachment with at least one caregiver is important for human survival. When the caregiver fails to provide support in affect regulation and meet the child's demands, the child forms its own coping mechanisms to regulate overwhelming arousal in insecurity (Bowlby, 1969, 1973, 1979, 1988). Then the child is led to an adaptation of a familial environment that they

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cannot escape from nor change but must survive (Goldsmith et al., 2004). Combining this with the nature of non-physical abuse forms that do not cause physical pain and that people may adapt to can lead to a lack of trauma awareness and under-reporting or no reporting in therapy. This is because children may separate adverse experiences from consciousness, especially from their significant others who betrayed them, in order to preserve attachment and survive (Goldsmith et al., 2004). Taking into account the possible lack of awareness that accompanies childhood traumatic experiences (Goldsmith et al., 2004), along with the lack of awareness in eating pathology, most notably in AN-R (Merwin et al., 2010), cognitive-focused techniques would be helpful in treatment. These techniques—emphasising awareness of thought processes, and their relationship to emotions and behaviour—will help patients understand how negative beliefs cause intolerable distress, keep their self-esteem low, maintain ED symptoms and reinforce their concept of a dangerous world (Follette et al., 1998). In other words, cognitive-focused techniques could help them understand the effects of recurrent non-physical abuse on self-esteem, emotion regulation and attachment and how all these factors operate in a cycle to maintain ED symptoms. This suggestion could resolve the alarming phenomenon whereby QoL does not improve in line with the reduction of ED symptoms, even after years of treatment. These findings in the literature regarding QoL (see Section 2.4.2 in Chapter 2) indicate that the factors for ED onset and maintenance have never been properly identified and targeted in treatment for any diagnostic subtype.

Because EDs are chronic in nature and resistant to treatment, along with the fact that they cause additional problems to already-vulnerable family dynamics, family interventions should be considered for adult patients as well. Rather than relying on traditional family therapy, which can be lengthy, a quicker approach could be considered, aiming at the

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psychoeducation of all the family members regarding the disorder, reducing the stress of caregiving, improving their communication and emotional expression skills, and their overall function as a group. This model (Falloon, 2003; Falloon et al., 1993) has been effectively used in major mental disorders, such as schizophrenia and bipolar disorder, but its flexible design can be adjusted to suit any mental or physical chronic illness. Given the transdiagnostic nature of the proposed models and their results in this thesis, this intervention can be applied to any ED type, as well as to families with a member at risk of developing an ED. It is important to mention that in both empirical studies, a significant number of patients and controls lived with their families, and this may have played a role in the results. However, family dynamics are powerful enough to affect people even when they leave the family home (Forward, 1989), and the financial ability to live independently from parents has changed over the last few years in Britain and the US (Bayrakdar & Coulter, 2018; Matsudaira, 2016; US Census, 2017). This indicates that familial relationships should be considered in adult psychotherapy instead of just in adolescent psychotherapy.

Future Research

It is important to note that the differences among the ED subtypes were discussed and acknowledged earlier in the thesis. All the studies recruited officially diagnosed ED patients, covering all the subtypes, to form the patient groups to test the proposed theoretical models. This indicates that the important developmental psychological factors mentioned and researched in this thesis may relate to the onset and maintenance of EDs, regardless of the subtype. An extension of this research would be to identify the specific factor(s) that lead to the development of a specific subtype rather than another. For instance, maybe the identity of the significant others responsible for the non-physical abuse (mother, father or both) plays a

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role in the subtype manifestation, as Study 3's participants emphasised their mothers. Additionally, the absence or presence of any other attachment figure that could shield the individual from the negative impact of stressful events (social buffering) could possibly play a role in the subtype or at least in the differentiation between the restrictive and purging behaviours. Furthermore, the traits of autism observed in AN could be a contributing factor for this subtype requiring further exploration. The consciousness of the origins of anger or lack of it could be another factor differentiating the ED symptoms. For instance, anger towards the weak self could be responsible for food restriction, and anger towards abusive significant others could be responsible for purging. Longitudinal studies based on the proposed psychological mechanism could offer valuable insight regarding its possible causal nature. Moreover, a psychometrically valid questionnaire measuring the continuation of non-physical abuse in adult life could be developed, as this would be helpful to both research and practice. In addition, further research on coupled emotions in EDs could elucidate the specific emotional couplings with the conscious origin of anger for each subtype. For example, the conscious origin of anger coupled with disgust could manifest as BN and BED, while the unconscious origin of anger coupled with sadness could manifest as AN. Disgust and sadness could be further explored as this thesis proposes that both emotions aim at the *imperfect* self which is created by low self-esteem. Finally, this thesis recommends revisiting the relationship between depression and EDs, and considering self-esteem when exploring mood states and associated emotions in ED patients. Based on the discussion about the relationship between depression and EDs (see Section 3.5.4 in Chapter 3), along with this thesis's findings, it could be suggested that the depressive mood observed in EDs is attributable to low self-esteem and that eating pathology is not the result of depression but the result of very low self-esteem.

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In conclusion, this thesis's combined results contribute clinically and theoretically to the field of EDs and abuse by highlighting the presence of non-physical abuse along with certain risk factors and exploring how these factors are common for all ED subtypes, contributing to both the onset and maintenance of the disorder. Future research could determine the similarities and differences of the proposed underlying psychological mechanism with respect to the ED subtypes. Furthermore, the fact that the same factors were identified in the non-clinical population may also indicate that the psychological mechanism proposed in this thesis is core and transdiagnostic regarding mental health and has value for prevention. These findings have important implications in directing research in clinical settings to gain a better understanding of the factors contributing to ED symptomology. Finally, this thesis's results constitute scientific evidence regarding the serious consequences of non-physical abuse in childhood and adulthood on mental health that could be used as a reference for stricter laws and policies.

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Appendix 1 - Ethics approval for Studies 1 and 2



26th February 2015

Kathryn Jane Gardner/Olga Theodoropoulou
School of Psychology
University of Central Lancashire

Dear Kathryn/Olga,

Re: PSYSOC Ethics Committee Application
Unique Reference Number: PSYSOC 185

The PSYSOC ethics committee has granted approval of your proposal application 'Nonphysical Forms of Childhood Abuse in Eating Disorder Patients: Effects on Psychosocial Quality of Life and Eating Pathology and the Role of Self-Esteem, Emotion Regulation and Attachment'. Approval is granted up to the end of project date* or for 5 years from the date of this letter, whichever is the longer. It is your responsibility to ensure that

- the project is carried out in line with the information provided in the forms you have submitted
- you regularly re-consider the ethical issues that may be raised in generating and analysing your data
- any proposed amendments/changes to the project are raised with, and approved, by Committee
- you notify roffice@uclan.ac.uk if the end date changes or the project does not start
- serious adverse events that occur from the project are reported to Committee
- a closure report is submitted to complete the ethics governance procedures (Existing paperwork can be used for this purposes e.g. funder's end of grant report; abstract for student award or NRES final report. If none of these are available use [e-Ethics Closure Report Proforma](#)).

Yours sincerely,

A handwritten signature in black ink that reads "K. Chantler".

Khatidja Chantler
Chair
PSYSOC Ethics Committee

Appendix 2 – Ethics approval for Study 3



19 November 2018

Kathryn Gardner / Olga Theodoropoulou
School of Psychology
University of Central Lancashire

Dear Kathryn / Olga

Re: PSYSOC Ethics Committee Application
Unique Reference Number: PSYSOC 185 Study 3

The PSYSOC ethics committee has granted approval of your proposal application 'Of life and eating pathology and the role of self-esteem, emotion regulation and attachment'. Approval is granted up to the end of project date.

It is your responsibility to ensure that

- the project is carried out in line with the information provided in the forms you have submitted
- you regularly re-consider the ethical issues that may be raised in generating and analysing your data
- any proposed amendments/changes to the project are raised with, and approved, by Committee
- you notify EthicsInfo@uclan.ac.uk if the end date changes or the project does not start
- serious adverse events that occur from the project are reported to Committee
- a closure report is submitted to complete the ethics governance procedures (Existing paperwork can be used for this purposes e.g. funder's end of grant report; abstract for student award or NRES final report. If none of these are available use [e-Ethics Closure Report Proforma](#)).

Yours sincerely

A handwritten signature in black ink, appearing to read "Christine Barter".

Christine Barter
Vice-Chair
PSYSOC Ethics Committee

* for research degree students this will be the final lapse date

NB - Ethical approval is contingent on any health and safety checklists having been completed and necessary approvals gained as a result.

Appendix 3 – Participant information sheet for Study 1

PARTICIPANT INFORMATION SHEET

Information about the researcher

My name is Olga Theodoropoulou and I am a PhD student in Psychology at the University of Central Lancashire, UK. I am a clinician and an external associate at the Eating Disorders Unit of Eginition hospital at the Medical School's 1st Psychiatric Clinic of Athens National and Kapodistrian University.

What the study is about

The present study is part of a doctorate in Psychology. This research is looking at food related behaviour and your participation is asked in order to better understand the factors that affect and disturb our relationship with food intake, as these factors contribute to the development of eating disorders and impact quality of life. During the past ten years eating disorders have increased and research in this field is needed in order to design more effective treatment programmes. Research findings indicate that family function and self-esteem have an effect on eating disorders development and on the quality of life of people with an eating disorder. The questionnaires you are asked to fill in measure those factors and their exploration will significantly contribute to the understanding of eating disorders.

What happens if I participate?

You will be asked to fill in 4 questionnaires that measure eating behaviour, quality of life, self-esteem and family functioning. You will need about 30-40 minutes for the completion. Your name will not be written on the questionnaires. Data will be collectively analysed and not individually, and will be seen only by myself, my project supervisor, markers of the PhD work and any other individual with a legitimate academic need (e.g., academics collaborating on the project). Data will be held confidential and will be stored in a secure place. It will be destroyed after 5 years. Some of the questions could possibly cause discomfort as they are asking about potentially sensitive topics such as your family. If you feel discomfort or distress you can call to 210-7289400 and book an appointment with a psychologist or a psychiatrist in a public hospital. Alternatively, you can find information about low cost therapy places on this website <http://www.psychotherapeia.net.gr/2-static/132-yphresies-dwrean-psyxologikhs-yposthrijhs> or by contacting this helplines: 197 or 210-7222333 or 210 - 5234737.

Do I have to take part?

No, participation is voluntary (there will not be any financial gain) and you may choose not to answer the questionnaires despite beginning to fill them in without offering an explanation. If this happens your data will be disregarded immediately.

What if I change my mind later?

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If you change your mind at a later point, you can withdraw your data by contacting me through an SMS in my mobile phone (6977 955 477) and giving me your code within 4 weeks from the questionnaires completion (the code is asked in the demographic information sheet for this reason and will consist of the first letter of your name, the first letter of your father's name, the first letter of your last name and the last two digits of your birth year), so I can delete your data. The latest you can withdraw is August 2015.

Who to contact for further information?

You can contact me in the following e-mail: otheodoropoulou1@uclan.ac.uk or my supervisor Dr. Jane Gardner at kjgardner@uclan.ac.uk

Who to contact in case of complaint?

Alternatively, if you have any concerns about the research that you wish to raise with somebody who is independent of the research team, you should raise this with the University Officer for Ethics: OfficerForEthics@uclan.ac.uk

Appendix 4 – Debriefing sheet for Study 1

DEBRIEFING FORM

During the past ten years eating disorders have increased and research in this field is essential in order to understand related factors that affect the onset of the eating disorders, as this will enable the design of more effective treatment programmes. Research findings indicate that family function and self-esteem have an effect on eating disorders development and on the quality of life of people with an eating disorder. The questionnaires you filled in measure those factors.

The exploration of these factors will contribute significantly to the understanding of eating disorders and it could not be achieved without your help. I would like to thank you for your decision to participate and contribute to the research field of eating disorders.

The present study is part of a doctorate in Psychology and is being conducted by a psychologist. You have the right to withdraw your data within 4 weeks from the completion of questionnaires. If you have any further questions or if you feel distressed from the procedure please contact the principal investigator Ms. Olga Theodoropoulou at: otheodoropoulou1@uclan.ac.uk

Supervisor: Dr. Kathryn Gardner; kjgardner@uclan.ac.uk

Some of the questions could possibly cause discomfort as they are asking about potentially sensitive topics such as your family. If you feel discomfort or distress you can call to 210-7289400 and book a free appointment with a psychologist or a psychiatrist. Alternatively, you can find information and support on this website <http://www.psychotherapeia.net.gr/2-static/132-yphresies-dwrean-psyxologikhs-yposthrijhs> or by contacting this helplines: 197 or 210-7222333 or 210 -5234737.

Alternatively, if you have any concerns about the research that you wish to raise with somebody who is independent of the research team, you should raise this with the University Officer for Ethics: OfficerForEthics@uclan.ac.uk

Appendix 5 – Participant information sheet for Study 2

PARTICIPANT INFORMATION SHEET

Information about the researcher

My name is Olga Theodoropoulou and I am a PhD student in Psychology at the University of Central Lancashire, UK. I am a clinician and an external associate at the Eating Disorders Unit of Eginition hospital at the Medical School's 1st Psychiatric Clinic of Athens National and Kapodistrian University.

What the study is about?

The present study is part of a doctorate in Psychology. It is looking at food related behaviour and your participation is asked in order to better understand the factors that disturb our relationship with food intake contributing to the development of eating disorders and impacting quality of life. During the past ten years eating disorders have increased and research in this field is needed in order to design more effective treatment programmes. Research findings indicate that childhood abuse, emotion regulation, temperament, attachment and self-esteem have an effect on eating disorders development and on the quality of life of people with an eating disorder.

What happens if I participate?

You will be asked to fill in 8 questionnaires measuring eating behaviour, quality of life, self-esteem, temperament, personality traits, emotion regulation, attachment and childhood abuse. You will need about 60 minutes for the completion. Your name will not be written on the questionnaires. Data will be collectively analysed and not individually, and will be seen only by myself, my project supervisor, markers of the PhD work and any other individual with a legitimate academic need (e.g., academics collaborating on the project). Data will be held confidential and will be stored in a secure place. It will be destroyed after 5 years. Some of the questions could possibly cause discomfort as they are asking about potentially sensitive topics such as self-harm, suicide and childhood abuse. If you feel discomfort or distress you can call to 210-7289400 and book an appointment with a mental health professional in a public hospital. Alternatively, you can find information about low cost therapy places on this website <http://www.psychotherapeia.net.gr/2-static/132-yphresies-dwrean-psyxologikhs-yposthrijhs> or by contacting this helplines: 197 or 210-7222333 or 210 -5234737.

Do I have to take part?

No, participation is voluntary (there will not be any financial gain) and you may choose not to answer the questionnaires despite beginning to fill them in without offering an explanation. If this happens your data will be disregarded immediately.

What if I change my mind later?

If you change your mind at a later point, you can withdraw your data by contacting me through an SMS in my mobile phone (6977 955 477) and giving me your code within 4 weeks from the questionnaires completion (the code is asked in the demographic information sheet for this reason and will consist of the first letter of your name, the first letter of your father's name, the first letter of your

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last name and the last two digits of your birth year), so I can delete your data. The latest you can withdraw your data is August 2016.

Who to contact for further information?

You can contact me in the following e-mail: otheodoropoulou1@uclan.ac.uk or my supervisor Dr. Jane Gardner at kjgardner@uclan.ac.uk

Who to contact in case of complaint?

If you have any concerns about the research that you wish to raise with somebody who is independent of the research team, you should raise this with the University Officer for Ethics: OfficerForEthics@uclan.ac.uk

Appendix 6 – Debriefing sheet for Study 2

DEBRIEFING FORM

During the past ten years eating disorders have increased and research in this field is essential in order to understand related factors that affect the onset of the eating disorders, as this will enable the design of more effective treatment programmes. Research findings indicate childhood abuse, emotion regulation, temperament, attachment and self-esteem have an effect on eating disorders development and on the quality of life of people with an eating disorder. The questionnaires you filled in measure those factors.

The exploration of these factors will contribute significantly to the understanding of eating disorders and it could not be achieved without your help. I would like to thank you for your decision to participate and contribute to the research field of eating disorders.

The present study is part of a doctorate in Psychology and is being conducted by a psychologist. You have the right to withdraw your data within 4 weeks from the completion of questionnaires. If you have any further questions or if you feel distressed from the procedure please contact the principal investigator Ms. Olga Theodoropoulou at: otheodoropoulou1@uclan.ac.uk

Supervisor: Dr. Kathryn Gardner; kjgardner@uclan.ac.uk

Some of the questions could possibly cause discomfort as they are asking about potentially sensitive topics such as childhood abuse, self-harm, suicide. If you feel discomfort or distress you can call to 210-7289400 and book a free appointment with a psychologist or a psychiatrist. Alternatively, you can find information and support on this website <http://www.psychotherapeia.net.gr/2-static/132-yphresies-dwrean-psyxologikhs-yposthrijhs> or by contacting this helplines: 197 or 210-7222333 or 210 -5234737.

Alternatively, if you have any concerns about the research that you wish to raise with somebody who is independent of the research team, you should raise this with the University Officer for Ethics: OfficerForEthics@uclan.ac.uk

Appendix 7 – Participant information sheet for Study 3

INFORMATION FOR PARTICIPATION IN RESEARCH

This is a research project on non-physical forms of abuse and your voluntary participation is very important and essential in order to understand better the factors that may/may not affect and disturb our relationship with food intake, as well as how they relate to eating disorders and quality of life. If you believe you have experiences of non-physical forms of abuse (emotional neglect, emotional/psychological abuse, physical neglect) both in childhood **and** nowadays in your adult life, you can participate in the research as the questions will move around in this context.

Your answers will be anonymous and a pseudo name will be used in the analysis. Yet, I am interested in your experiences and may use pseudonymised quotes in my work or publications. All of the information collected during the interviews will be kept confidential, and data will be transferred using a data key (i.e., a unique participant number) rather than your name. Audio recordings will be transferred to and stored on my password-protected computer and will be destroyed right after the completion of my PhD (immediately following transfer to the computer the recording will be securely deleted from the recording device and the tape will be destroyed, in accordance with UCLan's policy on the secure disposal of electronic data). The anonymised written transcript will be stored in my password-protected computer during the research. The consent forms with your name on will be stored in a locked cabinet in which I am the only person with access and linked only to your data by participant number. When the research is completed the transcript will be stored for a minimum of 5 years and then it will be destroyed. You will have the chance to see the transcript and feel assured that you are not identified in the text.

The members of your clinical team will not be aware of the interview content. There are a few exceptions to confidentiality though. For example, if you report information that suggests that you or somebody else is at risk of serious harm I may need to discuss this information with a member of your clinical team. We will also need to follow procedures to maintain your safety and the safety of others which may involve discussing the information with other professionals and making appropriate referrals, in accordance with the duty of care policy. If we do need to contact others about some of the information that you provide we will talk to you about this first.

Your participation will be voluntary and you may choose to withdraw from the interview on the spot. If you change your mind at a later point you can withdraw your data by contacting me via e-mail (otheodoropoulou1@uclan.ac.uk) giving your code (first letter of your name, first letter of your last name and your birth date) so I can delete your data within 7 days from the end of the interview. Withdrawal after 7 days will not be possible. If you accept participation please inform your therapist handing your signed consent and an appointment will be sent for the interview. If you have any further questions please contact the investigator Ms. Olga Theodoropoulou at otheodoropoulou1@uclan.ac.uk. This research has received ethical approval from the Ethics Committee of the University of Central Lancashire. Thank you for your participation.

Appendix 8 – Debriefing sheet for Study 3

DEBRIEFING FORM

During the last years the prevalence of eating disorders has increased and study in this field is essential in order to understand related factors that affect their onset so to design more effective treatment programmes. Relevant research suggests that non-physical abuse in childhood and adulthood may have an effect on quality of life of people with an eating disorder.

The exploration of these factors contributes significantly in the understanding of this disorder and it could not be materialized without your help. I would like to thank you for your decision to participate and contribute to the research field of eating disorders.

If some of these questions caused you distress you may contact the principal investigator Ms. Olga Theodoropoulou at otheodoropoulou1@uclan.ac.uk. If you wish, you can e-mail me 6 months after the interview to receive feedback on the results.

The present study is part of a doctorate in Psychology at the University of Central Lancashire and is conducted by a psychologist.

If you have any concerns about the research that you wish to raise with somebody who is independent of the research team, you should raise this with the University Officer for Ethics (OfficerForEthics@uclan.ac.uk).

Appendix 9 – Interview Schedule for Study 3

Screening: female adult ED outpatients currently in therapy that have stated experience of non-physical abuse both in childhood and in adult life. Duration 45-50 minutes and it will be arranged to have their session with their own therapist after the interview. It will take place in the outpatient unit and will be conducted in Greek.

Questions:

At first some questions on age, education, working status, duration of illness, age of ED onset (see last page).

1. Could you tell me about your experience of non-physical abuse in your adult life?
2. Is there something similar or different regarding your adult experience of non-physical abuse compared with your relevant experiences as a child/teenager?
3. Why do you believe this abuse happened when you were child/teenager?
4. Why do you think it is still happening?
 - What factors in your opinion, maintain this behaviour towards you?
 - How do you feel about the abuse?
5. How do you cope?
 - Is there something that makes it difficult/easy for you to cope?
 - Has therapy changed/not changed the way you see it?
6. A) Do you think the abuse is linked with your eating disorder symptoms now in anyway?
 - *In case they answer yes:* How do you think is it linked?
 - *In case of no answer:* what factors are linked with your eating disorder symptoms?B) Do you think the abuse as a child is linked with your experience of eating disorder development?
 - *In case they answer yes:* How do you think it is linked?
 - *In case of no answer:* What factors are linked with your eating disorder development?
7. Do you think this situation will change?

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- *In case of yes answer:* How do you think it will change?
- *In case of no answer:* what factors do you think will prevent this change?

The interview will close with a positive statement by the researcher according to the content of patient's speech during the interview. The positive statement will be relevant to how things that make people feel uncomfortable with could be improved through discussion in psychotherapy.

Demographic questions

Age:

Last education level:

Working status:

Age of ED onset:

Appendix 10 – Interview transcript Laura in Greek

1

- 1 Θα ήθελα να μου μιλήσετε για την εμπειρία που έχετε σχετικά με τη μη φυσική
2 μορφή κακοποίησης στην ενήλικη ζωή.
- 3 Καθώς μένω ακόμα με τους γονείς μου είναι μια προέκταση της ανήλικης ζωής μου.
4 Με κάποιους διαφορετικούς όρους, δεν ξέρω αν είναι καλύτεροι ή χειρότεροι, πάντως
5 ότι ζούσα ως παιδί το ζω και ως ενήλικη.
- 6 Μπορείτε να μου πείτε πιο συγκεκριμένα την εμπειρία τώρα ως ενήλικη?
- 7 Νιώθω ότι πολλές φορές σε κάποια βήματα που θέλω να κάνω υπάρχουν οι γονείς
8 μου που μου τραβούν το χαλί κάτω από τα πόδια ακυρώνοντας με είτε με προσβολές
9 λεκτικές είτε ακόμα και με σωματική βία. Στα πρώτα χρόνια της ενήλικης ζωής και
10 σωματική κακοποίηση.
- 11 Αυτό το έχετε βιώσει μόνο από τους γονείς?
- 12 Θεωρώ ότι αυτοί ήταν το πρώτο στάδιο και εφόσον είχα και επαφή μόνο με τους
13 γονείς μέσα στο σπίτι νομίζω ναί μόνο από τους γονείς.
- 14 Ως ενήλικη
- 15 Ως ενήλικη ναί.
- 16 Αυτό είναι κάτι που μπορεί να συμβεί είτε από φίλους στην παρέα είτε από
17 σύντροφο είτε από εργοδότη
- 18 Από εργοδότη ήταν ένα μεμονωμένο περιστατικό το οποίο δεν με πείραξε το έχω
19 αφήσει στο παρελθόν και δεν το συζητήσα καθόλας. Ήταν μόνο της ημέρας. Από
20 φίλους ή σύντροφο ή κάτι άλλο όχι. Δεν είχα κάτι.
- 21 Οπότε το κομμάτι της ενήλικης ζωής έχει να κάνει με ακύρωση
- 22 Ναί ακύρωση, προσβολές, λεκτική βία δηλαδή
- 23 Θέλετε να μου φέρετε κάποιο παράδειγμα?
- 24 Πολλά παραδείγματα, ας πούμε το τελευταίο χρονικό διάστημα που παίρνω και
25 φαρμακευτική αγωγή για κατάθλιψη και αγχώδεις διαταραχές προσπαθούν να μου
26 περάσουν ότι το μυαλό μου είναι πειραγμένο και γι αυτό παίρνω φάρμακα. Ότι
27 εκείνοι δεν έχουν καμία ευθύνη πάνω σε αυτό, ότι εγώ είμαι υπεύθυνη για πράγματα
28 που συμβαίνουν μέσα στο σπίτι.
- 29 Όπως? Για ποιο πράγμα θα μπορούσατε να είστε υπεύθυνη?
- 30 Τα τελευταία διάστημα που προσπαθώ να μένω στο σπίτι του φίλου με τον οποίο
31 είμαστε μαζί κάποιο διάστημα, ακόμα προσβολές για την ηθική μου.
- 32 Το ίδιο θα μπορούσατε να πείτε ότι συμβαίνει και από την αδερφή?

- 33 Όχι το ίδιο συμβαίνει και στην αδελφή μου από τους γονείς μας απλά η αδελφή μου
34 το διαχειρίζεται με διαφορετικό τρόπο. Μεταξύ μας έχουμε καλή σχέση και
35 κατανόηση.
- 36 **Κατάλαβα.**
- 37 **Η κακοποίηση εκφράζεται περισσότερο από τον πατέρα σας ή από τη μητέρα**
38 **σας ή είναι και οι δύο το ίδιο?**
- 39 Εκφράζεται και από τους δύο, ίσως πιο πολύ λεκτικά από τη μητέρα μου.
- 40 **Οπότε αυτό είναι το πλαίσιο της κακοποίησης στην ενήλικη ζωή.**
- 41 **Ναι**
- 42 **Υπάρχει κάτι όμοιο ή κάτι διαφορετικό ανάμεσα στην εμπειρία σας ως ανήλικη**
43 **και ως ενήλικη στη μη φυσική μορφή κακοποίησης?**
- 44 Στην ενήλικη ζωή θεωρούσα ότι δεν είμαι σε θέση ισχύος, ούτε τώρα θεωρώ ότι είμαι
45 σε θέση ισχύος, απλά τότε ως παιδί δεν μπορούσα να αντιδράσω με τον τρόπο που
46 ενδεχομένως μπορώ τώρα. Στην πράξη όμως επειδή θεωρώ ότι έμαθα σε αυτόν τον
47 τρόπο αντίδρασης ίσως εμποδίζομαι να αντιδράσω και τώρα. Δηλαδή δέχομαι πιο
48 παθητικά την κακοποίηση αυτή ή αντιδρώ με μία πολύ μεγάλη έκρηξη όπου η
49 αφορμή μπορεί να μην είναι τόσο μεγάλη αλλά σε εμένα λειτουργεί συσσωρευτικά.
- 50 **Κατάλαβα. Κάποια ομοιότητα ή διαφορά εκτός από την αντίδρασή σας? Η**
51 **αντίδρασή σας είναι ίδια από ό,τι κατάλαβα.**
- 52 Πλέον κάνω εκρήξεις θυμού.
- 53 **Ως ανήλικη δεν κάνατε εκρήξεις θυμού?**
- 54 **Όχι**
- 55 **Το κρατούσατε.**
- 56 **Ναι ναι να κρατούσα τον θυμό μέσα μου.**
- 57 **Ως κατάσταση από τους γονείς, εφόσον μου το έχετε κάνει συγκεκριμένα, τότε**
58 **και τώρα θα λέγατε ότι είναι ίδια? Αυτά που σας έλεγαν τότε σας λένε και τώρα?**
- 59 Για διαφορετικά θέματα. Τότε μπορεί να με ακόρωναν για την επίδοσή μου στα
60 μαθήματα η οποία αντί για 20 ήταν 19.
- 61 **Μάλιστα κατάλαβα**
- 62 **Η για το βάρος μου. Ότι «άλλες καπέλες είναι αδύνατες και εσύ δεν είσαι και πρέπει**
63 **να κόψεις το φαγητό». Ή ότι «κάποια άλλη πήρε 20 και εσύ πήρες 19». Συνεχείς**
64 **συγκρίσεις με άλλα παιδιά είτε σε θέμα βαθμών είτε σε αυτό που θεωρούσαν**
65 **σεβασμό απέναντί τους είτε προς την εμφάνισή μου (βάρος, χαρακτηριστικά**

- 66 προσώπου, σώματος κλπ). Κα θέματα ηθικής όταν ήμουν ανήλικη. Και επειδή ήθελα
67 να βγω στην ερηβεία μου που ποτέ δεν έβγαίνα γιατί ήταν κατακριτέο αλλά και στο
68 δημοτικό αν τύχαινε κάποιο αγόρι να εκδήλωνε κάποιο ενδιαφέρον – όσο ενδιαφέρον
69 μπορεί να δείξει ένα αγόρι στο δημοτικό- πάντα ήμουν εγώ αυτή που το προκαλούσε.
- 70 **Κατάλαβα. Το θέμα του σεβασμού που λέτε προς εκείνους?**
- 71 Όχι θα έπρεπε να τους σεβόμαστε εγώ και η αδελφή μου γιατί είμαστε στο σπίτι και
72 φροντίζουν για τη διατροφή μας και το μελόμαμά μας χωρίς να μας λείψουν κάποια
73 βασικά υλικά αγαθά που εκείνοι θεωρούν απαραίτητα.
- 74 **Από αυτό που μου περιγράφετε υπάρχει κάτι κοινό σήμερα? Τόιες ατάκες ίδιες
75 περιπτώσεις?**
- 76 Ναι ότι θα έπρεπε να πληρώνω ενόικιο για τη διαμονή μου στο σπίτι, ότι αργά να
77 γορίσω στο σπίτι, ότι άλλη που είναι στην ηλικία μου παντρεύτηκε και έχει δύο
78 παιδιά κι εγώ δεν έχω κάνει τίποτα ότι αυτοί στη θέση μου θα είχαν καταφέρει να
79 πάρουν αυτοκίνητο και πολλά άλλα.
- 80 **Κατάλαβα. Και το κομμάτι της ηθικής παραμένει με ένα διαφορετικό τρόπο.**
- 81 Σαφώς γιατί ενώ είμαι ανήλικη και έχω κάνει 2-3 σχέσεις στα 28 μου και μου λένε
82 «πόσο θα γράψει το κοντέρ? Πότε θα λέγεται κάποια γυναίκα ανήλικη αν όχι εσύ?»
- 83 **Υπάρχει κάτι το διαφορετικό σε αυτή την κατάσταση τότε και τώρα?**
- 84 Εκρήξεις θυμού. Η διαφορά δηλαδή είναι πιο πολύ στη δική μου αντίδραση με
85 εκρήξεις που δεν μπορώ να ελέγξω όταν νιώθω ότι με πνίγει το άδικο, πολλά
86 κλάματα, αναρωτιέμαι αν είμαι τρελή, αν έχουν άδικο, μήπως κάνω κάτι λάθος, μια
87 θυματοποίηση δηλαδή που τη μία λέω ότι δεν είμαι τρελή και εκείνοι έχουν άδικο και
88 με αυτά που μου λένε μου τραβάνε το χαλί κάτω από τα πόδια και δεν μπορώ να
89 πιστέψω στον εαυτό μου και να προχωρήσω αλλά από την άλλη αναρωτιέμαι μήπως
90 κάνω εγώ κάτι λάθος και είμαι καταδικασμένη γιατί συγκρούονται δύο πεποιθήσεις
91 μέσα μου.
- 92 **Αυτές τις σκέψεις που μου λέτε τώρα τις κάνατε και πιο μικρή όταν συνέβησαν
93 αυτά?**
- 94 Ναι τις έκανα και πιο μικρή, μήπως έχουν άδικο ας πούμε.
- 95 **Κατάλαβα**
- 96 Αλλά δεν αντιδρούσα, δεν υπήρχε έκφραση θυμού, θυμός υπήρχε. Υπήρχε και κλάμα
97 αλλά όχι τόσο έντονο όπως τώρα.
- 98 **Υπάρχει κάποια ομοιότητα η διαφορά με τα παραπάνω στη σχέση που έχετε με
99 τον σύντροφό σας?**
- 100 Τον ταρινό ή και προηγούμενες σχέσεις?

- 101 **Και με τις προηγούμενες. Κάτι πιο σταθερό μετά το οικογενειακό περιβάλλον**
 102 **είναι οι φίλοι, οι σύντροφοι, άνθρωποι που εργάζαστε συχνά σε επαφή που**
 103 **θεωρούνται σημαντικοί άλλοι.**
- 104 Στην πρώτη μου σχέση είχα επενδύσει πάρα πολύ και νόμιζα ότι ήταν ο ένας και
 105 μοναδικός και ότι με αυτόν θα κάνω οικογένεια για αυτό και με πείραξε όταν
 106 σταμάτησε αυτή η σχέση.
- 107 **Αυτή η σχέση είχε στοιχεία μη φυσικής κακοποίησης, στοιχεία απομάκρυνσης ή**
 108 **αδιαφορίας?**
- 109 Ναι απομάκρυνσης και αδιαφορίας και αυτός ήταν και ο λόγος που διέκοψα τη
 110 σχέση.
- 111 **Τη διακόψατε εσείς?**
- 112 Ναι
- 113 **Παρά το ότι τη θέλατε?**
- 114 Ναι παρά το ότι την ήθελα τη διέγραψα. Από εκεί και πέρα στην ταρτηνή μου σχέση
 115 βίωσα κάποια περιστατικά όπως «αν ήσουν τόσο καλά θα ήσουν καλύτερη» ή αν σε
 116 δω σε παραπάνω καλά μπορεί να μη μου αρέσεις», «πρέπει να γυμνάζεσαι» και κάτι
 117 τέτοια για την εμφάνιση. Στην αρχή με πείραζε αλλά τώρα το διαχειρίζομαι ή μάλλον
 118 το συνήθισα.
- 119 **Συνεχίζεται αυτό?**
- 120 Ναι και δεν θέλω από τη σχέση μου να βιώσω αυτά που βλέπω στη σχέση των γονιών
 121 μου μεταξύ τους ή αυτά που μου έκαναν οι γονείς μου. Γιατί πάλι μου μπαίνει η
 122 αμφιβολία μήπως είχαν δίκιο (οι γονείς) αφού το λέει κι αυτός (ο φίλος της)
- 123 **Κάτι άλλο στη συντροφική σχέση, πόσο και καιρό είστε μαζί?**
- 124 2.5 χρόνια περίπου
- 125 Είναι αρκετός καιρός για να έχουν αναπτυχθεί συμπεριφορές και οικειότητα και
 126 από τις δύο πλευρές. Θα λέγατε ότι υπάρχει κάποια συμπεριφορά που σας
 127 ενοχλεί?
- 128 **Με την έννοια της κακοποίησης?**
- 129 **Ναι όπως πχ φωνές, προσβολές.**
- 130 Ναι μπορεί να πει κάτι όπως το σκάρτετα εκείνη την ώρα σαν αυτά για την εμφάνιση
 131 (γυμναστήριο, καλά, τρόπος που ντύνομαι) και με εμένα να με πειράζει πολύ.
- 132 **Για πράγματα που κάνετε υπάρχει στήριξη ή υπάρχει ακύρωση?**

- 133 Υπάρχει το «κόκκοι τη γνώμη μου, αξιολόγησέ τη και αποφάσισε τι θα κάνεις». Όμως
134 επειδή έχουμε διαφορά ηλικίας δώδεκα χρόνια του βγαίνει ένα πιο πατρικό στολ.
- 135 **Αν εσείς κάνετε κάτι αντίθετο από τη συμβουλή του υπάρχει κάποιο πρόβλημα
136 μετά?**
- 137 Υπάρχει κριτική αλλά όχι με ένταση. Αποδίδει την επιλογή μου στην ηλικία μου.
138 Λέει ότι ίσως κι αυτός στην ηλικία μου να έκανε το ίδιο.
- 139 **Είπατε ότι από φίλους και συγγενείς δεν έχετε βιώσει κάτι τέτοιο.**
- 140 **Όχι.**
- 141 **Για ποιο λόγο πιστεύετε ότι συνέβη αυτή η κακοποίηση όταν ήσασταν ανήλικη?**
- 142 Νομίζω λόγω προσωπικών βιωμάτων και των δύο (των γονιών της εννοεί) και άλλων
143 θεμάτων και λόγω της κακής μεταξύ τους σχέσης. Δεν είχαν κάτι άλλο να
144 ασχοληθούν και στράφηκε με λανθασμένο τρόπο το ενδιαφέρον τους πάνω μας
145 (εννοεί και στην αδελφή της) και υπήρξαν όλες αυτές οι αντιδράσεις, ενδεχομένως
146 κάποιες ματαώσεις που είχαν δεχτεί οι ίδιοι δηλαδή από τους δικούς τους γονείς.
147 Έχω στο μυαλό μου και τη μητέρα μου που μπορεί να της συνέβη το ίδιο αλλά δεν το
148 σταμάτησε και το μετέφερε στα παιδιά της. Αυτός είναι και ο δικός μου φόβος
149 δηλαδή. Να σταματήσει εδώ. Αν κάνω παιδιά να μη συνεχίσει και το κάνω κι εγώ.
- 150 **Κατάλαβα. Οι παράγοντες λοιπόν έτσι όπως τους σκέφτεστε γι αυτό που συνέβη
151 δεν προέρχονται από εσάς, δεν νιώθετε ότι εσείς ευθύνεστε σε κάτι**
- 152 **Όχι δεν νομίζω ότι έκανα κάτι. Έχω μπει στη διαδικασία σύγκρισης με άλλα άτομα
153 της ηλικίας μου που είχαν πολύ χαμηλότερους βαθμούς για παράδειγμα και οι γονείς
154 τους, τους έταξαν διάφορα δώρα για να περάσουν την τάξη, το ποιο το θεωρώ επίσης
155 ακραίο, αλλά δεν είχαν φανές στο σπίτι επειδή υπήρξε ένα 18 στο τρίμηνο. Ούτε
156 επειδή στις πανελληνιες εις πούμε έβγαλα 18300 μόρια και όχι 19000 και ότι τι
157 ασοτυχία είναι αυτή...**
- 158 **Για ποιους λόγους πιστεύετε ότι αυτή η κατάσταση συμβαίνει ακόμα?**
- 159 Γιατί δεν μπορώ να αντιδράσω. Σε αυτό φταίω εγώ. Εκείνοι δεν έχουν αλλάξει, εγώ
160 το βλέπω αλλά δεν μπορώ να αντιδράσω. Δεν ξέρω πώς είναι και δεν έμαθα ποτέ να
161 βάζω όρια και να μη με θίγει κάποιος. Η πρώτη κουβέντα αν μιλήσω ανοιχτά γι' αυτό
162 σε κάποιο φίλο ή φίλη μου είναι «γιατί δεν φεύγεις»? κι όμως νιώθω ανάπηρη να το
163 κάνω. Με την κοριολεκτική σημασία. Νιώθω ότι θα γίνει χαμός. Έχω μπει σε μια
164 διαδικασία να αποφεύγω τη σύγκριση μαζί τους γιατί δεν την αντέχω καθόλου, με
165 χαλάει πάρα πολύ ψυχολογικά, και νιώθω αδύναμη να κάνω αυτά τα μεγάλα βήματα
166 που θα έπρεπε να έχω κάνει στην ηλικία μου. Όπως το να φύγω από το σπίτι και με
167 δεδομένο ότι έχω κάποιο στοιχειώδες εισόδημα. Θα μπορούσα ίσως φέτος να το
168 προσπαθήσω αφού δεν είμαι και καλά ψυχολογικά αλλά δυσκολεύομαι.

