AN INTEGRATIVE MODEL OF AGGRESSION: THE ROLE OF COGNITIONS IN RESPONSES TO STRESSORS IN FORENSIC AND NON-FORENSIC POPULATIONS

By

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ABSTRACT

This PhD aimed to further the understanding of aggression through the integration of research findings with theoretical models. As a result, a Stratified Integrated Model of Behavioural Aggression (SIMBA) that specifies and stratifies the roles of stress, cognitive structures and information processing was proposed. This may help guide therapeutic interventions aimed at the reduction of aggressive behaviour and inform risk assessment.

A systematic literature review of 77 papers was conducted to assess the relationship between stress systems' activity and aggression. The results showed that this relationship is likely present and can be both positive and negative. Thematic analysis of these papers identified six themes: 1) the impact of testosterone on the relationship between activity of stress response systems and aggression is undetermined; 2) the presence of sex differences in the relationship between stress response and aggression depends on the stress system and type of aggression; 3) specific disorders do not influence the relationship between stress and aggression; 4) experience of victimisation does not have a clear influence on the relationship between stress systems' activity and aggression; 5) the relationship between stress response markers and aggression differs among those with high stress exposure; and 6) history of aggression affects the relationship between stress response markers and anger-based aggression. These results highlighted that the stress-aggression relationship is present, but is likely to be indirect. However, the extraneous variables consistently affecting this relationship were not identified.

Addressing this issue, study one recruited 20 male students and 11 patients from a high secure hospital to establish the effects of aggression supportive cognitions and stress on aggressive behaviour. To assess aggression after a stress-evoking task, the Taylor Aggression Paradigm was used. It was predicted that while the presence of aggressive Implicit Theories

(ITs) would be positively associated with aggressive behaviour towards a stranger, the association of stress would differ between the samples. This was partially supported, as only one specific IT ("I am the law") was associated with aggression. Furthermore, only elevated skin conductance, but not changes in the heart rate, during the stress task was positively associated with aggression, and only among patients.

Study two involved 100 participants (49 men, 48 women, three not disclosed) with an average age of 29. It aimed to investigate the relationship between history of aggressive behaviour, affective states, and neutral and emotional information processing. Event Related Potentials (ERPs) during a Go/No-Go task were utilised to capture cognitive resources allocation, with a "supervisor – employee" laboratory paradigm used to assess aggression. Contrary to expectations, results showed that trait aggressiveness was only related to aggressive behaviour at higher levels of inhibitory processing. The hypothesis that artificially provoked changes in negative and positive affect would be related to aggressive behaviour was also not supported. However, as expected, feeling hostile was associated with short-lived aggressive behaviour, but only for those who had low response inhibition. Moreover, partially supporting expectations, a history of aggressive behaviour moderated the relationship between change in negative affect and aggressive behaviour. The last hypothesis, proposing emotional processing to be a mediator between response inhibition and aggressive behaviour, was also not confirmed.

Study three included 462 participants, of whom 300 were adults aged 26 or older (151 men, 149 women), and 162 representing transitional aged youth, aged between 18 and 25 (21 men, 141 women). This study aimed to identify direct and indirect effects exerted by aggression supportive cognitive structures, working memory problems, and stress on aggression by building a Structural Equation Model. It was expected that a direct cognitive pathway from aggression supportive cognitions directly to aggression would be identified. This hypothesis was supported. Meanwhile, the second hypothesis proposing an indirect relationship between

stress and aggression was only partially supported, with maladaptive coping style being the only mediator identified.

The current research demonstrated that aggression-supportive cognitive structures are the primary facilitators of aggressive behaviour. Meanwhile, the effect exerted by situational demands is contingent on the preferred coping style. Furthermore, despite the indirect nature, the influence of information processing was present for multiple precursors of aggression. Consequently, all these elements were included in the SIMBA and are suggested as primary targets for therapeutic aggression interventions. The results are discussed with attention to this proposed model, capturing further directions for future research.

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DEDICATION

This thesis is dedicated to Obraztsov Petr Borisovich. Forthright, responsible, and vigorous man who always had time and a good story. I am forever grateful for him teaching by example how to be a better person.

CHAPTER ONE: SETTING THE SCENE

Aggression is a form of behaviour that every person engages in throughout their life, albeit to varying degrees. Acts of aggression can have a significant effect on the lives of both individuals and communities. Consequently, understanding it, which leads to enhanced control over it, has a practical value in addition to epistemological worth. Since it is a mode of conduct, studying it is an extension of studying human actions that are the force that shapes the world.

Aggression has been defined as "behaviour that is intended to harm another person who is motivated to avoid that harm" (Allen & Anderson, 2017, p.1). There are several important features. First, aggression is a behaviour, meaning a physical act needs to take place. This excludes wishes, fantasies, feelings, other internal states, and cognitions. Moreover, the behaviour needs to be directed at another person. This separates suicide or self-harm and property damage from aggressive behaviour, unless these are used as means for psychological harm. The provided definition also states that the conduct needs to be purposeful, with an aim to hurt (Anderson & Bushman, 2002). Not only does this aspect remove accidental behaviour, but it also allows for unsuccessful attempts (e.g., throwing a punch at someone who evaded it is still considered aggression). Lastly, the motivation of the recipient highlights that not all harmful behaviours are aggressive (Bushman & Anderson, 2001). For example, tooth removal is always harmful, however if it is done by a dentist who was requested to do so, then it is not aggression. Likewise, instances when a person wants and consents to be harmed (e.g., masochism) cannot be considered aggression. Importantly, aggression can also take place when a victim is not in the vicinity of the aggression, where the intent of the aggression may not be evident to others, and/or when the identity of the aggressor is not known (e.g., the spreading of malicious gossip) (Allen & Anderson, 2017). This is referred to as indirect aggression

Similar to other forms of behaviour, aggression has been proposed to be governed by different principles. Socio-cognitive models emphasise the role of aggression-supportive

cognitive structures (DeWall, Anderson, & Bushman, 2012; Huesmann, 2016). According to these models, such structures reflect norms that condone or promote the use of aggression; contain associations between aggressive behaviour and valued outcomes and outline aggressive conduct as appropriate or efficient. These models are particular applications of cognitive models for behaviour. The reformulated *Social Information-Processing Model* (SIP) *of Social Adjustment* (Crick & Dodge, 1994) states that behaviour is preceded by six processes that can simultaneously interact with existing social schemas or knowledge. First, a person encodes internal and external cues, then they interpret these cues in terms of attributions. Afterwards, a goal for this situation is clarified, which is followed by the generation of a response. After that, the person makes a decision about the generated response and either enacts it or formulates a new one. However, all of these processes can simultaneously influence and be influenced by both the cognitions held by an individual and their selection. Similarly, the *Theory of Planned Behaviour* (TPB) (Ajzen, 1991) postulates that any behaviour results primarily from the intention to engage in it, and from the perceived control that affects both intention and behaviour. The attention TPB pays to perceived behavioural control is one of the indicators of the relevance of cognitive processes in influencing behaviour. The other indicator, according to Ajzen (1991), is that motivation for behaviour is partially determined by the existing attitudes towards it (favourable or unfavourable) and by subjective norms (is the behaviour approved or not?). These two constructs demonstrate that behaviour originates in the cognitions a person holds about it, which means that aggression as a form of behaviour also originates in cognitions supporting its use.

Although the influence of behavioural scripts[1] on aggressive conduct has been shown in previous research (Gilbert, Daffern, Talevski & Ogloff, 2013; Hosie, Gilbert, Simpson, &

[1] defined as cognitions, stored in memory, that contain information about what will occur in a situation, how one should react to it and what the results of these reactions will be (Huesmann, 1988).

Daffern, 2014; Musher-Eizenman et al., 2004), less attention has been paid to other forms of cognitive structures and physical aggression in particular. Furthermore, while these models are valuable, as they provide a comprehensive framework for aggression, they often lack detailed descriptions of the modifiers that are proposed to facilitate specific types or forms of aggression.

This is accounted for in the neurobiological models of aggression that postulate aetiological differences between reactive (also referred to as affective) and proactive (or instrumental) aggression (Fabian, 2010). Specifically, reactive aggression is posited to result from a failure to appropriately control aggressive responses to a stress-evoking environment, due to increased neural activation in the threat system (da Cunha-Bang et al., 2017; Farah, Ling, Raine, Yang, & Schug, 2018; White et al., 2015) and poor response inhibition (Chester & DeWall, 2015; da Cunha-Bang et al., 2017). Meanwhile, instrumental aggression, which is the selection of aggressive conduct as a means to a given end, is suggested to be rooted in poor ability to learn associations between behaviour and outcomes, decreased empathy and consequence evaluation (Blair, 2005; Ly et al., 2014; Morelli, Sacchet, & Zaki, 2015; White et al., 2013).

It is important to state that affective and proactive aggression represent typological and artificial approximations of one behavioural concept[2], rather than two different phenomena. In daily life, the motivation behind a given act of aggression can be mixed; for instance, aggression can be used with an aim of reducing stress (Bushman & Anderson, 2001; Blair, 2016). Moreover, despite the differences in the neural correlates of these two types of aggression, there are considerable similarities (Babcock, Tharp, Sharp, Heppner, & Stanford, 2014; Blair, 2004). Nevertheless, as the mechanisms identified for reactive and instrumental

[2] Further in the thesis, affective and proactive aggression are referred to as ‘types’.

aggression are related to this form of behaviour, they need to be incorporated into a general model.

This distinction also highlights an important aspect of aggressive behaviour, which is that it is likely to be affected by the demands that situations exert on an individual. Indeed, although research has shown that stress is associated with aggression, the results are conflicting as in some studies the relationship is reported to be positive and in others - negative (Gowin et al., 2013; Murray-Close et al., 2017; Sherwood et al., 2004; Verona & Curtin, 2006; Von Dawans, 2012). While this might indicate a difference in the effect of stress on aggression depending on the methods by which they are measured, it also points to the possibility of an indirect relationship. Regardless, in both cases, the variables that influence the stress-aggression relationship need to be identified and incorporated into the models explaining aggression.

Existing literature provides general models explaining aggressive behaviour and studies demonstrating specific mechanisms that facilitate or inhibit its types. Consequently, there is a need to synthesise and stratify this in order to arrive at an updated model. Factors related to repeated aggression across situations should be noted as the root causes of aggression. Meanwhile, those that affect aggression indirectly or only in specific circumstances should be placed in a separate category, reflecting their non-direct influence. In addition to improving the understanding of aggression, such an approach would also help in guiding therapeutic aggression interventions by highlighting the likely return on targeting specific aims.

Consequently, the current research programme aims to begin this process by proposing a model of aggressive behaviour that accounts for the roles of cognitions, information processing components and stress. Although existing models provide suggestions about the roles of these components, certain gaps remain. First, the socio-cognitive models, and specifically the General Aggression Model (GAM)(Anderson & Bushman, 2002), note that

arousal can contribute to aggressive behaviour. However, not only is the specific effect of arousal not clarified in these models, stress (which is the next stage of physiological response) is not accounted for. The effect of stress on aggression needs to be incorporated into a model, because it is conceptually related to the very definition of reactive aggression (Fabian, 2010). Although stress has been shown to be associated with aggressive behaviour, its changing effect highlights another important variable contributing to aggression: sex (Böhnke et al., 2010a; 2010b; Verona & Kilmer, 2007). While the socio-cognitive models (e.g., Anderson & Bushman, 2002; Huesmann, 2016) note that sex might affect the form of aggressive conduct (e.g., direct vs. indirect), they do not specifically address the possible differences in the same form of aggression between men and women.

Dual hormone theory suggests that aggressive behaviour is regulated by an interaction between cortisol and testosterone (Archer & Carré, 2016; Carré, Ruddick, Moreau, & Bird, 2017). Consequently, the varying intensity of aggressive behaviour shown by men and women (Böhnke et al., 2010a; 2010b) needs to be incorporated into an updated model. Furthermore, a meta-analysis by Mathur and VanderWeele (2019) showed that observational learning of aggression supportive behavioural scripts, which is emphasised in socio-cognitive models (Huesmann, 2016), has only a minor influence on aggressive behaviour. Consequently, the effect of experiential learning of aggression supportive cognitions on aggressive behaviour, which can be inferred from neurocognitive accounts of instrumental aggression (Blair, 2005), needs to be assessed and incorporated into a model.

Following recommendations of Gilbert, Daffern, and Anderson (2017), the current research also aims to investigate the effect of specific aggression supportive cognitions on aggressive behaviour. Building on retrospective cross-sectional research confirming the relationship between these two variables in forensic populations (e.g., Bowes & McMurran, 2013; Gilbert, Daffern, Talevski & Ogloff, 2013; Hosie, Gilbert, Simpson, & Daffern, 2014),

the current research employs an experimental approach. This allows for the direct assessment of the contribution of aggression supportive cognitions to observed aggressive behaviour. To further the understanding of whether the relationship between these two variables changes depending on the type of sample, the current thesis also employs a cross-sectional analysis with varying histories of aggressive behaviour and compares them to the transitional aged youth (TAY). Nevertheless, the main focus of this research is on the mechanisms behind aggression among people who have often engaged in it, such as patients of high secure forensic hospitals. They are the ones who are most likely to be the participants in therapeutic interventions targeting aggressive behaviour. The risk-need-responsivity (RNR) framework (Andrews, Bonta, & Hoge, 1990; Andrews, Bonta, & Wormith, 2011) suggests that offenders with the highest risk of violence should receive the most intensive treatment, and these programmes should address the criminogenic needs related to criminal behaviour. History of violence is placed among risk factors for such behaviour in the future (Douglas, Hart, Webster, Belfrage, Guy, & Wilson, 2014). Furthermore, Polaschek (2011) states that aggression interventions should be guided by theoretical models. Given that violence is an extreme form of aggression (Allen & Anderson, 2017), the models of aggressive behaviour should address it not only in community samples but also among those who exhibit it regularly. Understanding the principles behind aggression that are present in both of these populations will not only help to inform aggression interventions, but also help to guide preventive measures. Consequently, the current research aims to identify the mechanisms facilitating habitual aggressive behaviour, so that the targets for decreasing it or precluding its formation are clear.

In the following chapters, the noted models, their limitations and effects of stress on the variables suggested to influence aggressive behaviour will be elaborated and discussed in greater detail. This will be followed by a systematic literature review assessing the nature of the stress-aggression relationship, noting the influential third variables. Next, three empirical

studies establishing the pathways to aggressive behaviour will be presented. The findings will be discussed independently before being brought together in the final chapter where a tentative model will be proposed.

CHAPTER TWO: MODELS OF AGGRESSIVE BEHAVIOUR

2.1 Structure of the chapter

This chapter is devoted to models of aggressive behaviour, providing an overview of three frameworks: socio-cognitive, neurocognitive, and genetic representations of aggression. These models were selected as they provide the most comprehensive overview of the principles behind aggression that focus on several factors at once. These are explored, evaluated, and followed by a discussion highlighting the absence of irreconcilable contradictions, with a potential for integration provided. Afterwards, a metatheory of aggression is discussed with respect to the described models. The chapter concludes by outlining the two main factors facilitating aggressive conduct that are present across existing frameworks and suggesting a third factor.

2.2 Socio-Cognitive Models

2.2.1 Information Processing Model for the Development of Aggression

Information processing models of aggression are social-cognition models outlining how repeated patterns of aggressive behaviour are formed and the mechanisms that determine aggressive acts in given circumstances. One of the most referenced is the *Information Processing Model for the Development of Aggression* (IPM) (Huesmann, 1988), whose basic premise is that aggressive behaviour results from the selection of an aggressive behavioural script as the guide for dealing with a particular social situation. Behavioural scripts contain information about what will occur in a situation, how an individual should react and what the results of these reactions will be. They are thoughts that are stored in memory and evaluated before being enacted (Huesmann, 1988; 1998).

Although there is limited research on the relationship between aggressive scripts and aggression in the general population, the five studies reviewed by Gilbert & Daffern (2017)

show a positive correlation between these two variables. For instance, Egan and Campbell (2009) have shown that adult men and women who engage in negative fantasising were more likely to report more instances of physical aggression. However, Jouriles, Grych, Rosenfield, McDonald, and Dodson (2011) found a more specific pattern for male and female teenagers. Their results demonstrated that acceptance of teen dating violence, which is a part of the behavioural script for violence, requires aggressive automatic cognitions to be indicative of an increase in the perpetration of teen dating violence. Similarly, the belief that teen dating violence has negative consequences, which is an opposite belief about consequences to a behavioural script facilitating dating violence, interacted with aggression in automatic cognitions. This finding points to the dominant importance of overall cognitive structures supporting aggression rather than the specific scripts. Indeed, Musher-Eizenman and colleagues (2014) have shown that the cognitive recital of aggressive behaviour in the form of fantasy was only one of the cognitive mediators (others included: expected consequences and normative belief about aggression) for the effects of environment and affect on aggressive behaviour. A more macro-scale approach is reflected in the *General Aggression Model* (Anderson & Bushman, 2002), whereby behavioural scripts are included in a broader category of knowledge structures and into a cognitive route. This model is described in detail later in this chapter.