- 169 Ποιοι παράγοντες πιστεύετε ότι διατηρούν τη δική σας στάση και συμπεριφορά
170 όπως να μη βάζετε όρια και να μην φεύγετε από το σπίτι?
- 171 Η συνήθεια, το ότι έχω μάθει έτσι και ορισμένα πράγματα τα θεωρώ... ότι έτσι είναι
172 και δεν πρόκειται να αλλάξουν, είναι η συνήθεια. Όλοι έχουμε μάθει έτσι, εκείνοι
173 έχουν μάθει έτσι..εγώ έχω μάθει έτσι...
- 174 Είναι ένα μοντέλο που το γνωρίζετε δηλαδή, σας είναι γνωστή μια
175 καθημερινότητα..
- 176 Ναι ναι ναι
- 177 Και με έναν τρόπο σας είναι μια φυσιολογική καθημερινότητα που την ξέρετε
178 χρόνια
- 179 Ναι
- 180 Ο ένας παράγοντας είναι αυτός, ο άλλος που μου είπατε είναι η δική σας
181 αδυναμία αντίδρασης και της επιβολής ορίων. Όμως όπως μου είπατε έχετε
182 εκρήξεις θυμού άρα έχετε μια αντίδραση σε αυτό που συμβαίνει.
- 183 Ναι. Η έκρηξη θυμού δεν είναι απαραίτητα ότι έρχεται πάντα μετά από μια καθαρή
184 απτία... μπορεί η αφορμή να είναι ασήμαντη.
- 185 Οπότε να μην φαίνεται για ποιο λόγο αντιδράτε
- 186 Ναι. Στους άλλους εκείνη την ώρα φαίνεται ότι εγώ είμαι παράλογη. Εγώ σκέφτομαι
187 διάφορα εκείνη τη στιγμή αθροιστικά, συνολικά, η αντίδρασή μου είναι φοβερά
188 έντονη, δεν μπορώ να το ελέγξω, και στους άλλους μπορεί να μη φαίνεται για ποιον
189 λόγο γίνεται αυτό.
- 190 Στους γονείς σας στους οποίους απευθύνεται αυτή η αντίδραση δεν εξηγείτε το
191 λόγο της αντίδρασης σας αυτής?
- 192 Όχι στέκομαι στην αφορμή εκείνης της στιγμής.
- 193 Έχετε μιλήσει ποτέ στους γονείς σας για όλο αυτό?
- 194 Πάρα πολλές φορές.
- 195 Αρα το έχετε εκφράσει
- 196 Ναι ναι
- 197 Το κάνατε ανήλικη ή ενήλικη?
- 198 Και τα δύο.
- 199 Τί τους έχετε πει?

- 200 Ότι υπάρχει πρόβλημα, ότι κάτι δεν πάει καλά, ότι δεν είναι φυσιολογικό να γίνονται
 201 τέτοια πράγματα, ότι δεν είναι φυσιολογικές οι αντιδράσεις τους, δεν είναι
 202 υποστηρικτικά αυτά που κάνουν στα παιδιά τους αντίθετα τα σπράχνουν στον πάτο,
 203 τους είχα προτείνει να πάμε για οικογενειακή θεραπεία ώστε να μιλήσει όλη αυτή η
 204 κατάσταση και για να επιβεβαιωθώ κι εγώ ότι δεν είμαι τρελή κι ότι έχω δίκιο.
- 205 **Το οποίο δεν έγινε?**
- 206 Όχι
- 207 **Η δική τους στάση όταν εσείς τα λέγατε αυτά ποια ήταν?**
- 208 Ότι αυτά που λέω είναι δικαιολογίες για να δικαιολογήσω αυτά που κάνω-την
 209 ξέφρενη ζωή κατά τη γνώμη τους-ότι είμαι τρελή και το μυαλό μου είναι πειραγμένο
 210 κι ότι κάτι δεν κάνω εγώ σωστά στη ζωή μου. Ότι εγώ φταίω που γίνεται όλο αυτό.
 211 Ότι αν ήμουν ένα σωστό παιδί και έκανα αυτά που μου λένε θα ήταν πολύ καλύτερα
 212 τα πράγματα.
- 213 **Πώς νιώθετε για την κακοποίηση αυτή?**
- 214 Φριχτά..παίρνω αγωγή...τί άλλο να πω... Νιώθω θυμό, οργή, λύπη,
 215 απόγνωση...καταύκασμένη αποθνήσκω ορισμένες φορές. Κάνω τόσα πράγματα στη
 216 δουλειά μου κι όμως δεν το χαίρομαι γιατί υπάρχει ένα μελανό κομμάτι στη ζωή μου.
 217 Και είναι σημαντικό γιατί η οικογένεια είναι το πρώτο σκαλί για να προχωρήσει
 218 κάποιος και αυτή τη στιγμή το πρώτο σκαλί τρεμοπαίζει και από την αρχή δεν έστεκε
 219 καλά. Ως δασκάλα το βλέπω και πιο έντονα σε σχέση με άλλες οικογένειες.
- 220 **Πώς το αντιμετωπίζετε όλο αυτό? Υπάρχει κάτι που το κάνει πιο εύκολο να το
 221 αντιμετωπίσετε? Κάτι που δυσκολεύει την αντιμετώπιση?**
- 222 Σαφώς η συνύπαρξη το κάνει πιο δύσκολο γιατί φέρνει περισσότερες τριβές. Κάνω
 223 πολύ μεγάλη προσπάθεια να αποστασιοποιηθώ και να κάνω αυτό που θέλω εγώ κι
 224 ας γκρινιάζουν. Όποια ώρα και να γυρίσω θα γκρινιάζουν έτσι κι αλλιώς. Μου είναι
 225 πολύ δύσκολο να το κάνω αυτό. Δηλαδή και έξω που θα είμαι δεν θα το χαίρομαι. Το
 226 μυαλό μου θα είναι στο σπίτι. Κάνω προσπάθειες τη στιγμή που θα μου πουν κάτι να
 227 μην απαντήσω γιατί έτσι και μπω στη διαδικασία απάντησης θα γίνει έκρηξη.
 228 Κάποιες φορές έχω μπει και στη διαδικασία της λογικής απάντησης που όμως δεν
 229 βρίσκω να έχει αποτέλεσμα.
- 230 **Υπάρχει κάτι που σας δυσκολεύει ή σας διευκολύνει να το αντιμετωπίσετε?**
- 231 Η αδελφή μου με διευκολύνει. Είμαστε στο ίδιο σπίτι και έχουμε περάσει τα ίδια.
 232 Απλώς η κάθε μια το έχει διαχειριστεί διαφορετικά. Όχι ότι η αδελφή μου δεν έχει
 233 κατώλυτα αλλά νιώθω ότι έχω ένα συμπαριστάτη.
- 234 **Η σχέση με το σύντροφό σας έχει βοηθήσει στον τρόπο που το αντιμετωπίζετε
 235 τώρα?**

- 236 Δυστυχώς όχι. Το κομμάτι των οικογενειακών σχέσεων το θεωρώ πολύ ευαίσθητο και
 237 υπάρχουν κι άλλοι παράγοντες που δεν έχω ανοιχτεί. Δεν γνωρίζω όλα τα κομμάτια
 238 του παζλ. Σε κάποια πράγματα μπορεί να με βοηθάει εν αγνοία του και σε άλλα να με
 239 δυσκολεύει πάλι εν αγνοία του. Για παράδειγμα όταν θα μου πει για το βάρος μου,
 240 μου θυμίζει αυτά που λένε οι γονείς μου. Η διευκόλυνση είναι όταν μένω σπίτι του το
 241 οποίο με βοηθάει να λείπω από το σπίτι.
- 242 **Εσείς για ποιο λόγο νιώθετε ότι δεν μπορείτε να του ανοιχτείτε ενώ είναι πολλά**
 243 **χρόνια η σχέση?**
- 244 Ξέρει ότι είμαι σε ψυχοθεραπεία και καταλαβαίνει ότι το πρόβλημα προϋπήρχε της
 245 σχέσης όμως επειδή θεωρώ ότι αυτά που έχω βιώσει είναι πολύ έντονα και άσχημα
 246 νιώθω ότι κάπως θα χαλάσει η εικόνα μου. ίσως τα χρησιμοποιήσει εναντίον
 247 μου... ίσως επειδή δεν ξέρει ακριβώς τι γίνεται δεν θα καταλάβει... ή θα μου προτείνει
 248 να μείνουμε μαζί ενώ εγώ δεν θέλω γιατί υπάρχουν πράγματα στη σχέση που με
 249 ενοχλούν.
- 250 **Στη σχέση τί σας ενοχλεί και δεν μπορείτε να συγκατοικήσετε που θα ήταν μία**
 251 **λύση?**
- 252 Με αγχώνει λίγο η διαφορά ηλικίας, η αντίδραση των γονιών μου απέναντι σε αυτόν-
 253 απέναντι σε οποιονδήποτε βασικά-με προβληματίζει η σχέση του με τη μητέρα του
 254 που είναι πολύ εξαρτητική...και βέβαια ο αδελφός του που έχει ψυχολογικά
 255 προβλήματα.
- 256 **Αυτά που μου λέτε για τη μητέρα και τον αδελφό γιατί είναι πράγματα που**
 257 **πρέπει να λάβετε υπόψη σας για το πώς θα προχωρήσει αυτή η σχέση? Σε τί σας**
 258 **επιηρεάζουν?**
- 259 Με τον αδελφό του δεν έχουμε καμία επικοινωνία. Με έχει προσβάλλει και έχει
 260 άσχημη συμπεριφορά απέναντί μου από τότε που έμαθε ότι είμαι με τον αδελφό του.
 261 Τον γνώριζα από πιο πριν οπότε δεν ξέρω τί συναισθήματα έχει για μένα και γιατί
 262 βγάζει αυτή την επιθετική συμπεριφορά απέναντί μου. Η μητέρα του τον επιηρεάζει
 263 πολύ (τον συντροφοφ της) οπότε φοβάμαι ότι θα αναστατεύεται συνέχεια και επίσης με
 264 θεωρεί υπεύθυνη που δεν έχω καλή σχέση με τον αδελφό του. Επίσης έχει επικριτική
 265 στάση απέναντί μου και μου έχει πει ότι πρέπει να αρχίσω γυμναστήριο. Έχει και
 266 αυτή άποψη για την εικόνα του σώματός μου.
- 267 **Η ψυχοθεραπεία έχει αλλάξει καθόλου τον τρόπο που αντιμετωπίζετε αυτή την**
 268 **κατάσταση της κακοποίησης?**
- 269 Ναι. Σοζήτωνα κάποια πράγματα και παίρνω επιβεβαίωση ότι δεν είμαι τρελή και
 270 αντιμετωπίζω κάποια πράγματα πιο αποστασιοποιημένη. Είναι σα να ξέρω ότι θα
 271 φάω γροθιά αλλά προσπαθώ να σφίγγωμαι για να πονέσει λιγότερο.
- 272 **Πιστεύετε ότι η κακοποίηση τώρα σχετίζεται με τα συμπτώματα της ΔΠΤ με**
 273 **κάποιο τρόπο?**

- 274 Ξεκάθαρα. Όταν με βλέπουν να τρώω μου λένε ότι είμαι λαίμαργη και να σταματήσω
275 και ότι είμαι χοντρή και δεν θα μου κάνουν τα ρούχα μου. Εγώ συνεχίζω να βρίσκω
276 διέξοδο στο φαγητό και τρώω για να μην τους ακούω μέσα μου. Νιώθω χαρούμενη
277 όταν τρώω.
- 278 Πιστεύετε ότι η κακοποίηση στην ανήλικη ζωή σχετίζεται με την ανάπτυξη της
279 ΔΙΠ?
- 280 Ενδεχομένως να αλλά δεν μπορώ να το πω με σιγουριά. Μου έκαναν κριτική από
281 παιδί ότι έχω πάρει βάρος και είμαι χοντρή οπότε έτρωγα από αντίδραση και έπαιρνα
282 βάρος και μετά προσπαθούσα να το χάσω και συνέχεια γινόταν αυτό.
- 283 Με ποιο τρόπο μπορεί να συνδέεται η κακοποίηση με την έναρξη ΔΙΠ?
- 284 Όταν δεν μπορείς να κάνεις κάτι άλλο μέσα στο σπίτι...τρως, όταν απαγορεύονται
285 πολλά πράγματα δεν σου μένει κάτι άλλο.
- 286 Πιστεύετε ότι αυτή η κατάσταση θα αλλάξει?
- 287 Μόνο αν αλλάξω εγώ. Οι γονείς μου δεν πρόκειται να αλλάξουν. Μόνο αν πάρω εγώ
288 την κατάσταση στα χέρια μου θα αλλάξει κάτι. Αυτοί τα ίδια θα κάνουν αλλά εγώ θα
289 μπορώ να το αντιμετωπίσω καλύτερα. Δεν ξέρω όμως πώς θα γίνει αυτό. Θα ήθελα
290 να πατήσω ένα κουμπί και να μεταφερθώ σε μια πιο ήρεμη ζωή που θα μπορώ να
291 ρυθμίζω εγώ τα πράγματα και να μη νιώθω ότι είμαι αδύναμη να διαχειριστώ τη ζωή
292 μου όπως νιώθω τώρα. Σκέφτομαι ότι αν οι γονείς μου ήταν υποστηρικτικοί θα
293 αισθανόμουν βασίλισσα. Τώρα αισθάνομαι σκουπίδι. Στο μυαλό μου έχω εκείνο το
294 παιδί που υπέστη βία...δεν έχω μεγαλώσει. Περιμένω ακόμα την αγάπη και την
295 υποστήριξη που θεωρώ ότι θα έπρεπε να έχω πάρει. Ξέρω όμως ότι δεν θα συμβεί
296 αυτό.
- 297 Ποιοι παράγοντες πιστεύετε ότι θα εμποδίσουν την κατάσταση να αλλάξει?
- 298 Οι γονείς μου που δεν θα αλλάξουν και εγώ αν αφήσω τα πράγματα έτσι χωρίς να
299 κάνω τίποτα.
- 300 Κλείνοντας, είναι κάτι που θέλετε να προσθέσετε?
- 301 Αυτά που πέρασα ήταν ο λόγος που διάλεξα αυτή τη δουλειά για να υποστηρίξω τα
302 παιδιά όπως πιστεύω ότι θα έπρεπε να με έχουν υποστηρίξει οι γονείς μου. Επίσης
303 βλέπω από τη δουλειά μου τι αντίκτυπο μπορεί να έχει ένας κακός γονέας την
304 ψυχολογία του παιδιού. Και σε κάποια παιδιά μπορεί να βλέπω τον εαυτό μου και
305 άλλα να τα ζηλεύω γιατί εγώ δεν ήμουν σαν αυτά.
- 306 Θέλετε να μου εξηγήσετε?
- 307 Οι γονείς μου δεν έδιναν σημασία στα προσεγμένα ρούχα και να πάμε σχολείο με
308 κορδέλες και κάτι χαριτωμένο σαν κοριτσάκια. Υπήρχε έλεγχος και στο πότε θα μπει
309 πλήντηρο ενώ μπορεί να χρειαζόμουν κάτι νεότερα. Η πηγαίναμε στον γιατρό όταν

- 310 η κατάσταση είχε φτάσει στο απροχώρητο γιατί έλεγαν ότι δεν είναι τίποτα και θα
311 σου περάσει.
- 312 Κατάλαβα. Είναι κάτι άλλο που θέλετε να προσθέσετε?
- 313 Όχι.
- 314 Σας ευχαριστώ πολύ για τον χρόνο σας

Appendix 11 – Interview transcript Betty in Greek

1

- 1 Μπορείτε να μου μιλήσετε για την εμπειρία σας σχετικά με τη μη φυσική μορφή
2 κακοποίησης στην ενήλικη ζωή?
- 3 Έχει προκύψει πολλές φορές από διαφορετικά πρόσωπα, αρχικά από κάποια
4 πρόσωπα του οικογενειακού περιβάλλοντος και εν συνεχεία από συμμαθητές στο
5 σχολείο από δασκάλους στο σχολείο....το έχω υποστεί όλο αυτό πριν τα 18 μου σε
6 αρκετά μικρή ηλικία.
- 7 Θέλετε να μου μιλήσετε περισσότερο γι αυτό?
- 8 Ήταν σεξουαλική κακοποίηση αρχικά από τη θεία, εν συνεχεία από τη μητέρα,
9 σεματική κακοποίηση επειδή ήμουν άτακτη έχει γίνει από όλα τα μέλη της
10 οικογένειας και από παππού και γιαγιά.
- 11 Σε μη φυσική μορφή?
- 12 Σε μη φυσική μορφή εξακολούθη να συμβαίνει και τώρα. Bullying στο σχολείο ως
13 πόοιμε. Ήμουν χαμηλών επιδόσεων μαθήτρια και κάποιοι συμμαθητές
14 χρησιμοποιούσαν υποτιμητικές εκφράσεις προς εμένα. Το έκαναν και οι δάσκαλοι
15 επειδή δεν καταλάβαινα ή έλεγα λάθος την απάντηση (δεν προσπαθείς, δεν διαβάζεις,
16 είσαι τεμπέλα).
- 17 Από την οικογένεια?
- 18 Παρόμοια κατάσταση από τα μέλη της οικογένειας όταν με βοηθούσαν με τα
19 μαθήματα. Εδώ και δυο χρόνια έχω μάθει ότι έχω μαθησιακές δυσκολίες και έχω
20 δικαιολογήσει κάποια πράγματα για εκείνη την περίοδο. Το ανέφερα στην οικογένεια
21 και κανείς δεν μου ζήτησε συγνώμη.
- 22 Υπήρχε άσχημη συμπεριφορά όπως προσβολές?
- 23 Πάντοτε. Υποτιμητικά σχόλια για τους βαθμούς και όταν εγώ άρχιζα σταδιακά να
24 αντιδρώ και να απαντώ σε αυτό με χτυπούσαν.
- 25 Νιώθατε ότι υπήρχε παραμέληση?
- 26 Συναισθηματική παραμέληση ένιωθα ότι υπήρχε και από τους δύο μου γονείς. Από
27 τον πατέρα μου πάντα και από τη μητέρα μου από την ηλικία των 10.
- 28 Υπήρχαν προσβολές σε σχέση με την εμφάνισή σας?
- 29 Στην ενήλικη ζωή...δεν θα το έλεγα.
- 30 Αρα στο σπίτι η κακή συμπεριφορά με τι έχει να κάνει?
- 31 «δεν καταλαβαίνεις αυτά που διαβάζεις, συγκεντρώσου, είσαι τεμπέλα, είσαι
32 ακατάστατη»
- 33 Θα ήθελα να μου μιλήσετε για την εμπειρία σας με παραμέληση και ψυχολογική
34 κακοποίηση στην ενήλικη ζωή.

- 35 Μόλις ενηλικιώθηκα η θεία μου, μου πρότεινε χρήση ουσιών. Δεν το δέχθηκα και
 36 μου το πρότεινε κι άλλες φορές. Στη συνέχεια έγιναν πάρα πολλά. Από τη μητέρα
 37 μου υπάρχει διάκριση και υποτίμηση, έχω μια αδελφή μικρότερη, και κάνει σύγκριση
 38 με την αδελφή μου σε όλα. Και στην εμφάνιση (ότι είναι λιγότερα καλά, μπορεί και
 39 ελέγχει το φαγητό, έχει καλύτερες σπουδές).
- 40 **Ο πατέρας?**
- 41 Υπάρχει και τώρα συναισθηματική παραμέληση, αμφισβητεί το γεγονός ότι μπορεί
 42 να πάσχω από κάποιο νόσημα. Το ίδιο κάνει και η μητέρα μου.
- 43 **Από σύντροφο, φίλους, εργοδότες?**
- 44 Ναι έχει συμβεί. Είχα μιλήσει για προσωπικά θέματα σε σύντροφο και το
 45 χρησιμοποίησε για να με πλιγώσει. Έχουν γίνει και σχόλια για την εμφάνιση ότι
 46 πάχυνα (υπήρχε μεγάλη διαφορά ηλικίας με αυτόν). Επίσης έχω καταλάβει ότι δεν
 47 πήραν σε κάποια δουλειά λόγω παραπάνω καλών. Έχει τύχει να πάω σε γιατρό και να
 48 μου πει ότι αν αδυνατίσεις θα γίνεις πιο κομμητή ενώ αυτός ήταν παχύς.
- 49 **Από φίλους?**
- 50 Ναι έχει τύχει από μια φίλη να μου πει ότι πάχυνα ενώ ήμασταν σε γιορτή στο σπίτι
 51 μου γιατί είχα γενέθλια και το είπε μπροστά σε όλους.
- 52 Τα περισσότερα υποτιμητικά σχόλια στην ενήλικη ζωή έχουν να κάνουν με την
 53 εμφάνιση
- 54 Ναι γιατί ζούμε στην εποχή των εικόνων.
- 55 **Γιατί πιστεύεται ότι η μη φυσική κακοποίηση συνέβη όταν ήσασταν ανήλικη?**
- 56 Γιατί υπάρχει μιμητική συμπεριφορά. Τα άτομα που το έκαναν έχουν κακοποιηθεί κι
 57 αυτά.
- 58 **Γιατί πιστεύετε ότι συνεχίζει να συμβαίνει και στην ενήλικη ζωή?**
- 59 Γιατί ο κόσμος έχει ψυχολογικά προβλήματα
- 60 **Ποιοι παράγοντες πιστεύετε ότι διατηρούν αυτή τη συμπεριφορά απέναντί σας?**
- 61 Ευθύνομαι κι εγώ σε κάποια πρόσωπα, σε άλλους όχι. Όταν έχω επαφή με άτομα που
 62 είναι κακοποιητικά και το ξέρω, ευθύνομαι κι εγώ. Όμως μερικές σχέσεις μπορεί να
 63 είναι εθιστικές ή προαιγόνοες όπως μάνες-παιδιά, να μην μπορείς να τους αποφύγεις
 64 γιατί συμβιώνεις μαζί τους. Το ίδιο έχει συμβεί και σε σχέση, να μην μπορώ να φύγω
 65 ενώ έπρεπε.
- 66 **Πώς νιώθετε για την κακοποίηση?**
- 67 Είναι παράγοντας που επηρεάζει την εξέλιξή μου, μου δημιουργεί θόμο. Δεν υπάρχει
 68 μέριμνα για το ποιος μπορεί να κάνει παιδί. Οι γονείς μου ήταν ασταμάλλητοι για να

- 69 έχουν παιδί. Νιώθω απογοήτευση και ενοχές που δεν μπορώ σε κάποια πράγματα να
70 επιβληθώ, όπως στη μητέρα μου και να βάλω όρια.
- 71 Υπάρχει κάτι που σας δυσκολεύει/διευκολύνει στη διαχείριση?
- 72 Πάντοτε με δυσκολεύει.
- 73 Τι?
- 74 Το πώς θα επικοινωνήσω τα συναισθήματα μου, τις αποστάσεις που θα μπορούσα να
75 πάρω. Το οικονομικό με δυσκολεύει για να φύγω και από το σπίτι.
- 76 Υπάρχει κάτι που να σας διευκολύνει?
- 77 Δεν υπάρχει.
- 78 Πιστεύετε ότι η μη φυσική μορφή κακοποίησης συνδέεται με τα συμπτώματα
79 ΔΠΤ τώρα?
- 80 Φυσικά συνδέονται.
- 81 Πώς?
- 82 Με απογοήτευση και θυμό. Το φαγητό είναι κάτι εύκολο να το βρεις. Εκεί βγαίνουν
83 το άγχος το στρες η λύπη τα πλεγμένα συναισθήματα, πληγές...
- 84 Βρίσκετε διέξοδο εκεί?
- 85 Ναι. Κύκλος είναι.
- 86 Πιστεύετε ότι η μη φυσική μορφή κακοποίησης συνδέεται με την έναρξη ΔΠΤ?
- 87 Ναι. Οποδήποτε τραυματικό μπορεί να δημιουργήσει μια μορφή εθισμού. Υπήρχε και
88 μιμητική συμπεριφορά από την πλευρά μου σε ακραίες διατροφικές συμπεριφορές
89 μελών της οικογένειας.
- 90 Όπως?
- 91 Για παράδειγμα, η μητέρα μου τρώει ακατάστατα και πολύ.
- 92 Πιστεύετε ότι αυτή η κατάσταση της κακοποίησης θα αλλάξει?
- 93 Μπορούν να αλλάξουν κάποια πράγματα.
- 94 Πώς?
- 95 Εξαρτάται περισσότερο από εμένα, τον τρόπο που σκέφτομαι και αντιδρώ. Να πάρω
96 αποστάσεις από κακοποιητικούς ανθρώπους.
- 97 Ποιοι παράγοντες θα μπορούσαν να εμποδίσουν αυτή την αλλαγή?
- 98 Εξοικονομώ να πιστέω ότι είναι το οικονομικό. Για να φύγω από το σπίτι.

- 99 **Η ψυχοθεραπεία έχει αλλάξει τον τρόπο που το βλέπετε όλο αυτό?**
- 100 **Ναι** αναλύω τα συναισθήματά μου και αυτό με διευκολύνει
- 101 **Η κακοποίηση έχει επηρεάσει τον τρόπο που βλέπετε τον εαυτό σας?**
- 102 **Ναι** στην αυτοεκτίμησή μου. Στην προσέγγιση συντρόφων. Είμαι πολύ κλειστή και
103 ντροπαλή. Με έχει κάνει να νιώθω ανήμπορη και να κυριάζομαι εύκολα. Έχει
104 επηρεάσει την υγεία μου λόγω στρες και λυπής.
- 105 **Είναι κάτι άλλο που θέλετε να προσθέσετε?**
- 106 **Όχι**

Appendix 12-Interview transcript Jenny in Greek

1

- 1 Θα ήθελα να μου μιλήσετε για την εμπειρία που έχετε σχετικά με τη μη φυσική
2 μορφή κακοποίησης στην ενήλικη ζωή.
- 3 Μου έχει συμβεί κυρίως από ανθρώπους με τους οποίους είχα σχέση.
- 4 Μπορείτε να μου πείτε πιο συγκεκριμένα?
- 5 Έχω δεχθεί σχόλια για την εξωτερική εμφάνιση. Μου κάνουν κριτική..
- 6 Θέλετε να μου πείτε?
- 7 Τα μαλλιά μου, το σώμα, τα ρούχα... «πώς είσαι έτσι». Επίσης να με υποτιμούν και
8 να μου λένε τι να κάνω λες και είμαι χαζή και ο λόγος μου δεν μετρούσε πουθενά.
- 9 Αυτή ήταν η εμπειρία σας από όλους τους συντρόφους?
- 10 Ναι
- 11 Μπορείτε να μου πείτε πιο συγκεκριμένα που δεν μετρούσε ο λόγος σας?
- 12 Όταν συζητούσαμε για κάτι ήταν σα να μη μετρούσε η άποψή μου ή σα να ήταν
13 λάθος. Ένιωθα σα να μην είχα κάτι σημαντικό να πω.
- 14 Όπως?
- 15 Ακόμα και για του πού θα πάμε. Δεν πρότεινα ένα μέρος ή μια ταβέρνα που ήθελα γιατί
16 όλο ένιωθα ότι αυτά δεν θα ήταν ωραία για τον άλλο και θα τα απέρριπτε. Μια φορά
17 που το έκανα αυτό έγινε.
- 18 Αυτό το έχετε βιώσει μόνο από τους συντρόφους?
- 19 Κατά κύριο λόγο ναι. Όμως το βίωνω και στη δουλειά από μερικούς συναδέλφους.
- 20 Δηλαδή?
- 21 Μερικοί μου συμπεριφέρονται σα να μην υπάρχουν ή σα να τα κάνω όλα λάθος και να
22 χρειάζομαι οδηγίες. Άλλες φορές μου κάνουν σχόλια για τον χαρακτήρα μου.
- 23 Θέλετε να μου πείτε πιο συγκεκριμένα?
- 24 «Παραείσαι ντροπαλή. Δεν καταλαβαίνεις τι λέμε. Μην είσαι τόσο αδύναμη»
- 25 Σε ποιο πλαίσιο σας τα λένε αυτά?
- 26 Όταν είναι όλοι μαζί και νομίζουν ότι λένε κάτι αστείο αλλά στην πραγματικότητα
27 λένε κάτι χαζό. Νομίζουν ότι δεν καταλαβαίνω όταν αντρώω στις ηλίθιες συζητήσεις
28 τους.
- 29 Οι συζητήσεις τι θέμα έχουν?

- 30 Ποτέ κάτι σοβαρό. Συνήθως σχόλια για άλλους ανθρώπους σχετικά με την εμφάνισή
31 τους ή κοιτασομπολό για την προσωπική τους ζωή.
- 32 **Γιατί σας λένε αδύναμη?**
- 33 Είμαι αδύναμη. Δεν έχω αυτοεκτίμηση. Θέλω να πω φαίνεται γιατί όταν με ενοχλεί
34 κάτι δεν αντιδράω. Συνήθως φεύγω.
- 35 **Όποτε το κομμάτι της ενήλικης ζωής έχει να κάνει με ακύρωση?**
- 36 Ναι ακύρωση, προσβολές... υποτίμηση γενικότερα
- 37 **Υπάρχει κάτι όμοιο ή κάτι διαφορετικό ανάμεσα στην εμπειρία σας ως ανήλικη
38 και ως ενήλικη στη μη φυσική μορφή κακοποίησης?**
- 39 Στην ανήλικη ζωή αυτό γινόταν από τον πατέρα μου.
- 40 **Μπορείτε να μου πείτε πιο συγκεκριμένα?**
- 41 Είχε πολλά νεύρα. Μου φώναζε και με υποτιμούσε. Έπρεπε να είμαι υπάκουη.
- 42 **Δηλαδή?**
- 43 Να είμαι καλό παιδί, να μην κάνω φασαρία, να παίρνω καλούς βαθμούς. Γενικάς
44 ξεσπούσε τα νεύρα του πάνω μου με φωνές και προσβολές. Με έλεγε χαζή, χοντρή
45 και άσχημη.
- 46 **Αυτό το έχετε βιώσει μόνο από τον πατέρα σας?**
- 47 Κορίως ναι. Υπήρχε και στο σχολείο.
- 48 **Μπορείτε να μου πείτε γι' αυτό?**
- 49 Ένας καθηγητής είχε απότομη συμπεριφορά μαζί μου επειδή δεν είχα καλούς
50 βαθμούς. Δεν μου έδινε και σημασία σαν ό,τι είχα να πω θα ήταν λάθος.
- 51 **Από τους συμμαθητές?**
- 52 Και τα παιδιά είχαν χωριστεί σε καλούς και κακούς μαθητές και εγώ επειδή δεν είχα
53 πολύ καλούς βαθμούς βίωνα αδιαφορία. Σα να μην υπήρχα.
- 54 **Ποιος είχε αυτή τη συμπεριφορά απέναντί σας στο σχολείο?**
- 55 Και τα παιδιά και οι καθηγητές.
- 56 **Κατάλαβα. Κάποια ομοιότητα ή διαφορά στην εμπειρία σας ως ανήλικη και
57 ανήλικη? Η αντίδρασή σας για παράδειγμα.**
- 58 Η αντίδραση μου είναι η ίδια. Ούτε τότε μιλούσα ούτε τώρα μιλάω.
- 59 **Γιατί δεν μιλάτε?**

- 60 Δεν αισθάνομαι ότι έχω την αυτοπεποίθηση να υπερασπιστώ τον εαυτό μου. Δεν
61 μπορώ να εκφράσω τα συναισθήματά μου. Δεν μπορώ να επιτεθώ ενώ πρέπει.
- 62 **Γιατί πιστεύετε ότι συμβαίνει αυτό?**
- 63 Γιατί δεν έχω αυτοεκτίμηση. Έμαθα να μη μιλάω για να μην υπάρξει μεγαλύτερη
64 αντίδραση από τον άλλον.
- 65 **Κατάλαβα**
- 66 Αυτή ήταν η συμβουλή και της μητέρας μου σχετικά με τον πατέρα μου. Κι έτσι
67 συνήθισα να μη μιλάω και να μιλάω μόνο μέχρι εκεί που νιώθω ασφαλής γιατί δεν
68 ξέρω τί να κάνω αν ο άλλος αρχίσει να θυμώνει και να φωνάζει. Δεν ξέρω πώς να
69 αντιμετωπίσω τον θυμό του άλλου.
- 70 **Κατάλαβα.**
- 71 **Για ποιο λόγο πιστεύετε ότι συνέβη αυτή η κακοποίηση όταν ήσασταν ανήλικη?**
- 72 Δεν ξέρω. Νομίζω επειδή ο πατέρας μου είχε νεύρα. Δεν μπορώ να πω με σιγουριά.
- 73 Σκέφτεστε κάτι άλλο?
- 74 Και φταίει και η μητέρα μου που δεν έκανε κάτι για να με προστατεύσει από αυτό. Το
75 μόνο που έκανε ήταν να μου λέει να μη μιλάω. Όπως δεν μιλούσε κι αυτή. Στην
76 ουσία έμαθα να μη μιλάω.
- 77 **Για ποιους λόγους πιστεύετε ότι αυτή η κατάσταση συμβαίνει ακόμα?**
- 78 Γιατί δεν μπορώ να αντιδράσω. Σε αυτό φταίω εγώ. Όταν οι άλλοι βλέπουν έναν
79 άνθρωπο αδύναμο χωρίς αυτοεκτίμηση τους φαίνεται εύκολο θύμα για να βγάλουν τα
80 ψυχολογικά τους πάνω του.
- 81 **Σε τι πιστεύετε ότι σας έχει επηρεάσει η κακοποίηση?**
- 82 Σε όλα. Στο πώς είναι ο χαρακτήρας μου. Στο πώς νιώθω. Δεν μπορώ να νιώσω χαρά.
83 Είναι σα να κουβαλάω ένα βάρος. Είμαι πάντα στεναχωρημένη. Δεν προσπαθώ να
84 κάνω παραπάνω πράγματα. Δεν έχω αυτοεκτίμηση. Δεν πήρα την αγάπη που ήθελα
85 από τους γονείς μου. Η μητέρα μου δεν με στήριξε στη συμπεριφορά του πατέρα μου.
86 Δεν πιστεύω στον εαυτό μου. Κάνω μόνο αυτά που μπορώ και δεν προσπαθώ για
87 παραπάνω. Δεν θέλω ευθύνες. Δεν τις αντέχω. Νομίζω ότι δεν θα τα καταφέρω αν
88 προσπαθήσω για κάτι παραπάνω. Γι αυτό δεν σπούδασα κιόλας. Ήθελετε, λόγω των
89 ετών που δουλεύω θα μπορούσα να ζητήσω προαγωγή αλλά δεν το κάνω.
- 90 **Γιατί δεν το κάνετε?**
- 91 Δεν θέλω ευθύνες. Θέλω να κάνω τη δουλειά μου «αόρατα» και να φεύγω. Τα
92 πράγματα θα ήταν αλλιώς αν είχα αυτοεκτίμηση. Αν πίστευα στον εαυτό μου. Όταν
93 αυξάνονται οι ευθύνες θα πρέπει να έχει τις ικανότητες να αντιμετωπίσεις ό,τι συμβεί

- 94 και να έρθεις σε αντιπαράθεση με περισσότερους ανθρώπους. Αυτό δεν το θέλω.
95 Είναι δύσκολο.
- 96 Γιατί νιώθετε ότι σας είναι δύσκολο?
- 97 Γιατί δεν έχω μάθει να αντιμετωπίζω ανθρώπους και καταστάσεις. Έχω μάθει να μη
98 μιλάω και να φεύγω. Όταν είσαι έτσι δεν μπορείς να πάρεις θέση με ευθύνη.
- 99 Σε τί άλλο σας έχει επηρεάσει η κακοποίηση?
- 100 Κλείνομαι στο εαυτό μου. Δεν εκφράζομαι. Δεν μπορώ να πω αυτά που νιώθω. Δεν
101 εμπιστεύομαι τους άλλους. Δεν κάνω στενές σχέσεις. Μου αρέσει η απόσταση.
102 Νιώθω πιο ασφαλής όταν κρατάω αποστάσεις.
- 103 Σε σχέση με τους συντρόφους?
- 104 Δεν μπορώ να δεθώ. Δεν έρχομαι κοντά. Φοβάμαι την απόρριψη.
- 105 Και τι κάνετε?
- 106 Στην αρχή προσπαθώ να τους αρέσω και μετά φεύγω. Θέλω την ησυχία μου. Δεν
107 θέλω ευθύνες. Δεν μπορώ να μου λέει ο άλλος τί να κάνω. Δεν μπορώ να
108 τσακάνομαι.
- 109 Γιατί δεν μπορείτε να δεθείτε?
- 110 Για να μην πληγωθώ φυσικά. Όταν έρχεσαι κοντά με κάποιον γίνεται ευάλωτος.
111 Μετά αν πάει και στραβά θα πληγωθείς πολύ.
- 112 Ποιοι παράγοντες πιστεύετε ότι διατηρούν τη δική σας στάση και συμπεριφορά
113 όπως να μην αντιδράτε?
- 114 Η συνήθεια. Έτσι έχω μάθει. Να μην αντιδρά.
- 115 Μπορείτε να μου πείτε περισσότερα?
- 116 Φταίει ο τρόπος που μεγάλωσα. Όλη αυτή η κατάσταση με τον πατέρα μου με έκανε
117 να είμαι μαζεμένη. Να φοβάμαι να αντιδράσω. Να φοβάμαι να πω αυτά που νιώθω.
118 Δεν ένιωσα αποδοχή και αγάπη για να είμαι δυνατή και να φαίνεται αυτό στους
119 άλλους.
- 120 Είναι ένα μοντέλο που το γνωρίζετε δηλαδή, σας είναι γνωστή μια
121 καθημερινότητα...
- 122 Έτσι ακριβώς.
- 123 Πώς νιώθετε για την κακοποίηση αυτή?
- 124 Πολύ άσχημα. Νιώθω σαν θύμα και θυμάνω που είμαι τόσο αδύναμη και δεν μπορώ
125 να υπερασπιστώ τον εαυτό μου.

- 126 Πώς το αντιμετωπίζετε όλο αυτό? Υπάρχει κάτι που το κάνει πιο εύκολο να το
127 αντιμετωπίσετε? Κάτι που δυσκολεύει την αντιμετώπιση?
- 128 Δεν το αντιμετωπίζω. Απλώς δεν μιλάω όπως είπα και πριν.
- 129 Η ψυχοθεραπεία έχει αλλάξει καθόλου τον τρόπο που αντιμετωπίζετε αυτή την
130 κατάσταση της κακοποίησης?
- 131 Ναι. Με έχει βοηθήσει πολύ να καταλάβω πολλά για τον εαυτό μου.
- 132 Όπως?
- 133 Όπως ότι ο τρόπος που μεγάλωσα ευθύνεται για το ποια είμαι τώρα. Πώς σκέφτομαι
134 πώς αντρώω και πώς νιώθω.
- 135 Πιστεύετε ότι η κακοποίηση τώρα σχετίζεται με τα συμπτώματα της ΔΠΤ με
136 κάποιο τρόπο?
- 137 Έτσι νομίζω. Όταν είμαι στεναχωρημένη και έχω πράγματα που τα κρατώ μέσα μου
138 δεν τρώω. Κάπως σα να τιμωρώ τον εαυτό μου.
- 139 Δηλαδή?
- 140 Σα να μη μου αξίζει να φάω και κυρίως σα να μη μου αξίζει να φάω κάτι που μου
141 αρέσει. Σα να ξεσπάω εκεί. Στη σχέση μου με το φαγητό. Είναι και το μόνο που
142 μπορώ να ελέγξω.
- 143 Πιστεύετε ότι η κακοποίηση στην ανήλικη ζωή σχετίζεται με την ανάπτυξη της
144 ΔΠΤ?
- 145 Νομίζω ναι. Ο τρόπος που μεγάλωσα πρέπει να σχετίζεται με αυτό.
- 146 Με ποιο τρόπο μπορεί να συνδέεται η κακοποίηση με την έναρξη ΔΠΤ?
- 147 Όταν δεν είσαι καλά και δεν μπορείς να μιλήσεις μάλλον βρίσκεις έναν άλλο τρόπο
148 να το δείξεις. Έτσι νομίζω. Στρέφεται στον εαυτό σου και κλείνεται εκεί. Μόνο που
149 αυτός ο τρόπος είναι καταστροφικός τελικά. Δεν ξέρω πώς αλλιώς να το περιγράψω.
- 150 Πιστεύετε ότι αν μιλούσατε θα ήταν αλλιώς τα πράγματα? Δεν θα υπήρχε η
151 ΔΠΤ?
- 152 Ναι. Γιατί να υπήρχε? Αν μπορούσα να βγάλω όλα αυτά από μέσα μου δεν θα με
153 «έπρωγα».
- 154 Όταν λέτε «αυτά» τί εννοείτε?
- 155 Τα συναισθήματα φυσικά! Τον θυμό. Τη στεναχώρια για όσα συμβαίνουν και δεν
156 μπορώ να μιλήσω. Τη θλίψη. Αυτά δεν «πράννε» όλους τους ανθρώπους? Γι' αυτό ο
157 κόσμος αρρωσταίνει.

- 158 **Ο θυμός που απευθύνεται?**
- 159 Στον πατέρα μου που ήταν κακός μαζί μου. Στη μητέρα μου που δεν με προστατεύσει.
160 Που δεν με έμαθε να είμαι δυνατή.
- 161 **Κάπου αλλού?**
- 162 Στον εαυτό μου που δεν ήταν αρκετά δυνατός για να με προστατεύσει. Πολλές φορές
163 νιώθω θυμό για όλο τον κόσμο. Θυμώνω και όταν βλέπω άλλους που δεν μεγάλωσαν
164 έτσι. Τους ζηλεύω και αυτό με κάνει να θυμώνω μαζί τους. Γιατί αυτοί να είναι
165 δυνατοί κι εγώ όχι? Με θυμώνει αυτό.
- 166 **Η θλίψη?**
- 167 Μετά από σκέψεις που μου προκαλούν θυμό, νιώθω θλιμμένη. Θλιμμένη και
168 κοροιασμένη ταυτόχρονα. Ίσως θλιμμένη γιατί ξέρω ότι δεν είμαι ικανή να
169 αντιμετωπίσω όσα με θυμώνουν. Αυτός που με θυμώνουν. Όταν νιώθεis κάτι έντονα
170 κοροιάζεσαι, έτσι δεν είναι?
- 171 **Ναι. Πώς νιώθετε ότι όλα αυτά σας «τρώνε»?**
- 172 Είναι πιο δυνατά από εμένα. Γι αυτό φοβάμαι να μιλήσω. Δεν ξέρω τι θα γίνει αν τα
173 εκφράσω. Αντί να τα βγάλω έξω και να καταστρέψουν κάποιον άλλον τα αφήνω
174 μέσα μου και καταστρέφουν εμένα. Με τρώνε από μέσα. Κάπως έτσι μπορώ να το
175 περιγράψω. Βλέπω γύρω μου αυτούς που μπορούν και το κάνουν. Είναι μια χαρά δεν
176 δείχνουν λυπημένοι. Λένε ότι θέλουν και δεν τους νοιάζει τίποτα.
- 177 **Πιστεύετε ότι αυτή η κατάσταση θα αλλάξει?**
- 178 Μόνο αν αλλάξω εγώ. Πρέπει να γίνει πιο δυνατή και να υπερασπιζομαι τον εαυτό
179 μου.
- 180 **Θέλετε να μου πείτε πιο συγκεκριμένα?**
- 181 Να μιλάω όταν κάτι με ενοχλεί και να μην το επιτρέπω να συμβαίνει. Να μην
182 επιτρέπω στους άλλους να μου κάνουν σχόλια για τον εαυτό μου και να
183 παρεμβαίνουν στη ζωή μου. Να αποκτήσω αυτοπεποίθηση και να πιστέψω στον
184 εαυτό μου.
- 185 **Ποιοι παράγοντες πιστεύετε ότι θα εμποδίσουν την κατάσταση να αλλάξει?**
- 186 Εγώ θα είμαι το εμπόδιο αν δεν αλλάξω. Μόνο εγώ μπορώ να το αλλάξω αυτό. Μόνο
187 εγώ μπορώ να υπερασπιστώ τον εαυτό μου. Αλλά στη θεωρία είμαι καλή...στην
188 πράξη δεν μπορώ να το κάνω. Η αυτοεκτίμησή μου είναι χαλιά.
- 189 **Κλείνοντας, είναι κάτι που θέλετε να προσθέσετε?**
- 190 Όχι νομίζω ότι είπα όλα όσα ήθελα.
- 191 **Σας ευχαριστώ πολύ για τον χρόνο σας**

Appendix 13- Interview transcript Carol in Greek

1

- 1 Θα ήθελα να μου μιλήσετε για την εμπειρία που έχετε σχετικά με τη μη φυσική
2 μορφή κακοποίησης στην ενήλικη ζωή.
- 3 Μένω με τους γονείς μου οπότε δεν άλλαξε κάτι. Όπως μου φέρονταν πριν μου
4 φέρονται και τώρα.
- 5 Μπορείτε να μου πείτε πιο συγκεκριμένα την εμπειρία τώρα ως ενήλικη?
- 6 Με ακουράνουν. Είναι σα να μην κάνω τίποτα σωστά. Λεκτικά πιο πολύ.
- 7 Μπορείτε να μου δώσετε κάποιο παράδειγμα?
- 8 Όταν κάνω κάτι που νομίζουν ότι είναι λάθος ακούω «είσαι ηλίθια»? Και διάφορα
9 τέτοια αντίστοιχα. Κάνουν και σύγκριση με κόρες φίλων και με ξαδέλφες.
- 10 Δηλαδή?
- 11 Ότι τα έχουν καταφέρει καλύτερα στη ζωή τους επαγγελματικά και προσωπικά. Πιο
12 παλιά έκαναν σύγκριση για τους βαθμούς στο σχολείο.
- 13 Κατάλαβα
- 14 Δεν ξέρω πώς να το εκφράσω αλλά είναι σα να κάνω πάντα λάθος ή να περιμένουν
15 ότι θα κάνω λάθος. Και για το σώμα μου.
- 16 Θέλετε να μου πείτε?
- 17 Να μην τρώω πολύ και να κάνω γυμναστική. Τα ίδια μου έλεγαν και όταν ήμουν
18 μικρή. Μου έλεγαν ότι αν παχύνω θα με κοροϊδεύουν τα παιδιά στο σχολείο
- 19 Αυτό το έχετε βιώσει μόνο από τους γονείς?
- 20 Όχι μόνο. Και από αυτόν που έχω σχέση. Είναι πολύ κακοποιητικός. Σα να μη με
21 θέλει. Σα να είναι συνέχεια θυμωμένος μαζί μου. Δεν ξέρω. Δεν μπορώ να το
22 εξηγήσω.
- 23 Δηλαδή?
- 24 Είναι κάπως σα να φεύγω από τους γονείς μου και να πηγαίνω σε ένα ίδιο
25 περιβάλλον. Αντιδρά πολύ άσχημα και με προσβάλλει. Με κάνει να είμαι συνέχεια
26 στεναχωρημένη.
- 27 Θέλετε να μου εξηγήσετε?
- 28 Είμαι συνέχεια αγχωμένη μήπως και κάτι λάθος ή μήπως κάνω κάτι λάθος και αρχίζει
29 να μου φωνάζει. Μια φορά με είχε αφήσει σε μαγαζί και είχε φύγει επειδή είχαμε
30 ραντεβού και άργησα δέκα λεπτά. Με έκανε ρεζίλι και έφυγε. Μας κοιτούσε ο
31 κόσμος και ντροπέρισσα πολύ.
- 32 Κατάλαβα.

- 33 Είναι σα να βρίσκει συνέχεια αφορμές να τσικανόμαστε. Χωρίς λόγο. Με κάνει να
34 νιώθω ότι είμαι άχρηστη. Σα να μην αξίζω να είναι κάποιος μαζί μου. Σα να μην του
35 κάνει.
- 36 Γιατί μένετε σε αυτή τη σχέση αφού είστε συνέχεια στεναχωρημένη και δεν
37 φέρεται καλά?
- 38 Το έχω σκεφτεί και αυτό πρέπει να κάνει. Δεν ξέρω γιατί δεν μπορώ. Ίσως γιατί δεν
39 έχω πού να πάω. Όλες οι φίλες μου έχουν σχέση. Μόνο εγώ θα είμαι μόνη μου. Με
40 ποσόν θα βγαίνω? Κάποιος άλλος θα είναι καλύτερος? Κι αν δεν βρω? Αν μείνω για
41 πάντα μόνη μου?
- 42 Φοβάστε ότι θα μείνετε μόνη σας?
- 43 Ναι. Είναι πολύ δύσκολο να γνωρίσεις κάποιον και να θέλει σχέση. Όλοι ψάχνουν
44 μόνο σεξ τώρα. Τώρα με την καραντίνα είναι ακόμα χειρότερα. Όσοι έχουν ήδη μια
45 σχέση είναι τυχεροί. Ξέρω όμως ότι θα έπρεπε να φύγω.
- 46 Τι σας δυσκολεύει να φύγετε?
- 47 Δεν ξέρω. Ενώ με στεναχωρεί από την άλλη είναι μια κατάσταση που την ξέρω και
48 την έχω συνηθίσει. Μου είναι όλα γνωστά. Τα περιμένω. Μερικές φορές σκέφτομαι
49 ότι αν βρω κάποιον που μου φέρεται καλά δεν θα ξέρω τι να κάνω.
- 50 Ενώ σε μια κατάσταση που είναι κακοποιητική ξέρετε τί να κάνετε?
- 51 Ναι ξέρω τί να περιμένω. Δεν έχει εκπλήξεις
- 52 Οπότε στην ενήλικη ζωή η μη φυσική κακοποίηση σχετίζεται με ακύρωση
- 53 Ναι. Προσβολές και φωνές. Υπάρχουν πολλά κοινά με τους γονείς μου και τον φίλο
54 μου. Είναι κάτι που όταν το κατάλαβα σκαρίστηκα.
- 55 Θέλετε να μου φέρετε κάποιο παράδειγμα?
- 56 Κάποιες φορές που μαγειρεύω μπορεί να μου πέσει κάτι κάτω ή να λερώσω κάτι στην
57 κουζίνα και ο φίλος μου έχει ακριβώς την ίδια αντίδραση με την μητέρα μου. Ή θα
58 μου κάνει επαντικό σχόλιο για τις ικανότητές μου ή θα μου φωνάζει. Την πρώτη
59 φορά που έγινε αυτό στο σπίτι του φίλου μου πάγωσα. Ήταν σα να άκουγα τη μάνα
60 μου.
- 61 Κατάλαβα
- 62 Και τώρα που το λέω, καταλαβαίνω ότι κάθε φορά που μου φωνάζει παγώνει.
- 63 Γιατί παγώνετε?
- 64 Δεν μπορώ να αντιδράσω. Περιμένω να τελειώσει γιατί φοβάμαι να μιλήσω. Δεν
65 ξέρω τί να πω και πώς να το αντιμετωπίσω. Θυμάνω και στεναχωριέμαι. Δεν έχω

- 66 μάθει να απαντάω όταν κάποιος μου κάνει επίθεση. Δεν ξέρω πώς να το κάνω.
67 Επίθεση δεν είναι αυτό?
- 68 **Ναι. Είναι μια μορφή επιθετικής συμπεριφοράς. Η κακοποίηση εκφράζεται**
69 **περισσότερο από τον πατέρα σας ή από τη μητέρα σας?**
- 70 Εκφράζεται και από τους δύο, ίσως πιο πολύ λεκτικά από τη μητέρα μου. Είναι πολύ
71 επικριτική
- 72 **Οπότε αυτό είναι το πλαίσιο της κακοποίησης στην ενήλικη ζωή.**
- 73 Ναι
- 74 **Από άλλους ανθρώπους το βιώνετε αυτό πχ από φίλους?**
- 75 Όχι ιδιαίτερα. Δεν έχω να θυμηθώ κάτι από φίλους
- 76 **Υπάρχει κάτι ίδιο ή διαφορετικό στην εμπειρία σας ως ανήλικη και ως ενήλικη**
77 **στη μη φυσική μορφή κακοποίησης?**
- 78 Στην ανήλικη ζωή δεν μπορούσα να αντιδράσω και τα κρατούσα όλα μέσα μου. Το
79 ίδιο κάνω και τώρα.
- 80 Δηλαδή?
- 81 Δεν απαντάω γιατί αν απαντήσω νομίζω ότι μπορεί και να τους δείρω έτσι όπως
82 θυμάμαι. Έχω πολύ θυμό. Πολλές φορές σπάζω πράγματα όταν είμαι μόνη μου.
- 83 **Γιατί πιστεύετε ότι δυσκολεύεστε να αντιδράσετε όταν κάτι σας ενοχλεί και σας**
84 **προσβάλλει?**
- 85 Δεν ξέρω πώς να το κάνω. Δεν μπορώ να ελέγξω την αντίδρασή μου και δεν μπορώ
86 να ελέγξω και την αντίδραση των άλλων. Φοβάμαι πώς θα εξελχθεί.
- 87 **Ποιο είναι το χειρότερο που φοβάστε ότι θα γίνει?**
- 88 Ότι ο φίλος μου θα γίνει βίαιος ή θα χωρίσουμε.
- 89 **Σε σχέση με τους γονείς σας?**
- 90 Ότι δεν θα ξαναμιλήσουμε ποτέ. Καμιά φορά σκέφτομαι ότι μπορεί να έχουν δίκιο.
91 Είναι τυχερό που και οι γονείς και ο φίλος μου, μου φωνάζουν για τα ίδια πράγματα?
92 Από την άλλη καταλαβαίνω ότι δεν μπορεί να φταίω για όλα εγώ και η συμπεριφορά
93 τους δεν είναι αυτή που θα έπρεπε. Έχουν νευρά για τους δικούς τους λόγους και τα
94 βγάζουν πάνω μου.
- 95 **Είχατε την ίδια συμπεριφορά και από προηγούμενο σύντροφο?**
- 96 Δεν είχα άλλη σχέση πριν από αυτόν.
- 97 **Μάλιστα κατάλαβα. Πόσο καιρό είστε μαζί?**

- 98 Δύο χρόνια.
- 99 Έχετε την ίδια ηλικία?
- 100 Όχι αυτός είναι 8 χρόνια μεγαλύτερος.
- 101 Σας κάνει σχόλια και για την εξωτερική σας εμφάνιση?
- 102 Ναι.
- 103 Θέλετε να μου πείτε?
- 104 Κοιτάζει συνέχεια πόσο τρώω. Αν φάω πολύ μου λέει ότι θα παχύνω και μου προκαλεί εκνευρισμό αυτό.
- 105
- 106 Κατάλαβα.
- 107 Με αγχώνει ότι θα παχύνω. Παρατηρεί ότι πήρα βάρος και μου λέει ότι δεν θα μου κάνουν τα ρούχα μου.
- 108
- 109 Του έχετε πει ότι σας ενοχλεί αυτό που κάνει?
- 110 Όχι. Τι νόημα έχει? Δεν θα το κατάλαβει. Δεν μπορώ να τσακωθώ μαζί του. Δεν αντέχω άλλες φωνές και προσβολές.
- 111
- 112 Πώς αντιδράτε όταν σας κάνει τέτοια σχόλια?
- 113 Δεν αντρώ. Θυμάνομαι και στεναχωριέμαι ταυτόχρονα. Θέλω να φάω επειδή με έχει εκνευρίσει. Να φάω πολύ και να με βλέπει. Κάποιες φορές το έκανα αλλά μετά έκανα εμετό χωρίς να το ξέρει. Μου έλεγε «φάε έτσι και δεν θα χωράς να περάσεις την πόρτα». Ηβέλια να του ρίξω μπουλιά. Μακάρι να του έριχνα. Αλλά αντί να τον χτυπήσω συνέχισα να τρώω.
- 114
- 115
- 116
- 117
- 118 Γιατί συνεχίζετε να τρώτε αντί να του μιλήσετε?
- 119 Γιατί είναι πιο εύκολο για μένα να φάω από το να μιλήσω. Θα τσακωθούμε και θα φωνάζει. Δεν θέλω εντάσεις.
- 120
- 121 Για ποιο λόγο πιστεύετε ότι συνέβη αυτή η κακοποίηση όταν ήσασταν ανήλικη?
- 122 Δεν ξέρω. Νομίζω ότι έχει να κάνει με προβλήματα στον χαρακτήρα των γονιών μου.
- 123 Δηλαδή?
- 124 Έχουν δικιά τους θέματα και τα βγάζουν σε εμένα. Προφανώς δεν ξέρουν να φερθούν.
- 125 Η γιαγιά μου – της μητέρας μου η μαμά- ήταν πολύ αυταρχική. Μπορεί να φταίει αυτό που η μάνα μου είναι έτσι.
- 126
- 127 Κατάλαβα.
- 128 Δεν είναι όλοι οι γονείς έτσι. Έχει να κάνει με τον χαρακτήρα τους.

- 129 Για ποιους λόγους πιστεύετε ότι αυτή η κατάσταση συμβαίνει ακόμα?
- 130 Γιατί δεν αντιδρά. Σε αυτό φταίω εγώ και θυμάμαι με τον εαυτό μου. Αν αντιδρούσα
131 από μικρή δεν θα γινόταν αυτό. Είμαι αδύναμος χαρακτήρας. Αποφεύγω τη
132 σύγκρουση μαζί τους γιατί δεν το αντέχω καθόλου. Δεν μπορώ να τσακάνομαι. Αν
133 έμεινα μόνη μου θα ήταν καλύτερα πιστεύω. Αλλά δεν μπορώ τώρα.
- 134 Ποιοι παράγοντες πιστεύετε ότι διατηρούν τη δική σας στάση και συμπεριφορά
135 όπως να μη βάζετε όρια όταν κάποιος δεν σας φέρεται καλά?
- 136 Έχω συνηθίσει έτσι νομίζω. Έτσι είμαι δηλαδή. Σαν χαρακτήρας. Αυτό ξέρω να
137 κάνω. Οι άλλοι με βλέπουν σαν θύμα. Σαν αδύναμη εννοώ. Την επίθεση την κάνεις
138 σε κάποιον που φαίνεται αδύναμος όχι σε κάποιον που μοιάζει δυνατός
139 (αναστεναγμός)
- 140 Είναι μια κατάσταση που σας είναι οικεία δηλαδή.
- 141 Έτσι ακριβώς.
- 142 Πώς νιώθετε για την κακοποίηση αυτή?
- 143 Απάθεια. Σε να μην αξίζω τίποτα. Συνέχεια σκέφτομαι τι μπορώ να κάνω για να
144 φάνομαι καλύτερη. Να βρω καλή δουλειά, να γίνω όμορφη. Δεν ξέρω. Αν είμαι
145 πετυχημένη δύσκολα θα μου μιλήσει κάποιος υποτιμητικά. Έτσι νομίζω.
- 146 Συναισθήματα?
- 147 Θυμός. Και ζήλεια.
- 148 Ζήλεια προς ποιον?
- 149 Ζηλεύω τους άλλους που βλέπω ότι είναι πιο δυνατοί από εμένα ή που οι γονείς τους
150 είναι καλύτεροι.
- 151 Θυμός για ποιον?
- 152 Για όλους. Τους γονείς μου, τον φίλο μου, για μένα που δεν αντιδρά. Πολλές φορές
153 ξυπνάω έτσι με θυμό και νεύρα χωρίς κάποιο λόγο. Μου έρχεται να τα σπάσω όλα
154 και δεν έχει γίνει κάτι. Απλά ξυπνάω έτσι και με κοιτάζει αυτό.
- 155 Πώς το αντιμετωπίζετε όλο αυτό?
- 156 Νομίζω ότι δεν το αντιμετωπίζω. Απλά θυμάμαι πολύ μέσα μου. Έχω προσπαθή να
157 μην το δείχνω. Τρόω. Με ανακουφίζει αυτό εκείνη την ώρα αλλά μετά νιώθω
158 άσχημα.
- 159 Πιστεύετε ότι αν δείχνατε τον θυμό σας θα άλλαζε κάτι στη σχέση σας με το
160 φαγητό?

- 161 Δεν είμαι σίγουρη αλλά νομίζω να. Δεν θα χρειαζόταν να ανακουφιστώ από κάτι.
162 Αλλά δεν μπορώ να ξέρω σίγουρα.
- 163 Νιώθετε ότι το φαγητό σας ανακουφίζει από τον θυμό?
- 164 Ναι.
- 165 Με ποιον τρόπο?
- 166 Είναι σα να θέλω να κάνω κάτι έντονο εκείνη την ώρα αλλά δεν μπορώ. Τρώγοντας
167 είναι σα να ξεχνάω τον θυμό ή να τον πνίγω. Σα να τον καλύπτει το φαγητό και να
168 ξεχνάω. Αλλά τρώγοντας κάτι που μου αρέσει πολύ όχι απλά να φάω σιγήνους.
- 169 Υπάρχει κάτι που το κάνει πιο εύκολο να το αντιμετωπίσετε?
- 170 Η ψυχοθεραπεία με βοηθάει να εξηγήσω και να καταλάβω κάποια πράγματα για τον
171 εαυτό μου και τους άλλους. Αλλά χρειάζεται καιρός για να μπορέσω να αλλάξω κάτι.
172 Δεν μπορώ να γίνω άλλος άνθρωπος ξαφνικά.
- 173 Κάτι που δυσκολεύει την αντιμετώπιση?
- 174 Το ότι δεν μπορώ να μιλήσω όταν πρέπει. Αυτή η δυσκολία που έχω να μιλήσω.
175 Αυτό με τρώει. Που τα κρατάω όλα μέσα μου.
- 176 Η σχέση με το σύντροφό σας έχει παίξει κάποιο ρόλο στο πώς αντιμετωπίζετε
177 την κατάσταση?
- 178 Με έχει κάνει να είμαι ακόμα πιο αδύναμη νομίζω. Αν ήταν διαφορετικός μπορεί
179 αυτό να με βοηθούσε να δυναμώσω.
- 180 Πώς το εννοείτε αυτό?
- 181 Θέλω να πω ότι θα ένιωθα πιο καλά με τον εαυτό μου αν μου φερόταν καλά. Θα είχα
182 αυτοεκτίμηση και θα ήμουν πιο δυνατή. Θα απαντούσα. Θα αντιδρούσα! Σημαντικό
183 πράγμα να έχει κάποιος αυτοεκτίμηση. Οι άλλοι δεν του φέρονται σαν σκουπίδι. Δεν
184 τους παίρνει.
- 185 Η ψυχοθεραπεία έχει αλλάξει καθόλου τον τρόπο που αντιμετωπίζετε αυτή την
186 κατάσταση της κακοποίησης?
- 187 Έχει αλλάξει μέσα μου. Να καταλάβω κάποια πράγματα για μένα. Έξω δεν έχω
188 αλλάξει. Ίσως θέλει πολύ καιρό αυτό για να γίνει.
- 189 Πιστεύετε ότι η κακοποίηση τώρα σχετίζεται με τα συμπτώματα της ΔΠΤ με
190 κάποιο τρόπο?
- 191 Ναι.
- 192 Με ποιον τρόπο?

- 193 Όταν με βλέπουν να τρώω μου λένε ότι θα γίνω χοντρή και δεν θα μου κάνουν τα
 194 ρούχα μου εγώ συνεχίζω να τρώω! Με ηρεμεί αυτό και ξέρω καλάς ότι τους
 195 ενουερίζει να το κάνουν. Νιώθω ανασκούφιση όταν τρώω αλλά μετά αφήνω με τον
 196 εαυτό μου και θέλω να κάνω εμετό.
- 197 Πιστεύετε ότι η κακοποίηση στην ανήλικη ζωή σχετίζεται με την ανάπτυξη της
 198 ΔΠΠ?
- 199 Ίσως να, αλλά δεν μπορώ να ξέρω σίγουρα.
- 200 Με ποιο τρόπο μπορεί να συνδέεται η κακοποίηση με την έναρξη ΔΠΠ?
- 201 Όταν κάτι σε στεναχωρεί και δεν είσαι καλά και αυτό κρατάει καιρό θα επηρεάσει
 202 την ψυχολογία σου.
- 203 Με ποιον τρόπο θα την επηρεάσει?
- 204 Όταν κρατάς πράγματα μέσα σου... θυμό, στεναχώρια, παράπονα... αυτά πρέπει να
 205 βγουν. Θα βρουν τρόπο να βγουν αλλά αυτός ο τρόπος δεν θα είναι καλός. Δεν θα
 206 είναι υγιής.
- 207 Πιστεύετε ότι αυτή η κατάσταση θα αλλάξει?
- 208 Μόνο αν αλλάξω εγώ. Οι άλλοι δεν πρόκειται να αλλάξουν. Είναι αυτοί που είναι.
 209 Εγώ πρέπει να γίνω πιο δυνατή. Να υπερασπιζομαι τον εαυτό μου. Να έχω
 210 αυτοεκτίμηση και να φαίνεται αυτό. Να βάζω όρια. Να μιλάω. Χωρίς να με νοιάζει τί
 211 θα γίνει.
- 212 Ποιοι παράγοντες πιστεύετε ότι θα εμποδίσουν την κατάσταση να αλλάξει?
- 213 Εγώ αν δεν αλλάξω. Ο χαρακτήρας μου είναι το εμπόδιο. Πρέπει να αλλάξω
 214 χαρακτήρα. Να μην είμαι αδύναμη.
- 215 Κλείνοντας, είναι κάτι που θέλετε να προσθέσετε?
- 216 Οχι. Νομίζω τα είπα όλα...
- 217 Σας ευχαριστώ πολύ για τον χρόνο σας

Appendix 14- Interview transcript Kate in English

1

1 5th interview AN-purging type (former AN-R)

2 **Could you tell me about your experience with nonphysical abuse in minor life?**

3 To begin with, I was quite overweight and I was in a countryside environment where
4 people put a lot of emphasis on these things... I have only grown up with my mother
5 who was not worried about these matters so I had gained weight... so I was
6 essentially alone in everything I lived... bullying which was essentially criticism and I
7 also had very strong criticism from my social environment, uncles, aunts, teachers,
8 parents of friends, all those who were trying to do the job that my mother did not do
9 but not with very nice way.

10 **Would you like to explain to me?**

11 From a very young age I remember hearing bad things about my body, that is, my
12 aunt screaming at me that I have cellulite from the age of 12 or I was generally
13 accepting a lot of food shaming to the extent that I considered it normal, for example I
14 will not be eating at family dinners because I'm fat or we all gather together and I will
15 not eat because I have to diet. I was just eating secretly.

16 **Did they tell you that or did you do it because you were ashamed to eat?**

17 Up to one point they were telling me that (not to eat)... but they also made constant
18 comments... I think I was also hypersensitive so as soon as discussions about
19 silhouette and weight started and things like "let's watch what we eat" and they all
20 stared at me... you understand. And my friends at the time who had a lot anxiety
21 about their own body, even though they were very thin, they talked to me very
22 badly... they did not actually bully me but they would just say to me "I cannot look at
23 your hands because they are so fat". They believed it at the time I do not think they
24 were saying it out of malice, but it was all this paranoia at the time meaning it was
25 awful to have fat and extra weight in high school.

26 I understand. Were there any comments about your body? How was this
27 expressed, for example, by your relatives?

28 Yes... I was encouraged to diet... I was commented on both my body and my diet not
29 in the right way. They were trying to terrify me about everything... like 'this will
30 make you fat, it has 5000 calories'.

31 Your mother in all this, I imagine she would be present sometimes... was she
32 saying anything? Was she taking a position?

33 Uhm..

34 Did she say anything? Did she agree with them?

35 She was a little apathetic.

36 Did she say they were right? Did she tell you something later at home? Did she
37 say do not listen to them; How did she handle this issue? Because you said you
38 had it from everyone else but what about from your mother?

39 She did not handle it. When I complained to her that I was called fat and ugly..she
40 would say to me "ok it is bad that they call you ugly but objectively you are fat"
41 generally my mother is not good at supporting someone emotionally... she could not
42 help me at all in this whole thing. Telling someone that he is fat without helping him
43 does not help him much. But in general my mother was a bit uninvolved because it
44 was her style to be non-intrusive.

45 In other parts of your life was there support from your mother; In any other
46 problem you may have had?

47 It was not very easy to communicate with my mother ... she had grown up in a
48 different way, she could not understand my own problems. For her it was a problem
49 that they once dug in the field and we should be happy that we do not dig in the field

50 anymore. While she provided me with everything she was not good at interpersonal
51 support.

52 **Did you have any contact with your dad?**

53 No no. They divorced when I was born. I have no contact

54 **Any siblings?**

55 No, I am an only child

56 **I understand. Did you have friends to talk about what was troubling you?**

57 I had some girlfriends but they were a bit..how to say this... very authoritarian... I
58 was very easy going. I always had a friend who was very dynamic for whom I did
59 what she wanted me to do but I always got involved into such relationships that I was
60 the stepping stone..the boxing bag and it probably has to do with my personality

61 **Were you low profile?**

62 Very low profile

63 **What do you mean by saying they did to you what they wanted? An example?**

64 Uhm let me think..with one of my friends it was always expected that I will wait for
65 her, I will move her around or if I did not want to go to her house she would tell me
66 "since you want to lose weight, walking will be good for you" . Because it was so
67 obvious that I had an issue they were stepping on it. I did not understand and I thought
68 that the only solution not to accept such behavior would be to lose weight. Or I
69 thought it was reasonable to say to your friend who is overweight " you should walk
70 to my house so you can lose weight".