The need to account for more than behavioural scripts in explaining aggressive behaviour is evident from research in populations with histories of such behaviour. Studies (Gilbert, Daffern, Talevski & Ogloff, 2013; Hosie, Gilbert, Simpson, & Daffern 2014) investigating the effect of script rehearsal on aggressive behaviour among predominantly male patients of a forensic hospital with convictions for violent as well as non-violent offences have reported this effect to be present. However, script rehearsal was not the only significant factor in the tested models—it was accompanied by trait anger and favourable attitudes towards violence (Gilbert

et al., 2013) with comparable magnitude of effect (assessed via the standardised betas), or by trait anger and normative beliefs accepting of aggression (Hosie, et al., 2014). The relationships that aggression supportive attitudes and normative beliefs have with aggressive behaviour highlight that cognitive structures should be examined; yet, the influence of anger, which is an affective state, points to the relevance of affect.

However, the relationship between cognitive guides to aggression and behaviour can also be overshadowed by personality traits. Podubinski, Lee, Hollander and Daffern (2017) demonstrated that aggressive script rehearsal explains aggressive behaviour among psychiatric patients but only as a part of wider model. Their results showed that personality traits are better predictors of aggression than script rehearsal or aggression-favouring attitudes, because despite the significance of the overall model, script rehearsal and attitudes did not show significant effects independently. Similarly, Dunne, Lee, and Daffern (2019) found that the degree of fantasy about aggression is not a sole predictor of aggression. It is worth noting, however, that in the two latter studies (Dunne et al., 2019; Podubinski et al., 2017) script rehearsal was assessed using self-report measures, which raises concerns relating to impression management (Dunne et al., 2019). Moreover, when it comes to the practical application of risk estimation, the HCR-20 V3 includes an item "violent ideation", which reflects rehearsal of aggressive scripts. This inclusion suggests that behavioural scripts are recognised as one of the established potential risk predictors.

Given that these scripts are cognitive structures, Huesmann (1988) pays special attention to how they are acquired. The initial step is acquisition, which occurs by observing and encoding behaviours, and then rehearsing them in memory only or in play. The former is influenced by two factors. First, to convert behaviour of another into a script they need to be attended to and identified with. Huesmann and colleagues (2012) found that when presented with reports of violence, adolescents are more likely to associate themselves with those who

share their ethnicity, but to perceive the other group as engaging in more aggressive behaviours. The second influential factor is the evaluation of the observed script with respect to adopted norms and already held scripts (Huesmann, 1988; 1998). Consequently, scripts that match existing ones and are deemed appropriate are more likely to be encoded. A longitudinal study that followed 329 children supported these propositions. Of those who had watched violent TV shows between the ages of six and nine, children who associated themselves with fictional characters behaving aggressively, and who deemed such behaviour as realistic, were more likely to engage in similar behaviours as adults when compared to those who possessed neither or only one of these characteristics (Huesmann, Moise-Titus, Podolski, & Eron, 2003).

This evaluation of the appropriateness of a script for a given situation is considered to be dependent on three conditions (Huesmann, 1988). Firstly, there will be a prognosis about the outcome of the script. This judgement about the script's appropriateness will be affected by the individual's ability to consider this accurately as well as the proximity of the attended consequences. Secondly, this judgment will be influenced by an individual's sense of efficacy in enacting a given script. Lastly, whether or not a script is appropriate will depend on how it relates to the norms adopted by the person. The more it matches these norms, the more likely it will be adopted and used (Huesmann, 1988, 1998; Huesmann et al., 2003).

For a script to be readily available, it needs to be learned through one's own experience as well (Huesmann, 1988). Thus, further retention of the script is contingent on its outcome evaluation. One possibility is that while both positive and negative consequences of behaviour are considered by an actor, attention is devoted mostly to the positive consequences. It is also possible that aggression has been accepted as a norm and thus the outcome is perceived to match the expectation. In such cases, an individual can try to ensure that they are in an environment that shares their norms. In other words, if a person's environment does not

validate the use of aggression as appropriate, he or she might change their environment by entering a social group that shares those norms (Huesmann, 1988; 1998).

Huesmann (1988) also states that those who consistently enact aggressive scripts that are reinforced will not only continue to do so, but will also adopt aggression as a heuristic. Thus, they will be more likely to automatically consider it an effective solution to most problems, and thereby engage in it. Accordingly, Herrenkohl and colleagues (2001) demonstrated that for teenage men and women, engaging in antisocial behaviour during early adolescence and having parents with violence-favouring attitudes both increase the chances of engaging in at least one aggressive act after age 18. Similarly, a meta-analysis by Ttofi, Farrington and Losel (2012) showed that perpetration of bullying in adolescence is a consistent predictor of aggressive behaviour in adult life. Moreover, history of aggressive behaviour, operationalised as the number of prior convictions, has been demonstrated to be among the best predictors of violent recidivism, measured by re-convictions (Bonta, Law, & Hanson, 1998; Collins, 2010; Lund, Hofvander, Forsman, Anckarsäter, & Nilsson, 2013). Furthermore, Huesmann (1988) argues that observation of aggressive behaviour does not just present new scripts for potential adoption, it activates the already existing scripts for presented cues, thereby providing an opportunity for rehearsal.

Meanwhile, when it comes to particular situations, Huesmann (1988) argues that behaviour in any social interaction will be determined by individual or subjective factors in addition to objective aspects present in the situation. He outlines three points where the former can have a decisive effect. Firstly, individuals interpret situations differently. Secondly, there will be individual differences in particular scripts, the available number of them, and in the methods used to retrieve them from memory. Lastly, evaluation of each particular script that has been retrieved in relation to its perceived appropriateness and desirability of predicted outcome will vary between people. However, Huesmann (1988) presents this process of

selecting a behavioural script in the form a linear algorithm. This assumption required revision, as the *Reformulated Social Information-Processing Model (SIP) of Social Adjustment* (Crick & Dodge, 1994) suggested a process of generating a response to a social situation is nonlinear.

2.2.2 Reformulated Social Information-Processing Model (SIP) of Social Adjustment

As noted in the Chapter 1, according to the reformulated SIP (Crick & Dodge, 1994) any behaviour results from six processes. First, when a person enters a situation they encode external and internal cues. Then, using cognitive structures (e.g., scripts) stored in memory, the encoded cues are interpreted so that in the next step a person can select a goal or desired outcome for a situation. Importantly, as part of the cue interpretation an individual attributes intention to the actions of others. Consequently, as a fourth step, a person either chooses a response that brought the desired outcome in past situations with similar parameters, or creates a new response if a situation is novel. Afterwards, as a fifth step the created or chosen response is evaluated. If expected results are satisfactory with respect to goals, then the response is enacted; this is step six. In relation to the IPM, the reformulated SIP represents a broader framework as it includes the mechanisms described by Huesmann (1988, 1998) in the form of steps four, five and six.

Although Crick and Dodge (1994) suggest temporal order by numbering the steps within the model, the circular position of each of the steps shows that every time a given behaviour is enacted, the environment's response to it will affect how cues in a future situation will be encoded. Furthermore, the reformulated SIP positions "data base", which incorporates cognitive structures stored in memory such as schemata or normative beliefs, and which can influence each of the steps separately. Despite the lack of integration of non-cognitive factors (such as stress) into the model acknowledged by Crick and Dodge (1994), the reformulated SIP was a successful attempt to conceptualise cognitive processes leading to behaviours This

includes aggression, as non-linear and occurring in parallel. This approach was subsequently adopted by the General Aggression Model (Anderson & Bushman, 2002).

2.2.3 General Aggression Model

The General Aggression Model (GAM) is another socio-cognitive framework that outlines principles governing aggressive behaviour in particular social encounters and, like the reformulated SIP, accounts for non-linearity in the generation of behaviour (Anderson & Bushman, 2002). Not only does it suggest that resolution of a given situation informs the encoding and interpretation of cues in future, but it also suggests personal and situational structures have simultaneous effects on cue interpretation. The GAM supports the definition of aggression provided in the first chapter, as it retains the features of being directed towards others, proximate intention to inflict and understanding of harm, together with harm's avoidance by the victim(s). Since the GAM is a socio-cognitive theory, it emphasises the role of aggression supportive cognitions in the formation of such behaviour by an individual.

These cognitions are referred to as knowledge structures. They have four characteristic qualities (Anderson & Bushman, 2002). Firstly, they develop from both personal and observed experience. Furthermore, as they may include beliefs, affective states, behaviours, and scripts, employing them directs both apprehension of and behavioural reactions to social and physical stimuli. Moreover, they affect the construction of representations of physical and social environments. Lastly, with repeated application of these structures, their use requires less conscious awareness, in that they become more automated. These knowledge structures constitute what Huesmann (1988) referred to as individual factors. However, the GAM does not limit their influence to three moments of interaction; rather, it reflects the constant influence of cognitions suggested by the SIP (Anderson & Bushman, 2002).

According to the GAM, perceptual and person schemata and behavioural scripts are the main types of knowledge structures pertinent to aggressive behaviour (Anderson & Bushman, 2002). As described in the previous section, a script is a blueprint for conduct in a particular social situation (Huesmann, 1988; 1998). Meanwhile, perceptual and person schemata contain characteristics that are thought to be inherent to a particular object or situation (perceptual schemata) or to all members of a particular group of people (person schemata) (Anderson & Bushman, 2002).

According to this model, aggressive behaviour results from a processing of a social interaction that has three main aspects: inputs, routes and outcomes (Anderson & Bushman, 2002). During the inputs stage, a representation of a given interaction begins to form based on the situation itself and the person involved. While the situation reflects the physical objects, the environment and the observable behaviour of the others involved, the latter involves knowledge structures and personality characteristics that facilitate interpretation of the former. For instance, it has been shown that adolescents with callous-unemotional traits are likely to engage in aggressive behaviour (Lau & Marsee, 2013). Furthermore, a meta-analysis of 52 studies by Jones, Miller, and Lynam (2011) demonstrated that decreased levels of Agreeableness and Conscientiousness, together with increased levels of Neuroticism, are related to habitual aggression. This suggests that there are personality traits that increase the likelihood of aggressive behaviour across social situations. Another example of an individual's inputs that influence behaviour is how they interpret situations. A tendency to perceive others' motivations as hostile, referred to as Hostile Attribution Bias (HAB), has been consistently linked with aggressive conduct across situations (De Castro et al., 2002; Martinelli et al., 2018; Quan et al., 2019).

The HAB is corollary of the reformulated SIP, which highlighted that in any social encounter a person will try to attribute intentions to the actions of others. This will then

influence both the selection of the desired outcome for a given interaction and the selection of a response (Crick & Dodge, 1994). A meta-analysis of 41 studies with more than 6000 children (De Castro et al., 2002) showed that the tendency to perceive intentions of others as hostile rather than benign is consistently related to aggressive behaviour. Moreover, a systematic literature review of 27 studies by Martinelli and colleagues (2018) specified the nature of this relationship among children and adolescents further. They demonstrated that, among both males and females, HAB is more closely associated with aggression that is provoked by uncontrolled stress in the environment than it is with aggression used as a tool to achieve specific goals. Meanwhile, another systematic literature review (Tuente, Bogaerts, & Veling, 2019) demonstrated that the relationship between HAB and aggression is also consistently reported in adult samples, showing that it is not bound by age. This association was present among community samples as well as forensic patients, highlighting that the interpretation of situational cues influences aggressive behaviour regardless of history of aggression or presence of psychopathology. AlMoghrabi, Huijding and Franken (2018) presented participants with an unpleasant situation that was followed by reinforcement of its interpretation as either accidental or with intention to harm (provocation condition). They found that aggressive behaviour was higher in the provocation group than the accidental group. In other words, those who perceive situations as hostile are more likely to act aggressively.

Although the GAM is not restricted to cognitive factors, as Anderson and Bushman (2002) list further person factors that affect the interpretation of situation cues, such as long-term goals and biological predispositions. While this shows that their influence is acknowledged, the GAM does not provide a detailed description of their influence on resulting behaviour. Instead, only a general pathway is outlined.

Input of the internal and external information related to a given situation will inevitably create a certain internal state (Anderson & Bushman, 2002). Its creation is the second stage of

the GAM, which is subdivided into cognitive state, affective state, and arousal. Indeed, a literature review by Roberton, Daffern and Bucks (2012) highlights that while there is a consistent link between dysregulated anger (which is an affective state) and aggression, the role that is played by the regulation of other emotions is less frequently investigated. Furthermore, in a bimodal conceptualisation of aggression, reactive aggression is deemed to be aimed at reduction of arousal, thereby suggesting a close relationship between arousal and aggression (Meloy, 2006). Meanwhile, when it comes to cognitive states, Anderson and Bushman (2002) emphasise behavioural scripts[3], which were noted by Huesmann (1988; 1998), and hostile thoughts that are activated based on inputs. Indeed, DeWall, Twenge, Gitter, and Baumeister, (2009) demonstrated how men and women with hostile thoughts, evidenced by presence of HAB, are more likely to precede aggression after social rejection. However, it is important to note that the mediating role of HAB between social rejection and aggression was only present for aggression aimed at those involved in the rejection.

The GAM also includes beliefs about aggression and attitudes towards it as inputs. In a study with 44 male forensic patients from a secure psychiatric hospital, Ireland and colleagues (2019) found that believing aggression to be a norm increases engagement in it. Specifically, it was found that patients, who believed that intra-group aggression was an inevitable part of secure settings, had a higher chance of engaging in various forms of intra-group aggression by almost 40% compared to those who did not hold such a belief. Similarly, the belief that intra-group aggression can have positive consequences facilitated the likelihood of engaging in this behaviour. These results demonstrate that beliefs about aggression exert influence on behaviour even in highly monitored settings, where the costs

[3]Since behavioural scripts are placed within the category of knowledge structures and in the "cognition" route, further in this thesis the phrase 'cognitive structures' includes behavioural scrips.

and benefits of such behaviour differ from regular social interactions. Moreover, Gellman and Delucia-Waack (2006) demonstrated that male adolescents who hold attitudes forming a culture of violence within which the world is considered a perilous place, where one must constantly be on guard, were more likely to have more aggressive histories. Similarly, aggressive ideation, which corresponds to aggression supportive knowledge structures, has been at least moderately associated with aggressive behaviour in teenagers across time (Murray, Obsuth, Eisner, & Ribeaud, 2016). A small review of five studies by Bowes and McMurran (2013) extended this relationship to adults, as it revealed that among both university students and offenders, cognitions favouring the use of aggression and thereby normalising violence were associated with past aggressive behaviour. Their review also indicated that prisoners who engaged in intra-group aggression had a pronounced lack of victim supporting attitudes, indicating that it is not only attitudes about violence that affect use of aggression but also the attitudes towards potential victims.

It is worth noting, however, that only one of the studies reviewed by Bowes and McMurran (2013) had female participants, while the rest (Ireland et al., 2019; Gellman & Delucia-Waack, 2006) had exclusively male samples. This aspect significantly limits the generalisability of the results. However, at least within the male population, the influence of aggression related cognitions and attitudes appears to have a firm relationship with such behaviour. Furthermore, as HAB is a cognitive internal state (Anderson & Bushman, 2002) and its relationship with aggression was present in both men and women (Tuente et al., 2019), these reviews support the GAM's proposition that internal states affect aggressive behaviour across genders.

Although internal states are divided into three groups, Anderson and Bushman (2002) argue that they influence each other. Not only does this close relationship reflect the constant effect of cognitions on behavioural selection proposed by the reformulated SIP (Crick &

Dodge, 1994), but it also concurs with research suggesting a close interplay of affect and cognitions in their effects on aggression (Del Vecchio & O'Leary, 2008; Fives, Kong, Fuller, & DiGiuseppe, 2011; Roos, Hodges, Peets, & Salmivalli, 2016). Specifically, both anger and two types of knowledge structures show simultaneous and significant influences on aggressive behaviour among forensic patients (Gilbert et al., 2014). However, affect and cognitions can also interact. For instance, in children, anger can serve as an enhancer for the influence of the aggression-supportive cognitions on aggressive behaviour (Roos et al., 2016). Similarly, Del Vecchio and O'Leary (2008) demonstrated that anger serves as a mediator between mothers' cognitions and harsh responses to children's misbehaviour, including slapping a child.

Together, internal states represent routes that link the input information with the last stage—outcomes—where the conduct is selected (Anderson & Bushman, 2002). According to the GAM, in cases of scarce cognitive and temporal resources, assessment of the situation will be automatic, largely influenced by internal states and result in an impulsive action. However, when cognitive resources are available, time pressure is absent and a given social interaction is perceived as important, the situation can be re-appraised. This, in turn, can lead to alterations in the internal states, followed by the selection of a new script, ultimately resulting in a thoughtful action (Anderson & Bushman, 2002).

This division of script selection into 'automatic' and 'thoughtful' mirrors a renowned *Dual Process Model* of decision-making (Croskerry, 2009). This model posits decision-making and reasoning can be accounted for by the interaction between two types of processes called System 1 and System 2. While System 1 reflects largely automatic processes that are heuristically-driven, fast, cognitive resource-efficient and specific to a particular task, System 2 refers to effortful, conscious, reasoning-driven, slow processes that are abstract and generalisable (Evans, 2008). Thus, the last stage of the GAM (outputs) posits decision-making as a direct antecedent of aggressive conduct (Anderson & Bushman, 2002). Accordingly, in a

series of experiments DeWall, Baumeister, Stillman, and Gailliot, (2007) demonstrated that students who engaged in mildly cognitive demanding or monotonous tasks were more inclined to exhibit aggressive behaviour than those whose cognitive resources were untapped. This supports the GAM's proposition that when cognitive resources are low the appraisal of the knowledge structures guiding behaviour is impaired, resulting in poor response inhibition and impulsive actions.

The GAM provides an overarching framework that outlines processes through which aggressive behaviour emerges. However, its drawbacks are extensions of its benefits. Despite acknowledging the role of affective states and arousal, the GAM over-emphasises the role of cognitive processes. This is done at the expense of describing the role of stress, which has been shown to impact both aggressive behaviour (Murray-Close, Holterman, Breslend, & Sullivan, 2017) and cognitive processes such as decision-making (Starcke & Brand, 2012; 2016). Furthermore, as an overarching model that attempts to include all factors influencing aggressive behaviour, it synthesises general pathways underlying aggressive behaviour. However, such generalisability and overarching nature prevents it from providing a detailed description of each factor. Although another model of aggression—the *Integrated Information Processing* model (IIPM)(Huesmann, 2018)—does not address these specific shortcomings, it attempts to include even more general factors explaining aggressive behaviour.

2.2.4 Integrated Information Processing Model

In contrast to the earlier Information Processing Model for the Development of Aggression (IPM) (Huesmann, 1988; 1998), the IIPM (Huesmann, 2018) includes pathways that account for the influences of cognitive structures and internal states at each step. Given that the two previously described models of aggressive behaviour (the GAM and IPM) deem aggression an enactment of aggressive scripts and do not contain propositions that contradict those from the IIPM, it is useful to see them as part of one framework. Indeed, Huesmann

(2018) perceives the GAM as an *addition* to the principles of the IPM, because it formulates pathways for the development of aggressive behaviour and aggressive conduct in a particular situation. Thus, Huesmann (2018) proposes a further framework that unites socio-cognitive theories of aggression, the Integrated Information Processing Model (IIPM) (Huesmann, 2018).

For this model, Huesmann (2018) postulates four main tenets that correspond to the pathways proposed by the GAM. In the IIPM, aggressive behaviour is deemed a result of personal predispositions and situational factors. Repeated engagement in aggressive behaviour is proposed to typically begin in childhood, and to be a consistent predictor of aggression in adulthood. Severe aggression is considered a result of multiple environmental and biological factors, neither of which can be considered a sole cause. Lastly, learning is placed among the core processes that facilitate behavioural patterns, including aggressive ones. Within it, observational learning is suggested to play "the most important role" (Huesmann, 2018. p. 119).

Furthermore, both the GAM and IIPM emphasise the role of knowledge structures in facilitating aggressive behaviour (Anderson & Bushman, 2002; Huesmann, 2018). While they share behavioural scripts as one of the main knowledge structures, the other two also have similarities. While the IIPM highlights schemas about the world that facilitate assessment of social interaction, the GAM points to a broader perceptual schema that affects perception on all levels. So, in this regard, it appears that the former can simply be included in the latter, thereby making the difference between them quantitative rather than qualitative. Similarly, in terms of the third type of knowledge structures included in the IIPM and the GAM, this difference does not appear to be essential either. For the IIPM, the third type of structures are normative beliefs (Huesmann, 2018); In the GAM, they are person schemata (Anderson & Bushman, 2002). However, the former can be based on or even incorporate the latter, as beliefs

about what constitutes appropriate behaviour can include references to recipients of this behaviour.

Furthermore, both normative beliefs and person schemata can be seen as part of implicit theories. *Implicit theory* is another term that describes "beliefs about the properties of classes of objects, including humans" (Plaks, 2017, p. 261). In other words, this term refers to a complex cognitive structure that is based on one assumption (or a set of assumptions) about a particular object that are held true and from which an overarching representation of the social environment emerges. Compare this with the definition of normative beliefs in the IIPM, which describes them as an "individual's own cognition about acceptability or unacceptability of behavior" (Huesmann & Guerra, 1997, p. 409). Similarly, the definition of person schemata in the GAM is that it is a type of knowledge structure which "include beliefs about a particular person or groups of people" (Anderson & Bushman, 2002, p. 33). From this definition it is clear that person schemata are a specific subset of implicit theories.

Indeed, Harper and Bartels (2016) have shown that judgments about people in a particular group (sex offenders) are influenced by the general population's implicit theories about this group. Meanwhile, these normative beliefs must include references to a situation and to other actors in such a situation. Behaviour cannot be acceptable or unacceptable in a vacuum; there needs to be context for it. As soon as such context includes any object or people, the normative belief becomes informed, at least partly, by an implicit theory. Consequently, while person schemata are entirely included in implicit theories, normative beliefs stem from them.

Taking this into account shows that the influence of implicit theories on aggressive behaviour lends further support to socio-cognitive models of aggression. An analysis of interviews with violent offenders (n = 23) has identified that these individuals do possess implicit theories normalising the use of violence or adopting low self-efficacy in impulse control (Polaschek, Calvert, & Gannon, 2009). Similarly, an analysis of interviews with 23

sexual murderers by Beech, Fisher and Ward (2005) demonstrated that sexual murderers have implicit theories that normalise the use of aggression and shift blame to the victims. Furthermore, a literature review by Ruddle, Pina, and Vasquez (2017) highlighted that implicit theories approving and encouraging of aggressive conduct, and attributing the responsibility for it to victims, have been found among those who perpetrate domestic violence. Although this shows the existence of knowledge structures that condone the use of aggression, it does not yet mean that they have facilitated aggressive conduct. However, one study (Yeager, Trzesniewski, & Dweck, 2013) demonstrated that interventions aimed at changing implicit theories about oneself and potential victims decreases aggressive responses to provocation among both male and female adolescents aged 14 to 16—more than an intervention aimed at the creation of coping skills. Since it was the change in cognitive structures that produced a change in behaviour, but not the learning of new skills, it appears that cognitions influence aggressive conduct more than emotion regulation strategies do.

Huesmann's (2018) outline of a social interaction is similar to that from the GAM. He postulates that social interactions begin with evaluation of the situation itself, which is followed by retrieval of those scripts from memory that are deemed as matching the interpretation of the situation and one's inner state. Then, the applicability of the script is evaluated based on the value of its outcome and on adopted norms. Afterwards, an appropriate script is selected and enacted, and the outcomes are evaluated, again leading to retention or modification of the script.

However, the main challenge for the IIPM is similar to that of the GAM. Since it is proposed as an overarching framework accounting for all factors related to aggressive behaviour, it does not provide a detailed pathway for each factor. Moreover, as Huesmann (2018) states that no factor on its own can provide an in-depth explanation of aggressive behaviour, the model does not provide a comparison of individual factors that could show the

relative strength of the influence of each. This lack of outlined hierarchy of influential factors, together with emphasis on learning of aggression through observation, gives way to criticism that the IIPM, similar to the GAM, overvalues the impact of knowledge structures on aggression at the expense of other factors that are not explained. Consequently, the limitations of these models need to be discussed in greater detail.

2.3 Critical Evaluation of Socio-Cognitive Models

As noted, socio-cognitive models are primarily focused on the role of cognitive elements in the facilitation of aggression. Although they acknowledge the role of affective states and arousal, a lack of focus on these aspects has attracted criticism of these models. According to Ferguson and Dyck (2012), the paradigm that these models represent needs to be altered, because its core assumptions are invalid. However, contrary to their claim, even if most of their criticism is presumed valid, it does not undermine the credibility of socio-cognitive models of aggression. Instead, it points to the weaker aspects of these models that need to be amended or elaborated upon.

There are two lines of critique of the GAM that, if proven true, can significantly reduce the utility of this model. First, the focus on cognitions as an explanation of aggressive behaviour has been challenged. However, critics of the GAM concede that cognitions "may play some role" (Ferguson & Dyck, 2012, p. 224). Furthermore, the personal inputs in the GAM include biological factors, in addition to cognitive ones, and affective state together with arousal are suggested to serve as routes to aggression alongside cognitive states. Thus, the GAM does not focus exclusively on cognitive aspects.

Indeed, the importance of affective states and arousal can be seen in the outline of aggression intervention programmes based on the GAM. DeWall and colleagues (2011) place techniques aimed at reducing hostile affect among the first steps of their treatment programme.

Nonetheless, descriptions of the socio-cognitive models, the GAM or IIPM do not address the effects of stress or other non-cognitive factors in great detail. Taken together, this suggests that the premise for part of Ferguson and Dyck's (2012) criticism, namely over-focus on cognitions, is valid. Nevertheless, as cognitive structures supporting use of aggression have been shown to contribute to aggressive behaviour (Bowes & McMurran, 2013; Hoise et al., 2014; Tuente et al., 2019), this part of the model should not be abandoned. Instead, it should be used as a backbone for future models that incorporate other factors as well.

Another important line of critique is the GAM's lack of falsifiability, as this criterion is clearly argued as a necessity for scientific theory (Ferguson & Dyck, 2012). However, this criticism appears unwarranted. The base assumption of the GAM and of socio-cognitive models in general is that knowledge structures that contain and approve of aggressive behaviour increase the chances of such conduct in a given situation (Anderson & Bushman, 2002; Huesmann, 2018). This can be falsified by demonstrating how, with other variables held equal, aggression supportive knowledge structures do not increase the likelihood of aggressive conduct. Although some studies do show that inclusion of personality traits removes the effect of aggression-supportive cognitive structures (Dunne et al., 2019; Podubinski et al., 2017), other studies show the opposite pattern where such cognitive structures exert a significant effect (Bowes & McMurran, 2013: Gilbert et al., 2013; Hoise et al., 2014). Furthermore, the attribution of lack of falsifiability to the GAM, which is considered not just a model but a paradigm (Ferguson & Dyck, 2012), neglects the fact that a socio-cognitive paradigm of aggression represents an extension of the socio-cognitive paradigm of behaviour, of which aggression is one form.

Nevertheless, it is worth acknowledging that socio-cognitive models lack precision in describing the relationship between aggression supportive knowledge structures, as it can be both direct and indirect. For example, Podubinski, Lee, Hollander, and Daffern, (2017)

demonstrated that psychiatric patients' personality traits, which are viewed as solidified knowledge structures by Anderson and Bushman (2002), are more predictive of aggression than script rehearsal. Furthermore, they do not provide a strict hierarchy of factors that increase the chances of aggressive behaviour, which would demonstrate the magnitude of the influence of knowledge structures compared to other factors outlined in the model. For instance, as noted earlier, the results of Dunne, Lee, and Daffen (2019) shows that for prisoners, anger as an affective state is a better predictor of past aggressive behaviour. Similarly, Kramer, Jansma, Templeman, and Munte (2007) highlighted the role of negative emotions in contemplation of a response to provocation among general population. Similarly, the results of both the GAM and IIPM were proposed as integrative models (Anderson & Bushman, 2002; Huesmann, 2018), reflecting their attempt to unite existent theories rather than propose a radically new one.

Regardless, the criticism of Ferguson and Dyck (2012) rests on disproving the following assumptions attributed to the GAM. Firstly, they posit that the authors of the GAM have a moralising rather than scientific view on aggression. However, it appears that the importance of author views on adaptability of aggression is artificially inflated. Socio-cognitive models describe how an individual's knowledge structures influence their automatic or thoughtful appraisal of retrieved scripts. Consequently, it is the aggressor's belief about adaptability of aggression that is important, not the beliefs of the GAM's authors. Thus, even if the theorists view aggression as maladaptive, it does not refute the proposition of such models that an individual's knowledge structures approving or encouraging use of aggression increase the chances of this behaviour, especially since there are studies supporting this proposition (DeWall, Baumeister, Stillman, & Gailliot, 2007; Gellman, & Delucia-Waack, 2006; Ireland et al., 2019).

The GAM has also been challenged (Ferguson & Dyck, 2012) for its assumptions that aggression is learned behaviour and that exposure to real and fictional violence have equivalent effects on the learning of aggressive knowledge structures. Indeed, Fleckman, Drury, Taylor, and Theall, (2016) demonstrated that observing real life aggression was predictive of engaging in externalised behaviour, including aggression, among children aged four to 15. Moreover, they found that observation of aggression interacts with exposure to it from caregivers; specifically, observation has greater influence on behaviour when lower levels of exposure from caregivers are present. This suggests that witnessing aggression in reality facilitates acquisition of aggressive behavioural scripts. Moreover, a longitudinal study (Guerra, Huesmann, & Spindler 2003) found that among more than 4000 male and female children aged five to 12, exposure to real life violence increased the subsequent number of cognitions the children held, which normalised the use of aggression and also increased the frequency with which the children rehearsed those scripts. Furthermore, such aggression supportive cognitions and scripts were found to mediate the relationship between exposure to and engagement in aggressive behaviour.

Nevertheless, it needs to be acknowledged that the emphasis on observational learning of aggression-supportive cognitive structures by Huesmann (2018) might be disproportional. According to the GAM, playing violent video games allows for the learning of aggression-supportive knowledge structures (Anderson & Bushman, 2002). Given that in video games people do not physically enact behaviour but rather observe it on screen, this represents observational learning. Consequently, studies on the link between violent video games and aggressive behaviour can demonstrate the strength of observational learning of aggression-supportive cognitive structures. Although a recent meta-analysis by Mathur and VanderWeele (2019) demonstrated that violent video games do facilitate aggressive behaviour, the standardised effect size of influence was always minor (i.e., < .20). This suggests that

observational learning might not have precedence over enacting learning. Furthermore, in a longitudinal study whereby 90 adults played a violent or non-violent video game or did not play at all for a two month period (Kühn, Kugler, Schmalen, Weichenberger, Witt, & Gallinat, 2019), no effect on aggression was observed. Although this study raises further doubt about focus on observational learning, it is important to note that neither the GAM nor the IIPM differentiate between retrieval and application of differently acquired cognitive structures. In this sense, regardless of the source from which a script was acquired, be it real or fictional violence, the script can still be learned and later accessed (Huesmann, 2018).

Although socio-cognitive models do emphasise observational learning, they also describe the necessity of script enactment (Anderson & Bushman, 2002; Huesmann, 2018). Therefore, if it is the exposure to real (rather than fictional) violence that entrenches aggressive behaviour, the socio-cognitive models still stand, albeit requiring a shift in emphasis. Similarly, holding the premise that aggression is not learned behaviour as true does not refute the process of selecting a behavioural script proposed by these models.

Lastly, the assumption that aggression is automatic is challenged by Ferguson and Dyck (2012); nevertheless, it is unclear why this is attributed to the GAM. The last stage of the model includes decision-making and states that when there is a surplus of mental and temporal resources, a thoughtful action is chosen out of those options that were re-appraised. It is important to note that apart from the last objection, the criticism of Ferguson and Dyck (2012) rightly highlights the aspects that the socio-cognitive models have not outlined thoroughly. Although it does not refute them entirely, it warrants a further discussion of principles that govern aggressive behaviour.

Thus, in conclusion, although not without flaws, socio-cognitive models provide clear contributions to our understanding of aggression; the Information Processing Model and

Integrated Information Processing Model describe pathways by which aggressive scripts can be acquired and retained (Huesmann, 1988; 1998; 2018). Understanding their development helps identify the formation of repeated aggression. Furthermore, the notion that scripts guide aggressive behaviour have been applied to more specific offending behaviours, such as sexual offences (Gilbert & Daffern, 2017). The notions of scripts, rehearsal and formation of habitual aggression in childhood have been incorporated in current risk assessment instruments in the form of such items as "violent ideation or intent" and "history of problems with violence" (Douglas et al., 2013).