71 **So in minor life the comments were about weight and body image. I understand
72 that these bothered you. Did you speak?**

73 They bothered me a lot but I had no one to talk about it

74 **At that time, when someone was bothering you, did you tell him/her?**

75 No. I thought they had the right because I was giving it to them by being fat

76 **Did that create any feelings for you?**

77 Very low self-esteem. I was self-whipping endlessly, that is, I started dieting ... I felt
78 very bad when I ate. I ate secretly I had various emotional and self-regulation
79 difficulties.

80 **Apart from self-esteem, all this caused you any other emotions that were intense;**
81 I had negative feelings - I was not sure if it was right that I felt them. At the end of
82 high school I had decided to be gothic so to get the attention out of my weight because
83 everyone was concerned with the fact that I had green hair and wore black clothes. I
84 had gotten into such cliques that everyone was angry and delinquent — even though I
85 was a good student. I felt like we had something in common so I vented it (anger) a
86 bit like that.

87 **I understand. In adulthood was there anything that bothered you?**

88 After 18 I gained more weight and I started dieting. It was very difficult for me to
89 meet people and I blamed my weight for this. But now I understand that I was not
90 social and I would not meet people by staying in my house but at the time I thought I
91 looked like a monster but also some things had happened. I had a high school friend
92 who was bossy and had her own issues with her body and kept commenting on my
93 body. On top of that, I joined a university theater group and the director - I was in
94 normal weight at the time but it was established that I am the person who is being
95 bullied for her weight and they would tell me not to eat because "you will gain weight
96 again"- he (the director) had picked me up for an exercise and he was shooting me
97 from some angles and he was saying to me "what a nice face you have..if you only
98 didn't have this body" and he told me that a room full of people and I was a very shy
99 freshman and I remember coming home and crying for 5 hours. And I said that

100 everyone is right, I am a monster and I must definitely lose weight, otherwise nothing
101 good will ever happen to me in my life. There I started my first paranoid diet. I said I
102 would continue not to eat until the fat was completely gone.

103 **I see. Were there other such incidents?**

104 I was hypersensitive and I magnified everything.. someone on the street would see me
105 eating and tell me to diet I would take that comment very seriously.

106 **Have you had such comments from partners?**

107 During my first serious relationship I had anorexia so no, but with some guys I just
108 went out prior to the anorexia phase I had rude comments about my weight.

109 **Was there a boyfriend who was authoritarian, since you told me earlier that you
110 were engaging in such relationships due to your low profile?**

111 This personality died with anorexia because I decided to protect myself but in the
112 wrong way. I did not have authoritarian partners but one of them had bad outbursts
113 with a lot of yelling.

114 **How did you protect yourself with anorexia?**

115 I protected my vulnerable self. My low self-esteem self. Anorexia "helped" me lose
116 weight, become pretty, feel better about myself. It helped me "kill" the ugly
117 overweight girl that everyone mocked.

118 **I see.**

119 You see, anorexia started because I was mad at myself for being overweight. I got
120 angry at myself and I thought bullying was my fault because I was indeed fat. In my
121 mind, if I could lose weight and become thin and pretty bullying would be over. I
122 thought that by losing weight my self-esteem would dramatically improve and people
123 do not bully people with high self-esteem. That was the solution in my mind. It was
124 an unhealthy way to deal with it of course.

125 I see.

126 You know...self-esteem is visible. Others can see if you have it or not. And they treat
127 you accordingly. Low self-esteem equals an easy target. An easy victim. Weakness.
128 Anorexia was the easy way towards a better self-esteem. It's not that I woke up one
129 day and thought to myself "let's have anorexia"...no. I started dieting like crazy and at
130 some point I lost control.

131 I understand. Is there any difference or similarity with your experience in the
132 non-physical form of abuse as a minor and as an adult?

133 In my minor life I was told not to eat because I am fat and in adulthood to eat because
134 I am thin and I took the later as a compliment because of what I had gone through
135 with my weight.

136 Any similarities or differences in your reaction to the abuse?

137 Eh. in my minor life I was sad and I felt that I could not do anything. Later what I
138 could do was to lose weight and for the last 4 years with psychotherapy I realized that
139 I can answer back and I am not obliged to accept such behaviors. Now I have a hard
140 time with the idea of gaining weight but I am trying to work it through.

141 You told me that all this affected your self-esteem as a minor. Is it still the case
142 today?

143 In general, my self-esteem is not good. It created instability in how I see myself. I
144 have compulsions with weighingg myself and I think my self-esteem is related to the
145 outcome. That is, I try to stay at the weight I am now. Not to lose control and become
146 someone else again (fat). But with self-esteem I have issues.

147 I see. Did the abuse affect you elsewhere?

148 In how I connect with others. I do not know if this is because I am hypersensitive
149 though.

150 **What do you mean?**

151 I generally have an instability in my relationships. I find people who have issues and
152 they are bossy.

153 **Did the abuse affect you in how you connect with others?**

154 Now I am social but I avoid close relationships. I do not attach, I am afraid to attach

155 **What do you afraid of?**

156 The fact that I do not expect understanding. In the relationships I had I was trying to
157 keep a different face -I was not open to the problems I have with food - I was very
158 ashamed and I was trying to cover it all up and I was not myself.

159 **Why do you think child abuse took place?**

160 I think this happens to children who are overweight. People consider it normal to do
161 so. If you go to an adult to ask for help he will tell you to lose weight to resolve this.
162 But if someone has a big nose, an adult can say that it is not ok to make fun of it, but
163 for the weight they believe that it is your fault. I was also withdrawn and shy... I was
164 the kind of child you will bully. It's my fault that I was accepting the bullying and I
165 did not react.

166 **I see. It also had to do with your own reaction to it?**

167 Yes definitely

168 **Why do you think this happens in adulthood?**

169 Because I was born a woman. It is common to be stressed for appearance even when
170 you have a normal body. Especially in artistic circles it is normal to make comments
171 about the body and to be under pressure not to gain weight. I also had hypersensitivity
172 to this matter due to bullying at a young age and I accepted it as a victim so this was
173 also a reinforcing factor.

174 **Do you think this situation will change?**

175 Now I am thin and it has been a long time since I last heard something about my
176 weight. Now I can avoid it because I am thin but if I gain weight it will happen again.

177 **I see. Has this whole situation affected you somewhere?**

178 In everything. I have lost huge periods of time in my life while fighting with the ED. I
179 was late to finish college because I took a long break when I lost weight to go to work
180 in a bar because I thought it would boost my self-esteem. And there were times when
181 I did not leave the house and cried because I was ashamed that I was fat and depressed
182 because I was overweight. I had focused on the weight and I lost time from my life
183 mainly in the professional part and now at the age I am I should have done more
184 things and have a better CV. Now I have gaps in my CV.

185 **What about emotions?**

186 Clearly. It made me feel depressed.

187 **Anything else?**

188 Anger. I have a lot of anger. Over the last few years I have realized that I have a right
189 to be angry.

190 **The anger for whom do you have it? Where is it directed at?**

191 This is a very nice question because it really is like holding a hand grenade and not
192 knowing where I want to throw it. I mean, I blame myself, I blame my mother
193 because she let me gain weight, I blame society for being so oppressive with women
194 and weight, but most of all I burst out my anger with bulimic episodes. I do not know
195 who to blame first.

196 **I see. How did you handle your anger as a minor?**

197 I was eating. I suppressed my anger with food. I was told that I was fat and I ate to
198 feel better. When I was younger, I did not realize that I had the right to be angry.

199 **So you did not express your anger?**

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200 Not verbally. I was the good kid and I didn't talk. A couple of times I had put out
201 cigarettes on myself and another time I had taken a key and gone through all the cars
202 in the neighborhood to damage them. I had suppressed anger and so I took it out like
203 that.

204 **As an adult how do you express it? Is there anger?**

205 The whole eating problem had to do with anger because I punish myself with bulimic
206 episodes. And when I eat more I hit my hand on the wall because I am angry that I
207 ate. When I started with anorexia I blamed myself for everything. I believed that in
208 order to deserve better behavior I had to be thin. The whole phase of anorexia was as
209 if I had turned my anger on myself. I did not feel angry with others then. Now with
210 bulimia I understand how angry I am with others. I understood that with
211 psychotherapy. And now with the slightest reason I get very angry and throw things.
212 To come into direct conflict with someone... I have not done it. I'm afraid of conflict.

213 **Why are you afraid of coming into conflict?**

214 This is how I learned from my mother from a young age. That it is important not to
215 argue and not to have tensions. That the purpose in this life is not to argue. Now I
216 know this is not right but I find it very difficult to argue with someone. I have
217 difficulty with tensions. Even when I see others arguing, I get very nervous.

218 **In the anorexia phase you told me that you had turned your anger at yourself.**

219 **What about the bulimia phase;**

220 In bulimia I started to think that I was not to blame for the bullying and I started to
221 take out my anger. But this made me very confused because I thought I had organized
222 things in my head. Meaning that I am to blame for everything and I should not eat.
223 Then I realized that there were people who misbehaved and I did not know where to
224 turn my anger. I started thinking different things. I mean ok about my friends because

225 they were young and didn't know any better and they were also stressed about
226 appearance but the adults had to find a better way to talk to a child. I was helped by
227 what I read about feminism and that society has a responsibility for this by creating
228 wrong role models for women.

229 **So in anorexia the anger was clear and it was your fault but in bulimia you**
230 **thought it was others' fault. Do you think this change of mind played a role in**
231 **changing the symptoms?**

232 I think so but I cannot say exactly how it was done. However, when I started to think
233 differently and to know that it was not my fault, then bulimia came along. I think that
234 from one point onwards I saw bulimic episodes as a revolutionary act. But it's a bit
235 silly because in reality I was rebelling against myself. I made myself not eat but not
236 eating has been imposed to me by others through all these years of bullying.

237 **Do you vomit during the bulimia phase?**

238 I was taking some medicine to vomit. I ate once every 2 days a lot and then I took a
239 medicine to vomit. Now I am trying to fix all these in therapy.

240 **Has psychotherapy helped you?**

241 Definitely yes. I realized that it was not my fault and that I had the right to be angry. I
242 understood my relationship with food and I can explain it now.

243 **I see. Do you think that everything you have experienced is related to the ED**
244 **onset?**

245 Yes very much.

246 **How?**

247 I think if I did not have these experiences it (ED) might not have happened. I wouldn't
248 need to lose weight in order to be accepted and become obsessed with it. I am
249 generally sensitive to rejection. I cannot stand it at all. The rejection I had when I was

250 little had to do with my weight and my appearance. And now I try hard to avoid or
251 manage rejection through food.

252 **How do you mean that?**

253 When I was not accepted at a postgraduate programme I had a bulimic episode. When
254 I had to go to a job interview, 2 days before I did not eat anything as a preparation for
255 success. When I feel rejected I have bulimic episode. In order not to be rejected I do
256 not eat. Weight has been linked to rejection. The way I have learned to avoid rejection
257 is to limit food.

258 **Are you telling me that rejecting and continuing comments about appearance
259 keeps the symptoms going?**

260 Yes. They maintained them and strengthened them.

261 **So the symptoms now have to do with body reviews or rejection?**

262 Now with the rejection because I am thin. A rejection connection has been made with
263 the weight. It is automatic now.

264 **Out of all these, what do you think played the biggest role in starting the
265 disorder and then maintaining it?**

266 (Silence) It is a combination of social norms and bullying that lowered my self-
267 esteem. In order to have self-esteem I had to lose weight. I did not have self-esteem
268 from my home. My mother did not have self-esteem and so I probably learned not to
269 have one either. She did not care if she looked beautiful. She was very withdrawn and
270 closed to herself. I had no idea how a beautiful woman looks like. I did not have an
271 image as a guide to create my own one.

272 **If you had taken this picture from your mother what would this picture be like
273 do you think?**

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274 I do not know... she certainly could not let me gain so much weight and become so
275 ugly. This is neglect. This image for yourself and your weight follows you forever. To
276 put it correctly, your self-esteem follows you forever.

277 **What contributed to the maintenance of the disorder?**

278 My difficulty to attach. I use the disorder as a buffer in my relationship with others.
279 When I am closed inside the disorder I do not care much what others say and how bad
280 my relationship will be. It definitely has to do with my difficulty in forming
281 relationships.

282 **I see. Do you have anything to add?**

283 I think not, I told you everything I believe.

284 **Thank you very much for your time**

Appendix 15- Interview transcript Amanda in Greek

1

- 1 6th interview BED
- 2 Μπορείτε να μου μιλήσετε για την εμπειρία σας με φυσική μορφή κακοποίησης
3 στην ανήλικη ζωή?
- 4 Ξαν παιδιά ήμουν παχύσαρκα. Είχα πολλά καλά και αυτό ήταν κάτι που με έκανε να
5 είμαι εκτεθειμένη στα σχολία των άλλων. Σχόλια για την εμφάνιση. Πέρασα όλη μου
6 τη ζωή να είμαι η «χοντρή».
- 7 Θέλετε να μου πείτε? Ποιος σας έκανε αυτά τα σχόλια?
- 8 Από πολύ μικρή θυμάμαι να σχολιάζουν το βάρος μου και το σώμα μου. Τα παιδιά
9 στο σχολείο με έλεγαν χοντρή, βαρέλακα τέτοια πράγματα. Και οι καθηγητές μου
10 έλεγαν ότι πρέπει να χάσω βάρος. Ήταν πολύ ντροπιστικά. Ντρέπομαι να πάω στο
11 σχολείο. Από ένα σημείο και μετά θυμάμαι να ντρέπομαι να βγω έξω γενικά. Δεν με
12 καλούσαν και στα πάρτυ τα παιδιά. Είχαν κάνει και μια σύνδεση ότι όποιος είναι
13 χοντρός είναι και χαζός. Ότι το λίπος πάει και στον εγκέφαλο. κάπως έτσι.
- 14 Κατάλαβα. Οι γονείς πώς το αντιμετώπιζαν όλο αυτό?
- 15 Μου έλεγαν να μην τρώω τόσο πολύ. Αλλά δεν φταίω εγώ που έτρωγα. Στο σπίτι
16 είχαμε πάντα γλυκά και λιπαρά φαγητά. Και η μητέρα μου έτρωγε έτσι. Αυτό είχα
17 μάθει. Είχα αυτό το πρότυπο. Αλλά δεν είναι μόνο αυτό.
- 18 Τί άλλο ήταν?
- 19 Όταν ήμουν πολύ μικρή, 4-5 θυμάμαι τη μητέρα μου να προσπαθεί να με ταΐσει και
20 να υπάρχει η άποψη στο σπίτι ότι δεν έτρωγα. Και την έβλεπα να είναι
21 στεναχωρημένη που δεν έτρωγα και να προσπαθεί να βρει τρόπους να με ταΐσει. Από
22 ένα σημείο και μετά άρχισα να τρώω πολύ και δεν μπορούσα να σταματήσω.
- 23 Κατάλαβα
- 24 Όμως από τις φωτογραφίες μου σε αυτή την ηλικία του νηπιαγωγείου φαίνεται ότι
25 δεν ήμουν αδύνατη. Οπότε κατάλαβα ότι η μητέρα μου για κάποιο λόγο νόμιζε ότι
26 δεν τρώω. Αν δεν έτρωγα όπως μου έλεγαν θα έπρεπε να είμαι πολύ αδύνατη στις
27 φωτογραφίες. Αρχισα να τρώω για να μην είναι στεναχωρημένη. Την είχα ακούσει να
28 λέει στη θεία μου ότι είμαι δύσκολο παιδί. Όταν άρχισα να τρώω δεν μπορούσα να
29 σταματήσω.
- 30 Η σχέση με τους γονείς πώς ήταν? Νιώθατε να παίρνετε υποστήριξη σε αυτό?
- 31 Όχι.
- 32 Θέλετε να μου πείτε?
- 33 Ο πατέρας μου έλειπε πολύ λόγω δουλειάς. Η μητέρα μου δεν δούλευε και ήταν στο
34 σπίτι αλλά ήταν απόμακρη. Δεν έδειχνε τα συνασθήματα της. Δεν ήταν τρυφερή.
35 Ενώ με φρόντιζε με το φαγητό, δεν έκανε κάτι άλλο. Εννοώ συνασθηματικά. Δεν

- 36 ήταν άνθρωπος που αγκαλιάζε και έδειχνε τρυφερότητα. Δεν μπορούσα να της πω
37 κάτι όταν ήμουν λυπημένη γιατί φοβόμουν ότι θα τη στεναχωρήσω. Δεν είναι ότι δεν
38 με αγαπούσε αλλά σαν άνθρωπος ήταν έτσι. Ο χαρακτήρας της.
- 39 **Η μητέρα σας σε όλο αυτό με το bullying στο σχολείο έπαιρνε θέση?**
- 40 Μου έλεγε να μην τρώω και να είμαι καλή μαθήτρια. Θεωρούσε ότι είμαι αδύναμη
41 σαν χαρακτήρας. Και ο πατέρας μου έλεγε το ίδιο. Η μητέρα μου είχε κι αυτή
42 παραπάνω καλά κι έκανε συχνά διάλειψα χωρίς επιτυχία.
- 43 **Φύλους είχατε?**
- 44 Όχι ιδιαίτερα. Γενικά ήμουν απόμακρη. Δεν ήμουν κοινωνική. Δεν προσπαθούσα να
45 κάνω παρέες. Στο σχολείο δεν ήθελα να συμμετέχω σε ομαδικά παιχνίδια. Δεν
46 μπορούσα να ενταχθώ. Νόμιζα ότι και οι άλλοι δεν με ήθελαν στις παρέες. Όταν
47 προσπαθώ να μπω σε μια παρέα θα με απορρίψουν. Μου είχαν μάθει και από το
48 σπίτι ότι πρέπει να είμαι καλή σε όλα. Στη συμπεριφορά, στα μαθήματα. Γενικά
49 καλή σε όλα. Αυτό με άγχωνε. Ίσως φοβόμουν ότι δεν θα είμαι καλή σε όλα.
- 50 **Γιατί δεν θέλατε να προσπαθήσετε να μπείτε σε μια παρέα?**
- 51 Δεν είχα ποτέ αυτοεκτίμηση. Ποτέ. Έχασα χρόνια από τη ζωή μου να μην έχω
52 αυτοεκτίμηση. Όχι μόνο τότε και τώρα.
- 53 **Θέλετε να μου εξηγήσετε?**
- 54 Πίστευα ότι θα κάνω κάτι λάθος και θα με κοροϊδεύουν. Ότι δεν θα τα καταφέρω. Ότι
55 δεν ήμουν όμορφη. Ότι δεν ήμουν αρκετά καλή για τα ομαδικά παιχνίδια. Ότι δεν
56 ήμουν αρκετά καλή γενικά. Δεν ένιωθα και ποτέ άνετα να είμαι σε μια παρέα πάνω
57 από 2 άτομα. Δεν ένιωθα άνετα να μιλήσω. Ίσως νόμιζα ότι θα πω κάτι λάθος και θα
58 με κοροϊδέψουν.
- 59 **Κατάλαβα**
- 60 Δεν ξέρω... είναι πολύ κακό να μην έχεις αυτοεκτίμηση. Καθορίζει όλη τη ζωή. Τί θα
61 κάνεις στη ζωή σου. Ποιος θα είσαι... οι γονείς θα πρέπει να φροντίζουν τα παιδιά
62 τους να έχουν αυτοεκτίμηση. Αν δεν έχεις από παιδί δεν έχεις ποτέ. Αυτό έχω
63 καταλάβει. Κουβαλάς αυτό το φορτίο σε όλη σου τη ζωή.
- 64 **Δεν είχατε κάποιον να μιλήσετε για ό,τι σας απασχολούσε?**
- 65 Όχι. Ένιωθα μόνη μου. Νόμιζα ότι έφταγα εγώ για όλα. Επειδή είμαι αδύναμος
66 χαρακτήρας. Όταν σκεφτόμουν και με στεναχωρούσε το κρατούσα μέσα μου. Η παρέα
67 μου ήταν το φαγητό. Όταν έτρωγα κάτι που μου άρεσε ένιωθα καλά. Είχα συνέχεια
68 στο μυαλό μου να φάω. Όλο το φαγητό σκεφτόμουν.

- 69 **Οπότε στην ενήλικη ζωή τα σχόλια ήταν γύρω από το βάρος και την εικόνα**
 70 **σώματος. Κατάλαβαίνω ότι αυτά σας ενοχλούσαν. Μιλούσατε όταν σας τα**
 71 **έκαναν?**
- 72 **Με ενοχλούσαν πολύ αλλά δεν μιλούσα γιατί νόμιζα ότι είχαν δίκιο. Οντως ήμουν**
 73 **χοντρή οπότε τί να έλεγα? Προσπαθούσα να έχω καλούς βαθμούς για να μη με λένε**
 74 **χαζή.**
- 75 **Αυτό σας δημιουργούσε κάποια συναισθήματα?**
- 76 **Πολύ χαμηλή αυτοεκτίμηση...και θυμό. Θύμωνα με τον εαυτό μου που ήμουν χοντρή.**
- 77 **Εκτός από την αυτοεκτίμηση όλο αυτό σας προκάλεσε κάποια συναισθήματα που**
 78 **να ήταν έντονα?**
- 79 **Θυμό. Πολύ θυμό. Και για μένα που ήμουν άσχημη και για τους γονείς μου που δεν**
 80 **έκαναν κάτι για να βελτιωθεί η εμφάνισή μου.**
- 81 **Τον θυμό τον εκφράζατε?**
- 82 **Όχι. Δεν είχα μάθει να τον εκφράζω. Στο σπίτι δεν εκφράζαμε συναισθήματα. Δεν**
 83 **ξέραν γιατί. Πάντως έτσι έμαθα. Τον θυμό τον ανακούφιζα με το να τρέξω. Μόνο αυτό**
 84 **θυμάμαι να σας πω. Είναι σαν το τόσο πολύ φαγητό να έχει μπλοκάρει τις αναμνήσεις**
 85 **μου! (γελάει).**
- 86 **Τώρα σαν ενήλικη έχετε θυμό?**
- 87 **Ναι**
- 88 **Για ποιον?**
- 89 **Νομίζω για όλους. Σίγουρα για μένα που είμαι τόσο αδύναμη.**
- 90 **Κατάλαβα. Από συντρόφους είχατε τέτοια σχόλια?**
- 91 **Ναι είχα. Μου έλεγαν συνέχεια να αδυνατίσω γιατί ήμουν χοντρή. Με στεναχωρούσε**
 92 **αυτό. Κατάλαβα ότι ένας με απάτησε και μου είπε ότι αν ήμουν όμορφη δεν θα**
 93 **χρειάζοταν να βρει κάποια άλλη. Πάντα προσπαθούσα να αδυνατίσω αλλά δεν τα**
 94 **κατόφερνα. Οι δύο σύντροφοι που είχα με αντιμετώπιζαν σαν αδύναμη. Ήταν κάπως**
 95 **αυταρχικοί. Έπιαθα ότι δεν είχα γνώμη ή αν είχα θα ήταν λάθος. Κάπως έτσι...με**
 96 **έκαναν να νιώθω υποδεέστερη. Εβρισκα διέξοδο στο φαγητό κι αυτό δεν βοηθούσε**
 97 **γιατί συνέχιζα να είμαι χοντρή.**
- 98 **Τους μιλούσατε για αυτά που σας ενοχλούσαν?**
- 99 **Όχι. Δεν θέλω εντάσεις και φωνές. Οι συζητήσεις για συναισθήματα φέρνουν**
 100 **εντάσεις. Μπορεί να χαρίζαμε. Όταν δεν είσαι όμορφη είναι πολύ εύκολο να σε**
 101 **αφήσουν. Και δεν είναι εύκολο να βρεις κάποιον άλλο μετά.**
- 102 **Ο σύζυγός σας έχει την ίδια συμπεριφορά με τους πρώην συντρόφους?**

- 103 Δεν με προσβάλλει για την εμφάνιση αλλά μου λέει να αδυνατίσω. Όμως είναι κι
 104 αυτός απόμακρος σαν τη μητέρα μου. Αυτό με πειράζει. Δεν εκδηλώνει
 105 συνασθήματα. Δεν είναι τρυφερός. Για να σας δώσω να καταλάβετε, δεν τον είδα να
 106 κλαίει όταν πέθανε η μητέρα του με την οποία ήταν πολύ δεμένος.
- 107 **Κατάλαβα**
- 108 Θα ήθελα να είναι αλλιώς. Πιστεύω ότι με αγαπάει αλλά δεν μπορεί να το δείξει
 109 όπως θα ήθελα γιατί έτσι είναι ο χαρακτήρας του. Νιώθω παραμελημένη ενώ πιστεύω
 110 ότι με αγαπάει.
- 111 **Υπάρχει κάποια διαφορά ή ομοιότητα με την εμπειρία σας στη μη φυσική μορφή**
 112 **κακοποίησης μεταξύ ανήλικης και ενήλικης ζωής?**
- 113 Και στις δύο φάσεις τα σχόλια ήταν για το βάρος και την εξωτερική εμφάνιση. Και
 114 πάντα ένιωθα παραμελημένη. Πάντα ένιωθα μόνη μου.
- 115 **Κάποια ομοιότητα ή διαφορά στη δική σας αντίδραση?**
- 116 Και τότε έτρωγα και τώρα τρώω. Καταπίνω τα συνασθήματα με το φαγητό. Δεν
 117 είμαι αρκετά δυνατή για να χάσω κλά.
- 118 **Μου είπατε ότι όλο αυτό σας επηρέασε την αυτοεκτίμηση ως ανήλικη. Ισχύει το**
 119 **ίδιο και τώρα?**
- 120 Ναι. Δεν αλλάζει ποτέ αυτό νομίζω. Και στη δουλειά μου δεν έχω αυτοεκτίμηση.
 121 Κάνω μόνο αυτά που μου λένε και φοβάμαι να πάρω πρωτοβουλία μην κάνω λάθος.
 122 Είναι σα να φοβάμαι να ζήσω. Δεν ξέρω πώς αλλιώς να το περιγράψω. Δεν πιστεύω
 123 ότι η αυτοεκτίμηση αλλάζει ποτέ.
- 124 **Κατάλαβα. Σας έχει επηρεάσει η κακοποίηση κάπου αλλού?**
- 125 Είμαι απόμακρη.
- 126 **Αηλαδή?**
- 127 Δεν κάνω στενές σχέσεις. Φοβάμαι την απόρριψη. Αν είχα καλή αυτοεκτίμηση δεν θα
 128 φοβόμουν να δεθώ. Και τώρα δεν έχω φίλους.
- 129 **Πώς συνδέετε την αυτοεκτίμηση με το να δεθείτε με κάποιον?**
- 130 Όταν νιώθεις καλά για τον εαυτό σου δεν θα σε επηρεάσει η συμπεριφορά του άλλου
 131 και δεν θα πληγωθείς. Όταν μου λέει κάποιος κάτι άσχημο είναι σα να τρώω σφαίρα.
 132 Σα να πονάω. Αν σε ποροβλήσουν από κοντά θα πονάς πιο πολύ ενώ από μακριά
 133 δεν θα πονέσει τόσο.
- 134 **Κατάλαβα. Γιατί πιστεύετε ότι συνέβη η κακοποίηση στην ανήλικη ζωή?**
- 135 Πιστεύω συμβαίνει αυτό στα παιδιά που είναι παχύσαρκα. Οι άλλοι τα κοροϊδεύουν.
 136 Δεν είναι σωστό αλλά συμβαίνει. Φταίω κι εγώ που δεν ήμουν πιο δυνατή να

- 137 απαντήσα. Αν απαντούσα μπορεί να μην το ξηναϊόκαναν. Φταίνε και οι γονείς μου
138 που δεν με έμαθαν να μιλάω και να είμαι δυνατή.
- 139 **Κατάλαβα. Είχε να κάνει και με τη δική σας αντίδραση που το δεχόσασταν**
- 140 Έτσι πιστεύω
- 141 **Γιατί πιστεύετε ότι συμβαίνει και στην ενήλικη ζωή?**
- 142 Γιατί εξοικλοσθώ να είμαι χοντρή και να φάνομαι αδύναμη. Γιατί εξοικλοσθώ να
143 μην έχω αυτοεκτίμηση. Όταν έχεις αυτοεκτίμηση φαίνεσαι δυνατός και όταν
144 φαίνεσαι δυνατός οι άλλοι δεν σου κάνουν σχόλια. Δεν σε προσβάλλουν.
- 145 **Πιστεύετε ότι αυτή η κατάσταση θα αλλάξει?**
- 146 Μόνο αν αλλάξω εγώ και μπορώ να υπερασπιστώ τον εαυτό μου. Να γίνω πιο
147 δυνατή.
- 148 **Η ψυχοθεραπεία σας έχει βοηθήσει?**
- 149 Πολύ. Έχω κατάλαβε πολλά πράγματα για μένα και τη σχέση μου με το φαγητό.
- 150 **Κατάλαβα. Πιστεύετε ότι όλα αυτά που βιώσατε σχετίζονται με την έναρξη**
151 **ΔΠΤ?**
- 152 Ναι σίγουρα.
- 153 **Με ποιο τρόπο?**
- 154 Αν δεν είχα δυσάρεστες εμπειρίες στην παιδική ηλικία μπορεί να μην είχα
155 επικεντρωθεί τόσο στο φαγητό. Δεν θα έχανα τον έλεγχο.
- 156 **Θέλετε να μου εξηγήσετε?**
- 157 Αν οι γονείς μου, μου είχαν δώσει αυτοεκτίμηση δεν θα χρειαζόταν να βρω
158 παρηγοριά στο φαγητό. Θα ήμουν άλλος άνθρωπος. Δυνατή, κοινωνική. Δεν θα
159 περιστρεφόταν όλη μου η ζωή γύρω από το φαγητό. Δεν ξέρω αν τα λέω σωστά αλλά
160 έτσι νιώθω.
- 161 **Πιστεύετε ότι η συνέχεια της κακοποίησης παίζει ρόλο στη συνέχιση των**
162 **συμπτωμάτων?**
- 163 Ναι. Αφού συνεχίζω να τρώω όταν είμαι στεναχωρημένη, όταν κάτι με έχει πειράξει,
164 όταν νιώθω θυμό για τον εαυτό μου που δεν είμαι δυνατή, όταν νιώθω θυμό που
165 βρήκα ένα σύντροφο που μοιάζει με τη μάνα μου και δεν παίρνω αυτό που
166 θέλω...αρα σχετίζεται.

- 167 **Θέλετε να μου φέρετε κάποιο παράδειγμα?**
- 168 Όταν δεν κάνω κάτι καλά στη δουλειά μου και ο διευθυντής μου κάνει παρατήρηση
169 πηγαίνω στο φούρνο να πάρω να φάω ενώ δεν πεινάω.
- 170 **Κατάλαβα.**
- 171 Όταν ο άντρας μου είναι απόμακρος κι εγώ θέλω τρυφερότητα εκείνη την ώρα,
172 φτιάχνω κάτι λιπαρό που μου αρέσει και τρώω βλέποντας τηλεόραση. Σα να δίνω
173 ντάντεμα στον εαυτό μου με το φαγητό. Αρα δεν σχετίζεται? Εγώ έτσι πιστεύω. Αν
174 ήμουν δυνατή και είχα αυτοεκτίμηση θα χρειαζόταν να φάω για να νιώσω
175 ανακούφιση?
- 176 **Ναι κατάλαβα. Είπατε ότι δίνεται ντάντεμα στον εαυτό σας με το φαγητό.**
- 177 **Ναι.** Όταν θέλω μια αγκαλιά, προσοχή, τρυφερότητα, το βρίσκω στο φαγητό.
- 178 Πολλά από αυτά που σας λέω τα συνειδητοποιήσα μέσα από την ψυχοθεραπεία.
179 Πάντα όμως ήξερα...από μακριά...ότι το φαγητό αντικαθιστά κάτι που μου λείπει.
180 Αλλά δεν ήξερα να το εκφράσω σωστά. Αν με ρωτούσατε τα ίδια στα 15 δεν ήξερα
181 να σας τα πω όπως σας τα λέω τώρα.
- 182 **Από όλα αυτά τί πιστεύετε ότι έπαιξε τον μεγαλύτερο ρόλο στον να αρχίσει η
183 διαταραχή και μετά να διατηρηθεί?**
- 184 (Σιωπή) Θα σας πω. Όπως σας είπα, η μητέρα μου όταν ήμουν πολύ μικρή μου έλεγε
185 ότι είμαι δύσκολο παιδί και δεν τρώω. Νομίζω ότι ξεκίνησα να τρώω για να είναι
186 ευχαριστημένη και ικανοποιημένη από εμένα. Πίστευα ότι αν ήταν πιο
187 ευχαριστημένη θα ήταν και πιο τρυφερή μαζί μου. Ότι θα είχα την αποδοχή της. Ότι
188 θα μου έδειχνε ότι με αγαπάει. Νομίζω ότι εκεί βρίσκεται η απάντηση. Στην αποδοχή
189 και τρυφερότητα που είχα ανάγκη από τη μητέρα μου.
- 190 **Κατάλαβα.**
- 191 Πολλά πράγματα που γίνονται στο μέλλον έχουν τις ρίζες τους στη σχέση μας με τη
192 μαμά. Έτσι πιστεύω.
- 193 **Μάλιστα.**
- 194 Για αυτό προσπαθώ να είμαι τρυφερή με την κόρη μου. Να μην κάνω τα λάθη που
195 έκανε η μητέρα μου σε εμένα.
- 196 **Έχετε κάτι να συμπληρώσετε? Εγώ δεν έχω κάτι άλλο να ρωτήσω.**
- 197 **Νομίζω όχι...σας τα είπα όλα πιστεύω.**
- 198 **Σας ευχαριστώ πολύ για τον χρόνο σας**

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 16—Example of exploratory comments and emergent themes formation taken from Laura’s interview

Emergent themes	Original transcript-participant 1: Laura	lines	Exploratory comments
- Live as with parents	I would like you to tell me about the experience you have about the non-physical form of abuse in adulthood.	1	
- Abuse also in adulthood	As I still live with my parents it is an extension of my minor life. In some different terms, I do not know whether they are better or worse, though that I was living as a child living and as an adult.	2	Some type of abuse or adult
- Parents undermining her	Can you tell me more specifically about your experience now as an adult?	3	girl-child. Some kind of life for her
- Verbal and psychological abuse in adulthood	I feel that many times in some steps I want to do, there are my parents who pull me the carpet under the feet, invalidating me with either verbal insults or even physical violence. In the early years of adulthood there was also physical abuse.	4	Parents don't let her decide in her life with their restrictive behavior. Also physical abuse.
- Abuse in the home	Is this situation an experience you have only from your parents?	5	
	I think these were the first stage in life and since I only had contact with my parents at home I think yes only from the parents.	6	She however immediately from parents connecting them to her first stage in life
	As an adult yes.	7	
- Abuse by employer	This is something that can happen either from friends in a company or from a partner or an employer	8	
- Abuse from friends	From an employer it was an isolated incident that did not mind I left in the past and I did not even talk about it. It was only that day. From friends or partner or someone else no. I did not have anything like that.	9	Refers to isolated abusive incident from employer—not important factor mentions no such behavior from friends or partner as an adult
- negative speech	So the part of adult life has to do with invalidation	10	
- conditions	Do you want to give me an example?	11	
- psychological treatment	Many examples. Let's say the last time I take medication for depression and anxiety disorders try to get me that my mind is	12	Parents distort her need to reach therapy by blaming her mind for this changing