Socio-cognitive models also provide an outline of the mechanisms by which aggressive scripts become aggressive acts in a given social encounter. However, due to their focus on knowledge structures, their description of other factors influencing aggressive behaviour is limited. Moreover, although they discuss information processing that leads to the selection of aggressive scripts, they do not provide a thorough account of the neural structures that are related to this process. This in turn means that they neglect a neurocognitive dimension, findings from which can provide not only a specification of the influences of cognitions but also suggest the magnitude of effect exerted by other factors. Consequently, despite the absence of a formulated and unified model of aggression from neuropsychology, the findings on the aetiology of reactive and proactive aggression should be integrated with socio-cognitive models.

2.4 Neurocognitive models

For neurobiological models of aggression, a bimodal conceptualisation of aggression is important, as the modes are suggested to involve distinct brain structures (Fabian, 2010). While reactive aggression is characterised as a reaction to distressing situations with a goal of reducing stress, proactive aggression is described as a premeditated use of aggression that can

be used to achieve a distinct goal (Meloy, 2006). Consequently, affective aggression is held primarily as a failure to appropriately control aggressive responses to the environment. Accordingly, research has suggested that this type of aggression is underpinned by increased neural activation in the threat system (including basic amygdala, hypothalamus and periaqueductal grey [PAG]), coupled with decreased activity in frontal regions, which are associated with behaviour inhibition (Blair, 2016; Coccaro, Sripada, Yanowitch, & Phan, 2011). Meanwhile, proactive aggression is deemed a selection of aggressive conduct that results from a failure to learn the association between aggressive behaviour and negative outcomes (Blair, 2016). It is therefore suggested that this type of aggression is related to functional impairments in the amygdala and orbitofrontal cortex (Finger et al., 2012; Marsh et al., 2011).

Although there is a certain similarity in the structures involved in these two types of aggression, there is a difference in the patterns of their functioning. The amygdala plays an important role in a range of emotions and impacts both reactive and proactive aggression. In the case of the former, increased activation of the amygdala in response to provocation has been shown to be present among adult violent offenders and adolescents (aged 10 to 18) who were assessed as having a history of reactive aggression (da Cunha-Bang et al., 2017; White et al., 2015). Its heightened activation has also been used to explain variation in past reactive aggression of adult males from a general community sample (Farah, Ling, Raine, Yang, & Schug, 2018). Meanwhile, those who repeatedly engage in proactive aggression show lower levels of amygdala activation in response to distress compared to non-instrumentally aggressive individuals (Blair, Veroude, & Buitelaar, 2018; Lozier, Cardinale, VanMeter, & Marsh, 2014). Similarly, reduced amygdala volume has been linked with increased aggressive conduct persisting through development until adolescence (Pardini, Raine, Erickson, & Loeber, 2014). This importance of the functionality and structure of amygdala for aggression raises the

possibility that the influence of affective states and arousal noted in the socio-cognitive models of aggression can be of the same magnitude as that of cognitions.

However, for both affective and proactive aggression, the degree of amygdala activation does not provide a full explanation of the resulting conduct. For the reactive type, it needs to be coupled with a failure of the prefrontal cortex in inhibiting the impulses incoming from the threat system, which includes the amygdala (Coccaro et al., 2011). Indeed, research has supported this assumption, as those with the propensity for reactive aggression do show decreased fronto-limbic connectivity (Chester & DeWall, 2015; da Cunha-Bang et al., 2017). Meanwhile, in the case of proactive aggression, amygdala dysfunction is part of the explanation for the acquisition and maintenance of aggressive conduct, rather than facilitating aggression in a particular situation. Specifically, this impairment is postulated to prevent learning an association between stimulus and a negatively valenced outcome (Blair, 2005). Furthermore, Blair (2005) suggests that during development, cues demonstrating the distress of others should trigger an aversive emotional state. However, when there is an inability to form aversive associations, this unpleasant emotional state is not linked with the behaviour. This lack of association leads to evaluation of an aggressive act and its consequences without taking into account the harm caused to others. As a result, the value of aggressive behaviour is determined solely by the achieved benefit to the aggressor, regardless of the impact on others, which is usually of negative value.

Thus, proactive aggression is associated with a lack of empathy towards victims and is used solely due to its utility for the aggressor. Indeed, it has been shown that those who reported engagement in proactive aggression are less susceptible to emotional priming of their emotional response, via task-irrelevant pictures of happy or angry facial expressions, in a decision-making task with a monetary incentive attached to correct responses (Ly et al., 2014). Similarly, when presented with images of others in distress, those with psychopathic traits (a

disposition that is associated with proactive aggression) show less activation in the ventromedial prefrontal cortex (vmPFC)(Decety, Skelly, & Kiehl, 2013), a region associated with responsivity to reward (Morelli, Sacchet, & Zaki, 2015).

Taken together, this evidence supports Blair's (2016) proposition that dysfunctions in both amygdala and vmPFC underpin proactive aggression. While the low responsivity of amygdala undermines the ability to associate aggression with negative outcomes, the impaired vmPFC does not facilitate evaluation of the predicted outcomes of actions (White et al., 2013). This argument for a joint effect of amygdala and vmPFC dysfunction on aggressive conduct allows for the proposal of clear neural pathways for both steps noted in the GAM (Anderson & Bushman, 2002). The impaired amygdala functioning affects threat detection; for instance, by prioritising threatening cues (Bach, Hurlemann, & Dolan, 2015), which can skew situation perception (the first step in the GAM) towards hostility. Similarly, in terms of value assessment, impaired vmPFC is likely to affect the evaluation of applicability of a cognitive structure (the last step in the GAM) to a given situation.

Although this distinction has utility for studying the functional and structural networks of aggressive behaviour, there is one noteworthy aspect: in both reactive and proactive types, aggressive conduct results at least partly from failures in decision-making. Meanwhile, the centrality of decision-making processes to aggressive behaviour has been noted by the socio-cognitive models. In the case of reactive aggression, the impulses are not inhibited and are enacted; in the case of proactive aggression, such conduct is chosen as suitable. The prefrontal cortex is involved in both types (Blair, 2004). Furthermore, Blair (2016) argues that impairments in vmPFC can serve the same function in reactive aggression as in proactive. Specifically, since the vmPFC modulates the value of expected outcomes of the action, it can misrepresent the value of an aggression reaction to a provocative stimulus. Although this potential overlap does not render these two types of aggression equal, as they are suggested to

have aetiological differences, it does suggest potential for a unifying model of aggressive behaviour that has distinct origins, but is acted out through failures in decision-making.

The neurobiological models of reactive and proactive aggression clearly highlight distinct pathways that help to explain aggression, which is complementary to socio-cognitive models. The suggested role of the vmPFC in value representation (Blair, 2016) concurs with the idea of evaluation of behavioural scripts with respect to internalised norms (Huesmann, 1988; 2018). Similarly, the neural activation during reactive aggression has been interpreted within the GAM framework (Kramer, Jansma, Tempelmann, Munte, 2007). Moreover, the suggestion of a failure to learn the association between aggression and negative outcomes does echo Huesmann's (1988) propositions about entrenchment of aggressive scripts though flawed post-hoc evaluation of outcomes. Blair (2005) suggests that proactive aggression develops mainly due to the inability to attach negative value, stemming from the distress cues of others, to aggressive behaviour. Similarly, Huesmann (1998) suggests that aggressive behavioural scripts are solidified when they bring about the intended results. Thus, if a person cannot attach a negative outcome (representing harm to others) to their aggressive behaviour, their assessment of a script's utility is self-centred.

These overlaps suggest that neurobiological models provide more detail on the potential pathways for two specific types of aggression (reactive/proactive) that further, rather than dismiss, socio-cognitive explanations. Nevertheless, these two approaches have not yet been fully integrated. Furthermore, despite noting the role of genetics, they have not outlined how the interaction of genes and environment could potentially shape the mechanisms that facilitate aggressive conduct.

2.5 Genetic Models

There are two main genetic models of aggressive behaviour, which suggest pathways to such behaviour (Zhang, Cao, Wang, Ji, & Cao, 2016). Both posit that aggression, similarly to

other behaviours, is influenced by both genes and environment. The first is the *Diathesis-Stress model*, postulating that individuals who possess alleles associated with increased aggression can demonstrate habitually aggressive behaviour if they have suffered a harsh environment (Caspi et al., 2002). Within this model, certain people have a higher risk of engaging in aggressive behaviour when their environment is stressful. Necessarily, this implies that there are other people who will not engage in aggression in similar environments. In terms of interaction between genes and environment, the Diathesis-Stress Model favours genes, but specifies that for their expression, a specific environment is needed. The other genetic model is the *Differential Susceptibility model*, which proposes that people have alleles indicating their plasticity in responses to the environment, which they internalise (Belsky & Pluess, 2009). This approach can be exemplified by Belsky's (1997) proposition that for people, levels of aggression in their environment corresponds to levels of aggression they exhibit. In terms of gene and environment interaction, the Differential Susceptibility model clarifies that this interaction differs between people depending on the genes they have.

These two models do not have to be perceived as different paradigms, rather the Differential Susceptibility model can be seen as a further elaboration on the link proposed by the Diathesis-Stress model. Simons and colleagues (2012) demonstrated that individuals with more aggression-associated alleles behaved more aggressively when they experienced aggression rich environments, compared to those who have fewer such alleles. However, they were less aggressive when they were in less adverse environments.

This pattern of interaction, where genes impact aggression through their influence on reactions to the environment, which in turn shapes conduct, was confirmed with further research (Bakermans-Kranenburg & Van Ijzendoorn, 2011; Buchmann et al., 2014; Hygen et al., 2015; Simons et al., 2011; Zhang, Cao, Wang, Ji, & Cao, 2016). Specifically, a meta-analysis by Bakermans-Kranenburg & Van Ijzendoorn (2011) demonstrated that children

possessing less efficient dopamine-related genes were more affected by the environment than others. When the environment was negative, they were more likely than others to display adverse behaviours, including externalising. However, when the environment was positive, they were also more likely to display positive behaviour. Similarly, Hygen and colleagues (2015) showed that general population children carrying Catechol-Omethyltransferase Val158Met interacts with exposure to stressors in predicting aggressive behaviour. Meanwhile, Zhang and colleagues (2011) demonstrated a similar pattern among a community sample of adolescents, as their aggression was associated with an interaction between genes and environment. However, the effect of parenting differed depending on the presence of alleles indicating susceptibility to environmental influences. Buchmann and colleagues (2014) extended this work further into adults. Using a cohort study with the general population, they found that aggressive behaviour in 19-23 year olds was associated with having a seven-repeat allele of the dopamine D4 receptor (DRD4) gene and being exposed to prenatal maternal stress.

This pattern suggests that the relationship between genes and aggressive behaviour is indirect, which corresponds to the proposition of socio-cognitive models that aggression is an enactment of behavioural scripts that were acquired from the environment. Meanwhile, the Differential Susceptibility model specifies that genetic composition influences the ease with which the behaviours shown in the environment are internalised. Furthermore, analyses by Simons and colleagues (2011; 2012) show that aggression supportive cognitions (in the form of attitudes favouring aggressive conduct) act as mediators between the genetic response to environment and aggression. Thus, those who are more affected by the nature of their environment (hostile or non-hostile) are also more likely to internalise norms containing aggressive conduct as effective or appropriate from their environment and demonstrate aggressive behaviour. Given that socio-cognitive models of aggression provide a framework that explain the pathways through which aggression supportive cognitions emerge as conduct,

these findings support unification of the Differential Susceptibility and Integrated Information Processing models. An example of such an effort is the I^3 model (Finkel & Hall, 2018).

2.6 I^3 Model

The main way the I^3 ('I cubed') framework differs from other models discussed in this chapter is that it is suggested to be a metatheory. A metatheory is a theory that has other theories as the subject (Edwards, 2014; Van den Bos & American Psychological Association, 2007). However, Finkel (2014) extended this definition and proposes that a metatheory is a number of assumptions that are held as true and that can be used as a basis for the development of specific theories. Consequently, the I^3 model is posited as a guide for the creation of new theories explaining principles governing behaviour, including aggression (Finkel, 2014).

According to this model, aggression results from three main processes (Finkel & Hall, 2018), each of which can vary without affecting others. Instigation is defined as "the effects of exposure to a particular target object in a particular context that normatively affords a certain behaviour, with 'affords' referring to the target-object directed behavioural options that the target object furnishes the individual" (Finkel, 2014, p. 11). So, this process includes a range of responses to certain stimuli. It is important to immediately note that using the word 'object' in the definition of the process raises a question about an 'absent object'. For instance, when a person's expectations about behaviour in response to them are violated and others show no response, does this still constitute instigation? This question is not yet answered within the I^3 model.

In terms of previously described models, instigation includes the situation factors of the output stage of the GAM or the situational determinants from the IIPM. It also encompasses the provocation or stress from the environment that is posited to elicit reactive aggression. It is highlighted that a behaviour in a given situation partly results from cues or provocation by

others (Anderson & Bushman, 2002; Fabian, 2010; Finkel & Hall, 2018; Huesmann, 2018). However, while the GAM also includes non-external factors (e.g., pain or frustration) in the situational factors, the I^3 model separates such variables into a different category.

This next process is called impellance, which is posited to include "the effects of situational or stable factors that increase the likelihood that (or the intensity with which) the individual will experience a proclivity to enact the afforded behaviour when encountering that target object in that context" (Finkel, 2014, p. 12). These effects are suggested to occur either through affecting the psychological state of a person at the moment when they meet the instigator or through changing its perception afterwards. Given that Finkel (2014) further clarifies that while instigation concerns factors inherent to the specific object, impellance denotes factors inherent to the experience of the object. The provided description is that of subjective factors affecting perception and interpretation of the stimuli.

Similarly to instigation, this process can also be found in socio-cognitive, neurocognitive and genetic models. Impellance roughly corresponds to the person factors from the input stage of the GAM, together with the routes stage that includes affect, cognition and arousal influencing a person's psychological state. It is also similar to personal predispositions in the IIPM, and the knowledge structures that support aggressive behaviour emphasised in all socio-cognitive models. It is also reflected in the increased activity of the threat system argued to underpin reactive aggression (Blair, 2016; Coccaro, Sripada, Yanowitch, & Phan, 2011), and in the skewed evaluation of aggressive conduct's utility suggested to account for proactive aggression (Blair, 2004; 2005). Similarly, the Differential Susceptibility model states that there are specific alleles that correspond to the extent with which a person is affected by their environment and object within it (Belsky & Pluess, 2009). All of these factors represent the individual's biological or cognitive idiosyncrasies that affect how they perceive the objective

situation they are in. Consequently, these factors affect how the conduct for a given situation is selected.

The last process in the I^3 model is inhibition that "encompasses the effects of situational or stable factors that increase the likelihood that (or the intensity with which) people will override the effects of instigation and impellance, thereby reducing the likelihood or intensity of the behavior." (Finkel, 2014, p.13). This definition explains that despite the name, there can be reverse inhibitors that facilitate rather than inhibit a specific response. Thus, the last process of the I^3 model includes factors that can both amplify or deter intention to engage in aggressive behaviour.

With respect to the models described earlier, inhibition can be found in the socio- and neurocognitive frameworks. The GAM and the IIPM note the effect of cognitive resources on the appraisal and reappraisal of selected behavioural scripts. Similarly, reactive aggression is shown to be a result of poor impulse inhibition (Chester & DeWall, 2015; da Cunha-Bang et al., 2017), and proactive aggression is suggested to result from lack of such inhibitors as valuing the distress of others (Blair, 2005). Moreover, it appears that within I^3 model, the decision-making process that is suggested to immediately precede aggressive behaviour in the GAM (Anderson & Bushman, 2002) and neurocognitive models (Blair, 2016) is also part of the inhibition component, as it is during the decision-making stage that a behavioural script is chosen to be '(dis)inhibited'.

In addition to outlining the three processes, the I^3 model stresses that it is focused on processes that can have different operationalisations in empirical research (Finkel, 2014). It also highlights that the immediate predictor for aggression in a given situation is "behavioural proclivity," which is the impulse to behave in a certain manner. It is formed through an interaction between instigation and impellance, and whether or not it is manifested as behaviour

is contingent on its strength relative to inhibition (Finkel & Hall, 2018). This points to another aspect of the I^3, namely that the three described processes can have both direct and indirect (through interaction) effects on aggressive behaviour.

Although the I^3 metatheory has undeniable utility, it also has certain limitations. Its central postulations can be formulated as follows. Behaviour, including aggression, results from (dis)inhibition of desire to act in a certain manner that is shaped by the way an individual's idiosyncrasies influence perceptions of objective stimuli. In this form, this statement appears dangerously close to a truism, as it is a general statement reflecting an agreed-upon understanding of behaviour. However, using this metatheory as a foundation for future research might prove useful as it will lead to results existing within one system or paradigm. Nevertheless, on its own, due to very general terms and lack of specific operationalisation, the I^3 metatheory does not point to specific mechanisms which result in aggressive behaviour. Moreover, since according to the I^3 all three processes can have direct as well as indirect effects on aggression, every research finding about specific patterns of relationships is acceptable. Lastly, the use of the term 'inhibition' for the third process might be misleading. It implies that when aggressive behaviour occurs it is due to "disinhibition" or "weak inhibition," which in turn creates an inherently negative connotation. However, similarly to any behaviour, aggression is neither positive nor negative without context. Second, the term inhibition implies that the third process acts on another process, and this is what the I^3 model suggests. This, in turn, leads to an unanswered question of how the inhibition process can have a direct effect on behaviour, which is proposed in the I^3. By description it is an amplifier or a buffer, but it is not a cause. One way to reconcile both of these issues is to amend the term 'inhibition' in favour of 'decision-making' or 'evaluation processes'.

2.7 Concluding Comments

Aggression is a complex behaviour that is affected by different factors, ranging from the emotional state of a person in a particular situation (Robertson et al., 2012) to specific alleles (Simons et al., 2011; 2012). Although there are models that emphasise distinct factors, it appears possible to incorporate them into one framework by adopting cognitive structures and decision-making process as core facilitators of aggression. Socio-cognitive, neurocognitive and genetic models of aggression do not contain inherently unresolvable contradictions that make them incompatible. Instead, all of them support the principle that behaviour reflects an individual's cognitions, but they each shed light on different aspects of this process.

The potential for integration of the described models highlights two main principles governing aggression. Firstly, such conduct is a result of decision-making, which can be automatic, conscious or both (Anderson & Bushman, 2002; Blair, 2005; 2016). One of the main corollaries from this is that factors influencing decision-making will affect aggressive behaviour. The second principle is that aggression in any given situation is an enactment of knowledge structures that outline and approve of such conduct (Huesmann, 2018). A necessary consequence of this is that variables determining the formation of these structures will influence aggression.

There is capacity for a third principle, which can be derived from socio-cognitive and neurocognitive models. The GAM considers arousal and affective states as routes through which situational and individual inputs reach the appraisal stage. Similarly, the conceptualisation of reactive aggression highlights dysfunction in the threat system as being predictive of aggression. Given that stress is a high form of arousal involving the amygdala to a considerable extent (Di et al., 2016), it is possible that 'reactivity to stress' is the third principle. The addition of this third principle would also map onto the I^3 model. Since stress represents environmental demands on an individual, it reflects the instigation process of the I^3.

The definition of affective aggression as a reaction to stressors aimed at the reduction of arousal attests to the suggested link between stress and aggression (Fabian, 2010). This implies that stress has a direct influence on aggression. Moreover, stress has been shown to influence both decision-making and learning, specifically by altering sensitivity to rewards and punishment and facilitating habitual behaviours (Porcelli & Delgado, 2017). This suggests that since stress affects decision-making processes, from which aggression is suggested to result (Anderson & Bushman, 2002; Blair, 2016; Finkel & Hall, 2018), stress indirectly affects aggressive behaviour.

Furthermore, aggression-related behavioural scripts are proposed to be stored in memory (Gilbert & Daffern, 2017), and stress has been shown to facilitate memory consolidation but inhibit memory retrieval (Smeets, Otgaar, Candel, & Wolf, 2008). This, in turn, suggests that higher levels of stress facilitate the acquisition of aggressive scripts when they are enacted, as they will be encoded. If high stress inhibits recall, it can also impose limitations on the range of behavioural scripts that can be accessed in a given situation. Therefore, an individual with a wide array of aggressive scripts, but a limited range of non-aggressive scripts, might not be able to even consider the latter as they would not be retrieved. However, a person with low amount of aggression-supportive cognitions is less likely to behave aggressively under stress, as this person has different habits of behaviour. Given that even theoretically stress can have two different effects on aggressive behaviour that would be dependent on the presence or absence of aggression-supportive cognitive structures, stress is likely to have an indirect, rather than direct, influence on aggression.

Stress certainly appears to be interconnected with aggression, both during learning scripts for it, and when enacting them. It also has the potential to affect aggressive conduct through its influences on decision-making or learning (Porcelli & Delgado, 2017; Starcke & Brand, 2012; 2016). However, before postulating a principle by which stress affects aggression, there needs

to be a more in-depth exploration of what stress is. The next chapter begins with addressing this question and follows by showing that stress influences processes that precipitate and facilitate aggression.

CHAPTER THREE: THE EFFECTS OF STRESS RESPONSE ON PRECURSORS OF AGGRESSION

3.1 Structure of the Chapter

This chapter focuses on the effects of experiencing acute stress on the processes facilitating aggression. It begins with a short discussion about the concept of stress and its history, which is followed by a description of physiological stress response systems. Then, since Chapter 2 suggested that aggressive behaviour results from decision-making, the current chapter illustrates the influence of stress on this cognitive process. Furthermore, as behavioural scripts are thought to be stored in memory (Huesmann, 2016), the toll that stress takes on information retrieval will be discussed. This will be followed by an investigation of patterns in value-driven actions following acute stress exposure to account for the neurocognitive models of aggression (Blair, 2016). In addition, the relationship between stress and working memory capacity is explored, as the latter corresponds to the pool of available behavioural scripts that are retrieved from memory (Huesmann, 1998), evaluation of them and other aggression-supportive cognitive structures (Anderson & Bushman, 2002), and can act as an inhibition process noted in the I^3 model (Finkel & Hall, 2018). Lastly, the relevance of individual characteristics in affecting the response to a stressor is outlined based on the relationship between stress and anger. The chapter concludes by proposing an indirect effect of stress on aggression, occurring through aggression supportive knowledge structures and cognitive processes.

3.2 Brief History of the Concept of Stress

Claude Bernard (1865; as cited in Goldstein & Kopin, 2007) developed the idea of "milieu interieur" (internal environment), postulating that maintenance of the internal environment of the body is crucial for the autonomous survival of an organism (cited in Goldstein & Kopin, 2007). Developing this idea, Cannon (1939) introduced the term

"homeostasis" referring to a dynamic sustenance of given parameters of an organism within satisfactory limits. The main requirement for upholding the limits is negative feedback systems that can detect when the current value of a physiological parameter is incongruent with the satisfactory range, and subsequently initiate a process that restores the values. Consequently, Cannon argued that a "fight or flight" response is initiated by such a system when homeostasis is threatened. Meanwhile, Selye (1936) introduced the term "general adaptation syndrome" to describe the physiological reactions of rats that were subjected to potentially harmful stimuli. He suggested that the function of this syndrome was to facilitate the adjustment of the organism to new conditions. This was the foundation of his development and popularisation of the concept of stress, which he defined as "the nonspecific response of the body to any demands upon it" (Selye, 1974; as cited in Goldstein & Kopin, 2007).

Currently, stress is widely deemed to result from any threat to homeostasis, regardless of whether it is registered consciously or automatically (de Kloet, Joels, & Holsboer, 2005; Sandi & Haller, 2015). However, contrary to Selye's (1974) proposition, a stress response is now considered to be, to some extent, specific to the nature and perception of the threat and influenced by an organism's belief about their self-efficacy in dealing with it (Goldstein & Kopin, 2007). Further development of the concept involved introduction of "allostasis." This refers to establishing stability through changes in the satisfactory variation limits of the cardiovascular system's markers. While in the short term such changes can be adaptable, sustaining them in the long term produces negative results, the risk of which is the "allostatic load" (de Kloet, Joels, & Holsboer, 2005).

As stated previously, maintaining homeostasis rests on the ability to detect the distinction between current and set values and to then ensure that the values are brought within the needed range. "Homeostats" refer to such detectors, where the needed values are set by regulators and contrasted against the values obtained based on afferent information. In cases of a discrepancy,

effectors are triggered to reduce it (Goldstein & Kopin, 2007). Although this model allows us to conceptualise a response to stress, it is simplified. Not only are there usually several "effectors" for one variable, it is possible for one effector to be governed by several homeostats simultaneously. Lastly, it is important to note that distress is a cognitive form of stress based on the perception of a stressor's nature and strength and on an individual's perceived self-efficacy in coping. It is not an inherent part of every threat to homeostasis. However, in response to both stress and distress, two main systems of individual's organism are activated.

3.3 Stress Response Systems

The Sympathetic Nervous System (SNS) and Hypothalamic-Pituitary-Adrenal (HPA) axis are the two main systems in the human body that are engaged when stressors, defined as threats to homeostasis, are encountered (Carrasco & Van de Kar, 2003). Consequently, the corticotropin-releasing hormone (CRH) and norepinephrine are deemed to be the regulators of the stress systems (Chrousos, 2009). Encounters with threats to equilibrium trigger behavioural and physiological responses directed at restoring homeostasis, which are collectively referred to as the stress response (de Kloet, Joels, & Holsboer, 2005). As specified in Chapter 2, the increased activation of the threat detection system is associated with reactive aggression (Blair, 2016; Coccaro et al., 2011). Similarly, attribution of hostile intentions to others makes them more likely to be perceived as a threat and to elicit an aggressive response (Tuente et al., 2019). Thus, reactive aggression can be among the last stages of the stress response.

A first step in the stress response is an increase in the activity of the sympathetic nervous system that initiates a release of adrenaline and noradrenaline (catecholamines) from the adrenal medulla. This is followed by the release of CRH (also known as corticotropin releasing factor [CRF]) and vasopressin (AVP) from the hypothalamic paraventricular nucleolus (PVN), and of oxytocin from the posterior pituitary. Within the 5 to 10 seconds that follow, the pituitary secretes the adrenocorticotropin hormone (ACTH). At the same time, CRH and AVP engage

the HPA axis, leading to secretion of cortisol (corticosteroids); this facilitates energy metabolism, thereby preparing the body to respond to the stressor (Carrasco & Van de Kar, 2003; de Kloet, Joels, & Holsboer, 2005).

This cascade of neuroendocrine responses is also referred to as a 'fast stress system' that involves 'fight or flight' behaviours. In cases when the source for the threat to homeostasis are other people, aggression becomes an inherent part of the fight response. However, there is also the 'slow system,' which corresponds to recovery and adaptation after the initial response. Although corticosteroids are present in both systems, they act via different receptors. The fast stress system engages mineralocorticoid receptors (MR), suggested to be activated in the onset of stress response. However, the slow system incorporates glucocorticoid receptors (GR) which are triggered by abundant amounts of corticosteroid; they terminate the stress response and prepare for recovery (de Kloet, Joels, & Holsboer, 2005).

It is also important to note that CRH can be activated both by sensory stimuli passed to the Hypothalamic Paraventricular Nucleolus (PVN) via brain stem pathways, and by psychological stimuli triggering the limbic system (de Kloet, Joels, & Holsboer, 2005). This suggests the importance of the appraisal of a situation, and other psychological factors such as the perceived ability of prediction and control over the event that will follow. Consequently, three general categories of stressors can be outlined: 1) psychological stressors that are underpinned by a learned reaction to anticipation of negative states (e.g., fear); 2) a category corresponding to cardiovascular stressors that are mostly physical and threaten cardiovascular homeostasis (e.g., exercise); and 3) combined stressors where a physical stimulus is exacerbated by the psychological representation of it (e.g., pain) (Carrasco & Van de Kar, 2003).

Although aggression has been linked with all three types of stressors, stress does not have a direct immediate and unimpeded path to aggressive conduct. Barlett, Madison, DeWitt, and Heath, (2019) have demonstrated that in a community sample of men and women, tendency to fear retaliations from others was associated with aggression. They also specified that in a given situation, the fear of retaliation interacted with provocation in the form of negative feedback on their work (both are psychological stressors) in predicting aggressive behaviour. Similarly, Tiihonen, Halonen, Tiihonen, Kautiainen, Storvik, & Callaway, (2017) demonstrated that an increase in weather temperatures in Finland was associated with heightened rates of violent crime, thereby pointing to a link between physiological stress (temperature) and aggression. Nevertheless, they also specified that this association is likely accounted for by concurrent changes in serotonin (5-HT) transporter density, which has been linked to aggression via poor impulse inhibition (Fitzgerald, 2011). This relationship is also present for combined stressors such as pain. For instance, Kosson, Malec-Milewska, Gałązkowski, and Rzońca (2018) found an association between pain intensity and aggression among men and women attending a pain clinic.

However, investigating the path of stress's influence on aggression through the theoretical models clearly suggests that stress does not immediately result in aggression. The GAM (Anderson & Bushman, 2002), IIPM (Blair, 2016) and neurocognitive models (Huesmann, 2018) suggest that any external influence on an individual needs to go through decision-making to elicit aggressive conduct. Despite the presence of a direct path in the I^3 model from instigation (stressor) to aggressive behaviour, Finkel and Hall (2018) suggest that inclination to behave in a certain way resulting from instigation needs to be either inhibited or disinhibited to manifest as conduct. Nevertheless, in order to accurately describe the effect of a stress response on decision-making processes or other inhibitors from the I^3 model, the methods for eliciting such a response need to be discussed.

3.4 Induction of Stress Response

Currently, the concept of stress reflects systematic physiological changes evoked by a perceived threat to physical, psychological or social homeostasis (Sandi & Haller, 2015; Starcke & Brand, 2016). The two hallmarks of this response are activation of the sympathoadrenomedullary system (SAM), and then activation of the hypothalamic-pituitary-adrenal (HPA) axis. It is important to note that activation of the former without the latter can be sufficient for arousal, but together they constitute a full stress response.

Taking this double activation into account, it appears that for humans, specific psychosocial stressors are more threatening that purely physical ones. Performing a public speech to a non-responsive audience followed by performance of a cognitively taxing task, which are the main steps in the protocol of the Trier Social Stress Test (TSST), represents a psychological stressor with socioevaluative threat at its core (Kudielka, Hellhammer, Kirschbaum, Harmon-Jones, & Winkielman, 2007). Undergoing the TSST activates both SAM and HPA systems, thereby allowing it to be considered a standard stressor. At the same time, using only a physical threat to homeostasis, such as holding a hand in cold (0-5° C) water for a maximum of three minutes ('cold pressor task' [CPT]), can also be a stressor. However, although the CPT results in SAM activation, its effect on the HPA axis is less pronounced (Schwabe, Haddad, & Schachinger, 2008). This led to the addition of socioevaluative threat to the CPT, in the form of a non-responsive experimenter videotaping participants, which resulted in more robust activation of both SAM and HPA among healthy male students (i.e., 'Socially Evaluated Cold Pressor' [SECP], 'Maastricht Acute Stress Test' [MAST]) (Smeets, Cornelisse, Quaedflieg, Meyer, Jelicic, & Merckelbach, 2012). Thus, the socioevaluative component of psychological stressors appears to evoke the full stress response. The nature of psychological stressors cannot be inherent only to objective stimuli evoking stress, which is the definition of an instigator (Finkel, 2014). It is also influenced by an individual's idiosyncrasies, thereby

making some elements of stress a part of impellance processes. However, the stress response can also affect I^3 inhibition processes in form of decision-making.

3.5 Effects of Stress on Decision-making

In Chapter 2 it was argued that both reactive and proactive aggression result from a failure in decision-making, primarily due to impaired vmPFC functioning leading to misrepresented value of a behavioural outcome (Blaire, 2004; 2016). Given that stress has a documented effect on decision-making (Starcke & Brand, 2012; 2016), it appears plausible that through this cognitive process, stress will influence aggression. Therefore, it is worthwhile to first investigate the nature of stress's impact on decision-making, and then to assess it with regard to aggressive behaviour.

The aforementioned definition of the stress response suggests that its primary aim is to restore homeostasis. In order to achieve this, the stress response systems elicit physiological and psychological changes. Due to the activation of the SNS and release of catecholamines, arousal becomes an integral, yet not sufficient, part of the stress response. Consequently, its physiological markers, such as dilated pupils, increased heart rate and increased blood flow in muscles, appear as first reactions to a stressor. These changes provide an opportunity for an active 'fight or flight' response and are underpinned by the SAM system (Everly & Lating, 2019). Furthermore, they are followed by increased secretion of cortisol, which is activated by the other system responsible for reaction to stress: the HPA axis.

The joint functioning of the SAM system and HPA axis has been linked to the prefrontal cortex, amygdala and hippocampus (Dedovic, Duchesne, Andrews, Engert, & Pruessner, 2009; Joels & Baram, 2009), which are also associated with decision-making (Gowin, Mackey, & Paulus, 2013; Gupta, Koscik, Bechara, & Tranel, 2011; Weilbächer & Gluth, 2017). In a small-scale literature review of 17 studies, Starcke and Brand (2012) showed that stress can affect

decision-making processes on several levels. Experiencing stress influences the strategy used to make decisions, processing feedback from them (including the information about rewards and punishment associated with a decision) and the ability to make inferences based on pre-existing knowledge. However, in a subsequent meta-analysis of 26 studies, Starcke and Brand (2016) demonstrated that although stress plays a role in decision-making, it does not determine the outcome. Specifically, their results revealed that the nature of the situation in which a decision is made also affects the outcome. In circumstances where high risk-taking was not associated with losses, stress did not exert any effect on the outcome. These findings suggest that stress facilitates engagement of a particular strategy that is beneficial in one circumstance but disadvantageous in others.