<p>parents blame her for abuse and medication</p> <p>- Inmate's G sleep or boyfriend</p> <p>- Parents abuse sister as well</p> <p>- sister escapes</p> <p>- better than my sister</p> <p>- mother more abusive</p>	<p>"wrong" and that's why I take medication. That they have no responsibility for this and that I am responsible for things that happen in the house.</p> <p>Such as? What could you be responsible for?</p> <p>This period sometimes I sleepover at my boyfriend's house with whom we are together for some time, I hear insults regarding my ethics.</p> <p>So could you say that it's happening from my sister?</p> <p>Not the same thing is happening to my sister from our parents but my sister is managing it differently. Between us there is good relationship and understanding.</p> <p>I see. Abuse is expressed more by your father or your mother or is it the same?</p> <p>It is expressed by both, perhaps more verbally from my mother.</p> <p>So this is the context of adulthood abuse.</p> <p>Yes</p> <p>Is there something similar or something different between your experience as a minor and as an adult regarding non-physical abuse?</p> <p>In my childhood I thought I was not in a position of power, neither now I think I am in a position of strength, but as a child I should not react in the way that I can now. In practice, however, because I think I have learned in this way of reaction, I may be prevented from reacting now. That is, I accept more passively this abuse or I react with a very big anger explosion even when the cause may not be important but it works in me cumulatively.</p> <p>I see. Some resemblance or difference apart from your reaction;</p> <p>Now I am having anger explosions.</p> <p>As a minor you did not;</p>	<p>30 Their responsibility.</p> <p>31 They blame her for her psychological</p> <p>32 conditioned their abusive behavior.</p> <p>33</p> <p>34 Parents react to her in a way because</p> <p>35 she has a relationship with her sister,</p> <p>36</p> <p>37</p> <p>38 Parents behave the same way to her sister</p> <p>39 but she reacts better. She was a good</p> <p>40 girl or she was with her sister.</p> <p>41</p> <p>42 Both seem to have abusive behavior, mother</p> <p>43 more abusive.</p> <p>44</p> <p>45</p> <p>46</p> <p>47</p> <p>48</p> <p>49 As a child didn't feel strong enough to</p> <p>50 react - passive behavior. She was learned</p> <p>51 being passive and that also affects her</p> <p>52 behavior now. Either she is being passive</p> <p>53 or has bursts out of minor reasons</p> <p>54 because things are adding up inside her.</p> <p>55</p> <p>56</p> <p>57</p> <p>58 The difference between child and adult</p> <p>59 reaction is anger explosion now</p>
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3

<p>- Pressing out as a minor</p> <p>- no visible signs of abuse</p> <p>- compared her to other girls in the school</p> <p>- resulting comments on physical appearance</p> <p>- her father told her</p>	<p>No You were holding it. Yes Yes Yes As a situation (meaning the type of abuse) from parents, since you have made it specific, then and how would you say it is the same? What they were telling you then tell you now? For different reasons. Then they would invalidate me for my performance in the lessons which instead of 20 was 19. Oh, I see. Or for my weight. That "other girls are thin and you are not and you have to eat the food." Or that "someone else got 20 and you got 19". Continuous comparisons with other children, either in terms of grades or in what they regarded as respect to them or regarding my appearance (weight, physical characteristics, body image, etc.). And moral issues when I was a minor. And because I wanted to go out with friends during my adolescence I never did because it was not "right". Also in the elementary school if a boy was interested in me - as interested as a boy in the elementary school can be - I was always the one who caused it.</p>	<p>60 61 62 63 64 65 66 67 68 69 70 71 72 73 74 75 76 77 78</p>	<p>As a child she was being insensitive</p> <p>Parents were not satisfied with her grades at school over-rough supervisor or very good student. Being a child in a positive feedback.</p> <p>Parents compared her to other girls, commenting on grades, her weight and physical appearance as well as her morality. They didn't allow her to hang out with friends and were blaming her because a lot of school friend had a father if this was a bad thing especially their daughter.</p>
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NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 17 - Chronological list of emergent themes from Laura's interview

- 1 Living with parents
- 1 Abuse also in adulthood
- 1 Parents undermining her
- 1 physical abuse in adulthood
- 1 Abuse in the home
- 1 Abuse by employer once
- 1 No abuse from friends/boyfriend
- 1 Negative parental comments
- 1 Pharmacological treatment
- 1 Parents blame her for abuse and medication
- 1 Immoral to sleep at boyfriend's
- 2 Parents abuse sister as well
- 2 Sister copes better
- 2 Good relationship with sister
- 2 Mother more abusive
- 2 Passive reaction as child
- 2 Passive reaction as adult
- 2 Learned passivity
- 2 Burst outs as adult
- 2 Pressing anger as child
- 2 No positive parental feedback
- 2 Compared her to other in childhood
- 2 Insulting comments on physical appearance
- 3 Her fault a boy liked her

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 3 Parental respect demanded
- 3 Compared her to others in adulthood
- 3 Immoral for having boyfriends
- 3 Second thoughts on abuse
- 3 Cannot believe in herself
- 3 Feels like a victim
- 3 Doomed due to second thoughts
- 4 Neglected from first partner
- 4 Negative comments on appearance from current boyfriend
- 4 She is used to negative comments
- 4 Parents and boyfriend may be right
- 5 Patronization from partner
- 5 Partner undermining her choices
- 5 External attribution of abuse
- 5 Afraid not to do the same as a mother
- 5 Parents behaviour not justified
- 5 Feels weak to leave home
- 5 Cannot set limits
- 5 Avoids conflict
- 5 Internal attribution of abuse
- 5 Unable to react
- 6 Used to be abused
- 7 Tried to talk to parents
- 7 Negative emotions
- 7 Cannot enjoy accomplishments

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 7 Unstable family
- 7 Living with parents keeps abuse
- 7 Cannot enjoy being out
- 7 Avoids confrontation
- 8 Ashamed to talk about the abuse
- 8 Boyfriend's mother comments her weight
- 8 Psychotherapy helps
- 9 ED enhances abuse
- 9 ED relates to abuse onset
- 9 Only eating has left
- 9 She needs to change
- 9 Feels weak against life
- 9 Low self-esteem
- 9 Has not grown up
- 9 Still waits to be loved from parents
- 9 Abuse made her choose her profession
- 9 Sees herself in other kids
- 9 Neglect from parents

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 18 - Chronological list of emergent themes from Betty's interview

1 non-physical abuse from parents/classmates/teachers

1 physical and sexual abuse

1 non-physical abuse as adult

1 bullying at school for grades

1 insults at home for grades & homework

1 learning difficulties as child

1 emotional neglect from parents

2 aunt proposed drug use

2 mother compares her to sister

2 comment on weight from boyfriend

2 lost a job for weight

2 comments on weight from doctor

2 comments on weight from friend

2 abuse regarding weight as a adult

2 external attribution of abuse in childhood

2 external attribution of abuse in adulthood

2 unable to avoid abusive relationships

2 anger due to abuse

2 abuse affects her development

3 disappointment & guilt for not setting limits

3 cannot communicate emotions

3 cannot set limits

3 abuse related to ED now

3 disappointment and anger cope with food

NON-PHYSICAL ABUSE AND EATING DISORDERS

3 food alleviated stress, wounds and drowned emotions

3 abuse relates to ED onset

3 trauma leads to addictive behaviour

3 imitating unhealthy eating leads to ED

3 detaching from abusive people will stop abuse

3 psychotherapy helps

4 abuse affected self-esteem

4 feels helpless and tired

4 cannot open up and relate to partners

4 stress and sadness affected her health

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 19 - Chronological list of emergent themes from Jenny's interview

- 1 non-physical abuse from partners
- 1 negative physical appearance comments from partners
- 1 devaluation from partners
- 1 verbal abuse from partners
- 1 her opinion unimportant for partners
- 1 partners rejecting her opinion
- 1 she felt unimportant
- 1 partners rejecting her wishes
- 1 neglect and devaluation from colleagues
- 1 perceived as shy and weak from colleagues
- 1 does not like colleagues' jokes
- 1 colleagues think she is stupid
- 2 colleagues like gossiping
- 2 thinks she is weak
- 2 does not react to insults
- 2 insulting and devaluation in adulthood
- 2 abuse by father in childhood
- 2 father had a bad temper
- 2 yelling and devaluation from father
- 2 she had to be obedient
- 2 she had to be a good kid

NON-PHYSICAL ABUSE AND EATING DISORDERS

2 she had to be a good student

2 father insulted her

2 neglected and insulted by teacher

2 her opinion in class was unimportant

2 felt like she didn't exist in class

2 neglected and insulted by fellow students

2 she does not speak up

3 not confident to defend herself

3 cannot express feelings

3 cannot fight back

3 low self-esteem

3 learned not to speak

3 speaking make things worse

3 mother taught her not to speak

3 cannot handle others' reaction

3 cannot handle others' anger

3 attributes childhood abuse to father's temper

3 mother was passive

3 mother unable to protect her

3 still abused because she cannot react

3 it's her fault that she looks like a victim

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 3 she is seen as weak by others
- 3 her low self-esteem is visible
- 3 she looks like an easy victim
- 3 abuse shaped her character
- 3 abuse shaped her feelings
- 3 she cannot feel joy
- 3 feels like carrying a burden
- 3 she is always sad
- 3 she does not try for anything
- 3 does not try for more in life
- 3 does not have self-esteem
- 3 did not receive love from parents
- 3 mother not supportive
- 3 does not believe in herself
- 3 does not want responsibilities
- 3 believes she cannot succeed
- 3 felt she was not good enough for college
- 3 entitled for promotion but not claiming it
- 3 promotion means responsibilities
- 3 prefers to work invisibly
- 3 things would be different if she had self-esteem

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 3 higher responsibilities require skills
- 4 higher responsibilities mean confronting people
- 4 does not want to confront others
- 4 hard for her to confront others
- 4 learned not to speak and walk away
- 4 she closes to herself
- 4 does not express herself
- 4 does not trust others
- 4 does not want close relationships
- 4 she likes distance with people
- 4 feels safe when distant from others
- 4 cannot attach with others
- 4 afraid of partners' rejection
- 4 plays around with them and then leave
- 4 does not want responsibilities in relationships
- 4 does not want to be patronized from partners
- 4 does not want to fight with partners
- 4 if attached may be hurt
- 4 when close to others becomes vulnerable
- 4 if things go wrong she will be hurt
- 4 she is used to the abuse

NON-PHYSICAL ABUSE AND EATING DISORDERS

4 she is used to not react

4 afraid to speak and react

4 does not feel accepted and loved

4 she is weak and others see that

4 feels bad for the abuse

4 feels like a victim

4 angry at herself for being so weak

4 angry at herself for not defending herself

5 she does not confront abuse

5 the way she was raised shaped her personality

5 the way she was raised shaped her thoughts and feelings

5 abuse related to ED now

5 when sad and keeping things inside she does not eat

5 she punished herself

5 she does not deserve to eat

5 she does not deserve to eat something she likes

5 she is bursting out with food

5 food is the only thing she can control

5 the way she was raised is associated with ED onset

5 When you are not ok and cannot speak you will find another way to express it

5 you turn into your self

5 it's a catastrophic way to express your self

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 5 ED would not exist if she could speak
- 5 Feelings would not eat her if she could speak
- 5 Anger sadness and sorrow eat people
- 6 Anger for her father who was bad at her
- 6 Anger for her mother who did not protect her
- 6 Anger for her mother who did not teach her to be strong
- 6 Anger at her weak self who is not able to protect her
- 6 Anger at everyone who were raised with love
- 6 Jealous of people who were better raised than her
- 6 After having thoughts that cause her anger she feels sad and tired
- 6 sad because she cannot confront people that make her angry
- 6 Intense feelings make her tired
- 6 Feelings are stronger than her
- 6 She is afraid of her feelings
- 6 Her feelings can destroy others
- 6 She keeps feelings inside and they eat her
- 6 People who can speak up are happy
- 6 Things will change if she becomes stronger
- 6 Things will change if she defends herself
- 6 Things will change if she does not allow others to hurt her
- 6 Things will change if she acquires self-esteem
- 6 The obstacle will be her if she doesn't change
- 6 she good in theory but not in practice
- 6 bad in practice because her self-esteem is a mess

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 20 - Chronological list of emergent themes from Carol's interview

- 1 living with parents preserves abuse
- 1 devaluation from parents
- 1 parents think she makes mistakes
- 1 name calling
- 1 compare her to others
- 1 others are better in personal and professional life
- 1 parents compared her school grades to others
- 1 comments on her body appearance as an adult
- 1 parents tell her not to eat much
- 1 parents tell her to exercise
- 1 parents told her not to eat much and exercise
- 1 gaining weight would lead to bullying at school
- 1 boyfriend is abusive
- 1 feels boyfriend doesn't not want her
- 1 boyfriend always angry at her
- 1 feels with him the same way she feels with parents
- 1 same environment
- 1 boyfriend has a bad temper
- 1 boyfriend insults her
- 1 boyfriend always makes her feel sad
- 1 stressed not to say something wrong to him
- 1 stressed because he will yell at her
- 1 left her alone once because she was late
- 1 boyfriend made a public scene
- 1 felt embarrassed
- 2 boyfriend looks for reasons to argue

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 2 boyfriend makes her feel useless
- 2 boyfriend makes her feel unworthy to be with someone
- 2 she can't leave boyfriend
- 2 afraid to be alone
- 2 difficult to find a partner
- 2 she used to the situation with boyfriend
- 2 everything is expected
- 2 doesn't know what to do if someone treats her right
- 2 a known situation doesn't have surprises
- 2 insults and yelling in adulthood
- 2 common patterns between boyfriend and parents
- 2 shocked when realised commonalities
- 2 boyfriend has the same behaviour with mother
- 2 yelling and criticising are the same
- 2 hearing her boyfriend is like hearing her mother
- 2 when boyfriend yells she freezes
- 2 cannot react
- 2 afraid to speak
- 2 doesn't know how to handle boyfriend
- 2 she is upset and sad
- 3 she doesn't know what to do when attacked
- 3 mother more abusive and judgmental
- 3 no abuse from friends
- 3 could not react as a minor
- 3 cannot react as an adult
- 3 if she react she may beat parents
- 3 she has a lot of anger
- 3 break things when is alone

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 3 she doesn't know how to react when insulted
- 3 cannot control her reaction
- 3 cannot control others reaction
- 3 afraid of how things will develop when everyone is angry
- 3 afraid partner will become violent
- 3 afraid partner will break up with her
- 3 afraid parents will never speak to her again
- 3 wonders if parents and boyfriend are right
- 3 they yell at her for the same reasons
- 3 concludes their behaviour is not right
- 3 she is the victim of their bad temper
- 3 boyfriend is her first relationship
- 4 boyfriend older than her
- 4 boyfriend comments on how much she eats
- 4 boyfriend tells her she will become fat
- 4 boyfriend's weight comments irritate her
- 4 boyfriend causes her fatness anxiety
- 4 boyfriend will not understand if she talk to him
- 4 she cannot argue with him
- 4 she cannot afford yelling and insulting
- 4 she does not react to weight comments
- 4 sad and upset for weight comments
- 4 she wants to eat when boyfriend irritates her
- 4 she wants to eat in front of him to provoke him
- 4 when she eats more she vomits
- 4 she wants to punch him
- 4 eats instead of punching him
- 4 easier to eat than speak up

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 4 she does not want tension
- 4 attributes abuse to parents' issues
- 4 parents don't know how to behave
- 4 her mother had an authoritarian mother
- 5 abuse is ongoing because she cannot react
- 5 ongoing abuse is her fault
- 5 ongoing abuse makes her angry at her self
- 5 if she could react there would be no abuse
- 5 ongoing abuse due to her weak character
- 5 leaving home would make things better
- 5 she is used to the abuse
- 5 others see her as victim
- 5 abused because she looks weak
- 5 abuse is familiar
- 5 feels awful for the abuse
- 5 feels worthless
- 5 thinks how she can look better
- 5 find a better job
- 5 become pretty
- 5 if she becomes successful abuse will stop
- 5 anger and jealousy
- 5 jealous of people with better parents
- 5 anger for everyone
- 5 anger for parents
- 5 anger for boyfriend
- 5 wakes up feeling angry
- 5 cannot understand why she wakes up angry
- 5 she wants to break things

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 5 tired of waking up angry
- 5 keeps anger inside
- 5 tries not to show her anger
- 5 she eats
- 5 eating relieves her but feels awful later
- 6 displaying anger would save her from eating
- 6 no need to relief with food if anger expressed
- 6 food reliefs her from anger
- 6 when eating forgets or drowns anger
- 6 anger is covered by food she likes
- 6 therapy helps her understand herself
- 6 she needs time to change
- 6 she cannot become someone else at once
- 6 not speaking up makes things difficult
- 6 difficult for her to speak up
- 6 keeping things inside eats her
- 6 abuse by partner makes her weaker
- 6 if partner was different she would be stronger
- 6 if partner was kind she would feel better about herself
- 6 if partner was kind she would have self-esteem
- 6 if partner was kind she would be stronger
- 6 if partner was kind she could speak up
- 6 important to have self-esteem
- 6 others cannot treat people with self-esteem as garbage
- 6 abuse linked with current ED symptoms
- 7 weight comments make her eat more
- 7 eating calms her
- 7 provokes others by eating

NON-PHYSICAL ABUSE AND EATING DISORDERS

7 relieved when eating but disgusted with her self

7 when disgusted with herself she vomits

7 abuse may be linked with ED onset

7 keeping sadness inside affects psychology

7 negative feelings must find a way out

7 this way will not be healthy

7 she has to change

7 she has to become stronger

7 she has to defend herself

7 she needs to have self-esteem

7 self-esteem must be visible

7 she has to set limits

7 she has to speak up without caring for the consequences

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 21 - Chronological list of emergent themes from Kate's interview

- 1 she was overweight
- 1 social environment emphasised appearance
- 1 indifferent mother
- 1 she was alone
- 1 judgmental social environment
- 1 negative comments for her body
- 1 food shaming from others
- 1 not eating to family dinners
- 1 eating secretly
- 1 discouraged to eat in family dinners
- 1 friends anxious for their own body image
- 1 bullied from friends
- 1 fat was not allowed in high school
- 2 encouraged to diet
- 2 others terrified her about food
- 2 mother apathetic to the abuse
- 2 mother could not handle the abuse
- 2 mother justified the abuse
- 2 mother not supportive
- 2 mother not helpful
- 2 not good communication with mother
- 3 no contact with father
- 3 no siblings
- 3 authoritarian friends

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 3 always involved in bossy relationships
- 3 she has a low profile personality
- 3 bossy friend bullying her
- 3 she thought she had to lose weight
- 3 justified the abuse in childhood
- 3 she had none to talk
- 4 did not react to the abuse
- 4 very low self-esteem
- 4 starting dieting
- 4 eating secretly
- 4 emotional and self-regulation difficulties
- 4 negative feelings
- 4 questioned her negative feelings
- 4 adopted gothic style to draw out the attention from weight
- 4 joined angry groups to tolerate anger
- 4 started a diet at 18
- 4 blamed weight for not meeting people
- 4 she was not social
- 4 thought she looked like a monster
- 4 bullying in adulthood
- 4 a pattern to be bullied for weight
- 4 insulted in front of an audience
- 5 justified the abuse in adulthood
- 5 unless she loses weight nothing good will happen
- 5 dieting until the fat was gone

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 5 she was hypersensitive
- 5 bullied on the street
- 5 rude comments from people she dated
- 5 fragile self died through anorexia
- 5 partner with bad temper
- 5 anorexia helped her self-esteem
- 5 anorexia killed the overweight girl
- 5 anorexia came when she was angry at herself
- 5 bullying would end with weight loss
- 5 losing weight would improve self-esteem
- 5 high self-esteem people are not bullied
- 6 self-esteem is visible
- 6 low self-esteem makes you an easy target
- 6 anorexia can improve self-esteem
- 6 lost control when dieting
- 6 told not to eat as a minor
- 6 told to eat as an adult
- 6 viewed as thin was a compliment
- 6 sad in childhood
- 6 psychotherapy helped
- 6 hard to gain weight
- 6 low self-esteem created an unstable self
- 6 self-esteem linked to weight
- 6 doesn't want to gain weight
- 6 abuse affected her relationships

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 7 unstable relationships
- 7 afraid to attach
- 7 not being herself in relationships
- 7 overweight kids are abused
- 7 people are blamed for extra weight
- 7 she was withdrawn and shy
- 7 her fault for accepting bullying
- 7 she was born a woman
- 7 society puts pressure on women
- 7 victimisation was a reinforcing factor
- 8 she needs to remain thin
- 8 lost time with ED
- 8 worked in a bar to elevate self-esteem
- 8 not leaving the house because she was overweight
- 8 focusing on weight pushed her back
- 8 a lot of anger
- 8 doesn't know where to throw her anger at
- 8 she blames everyone
- 8 anger relief through bulimic episodes
- 8 suppressing anger with food in childhood
- 9 anger not verbally expressed
- 9 self-injury to relief anger
- 9 delinquent behaviour to relief anger
- 9 punishing herself through bulimic episodes
- 9 angry for eating

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 9 turning anger at herself created anorexia
- 9 turning anger at others created bulimia
- 9 throwing things when angry
- 9 afraid of direct conflict
- 9 learned not to argue
- 9 difficult for her to argue
- 9 with bulimia she took her anger out
- 9 changing her anger direction confused her
- 10 justifying bullying from friends
- 10 reading about feminism helped
- 10 bulimia came when she viewed things differently
- 10 bulimic episodes a revolutionary act
- 10 not eating has been imposed from others
- 10 purging episodes
- 10 psychotherapy helped her understand her relationship with food
- 10 if there was no abuse there would be no ED
- 10 cannot tolerate rejection
- 11 handles rejection through food
- 11 rejection triggers ED behaviour
- 11 rejection maintains and strengthens ED symptoms
- 11 low self-esteem important contributor to ED
- 11 didn't have a beautiful role model
- 12 mother letting her gain weight is neglect
- 12 self-esteem stays forever
- 12 attachment difficulty maintains the disorder

NON-PHYSICAL ABUSE AND EATING DISORDERS

12 hiding in the disorder protects her

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 22 - Chronological list of emergent themes from Amanda's interview

- 1 Exposed to comments because she was overweight
- 1 Comments on her physical appearance
- 1 Spent her life being the fat one
- 1 Comments on body and weight from a young age
- 1 Kids called her fat and barrel
- 1 Teachers told her to lose weight
- 1 Ashamed to go to school
- 1 Ashamed to go out in general
- 1 Not invited to parties
- 1 Called her stupid because fat goes to brain
- 1 Parents told her not to eat so much
- 1 Always fatty food in the house
- 1 Her mother ate a lot
- 1 She had that eating model form her mother
- 1 Her mother told her she was difficult with eating
- 1 Her mother was sad because she was not eating
- 1 She started eating a lot and couldn't stop
- 1 Her toddler photos show she was overweight
- 1 Her mother telling her she didn't ate and her photos do not match
- 1 She started eating a lot to please her mother
- 1 No support from parents regarding bullying
- 1 Father worked a lot
- 1 Mother was distant
- 1 Mother didn't display emotions
- 1 Mother was not affective
- 1 Mother provided food but no emotional support
- 2 Mother didn't hug her
- 2 She couldn't tell her mother she was sad not upset her
- 2 Mother told her not to eat and be a good student
- 2 Father told her not to eat and be a good student

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 2 Mother was overweight engaging in unsuccessful diets
- 2 She didn't have friends
- 2 She was distant and not social
- 2 She didn't try to make friends
- 2 She didn't want to participate in group games
- 2 She couldn't fit in
- 2 She thought others would not want her
- 2 She thought others would reject her
- 2 Parents told her she should be good at everything she does
- 2 Parents advise stressed her
- 2 She was afraid she wouldn't be good at everything
- 2 She never had self-esteem
- 2 She lost years from her life not having self-esteem
- 2 No self-esteem both as a minor and adult
- 2 She thought she would make a mistake and be ridiculed
- 2 She thought she wouldn't succeed
- 2 She thought she wasn't pretty
- 2 She thought she wasn't good enough for group games
- 2 She though she wasn't good at everything
- 2 She didn't feel comfortable being with more than two people
- 2 She didn't feel comfortable talking to others
- 2 She thought she would say something wrong and be laughed at
- 2 She felt alone
- 2 She thought she was to blame for everything
- 2 She is a weak character
- 2 Kept thoughts and feelings inside
- 2 Food was her company
- 2 Eating something she liked made her feel good
- 2 Her mind was always on food
- 2 All she could think about was food
- 3 She was bothered from negative body comments but thought they were right
- 3 She was fat so she couldn't say something back

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 3 Tried to have good grades so not to be called stupid
- 3 Very low self-esteem and anger
- 3 Anger at herself for being fat
- 3 A lot of anger at herself for being ugly
- 3 A lot of anger at her parents who didn't help her improve her appearance
- 3 She didn't express anger
- 3 She hadn't learn to express anger
- 3 Emotions were not expressed in her house
- 3 Her anger was relieved through eating
- 3 Feels like food has blocked her memories
- 3 Angry as an adult
- 3 Angry at everyone
- 3 Angry at herself for being weak
- 3 Partners told her to lose weight
- 3 Partners told her she was fat
- 3 Partners' comments made her sad
- 3 One partner cheated on her
- 3 Cheating partner blamed her for not being beautiful
- 3 She was trying to lose weight but couldn't succeed
- 3 Partners viewed her as weak
- 3 Partners were bossy
- 3 She felt her opinion would be wrong
- 3 Partners made her feel inferior
- 3 She found her way out through food but didn't help
- 3 She didn't talk her feelings to partners
- 3 She doesn't want tension and yelling
- 3 Conversations about emotions bring tension
- 3 If she talked boyfriends could break up with her
- 3 When you are not pretty it's easy to be dumb
- 3 When you are not pretty it's not easy to find someone else
- 4 Her husband tells her to lose weight
- 4 Her husband is distant like her mother

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 4 Her husband being distant bothers her
- 4 Husband doesn't display emotions
- 4 Husband is not tender
- 4 She wishes her husband was different
- 4 Her husband loves her but cannot show it
- 4 She feels neglected from husband
- 4 Felt neglected both as a minor and adult
- 4 Felt alone both as a minor and adult
- 4 Eating a lot as a reaction both as a minor and adult
- 4 She is not strong enough to lose weight
- 4 Low self-esteem never changes
- 4 She doesn't have self-esteem in her job
- 4 She only does what she is told
- 4 She is afraid to take initiations and make a mistake
- 4 She is afraid to live
- 4 Abuse made her distant
- 4 She doesn't engage in close relationships
- 4 She is afraid of rejection
- 4 If she had good self-esteem she wouldn't be afraid to come close to others
- 4 She doesn't have friends
- 4 Feeling good about yourself protects you from getting hurt from comments and behaviour
- 4 When someone tells her something bad it's like being shot
- 4 Being shot from close distance will hurt a lot
- 4 Being shot from long distance will not hurt that much
- 4 Overweight kids are abused
- 4 Her fault for not being strong enough to react
- 5 Her parents' fault for not teaching her to be strong and answer back
- 5 Abused now because she is still fat and weak
- 5 Abused because she doesn't have self-esteem
- 5 People with self-esteem look strong and others can't insult them
- 5 Psychotherapy helped her understand herself and her relationship with food
- 5 Abuse relates to ED onset

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 5 Negative childhood experiences made her focus on food
- 5 Negative childhood experiences made her lose control
- 5 If her parents had given her self-esteem she wouldn't need to use food for comfort
- 5 If her parents had given her self-esteem she would be a different person, strong and social
- 5 Ongoing abuse relates to current ED symptoms
- 5 Eats when sad and angry
- 5 Eats when angry at herself for not being strong
- 5 Eats when angry at herself for having a neglectful partner
- 6 Eats when her manager makes her remarks
- 6 Eats when her husband is distant and she needs a hug
- 6 She is nurturing herself with food
- 6 If she had self-esteem and was strong she wouldn't need food to comfort her
- 6 When she needs hug, attention and tenderness she eats
- 6 She started eating to please her mother
- 6 If her mother was satisfied she would be tender with her
- 6 Mother would accept her and show love
- 6 The answer is the acceptance and tenderness she needed from her mother
- 6 Things that happen later are rooted in the relationship with mother
- 6 She tries to be tender with her own daughter
- 6 Tries not to make the same mistakes with her daughter

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 23 - The superordinate themes and their emergent themes of Laura's interview

Non-physical abuse as child

- 1 Abuse in the home
- 2 Mother more abusive
- 2 No positive parental feedback
- 2 Compared her to others in childhood
- 2 Insulting comments on physical appearance
- 3 Her fault a boy liked her
- 3 Parental respect demanded
- 3 Compared her to others in adulthood
- 7 Unstable family
- 9 Neglect from parents

Non-physical abuse as adult

- 1 Abuse also in adulthood
- 1 Parents undermining her
- 1 Abuse by employer once
- 1 No abuse from friends/boyfriend
- 1 Negative parental comments
- 1 Parents blame her for abuse and medication
- 1 Immoral to sleep at boyfriend's
- 2 Parents abuse sister as well
- 3 Immoral for having boyfriends
- 4 Neglected from first partner
- 4 Negative comments on appearance from current boyfriend

NON-PHYSICAL ABUSE AND EATING DISORDERS

5 Patronization from partner

5 Partner undermining her choices

8 Boyfriend's mother comments her weight

ED and non-physical abuse

9 ED enhances abuse

9 ED relates to abuse onset

9 Only has eating left

Childhood non-physical abuse consequences in adult life

1 Pharmacological treatment

5 Afraid not to do the same as a mother

7 Cannot enjoy accomplishments

7 Cannot enjoy being out

8 Ashamed to talk about the abuse

9 Has not grown up

9 Abuse made her choose her profession

9 Sees herself in other kids

Emotion regulation

2 Sister copes better

2 Passive reaction as child

2 Passive reaction as adult

2 Learned passivity

2 Burst outs as adult

NON-PHYSICAL ABUSE AND EATING DISORDERS

2 Pressing anger as child

5 Cannot set limits

5 Avoids conflict

5 Unable to react

7 Negative emotions

7 Avoids confrontation

Self-esteem

3 Cannot believe in herself

3 Feels like a victim

3 Doomed due to second thoughts

3 Second thoughts on abuse

4 Parents and boyfriend may be right

4 She is used to negative comments

5 Feels weak to leave home

6 Used to be abused

9 Low self-esteem

9 Feels weak against life

Attachment

2 Good relationship with sister

8 cannot open up to partner

9 Still waits to be loved from parents

NON-PHYSICAL ABUSE AND EATING DISORDERS

Explaining non-physical abuse

1 Living with parents

5 Internal attribution of abuse

5 External attribution of abuse

5 Parents behaviour not justified

7 Tried to talk to parents

7 Living with parents keeps abuse

8 Psychotherapy helps

9 She needs to change

Other

1 physical abuse in adulthood

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 24 - The superordinate themes and their emergent themes of Betty's interview

Non-physical abuse as child

- 1 non-physical abuse from parents/classmates/teachers
- 1 bullying at school for grades
- 1 insults at home for grades & homework
- 1 emotional neglect from parents

Non-physical abuse as adult

- 1 non-physical abuse as adult
- 2 emotional neglect
- 2 aunt proposed drug use
- 2 mother compares her to sister
- 2 comment on weight from boyfriend
- 2 lost a job for weight
- 2 comments on weight from doctor
- 2 comments on weight from friend
- 2 abuse regarding weight mostly

Explaining non-physical abuse

- 2 external attribution of abuse in childhood
- 2 external attribution of abuse in adulthood
- 3 psychotherapy helps
- 3 detaching from abusive people will stop abuse

Childhood non-physical abuse consequences in adult life

- 2 abuse affects her development in life

NON-PHYSICAL ABUSE AND EATING DISORDERS

4 stress and sadness affected her health

ED onset and non-physical abuse

3 abuse relates to ED onset

3 trauma leads to addictive behaviour

3 imitating unhealthy eating leads to ED

ED maintenance and non-physical abuse

3 abuse relates to ED now

3 disappointment and anger cope with food

3 food alleviated stress, wounds and drowned emotions

Emotion regulation

2 anger due to abuse

3 disappointment & guilt for not setting limits

3 cannot communicate emotions

3 cannot set limits

Self-esteem

4 abuse affected self-esteem

4 feels helpless

Attachment

2 unable to avoid abusive relationships

4 cannot open up and relate to partners

Other

1 physical and sexual abuse in childhood

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 25 - The superordinate themes and their emergent themes of Jenny's interview

Non-physical abuse in childhood

- 2 abuse by father in childhood
- 2 yelling and devaluation from father
- 2 she had to be obedient and good student
- 2 father insulted her
- 2 neglected and insulted by teacher
- 2 neglected in class
- 3 mother was passive to her abuse
- 3 did not receive love from parents
- 3 mother did not protect her

Non-physical abuse in adulthood

- 1 non-physical abuse from partners
- 1 negative physical appearance comments from partners
- 1 verbal abuse and devaluation from partners
- 1 partners rejecting her opinion
- 1 partners rejecting her wishes
- 1 neglect and devaluation from colleagues
- 1 perceived as shy and weak from colleagues
- 1 colleagues think she is stupid
- 2 insulting and devaluation in adulthood

NON-PHYSICAL ABUSE AND EATING DISORDERS

Self-esteem

- 2 weak with no self-esteem
- 3 cannot defend herself due to low self-esteem
- 3 low self-esteem keeps her back
- 3 things would be different if she had self-esteem
- 3 avoiding responsibilities

Attachment

- 4 avoids close relationships / trust
- 4 avoids intimate relationships / rejection
- 4 attachment makes you vulnerable

Non-physical abuse and current ED

- 5 restricts food for punishment when sad
- 5 she does not deserve to eat
- 5 she does not deserve to eat something she likes
- 5 she is bursting out with food
- 5 food is the only thing she can control

Non-physical abuse and ED onset

- 5 the way she was raised is associated with ED onset
- 5 difficulty to speak up leads to destructive behaviour
- 5 ED would not exist if she could speak
- 5 Feelings would not eat her if she could speak
- 5 Anger sadness and sorrow eat people

NON-PHYSICAL ABUSE AND EATING DISORDERS

Non-physical abuse consequences

3 abuse shaped her character and feelings

3 she cannot feel joy

3 feels like carrying a burden

3 she is always sad

4 does not feel accepted and loved

5 the way she was raised shaped her personality, thoughts and feelings

Feelings for non-physical abuse

4 victimisation and anger at herself

6 Anger at her weak self who is not able to protect her

6 Anger and jealousy for other people

Dealing with non-physical abuse

2 does not react

2 she does not speak up

3 learned not to speak

3 speaking make things worse

3 mother taught her not to speak

4 learned not to speak and walk away

4 she closes to herself

NON-PHYSICAL ABUSE AND EATING DISORDERS

Emotion regulation

3 cannot express feelings

3 cannot handle others' anger

4 does not express herself

4 afraid to speak and react

6 angry thoughts make her sad and tired

6 Feelings are destructive and stronger than her

6 People who can speak up are fine

Explaining non-physical abuse

3 attributes childhood abuse to father's temper

3 still abused because she cannot react and looks like a victim

4 used to be abused and not react

5 she does not confront abuse

6 Things will change if she becomes stronger

6 Things will change if she does not allow others to hurt her

6 Things will change if she acquires self-esteem

6 The obstacle will be her if she doesn't change

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 26 - The superordinate themes and their emergent themes of Carol's interview

Non-physical abuse in childhood

- 1 parents compared her school grades to others
- 1 parents told her not to eat much and exercise
- 1 gaining weight would lead to bullying at school

Non-physical abuse in adulthood

- 1 living with parents preserves abuse
- 1 devaluation from parents
- 1 name calling and comparison from parents
- 1 parents expect her to make mistakes
- 1 comments on her body by parents
- 1 boyfriend is abusive
- 1 boyfriend and parents create the same environment
- 1 boyfriend insults her and makes her sad
- 1 stressed not to say something wrong to him
- 1 boyfriend made a public scene
- 2 boyfriend looks for reasons to argue
- 2 boyfriend makes her feel worthless
- 2 common abusive patterns between boyfriend and parents
- 3 mother more abusive and judgmental
- 3 no abuse from friends
- 4 boyfriend comments on her weight and how much she eats