The primary characteristic of the decision-making strategy during acute stress is its altered sensitivity to rewards and punishment, as compared to a non-stressed strategy (Starke & Brand, 2012; 2016). Indeed, Preston, Buchanan, Stansfield, and Bechara (2007) demonstrated that adult male participants, who were informed that they would have to deliver a speech describing what they disliked about their appearance to people behind a two-way mirror, took more time to learn an efficient strategy in a gambling task than male participants who received no such instructions and female participants in both conditions. This suggests that after encountering a stressor, individuals are less efficient in learning the rewards and punishments associated with the choices available to them. However, there is a potential problem with this study. Specifically, the manipulation check for the stressor was done only via heart rate (HR), which signals SAM system activation but not necessarily that of the HPA axis. Moreover, they did not investigate the relationship between heart rate and performance.

Nevertheless, their limitations were corrected by Van den Bos, Harteveld, and Stoop (2009) who, in addition to using the standard TSST procedure before the gambling task, measured cortisol as a marker of HPA axis activation in a sample of adult men and women.

They replicated the previous findings by demonstrating slower learning of losses and gains associated with choices in adult men with higher cortisol response to the stressor, in contrast to those who were not stressed. Furthermore, Van den Bos and colleagues (2009) demonstrated that cortisol response has a linear relationship with choosing the options associated with high rewards and higher risk, but which result in an ultimate loss, rather than options with smaller immediate gains and losses, but with a net profit. Similarly, Wemm and Wulfert (2017) provided evidence for the linear relationship between heart rate, evoked by the TSST, and selection of risker options in the task. In other words, experiencing acute stress, evidenced by activation of both the SAM system and HPA axis, facilitates high risk/reward decisions in men.

In a systematic literature review of 16 studies, Kuin, Masthoff, Kramer, and Scherder, (2015) showed that increased risk-taking in decision-making is associated with aggression for both men and women. Consequently, this highlights that altered decision-making processes might mediate the relationship between stress and aggression. Fishbein and colleagues (2009) further demonstrated that, among male inmates, low executive functioning (encompassing risky decision-making) was associated with lower rates of non-impulsive and non-aggressive reactions to provocation after a Cognitive Behavioural Therapy-based program. This highlights that stress might impair attempts to change behaviour from aggressive to non-aggressive through altering decision-making. From a theoretical standpoint, this resembles the proposed influence of arousal as an internal state on (re)appraisal of behavioural scripts in the GAM (Anderson & Bushman, 2002). Moreover, in terms of the I^3 metatheory, changes associated with the stress response become part of the impellance processes that interact with the inhibition process (Finkel & Hall, 2018).

It is important to note, however, that this preference for risk under acute stress has been shown to be sex dependent (Preston et al., 2007; Van den Bos et al., 2009; Wemm & Wulfert, 2017). Specifically, while poor risk associated decision making and acute stress had a linear

relationship in males, it had an inverted U shape in females. In other words, for women, the initial stress response facilitated less risky decision-making that led to net gain; however, this effect deteriorated with time. Nevertheless, Mather and Lighthall (2012) suggest that both risk-taking trajectories are underpinned by activation of the dopaminergic system, which highlights potential rewards at the cost of overshadowing potential losses.

Indeed, neuroimaging studies have demonstrated that the experience of acute psychosocial stress increases dopamine levels in ventromedial PFC in both males and females (Lataster et al., 2011; Nagano-Saito et al., 2013). At the same, the overall neural activation of vmPFC has been shown to be reduced by exposure to a stressor, suggesting impairment in cognitive control (Ossewaarde et al., 2011). This can be explained by the results of animal studies that show PFC impairment due to excessive stimulation of dopamine D1 receptors (Hermans, Henckens, Joëls, & Fernández, 2014). Furthermore, Uy and Galvan (2017) showed that following daily (rather than induced) stressors, adolescent and adult males making uncertain decisions expecting either net gain, net loss or equal probability for both have different patterns of neural activity in the PFC. The adolescent males showed an increase in risk-taking decisions associated with both net losses and gains, yet a decrease in PFC (specifically frontal pole) activity proportionate to the level of stress. Meanwhile, adults with a high stress response showed a decrease in risky decisions associated with loss when compared to those with a low stress response; however, their PFC activation remained similar to those with a low stress response, which was higher for gain-associated risks than loss-associated ones. Uy and Galvan (2017) suggest that adolescents' continuation of loss-associated risk-taking is due to impaired cognitive control, corresponding to decreased PFC activity.

This relationship between exposure to stress and PFC functioning has been suggested to be underpinned by norepinephrine and dopamine and to have an inverted U-shape (Arnsten, 2009). In other words, both increased and decreased norepinephrine and dopamine impair the

top-down control functions of the PFC. Furthermore, while the stress response diminishes functioning of the PFC, it has an opposite effect on the amygdala (Arnsten, 2015). The initial release of catecholamines and glucocorticoids is initiated by the amygdala's projections to the hypothalamus, and also forms a positive feedback loop through which amygdala activity is increased further (Arnsten, 2009; 2015). Accordingly, the experience of acute stress has been shown to be associated with increased connectivity between the vmPFC and amygdala, but reduced connectivity between the vmPFC and dorsolateral PFC (Maier, Makwana, & Hare, 2015). Thus, the stress response is associated both with impairment of neural structures linked with executive control, and with facilitation of neural structures related to immediate reward salience.

This pattern of neural activation during acute stress corresponds to the areas implicated in reactive aggression. Those who engage in reactive aggression demonstrate lower fronto-limbic connectivity, together with increased activation of the amygdala and decreased activation of the frontal cortex (Coccaro et al., 2011; da Cunha-Bang et al., 2017; Farah et al., 2018). Given such similarities, the stress response needs to be accounted for in models of aggressive behaviour, as it is a likely candidate for an impellance process that interacts with inhibition (Finkel, 2014).

Although the full stress response facilitates risky decision-making, particularly in males, it is important to note that this does not equate stress with poor decision-making, as high risk/rewards options are not inherently flawed. Similarly, applying this decision-making tendency to social encounters suggests that acute stress will most likely facilitate choosing a script that will yield high rewards, as their values will be outstanding. Nevertheless, for this particular choice to be aggressive, such a script needs to be among the possible options. Indeed, only one of the studies included in the literature review by Kuin and colleagues (2015) had a non-offender sample. Given that violent offenders are likely to have aggressive behavioural

scripts among possible options, the results of the review might be indicative that risky decision-making is associated with aggression when aggressive conduct is among the possible choices. An individual needs to have an aggressive script in their memory and retrieve it in order to act on it (Huesmann, 2016). Thus, the potential effect of stress on aggression through altered decision-making will be contingent on memory processes.

3.6 Effects of Stress on Memory

As stated in Chapter 2, aggression results from the selection of an aggressive behavioural script, which was stored in and then retrieved from memory (Huesmann, 2018; Gilbert et al., 2013). While acquisition of such scripts is a process that takes place outside of a given social encounter, its retrieval occurs during it. Therefore, despite the documented effect of stress on the multiple memory process encoding and retrieval (Schwabe, Joëls, Roozendaal, Wolf, & Oitzl, 2012; Wolf, 2009), the focus will be on encoding and retrieval, as they correspond to how aggression supportive cognitive structures can be acquired and committed to memory and then retrieved from it.

Smeets, Otgaar, Candel, & Wolf (2008) have demonstrated that although experiencing stress facilitates encoding of emotional information by male and female students, it has a negative effect on the retrieval of emotional memories. Although their study used the CPT for stress induction, markers of both the SAM system and HPA axis were increased and were associated with impaired word recall. The reason for the polar opposite effects on memory processes is suggested to be due to the enhancing effect of amygdala activation that prevented efficient encoding. Similarly, the impairing effect of stress on memory retrieval in form of free recall was confirmed in a subsequent study that used the Socially Evaluated Cold Pressor Test (SECPT), which adds socioevaluative threat to a physical one (Smeets, 2011). Male and female students who experienced acute stress have shown poorer free recall than those who did not experience it.

Schwabe and Wolf (2014) specified that the impairment in retrieval is time-dependent. Specifically, their results show that accuracy in the recognition of words immediately after stress was not different to those who were not stressed, but with the passing of time that corresponded to cortisol increases, accuracy rates decreased. This pattern was not confirmed by a meta-analysis of 113 studies, 33 of which investigated the effect on encoding, performed by Shields, Sazma, McCullough, and Yonelinas (2017). They found neither the time between stress onset and retrieval, nor the cortisol level, to be a significant moderator. However, the results of Shields and colleagues (2017) revealed that the impairing influence of stress on retrieval was consistent across studies. It is possible that the absence of the temporal effect in the meta-analysis was due to the lack of research investigating the effect of the delay between stressor and retrieval. Moreover, it is possible that the results of Schwabe and Wolf (2014) are partly due to the use of a recognition rather than free recall task. Nevertheless, the results of Shields and colleagues' (2017) analysis do not show a moderating effect of the task type.

Furthermore, the explanation behind lack of immediate effect of stress is relatively inconsistent with the proposed "memory formation mode," Schwabe, Joels, Roozendaal, Wolf, and Oitzl, (2012) suggest that the joint increase in fast non-genomic glucocorticoids and noradrenergic activity, particularly in the amygdala, attunes the prefrontal cortex and hippocampus for encoding of information. They call this initial reaction a "memory formation mode" that focuses cognitive resources on engaging with new material at the expense of other processes, including memory retrieval (Schwabe et al., 2012). However, the joint activation of both the SAM system and HPA axis appears to be essential for this mode, as activity of the former without the latter in the very beginning of a stressful situation was shown to have a positive effect on retrieval (Schönfeld, Ackermann, & Schwabe, 2014). Meanwhile, activation of both the SAM system and HPA axis has been linked to increased encoding and decreased

retrieval (Smeets et al., 2008). This, in turn, further supports the generally impairing effect of stress response on retrieval.

In the context of social interaction, this means that during or shortly following acute stress, a person will focus more on encoding situational cues than retrieving a number of varying behaviour scripts. This in turn means that those who possess large quantities of readily available aggressive cognitive structures are likely to only retrieve them without alternative non-aggressive cognitive structures, as both are thought to be stored in memory (Anderson & Bushman, 2002; Gilbert & Daffern, 2017; Huesmann, 1988; 1998; 2018). Consequently, by limiting the possible options of cognitive structures to those that support aggression, the stress response can facilitate such conduct through its effect on memory retrieval. However, it will be contingent on an individual possessing these structures.

With the passing of time after the stressor, the modus operandi of memory is argued to change to "memory storage mode," which is underpinned by activity of slower genomic glucocorticoids and decreases in catecholamines, as compared to immediate stress response (Schwabe et al., 2012). This shift in priorities can be further explained by the brevity of the hippocampal activation in the stress response (Cadle & Zoladz, 2015). Accordingly, the meta-analysis by Shields and colleagues (2017) demonstrated that the effect of stress on encoding is significantly influenced by time. The enhancing influence of the stress response was found to be dependent on the short time interval (approximately 11 minutes) between encountering the stressor and encoding. An additional increase in performance was present when the presented information was relevant for the stressor. Unless these conditions were met, stress was found to impede encoding. Together, these findings provide support for an immediate "memory formation mode" (Schwabe et al., 2012).

Although the immediate effect of the stress response on memory retrieval requires further clarification, activity of both the HPA axis and SAM system appears to predispose individuals to the acquisition of stressor-relevant information (Shields et al., 2017). This is achieved by the prioritisation of encoding, which is followed by a focus on sustaining the obtained material. In terms of behavioural scripts, this pattern has an important implication. Impaired retrieval following an encounter with a stressor would suggest that a restricted pool of behavioural scripts is available, because fewer of them can be retrieved from memory (Daffern & Gilbert, 2017; Huesmann, 2018).

This restriction does not automatically mean that aggressive scripts will be in such a pool. However, when they are, the impaired retrieval decreases the chances that alternatives will be retrieved from memory. For example, if an individual has a tendency to attribute hostile intent to others, the first cognitive structures to be retrieved would be those that condone aggression (Anderson & Bushman, 2002; Tuente et al., 2019). In this case, an experience of acute stress before a particular situation would impair the individual's ability to retrieve further knowledge structures during the situation (Anderson & Bushman, 2002; Shields et al., 2017), making aggressive behaviour more likely and thereby becoming a risk factor for aggression.

At the same time, the suggested beneficial influence of the SAM system (Schönfeld et al., 2014), which initiates the stress response, can provide a small window of a reversed effect. In other words, immediately following exposure to a stressor, there might be a relatively short time period where more behavioural scripts can be retrieved. If this is the case, then the autonomic arousal can have a beneficial effect on resolution of social encounters, as it increases the number of behavioural options. However, while in the memory testing studies (Schönfeld et al., 2014; Schwabe & Wolf, 2014; Shields et al., 2017; Smeets, 2011; Smeets et al., 2009), material that is recalled after acute stress is provided to participants by the researchers; in social encounters, this material is experience-based. This, in turn, highlights the relevance of personal

history in terms of behaviour. The more aggression supportive cognitive structures the person has acquired from the experience of observation (Anderson & Bushman, 2002; Huesmann, 2018), the more likely they are to have them retrieved during acute stress and the less likely they are to retrieve alternative structures after acute stress. Thus, the effect of stress on aggression through memory is contingent on the contents of the memory, specifically the amount of aggression supportive cognitive structures. One of the instances when a person has a wide range of such structures is when aggression is their habitual behaviour (Huesmann, 1998). Consequently, if the reasoning provided above is correct regarding stress's effect on retrieval influencing the behavioural script selection, then acute stress should facilitate habitual rather than novel behaviour.

3.7 Effects of Stress on Goal-Oriented Behaviour

Aggressive behaviour is argued to result from enactment of aggressive behavioural scripts that are evaluated as appropriate and leading to a valued outcome (Blair, 2016; Huesmann, 2016). The formation of an association between behaviour and outcome is denoted as instrumental learning (Schwabe & Wolf, 2011). The classical explanation for it rests on the stimulus—response pair that is solidified by the rewards following the response and is deemed to underpin habitual behaviour (Schwabe & Wolf, 2011). In terms of aggressive behaviour, an exemplary pair can be: receiving an offensive remark from a person (stimuli) followed by hitting this person (response), resulting in feeling able to defend oneself or feeling pleasure from physical superiority over the other person (reward).

Meanwhile, for a response to be goal-directed rather than habitual, in addition to stimulus—response pairing, two processes need to take place: 1) an individual needs to learn that there is a causal relationship between a given action and its consequence; and 2) they need to attach instrumental value to the consequence (Schwabe & Wolf, 2011). Returning to the previous example, an instance of goal-oriented behaviour would be if a person who has a habit

of responding with hitting those who make offensive remarks, would be trying to instead respond by being assertive or leaving the situation, obtaining a newly valued consequence of peaceful resolution.

Plessow, Kiesel and Kirschbaum (2012) have demonstrated that individuals exposed to acute stress show lower cognitive control compared to those who were not stressed. Specifically, the participants undergoing a stress response showed higher error rates during cognitive tasks than non-stressed participants. Similarly, a growing body of literature shows that those experiencing acute stress favour habitual over goal-oriented behaviour (Fournier, d'Arripe-Longueville, & Radel, 2017; Smeets, van Ruitenbeek, Hartogsveld, & Quaedflieg, 2019; Schwabe & Wolf, 2010; 2011). Consequently, for those who habitually engage in aggressive behaviour, experiencing acute stress might be a risk factor preventing them from changing to more peaceful behaviour.

Jusyte and colleagues (2017) demonstrated that male prisoners with habitual rule-breaking behaviour showed lower cognitive effort in breaking new arbitrary rules than those from the community without histories of rule breaking behaviour. Given that habitual behaviours require less cognitive effort (Schwabe & Wolf, 2011), and extending the findings of this study to aggression, acute stress might make habitual aggression requiring low cognitive effort a more frequently-chosen option than newly acquired non-aggressive responses that require more effort. In order to establish whether or not this is the case, a more detailed discussion about the consistency and conditions in which stress facilitates habitual behaviour is needed.

An initial study by Schwabe and Wolf (2010) showed that participants who underwent SECPT after the consequence of their habitual behaviour was devalued still engaged in the habitual behaviour despite valuing the outcome of different behaviour more. It is interesting that this tendency for habitual behaviour was present among stressed participants despite their

expressed preference for the newly valued choice. The results were replicated in a study of Fournier and colleagues (2017) even though they used a slightly different paradigm, where the old choice was devalued not through satiety, but through an instruction stating that one of the choices previously paired with scoring points would now lead to their loss, and one of the choices previously associated with loss of points would result in gain. Regardless of this change, people who performed the Trier Social Stress Test (TSST) responded more habitually than non-stressed participants. Meanwhile, the results of Smeets and colleagues (2019) specified that engaging in behaviour which used to be linked with a valued outcome, instead of taking actions that are linked to a currently-valued outcome, is predicated on high cortisol responsivity. Although participants who were administered the Maastricht Acute Stress Test (MAST) and those from the no-stress group were not different in choosing options that acquired new values, there was a significant difference between those who responded to the stressor with high cortisol levels and non-stressed participants, as well as those who did not respond to the stressor with elevated cortisol levels.

Taken together, these results suggest that those experiencing a stress response, as evidenced by the activity of the HPA axis, behave habitually rather than in a goal-oriented way. Furthermore, this effect appears to be present even when they know that the outcome they used to expect will not occur. A potential explanation behind such an outcome relies on the increase in connectivity between the amygdala and dorsal striatum, combined with a decreased connectivity between the amygdala and hippocampus during the stress response (Vogel et al., 2014; Wirz, Bogdanov, & Schwabe, 2018).

Moreover, acute stress has been shown to be associated with impaired functioning of the PFC (Hermans et al., 2014), which is deemed to be linked with goal-oriented behaviour. Specifically, with vmPFC that is associated with value representation (Ossewaarde et al., 2011). The relevance of the PFC in the regulation of goal-oriented behaviour following acute

stress is supported by the finding that the shift to habitual acts occurred only in people with low working memory capacity (Quaedflieg, Stoffregen, Sebalo, & Smeets, 2019). This suggests that implementation of newly learned outcome values for familiar choices requires a high degree of cognitive control. Consequently, the enhancement of habit-related structures and pathways, combined with simultaneous inhibition of those related to executive control, provide fertile ground for routine-oriented behaviour.

Dysfunctions in the amygdala and vmPFC with regard to value formation are also highlighted as underpinning proactive aggression (Ly et al., 2014). Blair (2004) also suggests that these impairments lead to proactive aggression, because a person has not formed an association between aggression and a negative outcome in the form of others' distress. In other words, for them, proactive aggression is the habitual behaviour.

Consequently, it is plausible to presume that those with a history of proactive aggression that follows acute stress are more likely to behave aggressively (i.e., respond habitually). Furthermore, when a social situation resembles that which a person has experienced previously, they will be guided by the favourable outcomes from previous experiences (Fournier et al., 2017). This, in turn, suggests that stress has the potential to lead those who completed aggression interventions into a relapse (Fishbein et al., 2009). The habit-based responding will likely occur regardless of the particular circumstances of a given encounter, as long as an option for behaviour associated with a beneficial outcome is present (Schwabe & Wolf, 2010). In terms of aggression, this means that an increased chance for enactment of such behavioural scripts is contingent on the presence of an embedded association between aggressive acts and a valuable outcome, which is part of the definition of an aggressive behavioural script (Huesmann, 1988). In other words, the relationship between the stress response and aggression appears to be indirect, as they are dependent on other individual characteristics such as a history

of habitual aggression. It is likely that another aspect is working memory functioning, decreases in which facilitate habitual behaviour under stress (Quaedflieg et al., 2019).

3.8 Effects of Stress on Working Memory

The potentially restrictive effect of the stress response on the pool of available behavioural scripts was discussed in relation to memory processes. However, there is a more relevant concept that represents part of executive functions; namely, working memory. This refers to a system that holds and processes a limited amount of information used for cognitive tasks or to guide behaviour (D'Esposito & Postle, 2015). The stress response impairs working memory (Schoofs, Preuß, & Wolf, 2008; Schoofs, Wolf, & Smeets, 2009) and, as stated, appears to have a pronounced effect on behavioural responses following an acute stress (Quaedflieg et al., 2019).

Aggression is thought to result from decision-making processes where after evaluation of cognitive structures, that which supports aggressive conduct is selected (Anderson & Bushman, 2002; Huesmann, 2018). If stress has a consistent effect on working memory, then through it stress can affect aggressive behaviour as well. Sprague, Verona, Kalkhoff, and Kilmer (2011) found that for men and women from the general population, executive functioning, which includes working memory, moderated the relationship between self-reported stress and aggression. Participants with lower levels of executive functioning showed a stronger relationship between perceived stress and aggressive behaviour than those with higher levels of executive functioning. These results place working memory into the inhibition processes for aggression in the I^3 model (Finkel & Hall, 2018). Consequently, the effect of stress response on working memory would represent an interaction between impellance and inhibition processes leading to aggressive behaviour.

Unfortunately, compared to the influence of stress on memory formation and retrieval, its effect on working memory has received less attention and results are more conflicting

(Shields, Bonner, & Moons, 2015; Shields, Sazma, & Yonelinas, 2016). Oei, Everaerd, Elzinga, van Well, and Bermond (2006) demonstrated that working memory performance (measured by matching a letter presented on the screen with the ones presented before) following exposure to the TSST was poorer compared to that after no stress. This effect was present only for the task with high taxation of working memory, specifically when participants needed to keep eight or more letters in memory for later matching. However, the results of Schoofs and colleagues (2008) showed that following an acute stressor, in the form of the TSST, men performed more poorly on working memory tasks in general compared to those who were not stressed. Although Schoofs et al. (2008) used a different task where participants needed to recognise whether a number was presented a certain amount of trials before, the impairing effect was found for tasks asking to match the current number with the one up to three trials before. This indicates that following acute stress, men struggle to hold more than two units of information as immediately available (i.e., in the working memory). Furthermore, in yet another task where men recalled previously-presented numbers either forward or backwards, Schoofs and colleagues (2009) found that the impairing effect of stress was present only for the reversed recall, which is considered to be a harder task. Similarly, they found that following stress, accuracy in judging solutions to equations was lower following a CPT rather than following a non-stressful experience.

A meta-analysis of 34 studies by Shields and colleagues (2016) confirmed that stress impairs working memory. Their results revealed that this influence becomes stronger with the increase in task difficulty, expressed by the taxation of working memory, and as time progresses. Interestingly, the increase in the impairing effect as time progresses appeared contrary to the previous meta-analysis of 18 studies (Shields et al., 2015) that demonstrated this effect only following acute administration of cortisol and its recession as the time passes. However, rather than being a contradiction, this distinction suggests that the impact of stress

on working memory is modulated not only by cortisol. Indeed, Elzinga and Roelofs (2005) showed that although people who responded with high levels of cortisol to the TSST had worse working memory performance than those who did not experience stress or those who did not respond to stressor with high levels of cortisol, their performance was not distinguishable during recovery phase. Taken together, these results indicate that both the SAM system and HPA axis responses are necessary for the inhibiting influence on working memory.

In social encounters, the constraints imposed on working memory by the stress response are likely to have several consequences. Due to the function of working memory as a buffer (D'Esposito & Postle, 2015), working memory impairment suggests a restricted pool of behavioural scripts that will be assessed for a given situation (Anderson & Bushman, 2002; Huesmann, 1998). If an individual has aggressive cognitive structures in larger quantities and they are more readily available than non-aggressive ones, then for them impairment of working memory by stress ensures focus on aggressive cognitive structures. Furthermore, as working memory is a component of executive functioning, its impairment following the stress response will likely lead to poor ability to implement any behaviour requiring a high degree of cognitive control, such as goal-oriented behaviour instead of habitual behaviour (Quaedflieg et al., 2019). It would also mean poor (re-)evaluation of cognitive structures (Anderson & Bushman, 2002), as impaired working memory means fewer components of the situation can be evaluated (Schoofs et al., 2008), such as the outcome for the actor or for others. For those with habitual aggression, restriction of the number of available behavioural scripts increases the likelihood of aggressive conduct.

Although there are several paths by which stress can impact aggressive behaviour through its effect on working memory, this effect is still contingent on personal factors, such history of habitual aggression or presence and availability of aggression supportive cognitive

structures. The relevance of individual characteristics for the behavioural outcome following an encounter with a stressor can be clearly exemplified by the affective response during stress.

3.9 Effects of Stress on Affective Response

Affect is defined as "any experience of feeling or emotion" (Van den Bos & American Psychological Association, 2007). It can be divided into positive affect, which refers to feelings of satisfaction, goal achievement or threat avoidance, or negative affect, encompassing feelings of dissatisfaction or failure (Van den Bos & American Psychological Association, 2007). Consequently, the affective response to a stressor corresponds to the emotions and feelings experienced during or after acute stress.

In terms of aggression, among the most relevant emotions is anger. A meta-analysis of 61 studies (Birkley & Eckhardt, 2015) demonstrated that anger, together with internalising negative emotions, has a robust association with aggression in the form of intimate partner violence. Anger has also been shown to have a slightly larger effect on aggression than aggressive behavioural scripts and attitudes (Gilbert et al., 2013; Hoise et al., 2014). Moreover, conceptually, affective aggression is argued to be the manifestation of anger (Blair, 2012). Similarly, the GAM and IIPM also highlight that anger contributes to aggressive behaviour (Anderson & Bushman, 2002; Huesmann, 2018) and within the I^3 model anger it constitutes part of the impellance process. Consequently, if stress can alter the valence or intensity of affective response to situational cues, then it can also influence the extent of aggressive behaviour. However, since anger has a prominent relationship with aggression, the focus will be on the effects that the stress response produces in terms of anger.

The experience of acute stress has been shown to divert resources from cognitive to affective processes (Oei et al., 2012). However, emotions can differ not only in their valence, such as positive or negative, but also in the associated behavioural motivation, such as approach or avoidance. Similarly, after a stimulus is appraised as stressful, which is a necessary

component of psychological stressors (Everly & Lating, 2019), its perception can differ in assigned valence. It can be deemed a challenge leading to potential gain or a threat associated with harm.

The Biopsychosocial (BPS) Model of Challenge and Threat posits that prevalence of situational demands over an individual's resources would result in a perception of a threat, whereas an excess of resources in the presence of low demands would be deemed a challenge (Seery, 2013). Furthermore, it is suggested that while both of these appraisals are accompanied by the activation of the SAM system, perceived threat is associated with greater HPA activation (Jamieson, Hangen, Lee, & Yeager, 2018). In other words, this conceptualisation proposes that appraising a situation as threatening would initiate a stronger stress response than appraising it as challenging. This, in turn, suggests that experiencing acute stress is predicated on the activation of the threat system.

The main role in this system responsible for detection of potential threats to physical well-being is played by the amygdala (Dedovic et al., 2009). It triggers the response to a stressor by classifying a stimulus as threatening. Importantly, heightened activity of the amygdala and other parts of the threat system are associated with the tendency to engage in reactive aggression (Blair, 2016; Coccaro et al., 2011; da Cunha-Bang et al., 2017; White et al., 2015). This highlights that acute stress and reactive aggression share certain neural structures. The increase in amygdala activation during acute stress (Arnsten, 2009; 2015) reflects the neural activation among violent offenders in response to provocation (da Cunha-Bang et al., 2017). Specifically, there is an increase in connectivity between the amygdala and dorsal striatum, combined with a decreased connectivity between the amygdala and hippocampus (Vogel et al, 2014; Wirz et al., 2018). While this resource re-allocation provides an explanation for the preference for automatic habitual behaviour under stress, the regulatory role of the amygdala can also elucidate emotional responses to stress.

Given that the full stress response appears to be contingent on detection of a threat, it is plausible to assume that it would evoke or strengthen emotions that will help deal with such circumstances. A stronger stress response, expressed though heightened heart rate and cortisol, is associated with anger, which is an approach-motivated emotion (Lupis, Lerman, Wolf, 2014; Moons, Eisenberger, & Taylor 2010). Moons and colleagues (2010) demonstrated that fear, representing avoidance-associated emotion, and anger as an approach-motivated one, are associated with stress levels in response to the TSST. These relationships were opposite to each other; namely, increased anger was correlated with higher cortisol response, and fear with lower one. However, both anger and fear were assessed using a retrospective self-report questionnaire administered after exposure to the stressor.

The potential challenge with such methodology was highlighted by Lupis and colleagues (2014), who assessed emotions during stress by monitoring participants' facial expressions as well as by retrospective self-report. They found a lack of relationship between these two measures, thereby raising questions about the validity of retrospective self-report. Nevertheless, Lupis and colleagues (2014) replicated and elaborated upon previous results by demonstrating the association between increased anger and both the SAM system and HPA axis activation, and the inverse relationship between fear and cortisol response. The direction of the analysis by Lupis and colleagues (2014) suggests that feeling anger during acute stress extends the duration of the stress response among men, but not women. In terms of aggressive behaviour, this shows that if a psychosocial stressor is added to a situation where a man is experiencing anger, the stress responses and aforenoted impairments associated with it would last longer, thereby making it more likely that this person would engage in aggressive behaviour, if he has a history of such.

Although the suggested relationship between stress and anger seems intuitive, these results do not correspond with propositions from the BPS model (Jamieson et al., 2018).

According to this model, perception of threat should evoke avoidance-motivated emotions—thus, fear and not anger. Furthermore, the TSST is specifically designed to exert demands on participants through socioevaluative threat and does not offer an immediate gain for enduring it. However, it is important to highlight that the correlational nature of the described results (Lupis et al., 2014; Moons et al., 2010) prevents determining any causal relationship.

Additionally, the positive association between cortisol during the stressors and anger contradicts the Dual Hormone Hypothesis, according to which there is an interaction between cortisol and testosterone that regulates anger (Mehta & Prasad, 2015). This hypothesis states that cortisol moderates the effects of testosterone on behaviour, so that testosterone facilitates dominating behaviours when cortisol is low, but when cortisol is high testosterone leads to less dominant conduct. Indeed, more cognitive effort to control anger was required by those men who have high testosterone and low cortisol, than those with the opposite hormonal pattern (Denson, Ronay, von Hippel, & Schira, 2013).

The Dual Hormone Hypothesis is demonstrated in the studies assessing both cortisol and testosterone following the stressors. Romero-Martínez, González-Bono, Lila and Moya-Albiol (2013) and Romero-Martínez, Lila, Sariñana-González, González-Bono and Moya-Albiol (2013) have shown that higher levels of anger following acute stress are present among men who have no history of perpetrating IPV than among those who have such history, as they had higher testosterone-to-cortisol ratios. Since both groups showed an increase in anger following acute stress, this shows that while low and high responses to stressors can facilitate anger, when the low response coincides with high levels of testosterone, the effect is more pronounced. This, in turn, means that stress can facilitate aggression among those with low responsivity to it (Mehta, DesJardins, van Vugt, & Josephs, 2017) as well as among those with high responsivity to it (Gerra et al., 2007).

Nevertheless, the precise mechanisms of the interaction between testosterone and cortisol are not yet fully understood, thereby necessitating more research in this domain (Mehta & Prasad, 2015). Moreover, using a foraging game[4] where a threat is represented by a "predator," Vogel and Schwabe (2019) demonstrated that following acute stress, participants were more avoidant than non-stressed ones when the threat was close. However, within the stress group, participants who reported engaging in physical aggression showed more approach behaviours. This suggests that the motivational base of affect during stress response is not determined purely by activation of the SAM system and HPA axis and is likely to be contingent on other personal characteristics (Fricke & Vogel, 2020).

A systematic literature review by Fricke and Vogel (2020) demonstrated that one such characteristic can be trait aggressiveness, as individuals possessing it are consistently shown to engage in approach behaviours, particularly to aggressive cues. Taken together, this highlights that stress is likely to exacerbate habitual affective and behavioural responses to it. For those who have history of anger and aggression, it would increase them; meanwhile, for those who do not have such histories, it would not elicit them. Cackowski and colleagues (2017) demonstrated this pattern among female patients with borderline personality disorder (BPD) and attention hyperactivity disorder (ADHD). In their study, participants with BPD, who also had the highest trait aggressiveness, showed a higher increase in anger following acute stress compared to ADHD patients or community participants. Interestingly, Cackowski and colleagues (2017) did not find a significant relationship between stress induction and aggressive behaviour. While they explain this in relation to the specifics of the aggressive paradigm, it is also possible that while the direct relationship was non-significant, a path model

[4] This is an approach – avoidance computerised paradigm where participants need to navigate a 24x16 grid to collect tokens associated with monetary reward. Together with the participants is a scripted "predator" that is initially asleep. Once it wakes up, it chases the participant to eat them, so they lose collected tokens.

looking at stress-to-anger and anger-to-aggression relationships would have yielded different results.