NON-PHYSICAL ABUSE AND EATING DISORDERS

Self-esteem

6 boyfriend affects her self-esteem

6 important to have self-esteem

Attachment

2 she can't leave her boyfriend

2 afraid to be alone

Non-physical abuse and current ED

6 abuse linked with current ED symptoms

7 weight comments make her eat more

7 relieved when eating but disgusted with her self

Non-physical abuse and ED onset

7 abuse may be linked with ED onset

7 keeping sadness inside affects psychology

7 negative feelings must find a way out

Non-physical abuse consequences

2 used to be abused by boyfriend

2 abuse doesn't have surprises

6 abuse by partner makes her weaker

Feelings for non-physical abuse

2 upset and sad

3 anger

5 feels awful for the abuse

NON-PHYSICAL ABUSE AND EATING DISORDERS

5 anger and jealousy

5 anger for everyone

Dealing with non-physical abuse

2 when boyfriend yells she freezes

3 she could never react to abuse

3 she doesn't know how to react when insulted

4 cannot confront boyfriend

4 she does not react to weight comments

Emotion regulation

3 if she react she may beat parents

3 cannot control hers and others' reaction

3 afraid partner will become violent or leave her

3 afraid parents will never speak to her again

4 keeps eating when boyfriend irritates her

4 easier to eat than speak up

5 wakes up feeling angry

5 eating to relief anger

6 no need to relief with food if anger expressed

6 eating covers anger

Explaining non-physical abuse

3 mixed thoughts regarding abuse

4 attributes abuse to parents' issues

5 abuse is ongoing because she cannot react

5 leaving home would make things better

NON-PHYSICAL ABUSE AND EATING DISORDERS

5 others see her as victim

5 looking better will stop abuse

6 if partner was different she would be stronger

6 therapy helps her understand herself

6 not speaking up makes things difficult

7 she has to change to stop abuse

Other

3 boyfriend is her first relationship

4 boyfriend older than her

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 27 - The superordinate themes and their emergent themes of Kate's interview

Non-physical abuse in childhood

- 1 overweight in a judgmental environment
- 1 indifferent mother
- 1 bullying from social environment
- 1 negative body comments and food shaming
- 1 not eating in family dinners
- 1 discouraged to eat in family dinners
- 1 bullied from friends
- 1 fat was not allowed in high school
- 2 encouraged to diet through food terror
- 2 mother not supportive
- 2 mother could not understand her
- 3 no other family member to talk to
- 3 authoritarian friends
- 3 bossy friend bullying her
- 3 she had none to talk
- 12 mother letting her gain weight is neglect

Non-physical abuse in adulthood

- 4 bullied from friend
- 4 insulted in front of an audience
- 5 bullied on the street
- 5 rude comments from people she dated

NON-PHYSICAL ABUSE AND EATING DISORDERS

5 partner with bad temper

Attachment

3 always involved in bossy relationships

7 unstable relationships

7 afraid to attach

7 not being herself in relationships

10 cannot tolerate rejection

Self-esteem

4 very low self-esteem

6 self-esteem is visible

6 low self-esteem makes you an easy target

6 anorexia can improve self-esteem

6 low self-esteem created an unstable self

6 self-esteem linked to weight

8 worked in a bar to elevate self-esteem

12 self-esteem stays forever

Non-physical abuse and ED onset

5 anorexia could stop bullying

5 losing weight would improve self-esteem

10 abuse not being her fault created bulimia

10 if there was no abuse there would be no ED

11 low self-esteem important contributor to ED onset

NON-PHYSICAL ABUSE AND EATING DISORDERS

Non-physical abuse and current ED

- 11 handles rejection through food
- 11 rejection triggers ED behaviour
- 11 rejection maintains and strengthens ED symptoms
- 12 attachment difficulty maintains the disorder
- 12 hiding in the disorder protects her

Feelings for non-physical abuse

- 4 negative feelings
- 6 sad in childhood
- 8 a lot of anger
- 8 angry at everyone

Non-physical abuse consequences

- 4 blamed weight for not meeting people
- 6 doesn't want to gain weight
- 6 abuse affected her relationships
- 8 lost time with ED
- 10 not eating has been imposed
- 10 tries to keep food

Dealing with non-physical abuse

- 1 eating secretly
- 3 she thought she had to lose weight
- 4 did not react to the abuse

NON-PHYSICAL ABUSE AND EATING DISORDERS

4 dieting and eating secretly

4 adopted gothic style to draw out the attention from weight

5 dieting until the fat was gone

5 anorexia killed the low self-esteem overweight girl

6 perceived as thin was a compliment

6 psychotherapy helped

8 she needs to remain thin

10 bulimic episodes a revolutionary act

Emotion regulation

4 emotional and self-regulation difficulties

4 joined angry groups to tolerate anger

8 suppressing anger with food in childhood

9 anger not verbally expressed

9 self-injury to relief anger

9 delinquent behaviour to relief anger

9 punishing herself through bulimic episodes

9 turning anger at herself created anorexia

9 turning anger at others created bulimia

9 throwing things when angry

9 afraid of direct conflict

9 learned not to argue

9 arguments make her nervous

9 with bulimia she took her anger out

9 changing her anger direction confused her

NON-PHYSICAL ABUSE AND EATING DISORDERS

Explaining non-physical abuse

3 low profile personality

3 justified the abuse in childhood

5 she was hypersensitive

7 overweight kids are abused

7 people are blamed for extra weight

7 her fault for accepting bullying

7 being a woman maintains abuse in adulthood

7 victimisation reinforces abuse

10 justifying bullying from friends

10 reading about feminism explained things

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 28 - The superordinate themes and their emergent themes of Amanda's interview

Non-physical abuse in childhood

- 1 she was always the "fat one"
- 1 Comments on body and weight from a young age
- 1 others kids didn't want her
- 1 Parents told her not to eat so much
- 1 parents not supportive and emotionally distant
- 2 Parents though she was weak and ate a lot

Non-physical abuse in adulthood

- 3 Partners told her to lose weight
- 3 Cheating partner blamed her appearance
- 3 Partners viewed her as weak
- 4 Husband tells her to lose weight
- 4 Her husband is emotionally distant

Self-esteem

- 2 Parents told her she should be good at everything she does
- 2 She never had self-esteem
- 2 She thought she was not good enough
- 2 Not comfortable around people
- 2 Low self-esteem never changes
- 3 When you are not pretty it's easy to be dumb
- 4 She doesn't have self-esteem in her job
- 4 She is afraid to live due to low self-esteem
- 4 self-esteem protects from being hurt
- 6 If she had self-esteem she wouldn't rely on food

Attachment

- 4 avoiding close relationships
- 4 good self-esteem would help attachment
- 4 Being shot from close distance will hurt a lot

NON-PHYSICAL ABUSE AND EATING DISORDERS

Non-physical abuse and current ED

- 5 Ongoing abuse relates to current ED symptoms
- 5 Eating when sad and angry
- 6 Eating when her manager makes her remarks
- 6 Eating when she feels neglected

Non-physical abuse and ED onset

- 1 Always fatty food in the house
- 1 eating model from her mother
- 1 Her mother was telling her to eat more
- 1 Her mother telling her she didn't eat probably not true
- 1 She started eating a lot to please her mother
- 5 Abuse relates to ED onset
- 5 Negative childhood experiences made her focus on food
- 5 If she had self-esteem from parents she wouldn't need food
- 6 Started eating to gain her mother's acceptance and tenderness
- 6 Relationship with mother is important

Non-physical abuse consequences

- 2 She didn't have friends
- 6 She tries to be tender with her own daughter
- 6 Tries not to make the same mistakes with her daughter

Feelings for non-physical abuse

- 1 Ashamed to go out
- 2 She felt alone
- 3 Very low self-esteem and anger
- 3 Anger for being ugly
- 3 Angry at everyone and at her weak self
- 4 She wishes her husband was different
- 4 She feels neglected from husband
- 4 Always felt neglected and lonely

Dealing with non-physical abuse

- 2 Kept thoughts and feelings inside
- 2 Food was her company
- 3 She did not react to the abuse
- 3 Tried to have good grades
- 3 She was trying to lose weight
- 3 used food as a way out
- 4 Eating a lot as a reaction both as a minor and adult

NON-PHYSICAL ABUSE AND EATING DISORDERS

- 5 she has to change
- 5 psychotherapy helped

Emotion regulation

- 3 She didn't express anger
- 3 Her anger was relieved through eating
- 3 Conversations about emotions bring tension
- 3 shallowing emotions with food

Explaining non-physical abuse

- 2 She thought abuse was her fault
- 4 Overweight kids are abused
- 4 Her fault for not reacting
- 5 Her parents' fault
- 5 Abused now because she is still fat and weak
- 5 Abused because of low self-esteem

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 29 – Table of master themes, superordinate themes and emergent themes of Laura’s interview

Themes	Page/Line	Key words
NON-PHYSICAL ABUSE IN A CONTINUUM		
Non-physical abuse in childhood		
2 No positive parental feedback	2/59-60	They were devaluating me for school grades not being 20 but 19.
2 Compared her to others in childhood	2/63-64	... constant comparison to other kids on grades and physical appearance (weight, facial and body characteristics).
2 Insulting comments on physical appearance	2/62-63;2/65-66	Other girls are thin and you are not. You need to reduce food
3 Her fault a boy liked her	3/68-69	If a boy liked me in elementary school...I was the one causing it
3 Parental respect demanded	3/71-73	Me and my sister should respect them because they provide for us
7 Unstable family	7/217-219	Family is the first step for someone to move on in life and this first step was trembling from the beginning in my case
9 Neglect from parents	9/306-309	My parents did not pay attention in neat clothing and us going to school looking girly
Non-physical abuse in adulthood		
1 Living with parents	1/3	As I still live with my parents it is (abuse) a continuation of my childhood
1 Abuse also in adulthood	1/5	Whatever I was experiencing as a child I now experience as adult as well
1 Parents undermining her	1/7-9	In some things I want to do my parents undermine me
1 Verbal and physical abuse in adulthood	1/9-10	Verbal and sometimes physical abuse in adulthood

NON-PHYSICAL ABUSE AND EATING DISORDERS

1 Abuse in the home	1/12-13	I think yes, only from parents
1 Abuse by employer once	1/18-19	An isolated incident from employer that it didn't bother me
1 No abuse from friends/boyfriend	1/20	From friends or partner or someone else... I didn't have anything (abuse incidence)
1 Negative parental comments	1/22	Devaluation, insulting, verbal abuse
1 Parents blame her for abuse and medication	1/25-28; 7/208-212	They try to convince me that my mind is <i>faulty</i> and this is why I am on medication and they have no responsibility for this;...that I am crazy and I am doing something wrong and it is all my fault...if I was a <i>good</i> kid doing whatever they say there would be no problem
1 Immoral to sleep at boyfriend's	1/30-31	Lately I sometimes sleep over at my boyfriend's and I hear insulting comment about my ethics (from parents)
2 Parents abuse sister as well	2/33	The same happens to my sister from our parents
2 Mother more abusive	2/39	Mother is more verbally abusive
3 Compared her to others in adulthood	3/77-79	Other girls at my age are married and have kids and when they (my parents) were at my age they had a car
3 Immoral for having boyfriends	3/81-82	I am a 28 year old who had 2-3 relationships and they tell me "when a woman should begin been characterised as immoral"?
4 Neglected from first partner	4/109-110	Neglect was the reason I ended the relationship
4 Negative comments on appearance from current boyfriend	4/115-117; 130-131	"if you were x kilos you would look better, if you gain more weight I may not like you, you need to work out"; he may make comments regarding my appearance and this hurts my

NON-PHYSICAL ABUSE AND EATING DISORDERS

		feelings
5 Patronization from current partner	5/133-134	As he is 12 years older than me he uses a patronising style
5 Current partner undermining her choices	5/137-138	He attributes my choices to my age
8 Current partner's mother comments on her weight	8/263-265	She says I have to work out and she has an opinion about my body image
Non-physical abuse consequences		
1 Pharmacological treatment	1/24-25	Lately I take medication for depression and anxiety
5 Afraid not to do the same as a mother	5/148-149	I think of my mother who probably had the same experience (abuse) but didn't stop it and passed it on to her children. That is my fear too. To stop it here. If I have kids not do the same.
7 Cannot enjoy accomplishments	7/215-216	I do so many things in my job and yet I don't enjoy it because there is a black part in my life.
7 Cannot enjoy being out	7/224-225	Whatever time I come back they will make a scene anyway. I cannot enjoy being out.
9 Weak to handle and control her life	9/289-291	I would like to push a button and move on to a more quiet life where I can adjust things myself and not feel that I am unable to manage my life as I feel now
9 Has not grown up	9/293	In my mind I have that child who suffered violence... I have not grown up.
9 Abuse made her choose her profession	9/300-301	What I went through was the reason I chose this job, to support the children as I think my parents should have supported me.
9 Sees herself in other kids	9/303-304	In some kids may see myself and I am jealous of others because I wasn't like them.
Feelings for non-physical abuse		
3 Feels like a victim	3/87	I wonder if I am crazy, if

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		they are right, if I am doing something wrong, a victimisation so to speak
3 Doomed due to second thoughts	3/90-91	I wonder if I'm doing something wrong and I'm doomed because two beliefs conflict within me.
7 Negative emotions	7/214-215	I feel anger, rage, sadness, despair... I feel doomed sometimes.
8 Ashamed to talk about the abuse	8/245-246	I find that what I have experienced is very intense and bad and I feel that my image will somehow be damaged
Dealing with non-physical abuse		
2 Sister copes better	2/34	...but she copes better
2 Passive reaction as child	2/44-46	In adolescence I used to think that I was not in a position of power, nor now do I feel in a position of power, just then as a child I could not react in the way I possibly can now
2 Passive reaction as adult	2/47-48	I feel that I have learned this way of reaction (passivity), maybe I am prevented from reacting now. That is, I am more passively accepting this abuse or reacting with a very big anger outburst
2 Learned passivity	2/46-47	I have learned this way of reaction (passivity)
3 crying a lot as an adult	3/86	A lot of crying
3 cried as a child	3/96-96	There was crying but not as much as now
4 She is used to negative comments on her appearance	4/117-118	At first it felt annoying but now I have used to it
5 Feels weak to leave home	5/165-166	I feel weak to do these big steps that I should have done at my age
5 Unable to react	5/160	They (parents) haven't changed, I see it but I can't react.
7 Tried to talk to parents	7/200-202	That there is a problem, that something is wrong, that it is

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		not normal to do such things, that their reactions are not normal, that they are not supportive and they are pushing us down..
7 Tried to find a solution	7/203-204	I have suggested that we go for family therapy to talk about this whole situation and to confirm that I am not crazy and that I am right.
7 Avoids confrontation	7/226-227	I make an effort when they tell me something not to answer because I will have a burst out.
8 Psychotherapy helps	8/268-270	Yes. I'm discussing some things and getting confirmation that I'm not crazy and dealing with some things more distant. It's like I know I'm going to get punched but I'm trying to tighten up to hurt less.
Explaining non-physical abuse		
3 Second thoughts on abuse-her fault	3/86; 4/121-122	I wonder if I am crazy, if they are right, if I am doing something wrong; I have this doubt that maybe they were right (the parents) because he (her partner) says it too
5 External attribution of abuse	5/142-148	I think due to their (parents) personal unresolved issues. They showed their interest in a wrong way maybe due to rejection from their own parents. I am thinking that my mother had the same experience with abuse but she continued this situation to her children.
5 Parents behaviour not justified	5/152-157	I don't think I did something wrong. I've been compared to other people of my age who were had lower grades, for example, and their parents were giving them various gifts to pass the class
5 Internal attribution of abuse	5/159-160	Because I cannot react and it

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		is my fault. I don't know how it is to set limits so not to be insulted as I never learnt to do it
6 Used to be abused	6/171-173	The habit, that I have learned this way and I believe that some things are not going to change and they are what they are... it is the habit. We all have learned this way, they have learned this way..I have learned this way..
7 Living with parents keeps abuse	7/222	Living with them obviously makes it more difficult because it brings more friction
9 She needs to change	9/286-288	Only if I change. My parents are not going to change. Only if I take the situation in my own hands will something change. They will keep doing the same things but I can handle it better
NON-PHYSICAL ABUSE AND ED		
Non-physical abuse and current ED		
9 ED enhances abuse	9/273-276	When they (her parents) see me eating they tell me that I am greedy and to stop and that I am fat and I will not fit to my clothes.
9 eating helps her stop hearing her parents	9/275-276	I continue to find a way out in food and eat to avoid listening to them.
9 eating makes her happy	9/276-277	I feel happy when I eat.
Non-physical abuse and ED onset		
9 eating as a reaction to negative weight comments	9/280-281	I was criticised by them (her parents) as kid for being overweight and being fat so I was eating out of reaction
9 a vicious cycle	9/281-282	I was gaining weight and then I was trying to lose it and then this kept happening.
9 She only had eating	9/283-284	When you can't do anything else at home... you eat. when

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		many things are forbidden you have nothing left but eating.
DEVELOPMENTAL FACTORS AND NON-PHYSICAL ABUSE		
Self-esteem		
3 Cannot believe in herself	3/89	I cannot believe in myself and move on
9 Low self-esteem	9/291-292	If my parents were supportive I would feel like a queen. Now I feel like trash
Attachment		
2 Good relationship with sister	2/34-35	We have a good relationship and understanding
8 cannot open up to partner	8/237-238	I have not opened up...He does not know all the pieces of the puzzle.
9 Still waits to be loved from parents	9/293-294	I am still waiting for the love and support that I feel I should have received. But I know that won't happen.
Emotion regulation		
2 Burst outs as adult	2/48-49	I react with a very big anger outburst where the cause may not be significant but it works to me in a cumulative way.
2 suppressing anger as child	2/53-56	I was holding anger inside as a child
3 uncontrollable anger outbursts	3/84-85	Anger outbursts. Outbursts that I cannot control
3 anger not expressed in childhood	3/96	There was not expression of anger but there was anger
5 Cannot set limits	5/160-161	I don't know how it is to set limits, I never learned it
5 Avoids conflict	5/164	I avoid conflict with them (parents) because I can't stand it at all
6 intense anger reaction for minor reasons	6/183-184	The anger outburst does not always come from a clear cause...the reason could be insignificant
6 cumulative thoughts lead to uncontrollable anger outburst	6/187-189	I think many things at the moment cumulatively, overall, my reaction is

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		terribly strong, I cannot control it, and others may not see why this is happening.
Other		
1 physical abuse in adulthood	1/9-10	There was physical abuse in early adulthood (by parents)

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Appendix 30 – Table of master themes, superordinate themes and emergent themes of Betty’s interview

Themes	Page/Line	Key words
NON-PHYSICAL ABUSE IN A CONTINUUM		
Non-physical abuse in childhood		
1 non-physical abuse from parents/classmates/teachers	1/3-6	It has often come from different people, first from some of the family members and then from schoolmates to schoolmasters... I had all this before my 18th at a very young age.
1 bullying at school for grades	1/13-16	Bullying at school let’s say. I was a low performing student and some classmates used derogatory expressions on me. The teachers did it also because I didn’t understand or I was giving the wrong answer “you are not trying, you are not reading, you are lazy”.
1 insults at home for grades & homework	1/23; 1/31-32	[...]Degrading comments on grades; "You don’t understand what you read, concentrate, you are lazy, you are messy"
1 emotional neglect from parents	1/26-27	I felt that there was emotional neglect from both my parents. From my father always and from my mother from the age of 10.
Non-physical abuse in adulthood		
1 non-physical abuse as adult	1/12	In non-physical form it happens now as well
2 aunt proposed drug use	2/35-36	When I reached adulthood my aunt proposed me drug use. I didn’t accept it and she kept proposing it
2 mother compares her to sister	2/36-39	From my mother there is discrimination and underestimation. I have a younger sister and she

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		compares to my sister in everything. In appearance (that she is thinner, that she can control eating, her studies were better).
2 emotional neglect	1/41-42	There is also emotional neglect now, denying the fact (her father) that I may have a disease. So does my mother.
2 comment on weight from boyfriend	2/45-46	He commented that I have become fat. He is older than me.
2 lost a job for weight	2/46-47	I have also understood that I didn't get a job because I had extra weight
2 comments on weight from doctor	2/47-48	A doctor once told me that if I lose weight I will become more elegant. The doctor himself was fat..
2 comments on weight from friend	2/50-51	A friend told me in front of everyone that I have become fat and we were in my house celebrating my birthday.
2 abuse regarding weight in adulthood	2/52-54	Yes because we live in the era of images (asked if the non-physical abuse in adult life has to do with weight mostly)
Non-physical abuse consequences		
2 abuse affects her development in life	2/67	It is a factor that affects my development
4 stress and sadness affected her health	4/103-104	It has affected my health due to stress and sadness
Feelings for non-physical abuse		
2 anger due to abuse	2/66-67	It makes me angry. There is no concern about who can have a child. My parents were unfit to have a child.
3 disappointment & guilt for not setting limits	3/69-70	I feel frustration and guilt that I cannot impose certain things to others, like on my mother, and set limits
4 helpless and tired	4/103	It (the abuse) has made me feel helpless and easily tired

NON-PHYSICAL ABUSE AND EATING DISORDERS

Dealing with non-physical abuse		
4 psychotherapy helps	4/100	Yes I analyse my feelings and this helps me
Understanding non-physical abuse		
1 learning difficulties in childhood	1/19-21	Over the past two years I have learned that I have learning difficulties and I have justified some things regarding that period of time (the devaluating comments from parents regarding school performance). I mentioned it to my family and no one apologized
2 external attribution of abuse	2/55-57-59	Because there is mimic behaviour (answering why she think it happened to her). The people who did this have also been abused. Because people have psychological issues (answering why it is still happening)
2 relating to abusive people preserves abuse	2/61-62	When I have contact with people who are abusive and I know it, I am responsible too.
2 her parents were unfit to have a child	2/68	My parents were unfit to have a child
3 detaching from abusive people will stop abuse	3/95-96	It depends more on me, the way I think and react. To distance myself from abusive people
NON-PHYSICAL ABUSE AND ED		
Non-physical abuse and current ED		
3 abuse relates to ED now through negative emotions	3/78-82	Of course they are related (answering if she believes continuing abuse and ED maintenance are related). With frustration and anger. Food is easy to find

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3 food alleviates stress, wounds and <i>drowned</i> emotions	3/82-85	That's where the stress, the stress, the sadness, the "drowned" feelings, the wounds come out. It is a cycle
Non-physical abuse and ED onset		
3 trauma leads to addictive behaviour	3/86-87	Anything traumatic can create a form of addiction
3 imitating unhealthy eating leads to ED	3/87-89	There was also mimetic behaviour on my part in extreme eating behaviours of family members. For example, my mother eats too much and in a messy way
DEVELOPMENTAL FACTORS AND NON-PHYSICAL ABUSE		
Self-esteem		
4 abuse affected self-esteem	4/102	Yes on my self-esteem (where non-physical abuse affected her).
Attachment		
2 important relationships can be addictive	2/62-64	Some relationships can be addictive or primitive like mother-child, you cannot avoid them because you live with them.
2 unable to avoid abusive relationships	2/64-65	The same thing has happened in a relationship, I couldn't leave when I should.
4 cannot open up and relate to partners	4/102-103	[...] on my partners' approach (where non-physical abuse affected her). I am very closed and shy
Emotion regulation		
3 cannot communicate emotions	3/71-74	(difficulty on) how to communicate my feelings
3 cannot set limits	3/74-75	(difficulty on)the distances I could get (from others)
Other		
1 physical and sexual abuse in childhood	1/8-10	It was sexual abuse first by the aunt, then by the mother, and physical abuse, because I was naughty, by all members of the family and by grandparents.

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 31 – Table of master themes, superordinate themes and emergent themes of Jenny’s interview

Themes	Page/Line	Key words
NON-PHYSICAL ABUSE IN A CONTINUUM		
Non-physical abuse in childhood		
2 abuse by father in childhood	2/39	In my minor life this was done by my father
2 yelling and devaluation from father	2/41	He had a lot of nerves. He shouted at me and underestimated me.
2 she had to be obedient and good student	2/41-43	I had to be obedient.. be a good kid, not to make a fuss, to get good grades
2 father insulted her	2/44-45	He called me stupid, fat and ugly.
2 neglected and insulted by teacher	2/49-50	One teacher was abusive to me because I did not have good grades. He did not care about me as if what I had to say would be wrong.
2 neglected in class	2/52-53	The children were also divided into good and bad students and because I did not have very good grades, I experienced indifference. As if I did not exist
3 mother was passive to her abuse	3/74-75	It’s my mother’s fault that she did nothing to protect me from this. All she did was tell me not to speak. As she did not speak either
3 did not receive love from parents	3/84-85	I did not get the love I wanted from my parents.
3 mother did not protect her	3/85	My mother did not support me against my father’s behaviour

NON-PHYSICAL ABUSE AND EATING DISORDERS

Non-physical abuse in adulthood		
1 non-physical abuse from partners	1/3	It has happened to me mainly from people with whom I had a relationship
1 negative physical appearance comments from partners	1/5	I have received comments about my appearance. They criticise me
1 verbal abuse and devaluation from partners	1/7-8	My hair, my body, my clothes... "how are you like that". Also to underestimate me and tell me what to do as if I am stupid and my opinion did not count anywhere
1 partners rejecting her opinion	1/12-13	When we talked about something it was as if my point of view did not count or as if it was wrong. I felt like I had nothing important to say
1 partners rejecting her wishes	1/15-17	I did not suggest a place or a movie that I wanted because I always felt he would reject them. Once that I did suggest something this is what happened.
1 neglect and devaluation from colleagues	1/19-22	But I also experience it at work from some colleagues. Some treat me as if I do not exist or as if I do everything wrong and I need guidance. Other times they comment on my character.
1 perceived as shy and weak from colleagues	1/24	"You are shy. You do not understand what we are saying. Do not be so weak"
1 colleagues think she is stupid	1/27-28	They think I do not understand when I react to their stupid conversations.

NON-PHYSICAL ABUSE AND EATING DISORDERS

2 insulting and devaluation in adulthood	2/36	Yes. Invalidation, insults. Devaluation in general
Non-physical abuse consequences		
3 abuse shaped her character and feelings	3/82	On how my character was shaped. On how I feel
3 she cannot feel joy	3/82	I cannot feel joy
3 feels like carrying a burden	3/83	It's like carrying a burden
3 she is always sad	3/83	I am always sad
4 does not feel accepted and loved	4/118-119	I did not feel acceptance and love so to be strong and show this to others
5 the way she was raised shaped her personality thoughts and feelings	5/133-134	The way I grew up is responsible for who I am now. How I think, react and feel.
Feelings for non-physical abuse		
4 victimisation and anger at herself	4/124-125	Very bad. I feel like a victim and I am angry that I am so weak and I cannot defend myself
6 Anger for her parents	6/159-160	For my father who was bad with me. For my mother who did not protect me. Who did not teach me to be strong
6 Anger for her weak self	6/162	For myself who was not strong enough to protect me.
6 Anger and jealousy for other people	6/163-165	Many times I feel angry for the whole world. I also get angry when I see others who did not grow up that way. I envy them and that makes me angry with them. Why should they be strong and I cannot? This makes me angry.
Dealing with non-physical abuse		

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2 does not react	2/33-34	When something bothers me I do not react. I usually leave.
2 she does not speak up	2/58	My reaction is the same. I did not speak then nor do I speak now
3 learned not to speak	3/63-64	I learned not to speak so that there would be no bigger reaction from others.
3 mother taught her not to speak	3/66	That was my mother's advice regarding my father
4 learned not to speak and walk away	4/97-98	I have learned not to speak and walk away
4 she closes to herself	4/100	I close to myself
Explaining non-physical abuse		
3 attributes childhood abuse to father's temper	3/72	I think because my father had a bad temper
3 still abused because she cannot react and looks like a victim	3/78-80	Because I cannot react. This is my fault. When others see a weak person without self-esteem, they look at an easy victim to put their psychological burden on him/her
4 used to be abused and not react	4/114	It's the habit. That's how I have learned.
5 she does not confront abuse	5/128	I don't confront it. I just don't speak like I said earlier.
6 Things will change if she becomes stronger	6/178-179	Only if I change. I have to become stronger and defend myself.
6 Things will change if she does not allow others to hurt her	6/181-183	To speak when something bothers me and not allow it to happen. Do not allow others to comment on me and interfere in my life
6 Things will change if she gains	6/183-184	To gain confidence and

NON-PHYSICAL ABUSE AND EATING DISORDERS

confidence		believe in myself.
6 The obstacle will be her if she doesn't change	6/186-188	I will be the obstacle if I do not change. Only I can change that. Only I can defend myself. But in theory I'm good; in practice I cannot do it. My self-esteem is a mess.
NON-PHYSICAL ABUSE AND ED		
Non-physical abuse and current ED		
5 restricts food as punishment when sad	5/137-138	I think so. When I am sad and I keep things inside I do not eat. It's like punishing myself
5 she does not deserve to eat	5/141-142	As if I do not deserve to eat and especially as if I do not deserve to eat something I like.
5 she is bursting out with food	5/141	Like bursting out there. In my relationship with food.
5 food is the only thing she can control	5/141-142	It's the only thing I can control
Non-physical abuse and ED onset		
5 the way she was raised is associated with ED onset	5/145	I think yes. The way I grew up must be related to this
5 difficulty to speak up leads to destructive behaviour	5/147-149	When you are not well and you cannot speak you probably find another way to show it. This is what I think. You turn into yourself and you shut yourself in there. But this way is destructive in the end. I do not know how else to describe it

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5 ED would not exist if she could speak	5/152	Yes. Why would it (ED) exist?
5 Feelings would not eat her if she could speak	5/152-153	If I could get all this out of me they would not "eat" me.
5 Anger sadness and sorrow eat people	5/155-157	Emotions of course! Anger. The sadness about what is happening and I cannot speak. The sorrow. Aren't these "eating" people? This is why people get sick
DEVELOPMENTAL FACTORS AND NON-PHYSICAL ABUSE		
Self-esteem		
2 weak with no self-esteem	2/33	I am weak. I don't have self-esteem.
3 cannot defend herself due to low-self-esteem	3/60-63	I do not feel that I have the confidence to defend myself. I cannot fight back when I have to.. Because I don't have self-esteem
3 low self-esteem keeps her back	3/83-84; 86-91	I'm not trying for more. I do not have self-esteem...; I do not believe in myself. I only do what I can and I do not try for more. I do not want responsibilities. I cannot stand them. I think I will not succeed if I try for something more. That's why I didn't go to college. You know, because of the years I have been working I could ask for a promotion but I am not doing it. I don't want responsibilities. I want to do my job "invisibly" and leave.
3 things would be different if she had self-esteem	3/92	Things would be different If I had self-esteem. If I believed in myself.

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3 avoiding responsibilities	3/93; 4/ 94-98	When responsibilities increase you should be able to deal with what is happening and come into conflict with more people. I do not want that. It's hard. Because I have not learned to deal with people and situations. I have learned not to talk and leave. When you are like that you cannot take a position with responsibilities
Attachment		
4 avoids close relationships / trust	4/101-102	I do not trust others. I do not have close relationships. I like distance. I feel safer when I keep my distance.
4 avoids intimate relationships / rejection	4/104-108	I cannot attach. I'm not coming close. I'm afraid of rejection. At first I make them like me and then I go away. I want my peace of mind. I do not want responsibilities. I cannot have someone telling me what to do. I cannot argue.
4 attachment makes you vulnerable	4/109-111	So as not to get hurt of course. When you get close to someone you become vulnerable. Then if something goes wrong you will be very hurt
Emotion regulation		
3 cannot express feelings	3/61	I cannot express my feelings
3 cannot handle others' anger	3/67-69	I have used not to speak and I only speak to the point where I feel safe because I do not know what to do if the other person starts to get

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		angry and shout. I do not know how to deal with the other person's anger
4 does not express herself	4/100	I cannot express myself. I cannot say what I feel
4 afraid to speak and react	4/116-117	This whole situation with my father made me withdrawn. To be afraid to react. To be afraid to say what I feel
6 angry thoughts make her sad and tired	6/167-170	After having thoughts that make me angry, I feel sad. Sad and tired at the same time. Maybe sad because I know I'm not capable of dealing with what makes me angry. Those who make me angry. When you feel something intensively you get very tired, don't you?
6 Feelings are destructive and stronger than her	6/172-175	They (emotions) are stronger than me. This is why I am afraid to speak. I do not know what will happen if I express them. Instead of taking them out and destroying someone else, I leave them inside me and they destroy me. They "eat me" from inside. This is how I can describe it.
6 People who can speak up are fine	6/175-176	I see around me those who can speak up. They are fine, they do not look sad. They say what they want and they do not care about anything.

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Appendix 32 – Table of master themes, superordinate themes and emergent themes of Carol’s interview

Themes	Page/Line	Key words
NON-PHYSICAL ABUSE IN A CONTINUUM		
Non-physical abuse in childhood		
1 parents compared her school grades to others	1/11-12	They used to compare my grades in school.
1 parents told her not to eat much and exercise	1/17-18	They told me the same things when I was little (not to eat much and exercise).
1 gaining weight would lead to bullying at school	1/18	I was told (by parents)that if I become fat, the children at school will make fun of me
Non-physical abuse in adulthood		
1 living with parents preserves abuse	1/3-4	I live with my parents so nothing has changed. As they treated me before, they treat me now
1 devaluation from parents	1/6	They devalue me. It’s like I am doing nothing right.
1 name calling and comparison from parents	1/8-11	When I do something they think is wrong I hear "are you stupid"; And things to this effect. They also make comparisons with friends’ daughters and cousins... That they have done better in their lives professionally and personally
1 parents expect her to make mistakes	1/14-15	I do not know how to express it but it is like I always make a mistake or they (her parents) expect me to make a mistake

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1 comments on her body by parents	1/15-17	And about my body. Not to eat much and exercise.
1 boyfriend is abusive	1/20-22	He is very abusive. As if he does not want me. As if he is constantly angry with me. I do not know. I cannot explain it.
1 boyfriend and parents create the same environment	1/24-25	It's like leaving my parents and going to the same environment
1 boyfriend insults her and makes her sad	1/25-26	He reacts very badly and insults me. He makes me constantly sad.
1 stressed not to say something wrong to him	1/28-29	I am constantly anxious not I say something wrong or not to do something wrong and he starts yelling at me
1 boyfriend made a public scene	1/29-31	He once left me in a shop alone because we had an appointment and I was ten minutes late. He embarrassed me and left. People were looking at us and I was very ashamed.
2 boyfriend looks for reasons to argue	2/33	It is as if he is constantly finding reasons to argue with me. Without a cause
2 boyfriend makes her feel worthless	2/33-35	He makes me feel worthless. As if I do not deserve someone to be with me. As if I am not good for him.
2 common abusive patterns between boyfriend and parents	2/53-60	There is a lot in common with my parents and my boyfriend. It's something I was shocked when I realised. Sometimes when I cook I can drop something or get dirty in the kitchen and my boyfriend has exactly the

NON-PHYSICAL ABUSE AND EATING DISORDERS

		same reaction as my mother does. He will either make an ironic comment about my abilities or shout at me. The first time this happened at my boyfriend's house I froze. It was like listening to my mother.
3 mother more abusive and judgmental	3/70-71	It is expressed by both, perhaps more verbally than my mother. She is very judgmental
3 no abuse from friends	3/75	Not particularly. I do not have anything to remember from friends
4 boyfriend comments on her weight and how much she eats	4/104-108	He constantly observes how much I eat. If I eat a lot he tells me that I will gain weight and that irritates me. He causes me anxiety that I will gain weight. He notices that I gained weight and he tells me that my clothes will not fit
Non-physical abuse consequences		
2 used to be abused by boyfriend	2/47-49	Even though it bothers me (the abuse), it is a situation that I know and am used to. I know everything. I'm waiting for them. Sometimes I think that if I find someone who treats me well I will not know what to do
2 abuse doesn't have surprises	2/50-51	I know what to expect. There are no surprises
6 abuse by partner makes her weaker	6/178-179	It (abuse by partner) has made me even weaker I think. If he was different he might have helped me get

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		stronger
Feelings for non-physical abuse		
2 upset and sad	2/65	It makes me upset and sad
3 anger	3/82	I have a lot of anger
5 feels awful for the abuse	5/143	Awful. As if I am not worthy of anything.
5 anger and jealousy	5/147-152	Anger and jealousy. I am jealous of others who are stronger than me or whose parents are better
5 anger for everyone	5/152	Anger for everyone. For my parents, for my boyfriend, for myself
Dealing with non-physical abuse		
2 when boyfriend yells she freezes	2/62-67	I understand that every time he shouts at me I freeze... I cannot react. I'm waiting for it to end because I'm afraid to talk. I do not know what to say and how to deal with it. I'm angry and upset. I have not learned to answer when someone attacks me. I do not know how to do it.. Isn't that attack?
3 she could never react to abuse	3/78-79	As a minor I could not react and I kept it all to myself. I do the same now
4 cannot confront boyfriend	4/109-111	No, what's the point? He will not understand. I cannot afford more yelling and insulting
4 she does not react to weight comments	4/112-113	I don't react. I get upset and sad at the same time.
Explaining non-physical abuse		
3 mixed thoughts regarding abuse	3/90-94	Sometimes I think they may be right. Is it a coincidence that both my parents and my

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		boyfriend shout at me for the same things? On the other hand I understand that I cannot be blamed for everything and their behaviour is not what it should be. They have nerves for their own reasons and they take them out on me.
4 attributes abuse to parents' issues	4/122-126	I think it has to do with problems in my parents' character. They have their own issues and they take them out on me. Obviously they do not know how to behave. My grandmother - my mother's mom - was very authoritarian. Maybe this is why my mother is like that.
5 abuse is ongoing because she cannot react	5/130-132	Because I do not react. This is my fault and I am angry with myself. If I had reacted from a young age, this would not have happened. I am a weak character. I avoid conflict with them because I cannot stand it at all. I cannot argue.
5 leaving home would make things better	5/133	If I was living alone it would be better I think. But I cannot now
5 others see her as victim	5/136-139	I'm used to it I think. That is how I am. As a character. That's what I know how to do. Others see me as a victim. Weak I mean. You attack someone who looks weak, not someone who looks strong (sigh)
5 looking better will stop abuse	5/143-145	I keep thinking about what I can do to look better. To find

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		a good job, to become beautiful. I do not know. If I am successful, it will be difficult for someone to speak in an insulting way to me. This is what I think.
6 if partner was different she would be stronger	6/178-179	If he was different (boyfriend) he could help be become stronger
6 therapy helps her understand herself	6/170-172; 6/177-178	Psychotherapy helps me explain and understand some things about myself and others. But it takes time for me to change something. I cannot become another person suddenly..... It (psychotherapy)has changed things inside me. To understand some things about me. Outside I have not changed. It may take a long time for this to happen.
6 not speaking up makes things difficult	6/174-175	The fact that I cannot speak when I have to. This difficulty I have to speak. This is “eating” me. That I keep everything inside me
7 she has to change to stop abuse	7/208-211	Only if I change. Others are not going to change. They are who they are. I have to become stronger. To defend myself. To have self-esteem and to show it. To set limits. To speak. Without caring what will happen.
NON-PHYSICAL ABUSE AND ED		
Non-physical abuse and current ED		
6 abuse linked with current ED symptoms	6/191	Yes

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7 weight comments make her eat more	7/193-194	When they (parents and partner) see me eating they tell me that I will become fat and my clothes will not fit, I continue eating! This calms me down and I already know that it irritates them when do it.
7 relieved when eating but disgusted with her self	7/195-196	I feel relieved when I eat but then I become disgusted with myself and want to vomit
Non-physical abuse and ED onset		
7 abuse may be linked with ED onset	7/199	I think it is related but I can't know for sure
7 keeping sadness inside affects psychology	7/201-202	When something upsets you and you are not well and it lasts for a long time it will affect your psychology
7 negative feelings must find a way out	7/204-206	When you keep things inside you... anger, sadness, complaints... they must come out. They will find a way out but this way will not be good. It will not be healthy
DEVELOPMENTAL FACTORS AND NON-PHYSICAL ABUSE		
Self-esteem		
6 boyfriend affects her self-esteem	6/181-182	I mean, I would feel better about myself if he (boyfriend) treated me better. I would have self-esteem and I would be stronger
6 important to have self-esteem	6/182-184	It is important to have self-esteem. Others do not treat people with self-esteem like rubbish. They can't.
Attachment		

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2 she can't leave her boyfriend	2/38-41	I have thought about (leaving boyfriend) and I have to do it. I do not know why I cannot. Maybe because I have nowhere to go. All my friends are in a relationship. I will be the only one alone. Who will I go out with? Will someone else be better than him? What if I do not find someone else? What If I be alone forever?
2 afraid to be alone	2/43-45	Yes. It is very difficult to meet someone for a relationship. Everyone is just looking for sex now. Now with the quarantine it is even worse. Those who already have a relationship are lucky. But I know I have to leave him
Emotion regulation		
3 if she react she may beat parents	3/81-82	I do not answer because if I answer I think I can hit them as I get angry. I'm very angry. Many times I break things when I am alone
3 cannot control hers and others' reaction	3/85-86	I cannot control my reaction and I cannot control others' reaction. I am afraid how things may develop.
3 afraid partner will become violent or leave her	3/88	He may become violent or break up with me
3 afraid parents will never speak to her again	3/90	That they may never speak to me again
4 keeps eating when boyfriend irritates her	4/113-117	I want to eat because he has irritated me. To eat a lot and so he can see me. Sometimes I did that but then I vomited without him knowing it. He told me "eat like this and you will not be able to go

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		through the door". I wanted to punch him. I wish I have done it. But instead of hitting him I continued to eat.
4 easier to eat than speak up	4/119-120	It is easier for me to eat than speak up. We will argue and he will shout at me. I do not want tensions
5 wakes up feeling angry	5/152-154	Many times I wake up with anger and nerves for no reason. I want to break everything even though nothing has happened. I just wake up like that and I get tired of it.
5 eating to relief anger	5/156-158	I'm just very angry inside. Outside I try not to show it. I eat. This relieves me at that time but then I feel bad.
6 no need to relief with food if anger expressed	6/161-164	<i>(Do you think that if you showed your anger something would change in your relationship with food?):</i> I'm not sure but I think so. I would not need to be relieved of anything. But I cannot know for sure. <i>(Do you feel that eating relieves your anger?):</i> Yes.
6 eating covers anger	6/166-168	It's like I want to do something intense at that time but I can't. Eating is like forgetting anger or drowning it out. As if the food covers anger and I forget. But eating something I really like not just eating anything
Other		
3 boyfriend is her first relationship	3/96	I never had another

NON-PHYSICAL ABUSE AND EATING DISORDERS

		relationship before him
4 boyfriend older than her	4/100	He is 8 years older than me

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 33 – Table of master themes, superordinate themes and emergent themes of Kate’s interview

Themes	Page/Line	Key words
NON-PHYSICAL ABUSE IN A CONTINUUM		
Non-physical abuse in childhood		
1 overweight in a judgmental environment	1/3-4	To begin with, I was quite overweight and I was in a countryside environment where people put a lot of emphasis on these things
1 indifferent mother	1/4-6	I have only grown up with my mother who was not worried about these matters (appearance) so I had gained weight... so I was essentially alone in everything I lived...
1 bullying from social environment	1/6-9	Bullying which was essentially criticism, and I also had very strong criticism from my social environment, uncles, aunts, teachers, parents of friends, all those who were trying to do the job that my mother did not do but not with very nice way.
1 negative body comments and food shaming	1/11-13	From a very young age I remember hearing bad things about my body, that is, my aunt screaming at me that I have cellulite from the age of 12 or I was generally accepting a lot of food shaming to the extent that I considered it normal.
1 not eating in family dinners	1/13-15	For example I will not be eating at family dinners because I’m fat or we all gather together and I will not eat because I have to diet.

NON-PHYSICAL ABUSE AND EATING DISORDERS

<p>1 discouraged to eat in family dinners</p>	<p>1/17-20</p>	<p>Up to one point they were telling me that (not to eat)... but they also made constant comments... I think I was also hypersensitive so as soon as discussions about silhouette and weight started and things like "let's watch what we eat" and they all stared at me... you understand..</p>
<p>1 bullied from friends</p>	<p>1/20-23</p>	<p>And my friends at the time who had a lot anxiety about their own body, even though they were very thin, they talked to me very badly... they did not actually bully me but they would just say to me "I cannot look at your hands because they are so fat".</p>
<p>1 fat was not allowed in high school</p>	<p>1/24-25</p>	<p>it was all this paranoia at the time meaning it was awful to have fat and extra weight in high school</p>
<p>2 encouraged to diet through food terror</p>	<p>2/28-30</p>	<p>I was encouraged to diet... I was commented on both my body and my diet not in the right way. They were trying to terrify me about everything...like 'this will make you fat, it has 5000 calories'.</p>
<p>2 mother not supportive</p>	<p>2/35; 2/39-44</p>	<p>She was a little apathetic..; She did not handle it. When I complained to her that I was called fat and ugly..she would say to me "ok it is bad that they call you ugly but objectively you are fat" generally my mother is not good at supporting someone emotionally... she could not help me at all in this whole thing. Telling someone that he is fat without helping him does not help him much. But</p>

NON-PHYSICAL ABUSE AND EATING DISORDERS

		in general my mother was a bit uninvolved because it was her style to be non-intrusive.
2 mother not good in understand her	2/47-51	It was not very easy to communicate with my mother... she had grown up in a different way, she could not understand my own problems. For her it was a problem that they once dug in the field and we should be happy that we do not dig in the field anymore. While she provided me with everything she was not good at interpersonal support.
3 no other family member to talk to	3/52-55	(asked about any contact with her father) No no. They divorced when I was born. I have no contact (asked about siblings) No, I am an only child
3 authoritarian friends	3/57-59	I had some girlfriends but they were a bit..how to say this... very authoritarian... I was very easy going. I always had a friend who was very dynamic for whom I did what she wanted me to do
3 bossy friend bullying her	3/64-67	Uhm let me think..with one of my friends it was always expected that I will wait for her, I will move her around or if I did not want to go to her house she would tell me "since you want to lose weight, walking will be good for you". Because it was so obvious that I had an issue they were stepping on it
3 she had none to talk	3/73	They bothered me a lot (the negative comments) but I

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		had no one to talk about it
12 mother letting her gain wait is neglect	12/274-275	She (her mother) certainly should not let me gain so much weight and become so ugly. This is neglect.
Non-physical abuse in adulthood		
4 bullied from friend	4/91-93	I had a high school friend who was bossy and had her own issues with her body and kept commenting on my body.
4 insulted in front of an audience	4/93-99	I joined a university theater group and the director - I was in normal weight at the time but it was established that I am the person who is being bullied for her weight and they would tell me not to eat because "you will gain weight again"- he (the director) had picked me up for an exercise and he was shooting me from some angles and he was saying to me "what a nice face you have..if you only didn't have this body" and he told me that a room full of people and I was a very shy freshman and I remember coming home and crying for 5 hours.
5 bullied on the street	5/104-105	someone on the street would see me eating and tell me to diet I would take that comment very seriously
5 rude comments from people she dated	5/107-108	<i>(asked about negative comments from partners)</i> During my first serious relationship I had anorexia so no, but with some guys I just went out prior to the anorexia phase I had rude comments about my weight
5 partner with bad temper	5/112-113	I did not have authoritarian

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		partners but one of them had bad outbursts with a lot of yelling.
Non-physical abuse consequences		
4 blamed weight for not meeting people	4/88-91	It was very difficult for me to meet people and I blamed my weight for this. But now I understand that I was not social and I would not meet people by staying in my house but at the time I thought I looked like a monster.
6 doesn't want to gain weight	6/139-140	Now I have a hard time with the idea of gaining weight but I am trying to work it through.
6 abuse affected her relationships	6/147-149	<i>(asked where the abuse affected her)</i> - In how I connect with others. I do not know if this is because I am hypersensitive though.
8 lost time with ED	8/178-184	<i>(asked where else this situation affected her)</i> - In everything. I have lost huge periods of time in my life while fighting with the ED.. And there were times when I did not leave the house and cried because I was ashamed that I was fat and depressed because I was overweight. I had focused on the weight and I lost time from my life mainly in the professional part and now at the age I am I should have done more things and have a better CV. Now I have gaps in my CV.
10 not eating has been imposed	10/235-236	I made myself not eat but not eating has been imposed to me by others through all these years of bullying

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10 tries to keep food	10/238-239	I was taking some medicine to vomit. I ate once every 2 days a lot and then I took a medicine to vomit. Now I am trying to fix all these in therapy.
Feelings for non-physical abuse		
4 negative feelings	4/81	I had negative feelings - I was not sure if it was right that I felt them
6 sad in childhood	6/137	Eh..in my minor life I was sad and I felt that I could not do anything.
8 a lot of anger	8/188-189	Anger. I have a lot of anger. Over the last few years I have realised that I have a right to be angry.
8 angry at everyone	8/191-195	<i>(asked about the direction of her anger)</i> - This is a very nice question because it really is like holding a hand grenade and not knowing where I want to throw it. I mean, I blame myself, I blame my mother because she let me gain weight, I blame society for being so oppressive with women and weight, but most of all I burst out my anger with bulimic episodes. I do not know who to blame first.
Dealing with non-physical abuse		
1 eating secretly	1/15	I was just eating secretly
3 she thought she had to lose weight	3/67-68	I did not understand and I thought that the only solution not to accept such behaviour would be to lose weight
4 did not react to the abuse	4/75	<i>(asked if she was saying something when it happened)</i> - No. I thought they had the right because I

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		was giving it to them by being fat
4 dieting and eating secretly	4/77-78; 4/88	I started dieting ...I felt very bad when I ate..I ate secretly; After 18 I gained more weight and I started dieting
4 adopted gothic style to draw out the attention from weight	4/81-83	At the end of high school I had decided to be gothic so to get the attention out of my weight because everyone was concerned with the fact that I had green hair and wore black clothes
5 dieting until the fat was gone	5/99-102	And I said that everyone is right, I am a monster and I must definitely lose weight, otherwise nothing good will ever happen to me in my life. There I started my first paranoid diet. I said I would continue not to eat until the fat was completely gone.
5 anorexia killed the low self-esteem overweight girl	5/111-117	This personality died with anorexia because I decided to protect myself but in the wrong way. I protected my vulnerable self. My low self-esteem self. Anorexia “helped” me lose weight, become pretty, feel better about myself. It helped me “kill” the ugly overweight girl that everyone mocked
6 perceived as thin was a compliment	6/133-135	In my minor life I was told not to eat because I am fat and in adulthood to eat because I am thin and I took the later as a compliment because of what I had gone through with my weight.
6 psychotherapy helped	6/138-139; 10/241-242	for the last 4 years with psychotherapy I realised that I can answer back and I am

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		not obliged to accept such behaviours; (<i>asked if therapy helped her</i>) Definitely yes. I realised that it was not my fault and that I had the right to be angry. I understood my relationship with food and I can explain it now.
8 she needs to remain thin	8/175-176	Now I am thin and it has been a long time since I last heard something about my weight. Now I can avoid it because I am thin but if I gain weight it will happen again
10 rebelled through bulimic episodes	10/233-236	I think that from one point onwards I saw bulimic episodes as a revolutionary act. But it's a bit silly because in reality I was rebelling against myself. I made myself not eat but not eating has been imposed to me by others through all these years of bullying.
Explaining non-physical abuse		
3 low profile personality	3/60-62; 7/163-164	It probably has to do with my personality. Very low profile; I was also withdrawn and shy... I was the kind of child you will bully.
3 justified the abuse in childhood	3/68-70	I thought it was reasonable to say to your friend who is overweight "you should walk to my house so you can lose weight".
5 she was hypersensitive	5/104	I was hypersensitive and I magnified everything
7 overweight kids are abused	7/160-161	I think this happens to children who are overweight. People consider it normal to do so
7 people are blamed for extra	7/161-163	If you go to an adult to ask

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weight		for help he will tell you to lose weight to resolve this. But if someone has a big nose, an adult can say that it is not ok to make fun of it, but for the weight they believe that it is your fault.
7 her fault for accepting bullying	7/164-165	It's my fault that I was accepting the bullying and I did not react.
7 being a woman maintains abuse in adulthood	7/169-171	Because I was born a woman. It is common to be stressed for appearance even when you have a normal body. Especially in artistic circles it is normal to make comments about the body and to be under pressure not to gain weight.
7 victimisation reinforces abuse	7/171-173	I also had hypersensitivity to this matter due to bullying at a young age and I accepted it as a victim so this was also a reinforcing factor.
10 justifying bullying from friends	10/224-226	I mean ok about my friends because they were young and didn't know any better and they were also stressed about appearance but the adults had to find a better way to talk to a child.
10 reading about feminism explained things	10/226-228	I was helped by what I read about feminism and that society has a responsibility for this by creating wrong role models for women.
NON-PHYSICAL ABUSE AND ED		
Non-physical abuse and current ED		
11 handles rejection through food	11/249-151	The rejection I had when I was little had to do with my weight and my appearance.

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		And now I try hard to avoid or manage rejection through food.
11 rejection triggers ED behaviour	11/253-257	When I was not accepted at a postgraduate programme I had a bulimic episode. When I had to go to a job interview, 2 days before I did not eat anything as a preparation for success. When I feel rejected I have bulimic episode. In order not to be rejected I do not eat. Weight has been linked to rejection. The way I have learned to avoid rejection is to limit food.
11 rejection maintains and strengthens ED symptoms	11/258-260	<i>(asked if rejecting and continuing comments on appearance preserve the ED symptoms)</i> - Yes. They maintained them and strengthened them.
12 attachment difficulty maintains the disorder	12/278	<i>(asked what contributed to ED maintenance)</i> - My difficulty to attach. I use the disorder as a buffer in my relationship with others.
12 hiding in the disorder protects her	12/279-281	When I am closed inside the disorder I do not care much what others say and how bad my relationship will be. It definitely has to do with my difficulty in forming relationships.
Non-physical abuse and ED onset		
5 anorexia could stop bullying	5/119-121	You see, anorexia started because I was mad at myself for being overweight. I got angry at myself and I thought bullying was my fault because I was indeed fat. In my mind, if I could lose weight and become thin

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		and pretty bullying would be over.
5 losing weight would improve self-esteem	5/122-124	I thought that by losing weight my self-esteem would dramatically improve and people do not bully people with high self-esteem. That was the solution in my mind. It was an unhealthy way to deal with it of course.
10 abuse not being her fault created bulimia	10/229-233	<i>(asked if the realisation that abuse was not her fault changed the ED symptoms)</i> - I think so but I cannot say exactly how it was done. However, when I started to think differently and to know that it was not my fault, then bulimia came along.
10 if there was no abuse there would be no ED	10/247-248	I think if I did not have these experiences (bullying) it (ED) might not have happened. I wouldn't need to lose weight in order to be accepted and become obsessed with it.
11 low self-esteem important contributor to ED	11/266-267	<i>(asked which was the most contributing and maintaining factor of ED)</i> - (Silence) It is a combination of social norms and bullying that lowered my self-esteem. In order to have self-esteem I had to lose weight.
DEVELOPMENTAL FACTORS AND NON-PHYSICAL ABUSE		
Self-esteem		
4 very low self-esteem	4/77	Very low self-esteem..I was self-whipping endlessly, that is, I started dieting
6 self-esteem is visible	6/126-127	You know...self-esteem is visible. Others can see if you

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		have it or not. And they treat you accordingly
6 low self-esteem makes you an easy target	6/127	Low self-esteem equals an easy target. An easy victim. Weakness
6 anorexia can improve self-esteem	6/128-130	Anorexia was the easy way towards a better self-esteem. It's not that I woke up one day and thought to myself "let's have anorexia"...no. I started dieting like crazy and at some point I lost control.
6 low self-esteem created an unstable self	6/143	In general, my self-esteem is not good. It created instability in how I see myself
6 self-esteem linked to weight	6/144-146	I have compulsions with weighing myself and I think my self-esteem is related to the outcome. That is, I try to stay at the weight I am now. Not to lose control and become someone else again (fat). But with self-esteem I have issues.
8 worked in a bar to elevate self-esteem	8/179-180	I took a long break (from studies) when I lost weight to go to work in a bar because I thought it would boost my self-esteem
11 connecting self-esteem to mother's image	11/267-271	I did not have self-esteem from my home. My mother did not have self-esteem and so I probably learned not to have one either. She did not care if she looked beautiful. She was very withdrawn and closed to herself. I had no idea how a beautiful woman looks like. I did not have an image as a guide to create my own one.
12 self-esteem stays forever	12/275-276	This image for yourself and your weight follows you forever. To put it correctly, your self-esteem follows you

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		forever
Attachment		
3 always involved in bossy relationships	3/59-60	...but I always got involved into such relationships that I was the stepping stone..the boxing bag and it probably has to do with my personality
7 unstable relationships	7/151-152	I generally have an instability in my relationships. I find people who have issues and they are bossy.
7 afraid to attach	7/154	Now I am social but I avoid close relationships. I do not attach, I am afraid to attach
7 not being herself in relationships	7/156-158	<i>(asked what she is afraid of)</i> The fact that I do not expect understanding. In the relationships I had I was trying to keep a different face -I was not open to the problems I have with food - I was very ashamed and I was trying to cover it all up and I was not myself.
10 cannot tolerate rejection	10/249	I am generally sensitive to rejection. I cannot stand it at all.
Emotion regulation		
4 emotional and self-regulation difficulties	4/78-79	I had various emotional and self-regulation difficulties.
4 joined angry groups to tolerate anger	4/84-86	I had gotten into such cliques that everyone was angry and delinquent — even though I was a good student. I felt like we had something in common so I vented it (anger) a bit like that.
8 suppressing anger with food in childhood	8/197-198	I was eating. I suppressed my anger with food. I was

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		told that I was fat and I ate to feel better. When I was younger, I did not realise that I had the right to be angry.
9 anger not verbally expressed	9/200	<i>(asked if she expressed anger)</i> - Not verbally. I was the good kid and I didn't talk.
9 self-injury to relief anger	9/200-201	A couple of times I had put out cigarettes on myself
9 delinquent behaviour to relief anger	9/201-203	Another time I had taken a key and gone through all the cars in the neighborhood to damage them. I had suppressed anger and so I took it out like that.
9 punishing herself through bulimic episodes	9/205-207	The whole eating problem had to do with anger because I punish myself with bulimic episodes. And when I eat more I hit my hand on the wall because I am angry that I ate.
9 turning anger at herself created anorexia	9/207-209	When I started with anorexia I blamed myself for everything. I believed that in order to deserve better behaviour I had to be thin. The whole phase of anorexia was as if I had turned my anger on myself. I did not feel angry with others then.
9 turning anger at others created bulimia	9/209-211	Now with bulimia I understand how angry I am with others. I understood that with psychotherapy.
9 throwing things when angry	9/211	And now with the slightest reason I get very angry and throw things.
9 afraid of direct conflict	9/212	To come into direct conflict with someone... I have not done it. I'm afraid of conflict
9 learned not to argue	9/214-216	This is how I learned from my mother from a young

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		age. That it is important not to argue and not to have tensions. That the purpose in this life is not to argue. Now I know this is not right but I find it very difficult to argue with someone.
9 arguments make her nervous	9/216-217	I have difficulty with tensions. Even when I see others arguing, I get very nervous.
9 with bulimia she took her anger out	9/220-221	In bulimia I started to think that I was not to blame for the bullying and I started to take out my anger.
9 changing her anger direction confused her	9/221-224	But this made me very confused because I thought I had organised things in my head. Meaning that I am to blame for everything and I should not eat. Then I realised that there were people who misbehaved and I did not know where to turn my anger.

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Appendix 34 – Table of master themes, superordinate themes and emergent themes of Amanda’s interview

Themes	Page/Line	Key words
NON-PHYSICAL ABUSE IN A CONTINUUM		
Non-physical abuse in childhood		
1 she was always the “fat one”	1/4-6	As a child I was obese. I had a lot of weight and that was something that made me exposed to the comments of others. Comments on my appearance. I spent my whole life being the "fat one".
1 Comments on body and weight from a young age	1/8-10	From a very young age I remember people commenting on my weight and my body. Kids at school used to call me fat, barrel, things like that. And my teachers were telling me that I had to lose weight.
1 other kids didn’t want her	1/11-13	The children did not invite me to their parties. They had also made a connection that whoever is fat is also a fool. That fat goes to the brain..something like that.
1 Parents told her not to eat so much	1/15	They (parents) were telling me not to eat that much
1 parents not supportive and emotionally distant	1/33-38	My father was not present most of the time because of work. My mother did not work and was at home but she was distant. She did not show her feelings. She was not tender. While she was taking care of my food, she did nothing else. I mean emotionally. She was not a person who hugged and

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		showed tenderness. I could not say anything to her when I was sad because I was afraid I would upset her. It is not that she did not love me but as a person he was like that. Her character.
2 Parents though she was weak and ate a lot	2/40-41	She (her mother) was telling me not to eat a lot and be a good student. She thought I have a weak character. And my father was telling me the same thing.
Non-physical abuse in adulthood		
3 Partners told her to lose weight	3/91-92	I was constantly told (by partners) to lose weight because I was fat. I was upset about that.
3 Cheating partner blamed her appearance	3/92-93	I realised that someone cheated on me and he told me that if I was beautiful he would not need to find someone else.
3 Partners viewed her as weak	3/94-96	The two partners I had treated me as a weak person. They were somewhat authoritarian. I felt that I had no opinion or if I had one it would be wrong. Something like that... they made me feel inferior.
4 Husband tells her to lose weight	4/103	He does not insult me for my appearance but he tells me to lose weight
4 Husband is emotionally distant	4/104-106	He is as distant like my mother. That bothers me. He does not display emotions. He is not tender. To give you an example, I did not see him crying when his mother, with whom he was very

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		close, died.
Non-physical abuse consequences		
2 She didn't have friends	2/44-47	I was generally distant. I was not social. I was not trying to make friends. At school I did not want to participate in group games. I could not fit in. I thought that others did not want me in the company. That if I tried to join them they would reject me.
6 Tries not to make the same mistakes with her daughter	6/194-195	I try to be tender with my daughter. So not to not make the mistakes that my mother made to me
Feelings for non-physical abuse		
1 Ashamed to go out	1/10-11	It was very embarrassing. I was ashamed to go to school. From one point on I remember being ashamed to go out in general
2 She felt alone	2/65	I felt alone
3 Very low self-esteem and anger	3/76	Very low self-esteem and anger. I was angry at myself for being fat
3 Anger for being ugly		Anger. A lot of anger. At myself for being ugly and at my parents who did nothing to improve my appearance
3 Angry at everyone and at her weak self	3/89	I think at just about everyone. Certainly at me for being so weak.
4 She wishes her husband was different	4/108	I wish he was different
4 She feels neglected from husband	4/109-110	I feel neglected even though I think he loves me
4 Always felt neglected and lonely	4/114	I always felt neglected. I always felt alone.
Dealing with non-physical abuse		

NON-PHYSICAL ABUSE AND EATING DISORDERS

2 Kept thoughts and feelings inside	2/66	Anything I was thinking and made me feel sad I kept it inside me
2 Food was her company	2/67-68	My company was food. When I ate something I liked I felt good. I always had food in my mind. All I was thinking about was food.
3 She did not react to the abuse	3/72-73	I did not speak because I thought they were right. I was indeed fat so what could I say?
3 Tried to have good grades	3/73-74	I was trying to have good grades so as not to be called stupid.
3 She was trying to lose weight	3/93-94	I always tried to lose weight but I could not succeed.
3 used food as a way out	3/96-97	I was finding a way out in food and which did not help because I continued to be fat
4 Eating a lot as a reaction both as a minor and adult	4/116	And then I ate a lot and now I eat a lot
5 she needs to change	5/146-147	Only if I change and be able to defend my self
5 psychotherapy helped	5/149	It helped me realise many thing about myself and my relationship with food
Explaining non-physical abuse		
2 She thought abuse was her fault	2/65-66	I thought I was to blame for everything. Because I am a weak character.
4 Overweight kids are abused	4/135-136	I think this happens to children who are obese. Others make fun of them. It's not right but it happens
4 Her fault for not reacting	4/136-137	It is also my fault that I was not able to answer. If I had

NON-PHYSICAL ABUSE AND EATING DISORDERS

		answered they might not have done it again.
5 Her parents' fault	5/137-138	It is also my parents' fault that they did not teach me to speak and to be strong.
5 Abused now because she is still fat and weak	5/142	Because I am still fat and I look weak
5 Abused because of low self-esteem	5/142-144	Because I still do not have self-esteem. When you have self-esteem you look strong and when you look strong others do not comment on you. They do not insult you
NON-PHYSICAL ABUSE AND ED		
Non-physical abuse and current ED		
5 Ongoing abuse relates to current ED symptoms	5/163	<i>(asked if current ED symptoms are related to ongoing abuse) Yes</i>
5 Eating when sad and angry	5/163-166	Since I continue to eat when I am sad, when something has bothered me, when I feel angry at myself for not being strong, when I feel angry because I found a partner who looks like my mother and I do not get what I want... then it (<i>abuse to current ED symptoms</i>) is related.
6 Eating when her manager makes her remarks	6/168-169	When I do not do something right at work and my manager is telling me off I go to the bakery to get something to eat even though I am not hungry
6 Eating when she feels neglected	6/171-173	When my husband is emotionally distant and I want tenderness at that time, I cook something fatty that I

NON-PHYSICAL ABUSE AND EATING DISORDERS

		like and I eat watching TV. Like nurturing about myself with food. So is it not related? I think it is. (<i>abuse and ED symptoms</i>)
Non-physical abuse and ED onset		
1 Always fatty food in the house/learned behaviour	1/15-16	It's not my fault that I ate a lot. At home we always had sweet and fatty foods.
1 eating model from her mother	1/16-17; 2/41-42	My mother ate like that (a lot). That was what I had learned. I had this pattern. But that is not the only reason (of the ED onset). ; My mother also gained weight and often went on diets without success.
1 Her mother was telling her she had to eat more	1/19-22	When I was very young, 4-5 years old, I remember my mother trying to feed me and there was this view at home that I was not eating. And I was seeing her being upset that I was not eating and she was trying to find ways to feed me. At one point and then I started eating a lot and I could not stop
1 Her mother telling her she didn't eat probably not true	1/24-27	But from my photos at this age of kindergarten it seems that I was not thin. So I realised that my mother for some reason thought I was not eating. If I did not eat as I was told, I would have to be very thin in the photos.
1 She started eating a lot to please her mother	1/27-29	I started eating so she (her mother) wouldn't be upset. I had heard her tell my aunt that I was a difficult child. When I started eating I could not stop.

NON-PHYSICAL ABUSE AND EATING DISORDERS

5 Abuse relates to ED onset	5/150-152	<i>(asked if she believes her experiences relate to ED onset). Yes for sure.</i>
5 Negative childhood experiences made her focus on food	5/154-155	If I had not lived unpleasant experiences in childhood I might not have focused so much on food. I would have not lost control
5 If she had self-esteem from parents she wouldn't need food	5/157-160	If my parents had given me self-esteem I would not need to find comfort in food. I would be a different person. Strong, social. My whole life would not revolve around food. I do not know if I'm right but that's how I feel.
6 Started eating to gain her mother's acceptance and tenderness		I think I started eating so my mother would be happy and satisfied with me. I thought that if I pleased her she would more tender with me. That I would gain her acceptance. That she would show me that she loves me. I think that is the answer. In the acceptance and tenderness I needed from my mother.
6 Relationship with mother is important	6/191-192	Many things that happen later in life are rooted in our relationship with mom. That is what I believe
DEVELOPMENTAL FACTORS AND NON-PHYSICAL ABUSE		
Self-esteem		
2 Parents told her she should be good at everything she does	2/47-49	I was also taught at home that I must be good at everything. In behaviour, in lessons. Generally good at everything. This stressed me.

NON-PHYSICAL ABUSE AND EATING DISORDERS

		Maybe I was afraid I would not be good at everything
2 She never had self-esteem	2/51-52	I never had self-esteem. Never. I lost years of my life not having self-esteem. Not only then but now as well
2 She thought she was not good enough	2/54-56	I thought I would do something wrong and they would make fun of me. That I will not succeed. That I was not beautiful. That I was not good enough for team games. That I was not good enough in general...
2 Not comfortable around people	2/56-58	I never felt comfortable being in a group of more than 2 people. I did not feel comfortable talking. Maybe I thought I would say something wrong and make fun of me
2 Low self-esteem never changes	2/60-63	I do not know..it is very bad not to have self-esteem. It determines all your life. What will you do in your life.. Who will you be... parents should make sure their children will have self-esteem. If you didn't have self-esteem as a child you will never have. This is what I have understood. You will carry this burden throughout your life.
3 When you are not pretty it's easy to be dumbed	3/100-101	When you are not beautiful it is very easy to be dumbed. And it's not easy to find someone else afterwards.
4 She doesn't have self-esteem in her job	4/120-121	And in my job I do not have self-esteem. I only do what I am told and I am afraid to take the initiative not to make a mistake.

NON-PHYSICAL ABUSE AND EATING DISORDERS

4 She is afraid to live due to low self-esteem	4/122-123	It's like I'm afraid to live. I do not know how else to describe it. I do not believe that self-esteem ever changes.
4 self-esteem protects from being hurt	4/130-131	When you feel good about yourself you will not be affected others' behaviour and you will not be hurt.
6 If she had self-esteem she wouldn't rely on food	6/174-175	If I was strong and I had self-esteem would I need to eat to feel relieved?
Attachment		
4 avoiding close relationships	4/127	I don't engage in close relationships. I am afraid of rejection
4 good self-esteem would help attachment	4/127-128	If I had a good self-esteem I wouldn't afraid to attach. Now I don't have friends
4 Being shot from close distance will hurt a lot	4/131-133	When someone says something bad to me, it's like eating a bullet. I hurt. If they shoot you from a close distance you will hurt more while from a long distance it will not hurt so much.
Emotion regulation		
3 She didn't express anger	3/-82-83	No. I had not learned to express it. At home we did not express emotions. I do not know why. Anyway, that's how I have learned
3 Her anger was relieved through eating	3/83-85	I relieved my anger through eating. That's all I remember to tell you. It's like so much food has blocked my memories! (laughs).
3 Conversations about emotions bring tension	3/99-100	(asked if she talked to partners) No. I do not want

NON-PHYSICAL ABUSE AND EATING DISORDERS

		tensions and yelling. Conversations about emotions bring tension. We may have split up
4 swallowing emotions with food	4/116-117	I swallow emotions with food. I am not strong enough to lose weight.

Appendix 35 – Greek translated version of CTQ

Childhood Trauma Questionnaire (CTQ)

Οδηγίες: Οι ερωτήσεις αφορούν κάποιες από τις εμπειρίες που μπορεί να είχατε μεγαλώνοντας ως παιδί και έφηβος/η. Για κάθε ερώτηση βάλτε σε κύκλο τον αριθμό που περιγράφει καλύτερα τη συχνότητα της κάθε δήλωσης: «*Ποτέ αληθές*» σημαίνει ότι ποτέ δεν συνέβη αυτό που αναφέρει δήλωση μέχρι το «*πολύ συχνά αληθές*» όταν η δήλωση συνέβαινε πολύ συχνά. Κάποιες δηλώσεις αναφέρονται συγκεκριμένα στο οικογενειακό περιβάλλον (συμπεριλαμβάνονται και τα αδέρφια & όποιος άλλος ζούσε μαζί σας στο σπίτι πχ γιαγιά, παππούς) και κάποιες άλλες είναι γενικές (πχ. πιστεύω ότι κακοποιήθηκα συναισθηματικά). Όταν δεν αναφέρει η δήλωση την οικογένεια σημαίνει ότι αυτό που περιγράφει η δήλωση μπορεί να συνέβη από οποιονδήποτε εντός ή εκτός οικογένειας. Παρόλο κάποιες από αυτές τις ερωτήσεις έχουν προσωπικό χαρακτήρα, παρακαλώ προσπαθήστε να απαντήσετε όσο πιο ειλικρινά μπορείτε. Οι απαντήσεις σας θα είναι απόρρητες.

Όταν μεγάλωνα,...	Ποτέ αληθές	Σπανίως αληθές	Μερικές φορές αληθές	Συχνά αληθές	Πολύ συχνά αληθές
1. Δεν είχα αρκετό φαγητό (δεν είχε μαγειρέψει κάποιος ή ήταν λιγότερο από ότι θα έπρεπε για να φτάσει για όλους).	1	2	3	4	5
2. Ήξερα ότι υπήρχε κάποιος να με φροντίζει και να με προστατεύει.	1	2	3	4	5
3. Μέλη της οικογένειάς μου με αποκάλεσαν «χαζό/ή» «τεμπέλη/α» ή «άσχημο/η» (οποιοσδήποτε στην οικογένεια).	1	2	3	4	5
4. Οι γονείς μου ήταν πολύ μεθυσμένοι ή «φτιαγμένοι (ουσίες)» για να φροντίσουν την οικογένεια.	1	2	3	4	5
5. Υπήρχε κάποιος στην οικογένειά μου που με βοήθησε να νιώσω σημαντικός/ή ή ξεχωριστός/ή.	1	2	3	4	5

NON-PHYSICAL ABUSE AND EATING DISORDERS

Όταν μεγάλωνα,...	Ποτέ αληθές	Σπανίως αληθές	Μερικές φορές αληθές	Συχνά αληθές	Πολύ συχνά αληθές
6. Έπρεπε να φοράω βρώμικα ρούχα (αναγκαζόμουν γιατί δεν έβρισκα πλυμένα/σιδερωμένα).	1	2	3	4	5
7. Ένωθα αγαπητός/ή.	1	2	3	4	5
8. Σκέφτηκα ότι οι γονείς μου ευχήθηκαν να μην είχα γεννηθεί ποτέ.	1	2	3	4	5
9. Χτυπήθηκα τόσο δυνατά από κάποιον στην οικογένειά μου που έπρεπε να δω γιατρό ή να πάω στο νοσοκομείο.	1	2	3	4	5
10. Δεν υπήρχε τίποτα που να ήθελα να αλλάξω σχετικά με την οικογένειά μου.	1	2	3	4	5
11. Μέλη της οικογένειάς μου με χτύπησαν τόσο δυνατά που μου άφησαν μελανιές ή σημάδια (οποιοσδήποτε στην οικογένεια μπορεί να το έκανε).	1	2	3	4	5
12. Τιμωρήθηκα με μία ζώνη, μία βέργα, ένα σχοινί ή κάποιο άλλο σκληρό αντικείμενο.	1	2	3	4	5
13. Τα μέλη στην οικογένειά μου πρόσεχαν ο ένας τον άλλον.	1	2	3	4	5
14. Μέλη της οικογένειάς μου, μου είπαν πληγωτικά ή προσβλητικά πράγματα.	1	2	3	4	5

NON-PHYSICAL ABUSE AND EATING DISORDERS

Όταν μεγάλωνα,...	Ποτέ αληθές	Σπανίως αληθές	Μερικές φορές αληθές	Συχνά αληθές	Πολύ συχνά αληθές
15. Πιστεύω ότι κακοποιήθηκα σωματικά.	1	2	3	4	5
16. Είχα τα τέλεια παιδικά χρόνια.	1	2	3	4	5
17. Χτυπήθηκα ή δάρθηκα τόσο άσχημα που το παρατήρησε κάποιος όπως δάσκαλος, γείτονας ή γιατρός.	1	2	3	4	5
18. Ένωσα ότι κάποιος στην οικογένειά μου με μισούσε.	1	2	3	4	5
19. Τα μέλη της οικογένειάς μου ένωθαν κοντά ο ένας στον άλλον.	1	2	3	4	5
20. Κάποιος προσπάθησε να με αγγίξει με σεξουαλικό τρόπο ή προσπάθησε να με κάνει να τον/την αγγίξω.	1	2	3	4	5
21. Κάποιος απείλησε να με βλάψει ή να πει ψέματα για μένα εκτός αν έκανα κάτι σεξουαλικό μαζί του/της.	1	2	3	4	5
22. Είχα την καλύτερη οικογένεια στον κόσμο.	1	2	3	4	5
23. Κάποιος προσπάθησε να με αναγκάσει να κάνω σεξουαλικά πράγματα (οτιδήποτε σεξουαλικής φύσης) ή να τα παρακολουθήσω.	1	2	3	4	5
24. Κάποιος με κακοποίησε σεξουαλικά.	1	2	3	4	5

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Όταν μεγάλωνα,...	Ποτέ αληθές	Σπανίως αληθές	Μερικές φορές αληθές	Συχνά αληθές	Πολύ συχνά αληθές
25. Πιστεύω ότι κακοποιήθηκα συναισθηματικά.	1	2	3	4	5
26. Υπήρχε κάποιος να με πάει στον γιατρό αν το χρειαζόμουν.	1	2	3	4	5
27. Πιστεύω ότι κακοποιήθηκα σεξουαλικά.	1	2	3	4	5
28. Η οικογένειά μου ήταν πηγή δύναμης και υποστήριξης.	1	2	3	4	5

Appendix 36 – Greek translated version of DERS

Κλίμακα Αυτορρύθμισης Συναισθημάτων

Παρακαλείστε να αναφέρετε πόσο συχνά ισχύουν οι ακόλουθες δηλώσεις για εσάς γράφοντας τον κατάλληλο αριθμό από την παρακάτω κλίμακα στη γραμμή δίπλα από κάθε ερώτηση.

1-----2-----3-----4-----5

Σχεδόν ποτέ Μερικές φορές Συνήθως Τις περισσότερες φορές Σχεδόν πάντα
(0-10%) (11-35%) (36-65%) (66-90%) (91-100%)

- _____ 1) Τα συναισθήματά μου, μου είναι ξεκάθαρα.
- _____ 2) Δίνω προσοχή στο πώς αισθάνομαι (παρατηρώ προσεκτικά πώς νιώθω).
- _____ 3) Βιώνω τα συναισθήματά μου ως συντριπτικά (με κατακλύζουν/δεν μπορώ να τα διαχειριστώ) και ανεξέλεγκτα.
- _____ 4) Δεν έχω ιδέα για το πώς αισθάνομαι.
- _____ 5) Έχω δυσκολία να βγάλω νόημα από τα συναισθήματά μου.
- _____ 6) Δίνω ιδιαίτερη προσοχή στα συναισθήματά μου.
- _____ 7) Ξέρω ακριβώς πώς αισθάνομαι.
- _____ 8) Νοιάζομαι για το τί αισθάνομαι.
- _____ 9) Είμαι μπερδεμένος/η σχετικά με το πώς αισθάνομαι.

NON-PHYSICAL ABUSE AND EATING DISORDERS

1-----2-----3-----4-----5

Σχεδόν ποτέ Μερικές φορές Συνήθως Τις περισσότερες φορές Σχεδόν πάντα
(0-10%) (11-35%) (36-65%) (66-90%) (91-100%)

- _____ 10) Όταν είμαι αναστατωμένος/η, αναγνωρίζω τα συναισθήματά μου.
- _____ 11) Όταν είμαι αναστατωμένος/η, θυμώνω με τον εαυτό μου που αισθάνομαι έτσι.
- _____ 12) Όταν είμαι αναστατωμένος/η, έρχομαι σε αμηχανία που αισθάνομαι έτσι.
- _____ 13) Όταν είμαι αναστατωμένος/η, δυσκολεύομαι να ολοκληρώσω μια δουλειά.
- _____ 14) Όταν είμαι αναστατωμένος/η, βγαίνω εκτός ελέγχου.
- _____ 15) Όταν είμαι αναστατωμένος/η, πιστεύω ότι θα παραμείνω έτσι για πολύ καιρό.
- _____ 16) Όταν είμαι αναστατωμένος/η, πιστεύω ότι θα καταλήξω να αισθάνομαι πολύ θλιμμένος/η.
- _____ 17) Όταν είμαι αναστατωμένος/η, πιστεύω ότι τα συναισθήματά μου είναι βάσιμα (είναι λογικό που αισθάνομαι έτσι) και σημαντικά.
- _____ 18) Όταν είμαι αναστατωμένος/η, έχω δυσκολία να επικεντρωθώ σε άλλα πράγματα.
- _____ 19) Όταν είμαι αναστατωμένος/η, αισθάνομαι ότι είμαι εκτός ελέγχου.
- _____ 20) Όταν είμαι αναστατωμένος/η, μπορώ να φέρω σε πέρας τις δουλειές μου.

NON-PHYSICAL ABUSE AND EATING DISORDERS

1-----2-----3-----4-----5

Σχεδόν ποτέ Μερικές φορές Συνήθως Τις περισσότερες φορές Σχεδόν πάντα
(0-10%) (11-35%) (36-65%) (66-90%) (91-100%)

_____ 21) Όταν είμαι αναστατωμένος/η, αισθάνομαι ντροπή για τον εαυτό μου που νιώθω έτσι.

_____ 22) Όταν είμαι αναστατωμένος/η, ξέρω ότι μπορώ να βρω έναν τρόπο για να αισθανθώ καλύτερα τελικά.

_____ 23) Όταν είμαι αναστατωμένος/η, νιώθω σαν να είμαι αδύναμος/η.

_____ 24) Όταν είμαι αναστατωμένος/η, νιώθω ότι μπορώ να διατηρήσω τον έλεγχο της συμπεριφοράς μου.

_____ 25) Όταν είμαι αναστατωμένος/η, αισθάνομαι ενοχές που νιώθω έτσι.

_____ 26) Όταν είμαι αναστατωμένος/η, έχω δυσκολία στη συγκέντρωση.

_____ 27) Όταν είμαι αναστατωμένος/η, έχω δυσκολία στο να ελέγξω τη συμπεριφορά μου.

_____ 28) Όταν είμαι αναστατωμένος/η, πιστεύω ότι δεν υπάρχει τίποτα που μπορώ να κάνω για να αισθανθώ καλύτερα.

_____ 29) Όταν είμαι αναστατωμένος/η, εκνευρίζομαι με τον εαυτό μου που νιώθω έτσι.

_____ 30) Όταν είμαι αναστατωμένος/η, αρχίζω να αισθάνομαι πολύ άσχημα για τον εαυτό μου.

_____ 31) Όταν είμαι αναστατωμένος/η, πιστεύω ότι το να παραμένω «κολλημένος/η» σε αυτό το συναίσθημα είναι το μόνο που μπορώ να κάνω.

NON-PHYSICAL ABUSE AND EATING DISORDERS

1-----2-----3-----4-----5

Σχεδόν ποτέ Μερικές φορές Συνήθως Τις περισσότερες φορές Σχεδόν πάντα
(0-10%) (11-35%) (36-65%) (66-90%) (91-100%)

_____ 32) Όταν είμαι αναστατωμένος/η, χάνω τον έλεγχο της συμπεριφοράς μου.

_____ 33) Όταν είμαι αναστατωμένος/η, έχω δυσκολία να σκεφτώ οτιδήποτε άλλο.

_____ 34) Όταν είμαι αναστατωμένος/η, παίρνω τον χρόνο μου για να καταλάβω τί αληθινά αισθάνομαι.

_____ 35) Όταν είμαι αναστατωμένος/η, μου παίρνει πολύ χρόνο για να αισθανθώ καλύτερα.

_____ 36) Όταν είμαι αναστατωμένος/η, τα συναισθήματά μου μοιάζουν συντριπτικά (με κατακλύζουν/δεν μπορώ να τα διαχειριστώ).

Δημιουργήθηκε από την Kim L. Gratz, PhD στις Η.Π.Α. Copyright 2004.

Προσαρμογή στην Ελληνική γλώσσα 2015: Όλγα Θεοδωροπούλου μετά από άδεια της Kim L. Gratz.

Appendix 37 – Greek translated version of ATQ

Παρακαλείσθε να παρέχετε τις ακόλουθες πληροφορίες τσεκάροντας την κατάλληλη απάντηση και συμπληρώνοντας το κενό.

Φύλο: Άνδρας _____ Γυναίκα _____

Είναι η Ελληνική γλώσσα η μητρική σας γλώσσα? Ναι _____ Όχι _____

Ηλικία: _____ **Τελευταία ολοκληρωμένη βαθμίδα εκπαίδευσης:** _____

Χώρα καταγωγής: _____

ΕΡΩΤΗΜΑΤΟΛΟΓΙΟ ΙΔΙΟΣΥΓΚΡΑΣΙΑΣ ΕΝΗΛΙΚΩΝ (ΕΚΔΟΣΗ 1.3)

Οδηγίες

Στις επόμενες σελίδες θα βρείτε μια σειρά από δηλώσεις που οι άνθρωποι μπορούν να χρησιμοποιήσουν για να περιγράψουν τον εαυτό τους. Δεν υπάρχουν σωστές ή λάθος απαντήσεις. Όλοι οι άνθρωποι είναι μοναδικοί και διαφορετικοί, και είναι αυτές οι διαφορές για τις οποίες προσπαθούμε να μάθουμε περισσότερες πληροφορίες. Παρακαλώ διαβάστε προσεκτικά κάθε δήλωση και δώστε την καλύτερή σας εκτίμηση για το πόσο καλά σας περιγράφει. Σημειώστε δίπλα από κάθε δήλωση τον κατάλληλο αριθμό από τις παρακάτω επιλογές για να υποδείξετε πόσο καλά σας περιγράφει η κάθε δήλωση.

Βάλτε το νούμερο:

Αν η δήλωση είναι:

- | | |
|---|----------------------------------|
| 1 | εξαιρετικά ψευδής για εσάς |
| 2 | αρκετά ψευδής για εσάς |
| 3 | ελάχιστα ψευδής για εσάς |
| 4 | ούτε αληθής ούτε ψευδής για εσάς |
| 5 | ελάχιστα αληθής για εσάς |
| 6 | αρκετά αληθής για εσάς |
| 7 | εξαιρετικά αληθής για εσάς |

Αν μια δήλωση δεν ισχύει για εσάς (για παράδειγμα, αν αναφέρεται σε οδήγηση αυτοκινήτου και εσείς δεν οδηγείτε), τότε βάλτε το “X” (δεν ισχύει για εσάς). Η απάντηση X δεν σημαίνει ότι δεν σας συμβαίνει καθόλου αυτό που διαβάζετε. Στην περίπτωση που δεν σας συμβαίνει ποτέ αυτό που λέει η πρόταση, βάζετε 1 σύμφωνα με τις παραπάνω οδηγίες.

Ελέγξτε για να βεβαιωθείτε ότι έχετε απαντήσει για κάθε δήλωση.

NON-PHYSICAL ABUSE AND EATING DISORDERS

Το ερωτηματολόγιο έχει μεταφραστεί από την Όλγα Θεοδωροπούλου (2015) μετά από άδεια χρήσης & μετάφρασης από το Mary Rothbart Lab, Bowdoin College, Brunswick, Maine, USA

- ___ 1) Τρομάζω εύκολα.
- ___ 2) Συχνά αργώ στα ραντεβού.
- ___ 3) Μερικές φορές μικρά γεγονότα με κάνουν να αισθάνομαι έντονη ευτυχία.
- ___ 4) Θεωρώ τους δυνατούς θορύβους πολύ εκνευριστικούς.
- ___ 5) Μου είναι συχνά δύσκολο να εναλλάσσω την προσοχή μου ανάμεσα δύο διαφορετικές εργασίες.
- ___ 6) Σπανίως αγανακτώ όταν πρέπει να περιμένω σε μια σειρά/ουρά που κινείται αργά.
- ___ 7) Δεν θα απολάμβανα την αίσθηση του να ακούω δυνατά μουσική σε ένα σόου με φώτα λείζερ.
- ___ 8) Συχνά κάνω σχέδια που δεν ολοκληρώνω την εφαρμογή τους.
- ___ 9) Σπανίως νιώθω λυπημένος/η όταν αποχαιρετώ φίλους ή συγγενείς.
- ___ 10) Σπανίως τραβούν την προσοχή μου οπτικές λεπτομέρειες που μετά βίας φαίνονται (δεν παρατηρώ ιδιαίτερα μικρές λεπτομέρειες στο χώρο γύρω μου).
- ___ 11) Ακόμη κι όταν αισθάνομαι μεγάλη ενέργεια, μπορώ να καθίσω ακίνητος/η χωρίς ιδιαίτερη προσπάθεια, αν αυτό είναι απαραίτητο.
- ___ 12) Κοιτάζοντας προς τα κάτω στο έδαφος από ένα εξαιρετικά ψηλό μέρος θα με κάνει να νιώσω άβολα.
- ___ 13) Όταν ακούω μουσική, συνήθως αντιλαμβάνομαι τις λεπτές συναισθηματικές αποχρώσεις (παρατηρώ τις πτυχές των μουσικών κομματιών που μεταφέρουν μικρές αλλαγές στο συναίσθημα).
- ___ 14) Δεν θα απολάμβανα να κάνω μια δουλειά που θα περιελάμβανε την εξυπηρέτηση κοινού (πχ σε τηλεφωνικό κέντρο, γκισέ τράπεζας κλπ).
- ___ 15) Μπορώ να εκτελέσω μια εργασία ακόμη κι αν θα προτιμούσα να μην το κάνω.
- ___ 16) Μερικές φορές δεν φαίνεται να είμαι ικανός/ή να νιώσω ευχαρίστηση από γεγονότα και δραστηριότητες που κανονικά θα έπρεπε να απολαμβάνω.
- ___ 17) Το βρίσκω πολύ ενοχλητικό όταν ένα κατάστημα δεν έχει στοκ από ένα προϊόν που θέλω να αγοράσω (πηγαίνω στο κατάστημα και μου λένε ότι τελείωσε).

NON-PHYSICAL ABUSE AND EATING DISORDERS

___ 18) Έχω την τάση να παρατηρώ συναισθηματικές πτυχές σε πίνακες ζωγραφικής και φωτογραφίες.

___ 19) Συνήθως μου αρέσει να μιλάω πολύ.

___ 20) Σπανίως λυπάμαι όταν παρακολουθώ μια λυπητερή ταινία.

___ 21) Έχω συχνά επίγνωση των ήχων των πουλιών στη γειτονιά μου.

___ 22) Όταν είμαι μέσα σε μικρούς χώρους, όπως ασανσέρ, αισθάνομαι νευρικότητα.

___ 23) Όταν ακούω μουσική, συνήθως μου αρέσει να αυξάνω την ένταση περισσότερο από τους άλλους ανθρώπους.

___ 24) Μερικές φορές φαίνεται να καταλαβαίνω τα πράγματα διαισθητικά.

___ 25) Μερικές φορές ασήμαντα γεγονότα μου προκαλούν έντονη θλίψη.

___ 26) Είναι εύκολο για μένα να συγκρατήσω το γέλιο μου σε μια κατάσταση όπου το γέλιο δεν θα ήταν κατάλληλη συμπεριφορά.

___ 27) Μπορώ να κάνω τον εαυτό μου να δουλέψει για ένα δύσκολο έργο, ακόμη και όταν δεν έχω όρεξη ούτε να προσπαθήσω.

___ 28) Σπανίως υπάρχουν μέρες που να μη βιώνω έστω και σύντομες στιγμές έντονης ευτυχίας.

___ 29) Όταν προσπαθώ να εστιάσω την προσοχή μου αποσπώμαι εύκολα.

___ 30) Πιθανόν θα απολάμβανα να παίζω ένα απαιτητικό και γρήγορο σε ρυθμό video game που κάνει πολύ θόρυβο και έχει πολλά έντονα φώτα που αναβοσβήνουν.

___ 31) Κάθε φορά που πρέπει να καθίσω και να περιμένω για κάτι (π.χ. σε μια αίθουσα αναμονής), εκνευρίζομαι.

___ 32) Συχνά ενοχλούμαι από το πολύ έντονο φως.

___ 33) Σπανίως παρατηρώ το χρώμα των ματιών κάποιου.

___ 34) Σπανίως λυπάμαι όταν ακούω για ένα δυσάρεστο γεγονός.

___ 35) Όταν με διακόπτουν ή μου αποσπούν την προσοχή, συνήθως μπορώ εύκολα να ξανασυγκεντρωθώ σε ό, τι έκανα πριν συμβεί αυτό.

___ 36) Βρίσκω μερικούς ήχους τριξίματος πολύ ενοχλητικούς (πχ κιμωλία στον πίνακα, τριβή μετάλλων, λιμάρισμα νυχιών).

___ 37) Μου αρέσουν οι συνομιλίες που περιλαμβάνουν πολλά άτομα.

NON-PHYSICAL ABUSE AND EATING DISORDERS

- ___ 38) Συνήθως είμαι υπομονετικός άνθρωπος.
- ___ 39) Όταν ξεκουράζομαι με κλειστά τα μάτια, μερικές φορές βλέπω οπτικές εικόνες.
- ___ 40) Μου είναι πολύ δύσκολο να συγκεντρωθώ σε κάτι όταν είμαι στενοχωρημένος/η.
- ___ 41) Μερικές φορές το μυαλό μου είναι γεμάτο από ένα ευρύ φάσμα σκέψεων και εικόνων που έχουν ασαφή σύνδεση μεταξύ τους.
- ___ 42) Τα πολύ έντονα χρώματα μερικές φορές με ενοχλούν.
- ___ 43) Μπορώ εύκολα να αντισταθώ στο να πεταχτώ και να μιλήσω χωρίς να είναι η σειρά μου, ακόμη και όταν είμαι ενθουσιασμένος/η και θέλω να εκφράσω μια ιδέα.
- ___ 44) Μάλλον δεν θα απολάμβανα μια γρήγορη και ξέφρενη βόλτα σε ρόδα/τραινάκι του λούνα παρκ.
- ___ 45) Μερικές φορές αισθάνομαι λυπημένος/η για περισσότερο από μία ώρα.
- ___ 46) Σπανίως απολαμβάνω να κοινωνικοποιούμαι με μεγάλες ομάδες ανθρώπων.
- ___ 47) Αν σκεφτώ κάτι που πρέπει να γίνει, συνήθως αρχίζω αμέσως να δουλεύω γι' αυτό.
- ___ 48) Δεν μου παίρνει πολύ να νιώσω ενοχλημένος/η ή εκνευρισμένος/η.
- ___ 49) Δεν χρειάζεται πολύ για να μου προκαλέσει κάποιος μια χαρούμενη αντίδραση.
- ___ 50) Όταν είμαι χαρούμενος/η και ενθουσιασμένος/η για ένα επερχόμενο γεγονός, δυσκολεύομαι να εστιάσω την προσοχή μου σε εργασίες που απαιτούν συγκέντρωση.
- ___ 51) Μερικές φορές έχω μια αίσθηση πανικού ή τρόμου χωρίς κάποιον προφανή λόγο.
- ___ 52) Συχνά παρατηρώ ήπιες μυρωδιές και αρώματα.
- ___ 53) Συνήθως έχω πρόβλημα να αντισταθώ στη λαχτάρα μου για ποτά, φαγητά, κ.λπ.
- ___ 54) Τα πολύχρωμα φώτα που αναβοσβήνουν με ενοχλούν (πχ διαφημιστικές πινακίδες, έντονα φώτα που αναβοσβήνουν σε club).
- ___ 55) Συνήθως ολοκληρώνω πράγματα πριν λήξει η προθεσμία τους (πχ πληρωμή λογαριασμών, εργασίες με προθεσμία παράδοσης κλπ).
- ___ 56) Αισθάνομαι συχνά λυπημένος/η.
- ___ 57) Συχνά έχω επίγνωση με ποιο τρόπο το χρώμα και ο φωτισμός ενός δωματίου επηρεάζουν τη διάθεσή μου.
- ___ 58) Συνήθως παραμένω ήρεμος/η χωρίς να ενοχλούμαι όταν τα πράγματα δεν εξελίσσονται ομαλά για μένα.

NON-PHYSICAL ABUSE AND EATING DISORDERS

___ 59) Η δυνατή μουσική μου είναι δυσάρεστη.

___ 60) Όταν είμαι ενθουσιασμένος/η για κάτι, είναι συνήθως δύσκολο για μένα να αντισταθώ στο να βιαστώ να ασχοληθώ με αυτό πριν να έχω εξετάσει τις πιθανές συνέπειες.

___ 61) Οι δυνατοί θόρυβοι μερικές φορές με τρομάζουν.

___ 62) Μερικές φορές ονειρεύομαι ζωνηρά, λεπτομερή τοπία που δεν έχουν καμία σχέση με οτιδήποτε έχω βιώσει όταν είμαι ξύπνιος/α.

___ 63) Όταν βλέπω ένα ελκυστικό αντικείμενο σε ένα μαγαζί, μου είναι συνήθως πολύ δύσκολο να αντισταθώ στην αγορά του.

___ 64) Θα απολάμβανα να παρακολουθήσω ένα σόου με λείζερ με πολλά έντονα πολύχρωμα φώτα που αναβοσβήνουν.

___ 65) Όταν ακούω για ένα δυσάρεστο γεγονός, αμέσως αισθάνομαι λυπημένος/η.

___ 66) Όταν παρακολουθώ μια ταινία, συνήθως δεν προσέχω τον τρόπο με τον οποίο το σκηνικό χρησιμοποιείται για να μεταφέρει τη διάθεση των χαρακτήρων.

___ 67) Συνήθως μου αρέσει να περνώ τον ελεύθερο χρόνο μου με ανθρώπους.

___ 68) Δεν με φοβίζει αν νομίζω ότι είμαι μόνος/η κάπου και ξαφνικά ανακαλύψω ότι κάποιος βρίσκεται κοντά μου (ενώ δεν τον είχα δει).

___ 69) Συχνά έχω συνειδητά επίγνωση για το πώς ο καιρός φαίνεται να επηρεάζει τη διάθεσή μου.

___ 70) Χρειάζονται πολλά για να με κάνει κάποιος να νιώσω αληθινά ευτυχισμένος/η.

___ 71) Σπανίως έχω επίγνωση της υφής των αντικειμένων που κρατάω.

___ 72) Όταν φοβάμαι για το πώς μπορεί να εξελιχθεί μια κατάσταση, συνήθως αποφεύγω να ασχοληθώ.

___ 73) Απολαμβάνω ιδιαίτερα τις συζητήσεις που μπορώ να πω πράγματα χωρίς να τα σκεφτώ πρώτα.

___ 74) Χωρίς να καταβάλω προσπάθεια οι δημιουργικές ιδέες παρουσιάζονται μόνες τους (δεν σκέφτομαι ιδιαίτερα για να μου έρθει μια δημιουργική ιδέα).

___ 75) Όταν προσπαθώ κάτι νέο, σπανίως ανησυχώ για την πιθανότητα να αποτύχω.

___ 76) Μου είναι εύκολο να αναστείλω μια διασκεδαστική συμπεριφορά που θα ήταν ακατάλληλη (πχ να γελάω μέσα σε εκκλησία, βιβλιοθήκη κλπ).

___ 77) Δεν θα απολάμβανα την αίσθηση που μου προκαλείται από το να φώναζα όσο πιο δυνατά μπορώ (δηλαδή την ένταση που προκαλείται στο σώμα όταν φωνάζουμε).

NON-PHYSICAL ABUSE AND EATING DISORDERS

Appendix 38 – Comparison of the means’ of the thesis scales to those of published studies (with a population as similar as possible to the population of the thesis). No available data was found regarding ED patients for: ECRS, WHOQoL-BREF, MNBS, ATQ

Comparison of means (SD) with other published studies for controls

Rosenberg Self-Esteem Scale	Author, Greeks (n = 154)	Garcia (2016), USA, n =25	Group comparison
Self-esteem	20.68 (4.05)	20.92 (1.78)	$t(177) = .29, p = .771$

Eating Disorders Examination Questionnaire	Author, Greeks (n = 154)	Carey et al., 2019, UK (n = 851)	Group comparison
Restraint	1.28 (1.16)	1.37 (1.34)	$t(1003) = .78, p = .434$
Eating concern	.64 (.71)	1.03 (1.11)	$t(1003) = 4.21, p < .001$
Shape concern	1.61 (1.29)	2.51 (1.58)	$t(1003) = 6.68, p < .001$
Weight concern	1.24 (1.20)	2.10 (1.57)	$t(1003) = 6.46, p < .001$
Global score	1.20 (.95)	1.75 (1.25)	$t(1003) = 5.20, p < .001$

Difficulties in emotion regulation scale	Author, Greeks (n = 188)	Gratz & Roemer, 2004, USA (n = 260)	Group comparison
Total score	81.16 (20.33)	77.99 (20.72)	$t(446) = -1.61, p = .108$

Family Assessment Device	Author, Greeks (n = 154)	Mansfield et al., 2015, USA (n = 155)*	Group comparison
Problem solving	2.06 (.36)	2.00 (.36)	$t(307) = -1.47, p = .144$
Communication	2.09 (.37)	2.05 (.38)	$t(307) = -.94, p = .349$
Roles	2.33 (.38)	2.22 (.34)	$t(307) = -2.68, p = .008$
Affective responsiveness	2.17 (.53)	1.97 (.47)	$t(307) = -3.51, p = .001$
Affective involvement	2.03 (.38)	2.02 (.42)	$t(307) = -.22, p = .827$
Behaviour control	2.00 (.30)	1.65 (.30)	$t(307) = -10.25, p < .001$
General functioning	1.92 (.38)	1.79 (.42)	$t(307) = -2.85, p = .005$

*families

NON-PHYSICAL ABUSE AND EATING DISORDERS

Experiences in Close Relationships Structures	Author, Greeks (n = 188)	Moreira et a., 2015, Portuguese (n = 169)	Group comparison
Global anxiety	2.03 (.96)	2.06 (1.04)	$t(355) = .28, p = .777$
Global avoidance	2.82 (.92)	2.27 (.78)	$t(355) = -6.06, p < .001$

Adult Temperament Questionnaire – Short Form	Author, Greeks (n = 188)	Gardner et al., 2011, UK (n = 97)*	Group comparison
Negative affect	4.12 (.65)	4.12 (.70)	$t(283) = 0.00, p = 1.000$
Extraversion surgency	4.56 (.73)	4.74 (.71)	$t(283) = 2.00, p = .048$
Orienting sensitivity	4.61 (.74)	4.63 (.68)	$t(283) = .22, p = .824$
Effortful control	4.38 (.74)	4.26 (.68)	$t(283) = -1.33, p = .184$

*80 females out of the 97 participants

WHOQOL-BREF	Author, Greeks (n = 188)	Chung et al., 2016, Chinese (n = 256)	Group comparison
Psychological health	14.24 (2.04)	14.36 (2.10)	$t(442) = .60, p = .547$
Social relationships	14.39 (3.15)	14.38 (2.04)	$t(442) = -.04, p = .968$

The Multidimensional Neglectful Behaviour Scale Adult Recall Version Short Form	Author, Greeks (n = 188)	Kitano et al., 2018, Japanese (n = 536)*	Group comparison
Multidimensional neglect	11.99 (3.17)	13.1 (3.4)	$t(722) = 3.92, p < .001$

*432 women out of the 536 participants

Childhood Trauma Questionnaire-SF	Author, Greeks (n = 188)	Sacchi et al., 2018, Italian (n = 341)	Group comparison
Emotional abuse	8.81 (3.87)	6.36 (2.60)	$t(527) = -8.67, p < .001$
Emotional neglect	9.11 (3.84)	9.06 (3.90)	$t(527) = -.14, p = .887$
Physical neglect	6.16 (1.58)	5.70 (1.40)	$t(527) = 3.45, p = .001$

NON-PHYSICAL ABUSE AND EATING DISORDERS

Comparison of means (SD) with other published studies for ED Patients

Childhood Trauma Questionnaire	Author, Greeks (n = 80)	Kong & Bernstein, 2009, Korean (n = 73)*	Group comparison
Emotional abuse	13.10 (5.62)	12.25 (4.62)	$t(151) = -1.02, p = .311$
Emotional neglect	12.10 (5.18)	13.88 (4.72)	$t(151) = 2.22, p = .028$
Physical neglect	7.38 (2.79)	10.27 (3.66)	$t(151) = 5.52, p < .001$

* 97.3% females (71 women, 2 men)

Difficulties in emotion regulation scale	Author, Greeks (n = 80)	Wolz et al., 2015, Spanish, (n = 134)	Group comparison
Total score	108.41 (25.80)	107.30 (27.69)	$t(212) = -.29, p = .771$

Family Assessment Device	Author, Greeks (n = 154)	Mansfield et al., 2015, USA (n = 46)*	Group comparison
Problem solving	2.37 (.54)	2.45 (.49)	$t(198) = .90, p = .369$
Communication	2.46 (.61)	2.38 (.43)	$t(198) = -.83, p = .408$
Roles	2.49 (.40)	2.30 (.39)	$t(198) = -2.84, p = .005$
Affective responsiveness	2.52 (.68)	2.30 (.58)	$t(198) = -.99, p = .048$
Affective involvement	2.26 (.52)	2.21 (.44)	$t(198) = -.59, p = .555$
Behaviour control	2.21 (.41)	1.93 (.40)	$t(198) = -4.09, p < .001$
General functioning	2.41 (.64)	2.31 (.49)	$t(198) = -.98, p = .330$

*families with a mental health diagnosis

Rosenberg Self-Esteem Scale	Author (n = 154), Greeks	Raykos et al., 2017, Australian (n = 306)*	Group comparison
Self-esteem	15.00 (5.55)	21.87 (5.67)	$t(458) = 12.35, p < .001$

*97.7% women

Eating Disorders Examination Questionnaire	Author (n = 154), Greeks	Raykos et al., 2017, Australian (n = 306)*	Group comparison
Restraint	2.98 (1.68)	3.74 (1.65)	$t(458) = 4.63, p < .001$
Eating	2.66 (1.51)	3.71 (1.39)	$t(458) = 7.43, p < .001$
Shape	3.84 (1.59)	4.74 (1.33)	$t(458) = 6.41, p < .001$
Weight	3.46 (1.56)	4.33 (1.48)	$t(458) = 5.84, p < .001$
Global score	3.23 (1.35)	4.06 (1.27)	$t(458) = 6.48, p < .001$

*97.7% women

Appendix 39– Proofreading Adherence Guidelines



Declaration of Proof reading Services

Confirmatory Statement of Acceptance

Name of Candidate Olga Theodoropoulou
Type of Award Doctorate of Philosophy in Psychology

I declare that I have read, understood and have adhered to UCLan's Proofreading Policy (Appendix 1) when proof reading the above candidate's research degree thesis.

Signature of Proof reader *Linda Rawlinson*

Print name: Linda Rawlinson

Name of Company Proofed Inc.

Contact Details

Telephone: +44 20 3966 5659

Email: linda@getproofed.com

Extract from Research Student Assessment Policies and Procedures Handbook

10. Policy on Proof-reading for Research Degree Programmes and the Research Element of Professional Doctorate Programmes

This policy is to clarify the use of third parties for proof-reading for student's written work for Research Programme Approval, Transfer from MPhil to PhD, the thesis (or synoptic commentary) and any work which later forms part of the final thesis. This applies to all written work or the thesis, whether draft or a final version, submitted for these assessments whether the proof-reading is for the whole or part of the work.

10.1 Principles

- (i) Each student's work must be solely his/her own work.
- (ii) Students at postgraduate level are expected to have developed their own proof-reading skills to a suitably advanced level for the award and be aware of the difference between proof-reading and editing. However, all students are encouraged to have their theses proof-read. But editing is the sole responsibility of the student.
- (iii) Students should receive advice and guidance on the drafting of any work and the thesis for submission from their supervisors and any designated advisors. Supervisors will assist with proof-reading.
- (iv) Students who consider they need assistance on the use of English should contact WIGER.
- (v) Students must not employ any person to write any parts or the complete work on his or her behalf, whether from professional companies, family, personal friends, other students or any other person except where an amanuensis has been appointed for the student as part of the student's disability support through UCLan's Disability Service. Inadequate skills in written English will not be justification for use of an amanuensis or a writer.
- (vi) Students must make all alterations to their work or their thesis themselves.
- (vii) Students are responsible for interpreting the advice of any proof-reader employed.

10.2 Engagement of third party proof-reading services

If a student employs a third party then the student is responsible for acknowledging the assistance with proof-reading. Any assistance must be acknowledged in a statement in the work or the thesis. The student is also responsible for clarifying the limits for the assistance. It is a requirement that:

- the student provides the third party with a copy of this policy and obtains a confirmatory statement of acceptance from that party;
- the student provides the third party with paper copies for annotation;
- students should retain the copy of the proof-reader's annotated work until the assessment process is complete.

Warnings: students are warned that any use of third party proof-reading services must not compromise their authorship of the work submitted, and, in particular, that the substance of work must remain the student's own. Students are also warned that they will be held responsible for work which they submit, and that the use of third party services will not be accepted in mitigation of any deficiencies in the work.

10.3 Unfair Means to Enhance Performance

Where a student does not follow the policy and is considered to have used a third party for non-permitted forms of assistance then the matter will be dealt with under the Unfair Means to Enhance Performance procedures.

Students must ensure they are aware of and abide by the regulations and policies.

10.4 Permitted assistance and advice

In the main text, tables, diagrams, footnotes, endnotes and illustrations proof-readers may suggest corrections with regard to:

- ✓ Spelling and punctuation
- ✓ Formatting
- ✓ Compliance with English conventions on grammar and syntax
- ✓ Consistency of page numbers, headings and footnotes

10.6 Non-permissible assistance and advice

Changing any text, table diagram, or illustration in the following ways by proof-readers (or as a result of their advice) is not permitted:

- X to clarify arguments or ideas
- X to develop arguments or ideas
- X to change arguments or ideas
- X to correct factual information
- X to translate work in to English
- X to reduce the length of the work
- X to assist with referencing

10.8 Method for third party advice

Access to the source document to be submitted for the assessment should remain solely with the student and not be passed to the third party.

The third party undertaking the proofreading should be given the advice by a means which provides a record showing the changes recommended.

The student must consider the changes advised, interpret them accordingly and undertake the changes personally. Students are responsible for ensuring that the advice given does not alter the intended meaning or use subject specific terminology in the wrong context.