In this light, the support for the link between the stress response and anger that was provided by Lieberman and colleagues (2015) needs to be interpreted with attention given to the nature of the sample and the stressor. Participants in this study were volunteers from the US Navy Survival, Evasion, Resistance and Escape School, who are likely to have more experience with aggression than the general population. Meanwhile, the procedure used to evoke a stress response was several days of "mock captivity" that included "mock interrogation." Such a stressor is something a participant was trained for, and enduring it is necessary for completion of the course; thus, there is considerable likelihood that personal resources of the participants were close to the external demands and that the potential gain was present. The results of Lieberman and colleagues (2015) demonstrate that although there is a general increase in anger during highly demanding situations, this increase is greater in those participants who received Tyrosine, which acts as precursor to norepinephrine.

Together, these results demonstrate a lack of heterogeneity in emotions following a stressful experience—even if they are operationalised in binary options of avoidance or approach motivation. In other words, for an emotional response following acute stress, the individual's personal characteristics and appraisal of the situation appear to be more influential than the simple presence of a stressor (Fricke & Vogel, 2020). In application to social encounters, this suggestion is straightforward: although the stress response does facilitate activation within affective processing brain structures, the nature of the response will be determined by person and situation factors. Violent offenders show increased limbic system responsivity to emotional tasks when compared to non-offenders (Siep, Tonnaer, van de Ven, Arntz, Raine, & Cima, 2019). Activation of the stress response is predicated on threat detection, partly corresponding to a similar system (Jamieson et al., 2018) which is also associated with

reactive aggression (da Cunha-Bang et al., 2017; White et al., 2015). Consequently, for those who have history of reactive aggression, activation of the threat system is likely to facilitate aggressive behaviour; meanwhile, for those who do not, threat system activation will not 'force' them to become aggressive.

3.10 Concluding Remarks

The current chapter has demonstrated that stress affects several important mechanisms facilitating aggressive conduct. The socio- and neurocognitive models posit that aggression results from decision-making processes during which aggression supportive cognitive structures are selected (Anderson & Bushman, 2002; Blair, 2016; Huesmann, 2018). Experiencing stress responses has been shown to affect decision-making processes by facilitating risk-oriented decision strategies (Starcke & Brand, 2012; 2016), which in turn is linked with aggressive behaviour (Kuin et al., 2015). It was also shown to affect encoding and retrieval memory processes (Shields et al., 2017), which in turn affects consolidation and retrieval of aggression supportive cognitive structures, as they are thought to be stored within memory (Gilbert & Daffern, 2017; Huesmann, 1998).

Clarifying the effect of stress on retrieval of behavioural scripts, it was shown that stress facilitates habitual conduct even when its outcome is no longer desired (Fournier et al., 2017; Schwabe & Wolf, 2010). Stress was also demonstrated to impair working memory capacity (Shields, 2015; 2016), clarifying why it acts as a moderator for the stress-aggression relationship (Sprague et al., 2011). Furthermore, the stress response is predicated on activation of the threat system, involving the amygdala and inhibiting the regulatory capacities of PFC, thereby mirroring the process suggested to underpin reactive aggression (Arnsten, 2015; Oei et al., 2012). Although it was not shown to facilitate a specific affective response, acute stress generally facilitates anger among men and women (Moons et al., 2010), which is an affective response repeatedly linked with aggression (Birkley & Eckhardt, 2015; Hoise et al., 2014).

However, further studies have demonstrated that the amplitudes of increased anger following stress is contingent on personal factors such as trait aggressiveness, testosterone to cortisol ratio and offending history (Cackowski et al., 2017; Romero-Martínez et al., 2013a; 2013b).

These differences in the intensity of the affective response following a stressor based on other variables underscore the core argument of this chapter. The specific behavioural and affective responses to stressors is likely to be determined not solely by the physiological reaction to them, but by person factors such as the presence of aggression supportive cognitive structures or a history of habitual aggressive behaviour. Given the influence that the stress response has on multiple processes associated with aggression, it is arguably an important impellent in terms of the I^3 model. However, its path to aggression is unlikely to be direct.

Unless a stressor is cardiovascular, it needs to be perceived as stressful in order to initiate activations of both the SAM system and HPA axis (Carrasco & Van de Kar, 2003). Given the prevalence of potential psychological and combined stressors in social encounters, the relevance of personal factors affecting the appraisal of the situation is evident. To a large extent, they will determine whether the full stress response occurs. However, there are stimuli that consistently engage both the SAM system and HPA axis. Nevertheless, even following such a standardised stressor, people differ in terms of approaching or avoiding a threat, based on their history of aggressive behaviour (Vogel & Schwabe, 2019). In other words, individuals' experiences determine the variation of motivational response, which corresponds to the association between stress response and habitual rather than goal-oriented behaviour (Smeets et al., 2019). Nevertheless, the nature of habitual behaviour will be contingent on the experience of the individual. Consequently, the relationship between stress and aggression appears to be indirect as stressor impacts cognitive processes, related to evaluation of behavioural scripts (Shields, 2015; 2016; Starcke & Brand, 2012; 2016). However, for this

orchestrated alteration in information processing to result in enactment of an aggressive script, it needs to be acquired, rehearsed, valued, available and be one among many (Anderson & Bushman, 2002; Huesmann, 2018).

Consequently, the effect of stress on aggressive conduct is likely to be indirect. Its investigation should also account for information processing capacities and aggression-supportive cognitions. The following chapter outlines how the current thesis aims to achieve this. Building on the information presented in Chapters 2 and 3, it will provide a rationale for a systematic literature review and a series of studies, findings from which will serve as the basis for a proposed integrated model of aggression.

CHAPTER FOUR: ADDRESSING THE RESEARCH PROBLEM

4.1 Structure of the Chapter

This chapter demonstrates how gaps and conflicts within the existing literature informed the aims of this thesis. It focuses on the relationships between cognitive structures and processes, stress response and aggression. The rationale for each of the studies is presented before specific predictions are then outlined.

4.2 Rationale for the Research

The main aim of this thesis is to investigate the factors related to cognitive structures, cognitive processing and stress that are associated with aggression and to construct a model of aggressive behaviour. Specifically, it aims to stratify the pathways to such behaviour and to establish how changeable variables affect aggressive behaviour. Cognitive structures and processes were selected because, as demonstrated in Chapter 2, aggression can be thought of as resulting from decision-making processes whereby aggression supportive cognitive structures are chosen as actions (Anderson & Bushman, 2002; Blair, 2016; Huesmann, 2018). Similarly, in Chapter 3 stress was shown to affect decision-making processes (Shields, 2015; 2016: Starcke & Brand, 2012; 2016) and memory (Shields et al., 2017), where the cognitive structures are thought to be stored and retrieved from (Gilbert & Daffern, 2017; Huesmann, 1998).

Consequently, stress was selected as another factor that needs to be included in the model. Thus, the studies presented include all three processes outlined in the I^3 model (Finkel, 2014). The stressors act as the instigators, as they represent external objects evoking reactions from an individual. The aggression supportive cognitive structures represent impellance processes, as they are individual idiosyncrasies that affect how objective stimuli are perceived and consequently change psychological states. Meanwhile, information processing capacity allows

to operationalise inhibition processes, as they are suggested to affect whether or not aggressive scripts are enacted (Anderson & Bushman, 2002; Huesmann, 2018; Sprague et al., 2011).

The integration of socio-cognitive and neurocognitive approaches, as illustrated in Chapter 2, resulted in the formulation of two principles governing aggression: 1) such conduct is a result of decision-making (Anderson & Bushman, 2002; Blair, 2004; 2016); 2) aggression is an enactment of cognitions, which outline and approve such conduct (Huesmann, 1988, 1998, 2016). This means that when entering a social interaction, a person makes a decision about which behavioural script to follow. Consequently, it is expected that while aggression supportive cognitive structures exert a primary and direct effect on aggressive conduct, the characteristics of information processing that reflect differences in decision-making act as inhibitors and disinhibitors.

Furthermore, Chapter 3 showed that stress affects both script selection processes and behaviour in general. On a fundamental level, experiencing stress leads to the activation of resources that can be used for energy-demanding actions (de Kloet, et al., 2005; Everly & Lating, 2019). One such type of action can be aggressive behaviour, especially the reactive type, which is argued to be motivated by an attempt to control situations that provoke stress (Fabian, 2010). Moreover, activation of the Sympathomedullary Pathway (SAM) and Hypothalamic-Pituitary-Adrenal (HPA) axis has been demonstrated to affect the decision-making processes during which behavioural scripts are selected and memory where they are thought stored (Anderson & Bushman, 2002; Huesmann, 1988; 2016; Shields et al., 2017; Starcke & Brand, 2012; 2016). Specifically, following acute stress, individuals (particularly men) show diminished working memory functioning (Shields et al., 2016), a preference for habitual behaviour (Smeets et al., 2019) and an overvaluation of rewards, even when they are associated with high risk (Starcke & Brand, 2012; 2016), and a preference for habitual behaviour (Smeets et al., 2019).

Thus, due to its influence on the cognitive processes that facilitate aggression, stress is likely an important secondary factor affecting such behaviour. Furthermore, there are two reasons to investigate its role closer. First, studies show conflicting evidence on the relationship between stress and aggression (Böhnke et al., 2010b; Gerra et al., 2001; 2007; Madden & Shaffer, 2019; Margittai et al., 2018; Mehta et al., 2017; Murray-Close et al., 2017; Verona & Curtin, 2006). Consequently, there is a need to assess whether this association is direct and, if not, to establish the intermediary variables.

Second, as was shown in Chapter 3 (specifically section 3.4), the addition of socioevaluative components to purely physical stressors increases the intensity of the stress response (Schwabe, Haddad, & Schachinger, 2008; Smeets et al., 2012). This highlights the role of perception in determining the magnitude of experienced stress, which in turn suggests that stress is a changeable factor that can be addressed through intervention. Indeed, research has shown that therapeutic techniques focused on cognitions can reduce stress among general, clinical and prisoner populations (Auty, Cope, & Liebling, 2015; Fjorback, Arendt, Ørnbøl, Fink, & Walach, 2011; Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012; Regehr, Glancy, & Pitts, 2013). Consequently, the systematic literature review further investigates the relationship between stress response markers and aggression. Its aims and predictions are outlined below.

4.2.1 Systematic Literature Review: The Relationship Between Stress Response Systems and Aggression

Aims.

To further investigate the relationship between stress and aggression suggested in Chapter 3, the systematic literature review aims to:

1. Establish the nature of the relationship between stress and aggression.
2. Identify influential third factors that affect the stress-aggression relationship.

Predictions.

Based on the effect that the stress response has on precursors of aggression discussed in Chapter 3, especially anger, it is expected that:

Both heightened and lowered activity of the stress response systems are expected to be associated with aggressive behaviour (Böhnke et al., 2010a; 2010b; Gerra et al., 2001; 2007; Madden & Shaffer, 2019; Verona et al., 2006).

4.2.2 Study 1: Investigating the Effects of Stress and Implicit Theories on Aggressive Behaviour

Following the literature review, a small-scale study will test the proposition of socio-cognitive models stating that aggression supportive cognitive structures facilitate aggressive conduct (Anderson & Bushman, 2002; Huesmann, 1988; 1998; 2016). In particular, the relationship between Implicit Theories (ITs), representing a cluster of core beliefs regarding aggression and its use (Polaschek, Calvert, & Gannon, 2009), and behaviour will be assessed using an aggression paradigm. Additionally, to further the results of the literature review, the study will investigate the association between stress response and aggressive behaviour and whether it differs between students and patients of a high secure forensic psychiatric hospital.

Aims:

1.1. Establish the relationship between Implicit Theories supporting aggression and aggressive behaviour towards a stranger.

1.2. Investigate whether the stress – aggression relationship differs between students and patients of a high secure hospital.

Predictions:

1.1. Aggression supportive cognitions will be positively associated with aggressive behaviour (Huesmann, 2016).

1.2. An increase in the heart rate and skin conductance level will be positively associated with aggression (Verona & Kilmer, 2007).

The findings from this study are expected to outline the main paths to aggressive behaviour in a given situation and, as such, point to the influential variables. A positive relationship between aggression supportive Implicit Theories and aggressive behaviour would support the basic proposition of socio-cognitive models: aggression is predicated on enactment of aggression supportive cognitive structures (Anderson & Bushman, 2002; Huesmann, 2018). Meanwhile, the association between the Sympathomedullary (SAM) system markers and aggression would highlight the role of stress in facilitating aggressive conduct.

4.2.3 Study 2: The Role of Information Processing in Facilitating Aggressive Behaviour

In order to provide more detailed pathways to aggression, Study 2 will focus on information processing capacity. This capacity affects decision-making processes, which are suggested to account for aggressive behaviour (Anderson & Bushman, 2002; Blair, 2004; 2016). Similar to Study 1, aggression will be measured using a laboratory paradigm. Specifically, it will use the P3 Event Related Potential (ERP) component during a linguistic Go/No-Go task, as ERP reflects cognitive processing of the situation that precedes the aforementioned "choice" of a given behavioural script and has been shown to be negatively related to aggression (Fanning, Berman, & Long, 2014; Jabr, Denke, Rawls, & Lamm, 2018; Verona & Bresin, 2015). Furthermore, in Chapter 3, stress was shown to amplify affective responses that are in turn determined by individual characteristics (Fricke & Vogel, 2020). Consequently, Study 2 shifts the focus from the stress response to the affective response and its relationship with aggressive behaviour.

Aims:

2.1. Identify the pathways from cognitive processing to aggressive behaviour via P3 amplitude.

2.2. Investigate the role of hostility in informing aggressive behaviour.

2.3. Establish the influences of positive and negative affect on aggressive behaviour.

Predictions:

2.1. An allocation of cognitive resources moderates the relationship between trait aggressiveness and aggressive behaviour (Fanning, Berman, & Long, 2014; Jabr, Denke, Rawls, & Lamm, 2018).

2.2. An allocation of cognitive resources moderates the relationship between past history of aggression and aggressive behaviour (Huesmann, 2018; Sprague, et al., 2011).

2.3. Participants who respond with increases in negative affect during provocation will show more aggressive behaviour than participants who responded with increases in positive affect during provocation (Megías, Gómez-Leal, Gutiérrez-Cobo, Cabello, & Fernández-Berrocal, 2018).

2.4. Participants who reported higher hostility following provocation will show more aggression than those who reported other forms of negative affect (Burt, Mikolajewski, & Larson, 2009).

2.5. Negative affect measured during provocation (pre to post mood induction) will be predictive of aggressive behaviour only among participants with high trait aggressiveness and a history of aggression (Donahue, Goranson, McClure, & Van Male, 2014; Pawliczek et al., 2013).

2.6. A disposition toward worry will have an indirect effect on aggressive responding that will be mediated by allocation of cognitive resources (lower P3 Go/No-Go Difference) (Sprague, Verona, Kalkhoff, & Kilmer, 2011).

2.7. Strength of the association between hostility and aggressive responding will increase as the allocation of cognitive resources decreases (Fanning, Berman, & Long, 2014).

2.8. Change in negative affect will have an indirect effect on aggressive behaviour mediated by allocation of cognitive resources (Donahue et al., 2014; Sprague et al., 2011).

2.9. Emotional vs Neutral P3 differences will mediate the relationship between lower P3 difference Go/No-Go and aggressive behaviour (Donahue et al., 2014; Sprague et al., 2011).

As the results of the Study 2 are expected to show associations between information processing and aggressive behaviour, they would serve to verify the proposition that aggression results from decision-making.

4.2.4 Study 3: Establishing Pathways from Cognitions and Stress to Aggressive Acts and Traits

Both Study 1 and Study 2 have experimental designs, where behavioural aggression is assessed through laboratory paradigms. However, in order to formulate a model of aggression, pathways identified in specific situations, represented by the laboratory paradigms, should be confirmed as present in daily life and across situations. Consequently, Study 3 utilises a cross-sectional design in an effort to identify the routes to aggressive behaviour in daily life rather than in laboratory paradigms. This unifies the theoretical propositions derived in the Chapters 2 and 3 and, given the noted influence of therapeutic techniques on the intensity of stress response, Study 3 adds coping styles as a mediator of the stress-aggression relationship. The reasoning behind addition of coping styles rests on previous research. While Whitman and Gottdiener (2015) reported direct association between maladaptive coping styles and

aggression, Gardner, Archer, and Jackson (2012) showed that coping skills mediate the relationship between borderline personality disorder traits, which include poor response to frustrations. Consequently, coping styles need to be considered as a potential mediator for stress – aggression relationship.

Aim:

3.1. Build a model that describes the relationships between aggression, aggression supportive cognitive structures, working memory problems, stress, and coping styles.

Predictions:

3.1. The effect of perceived stress on aggression will be mediated by adaptive and maladaptive coping styles (Gardner et al., 2012, Whitman & Gottdiener, 2015).

3.2. The effect of hostile attribution bias on aggressive behaviour and traits will be mediated by the criminal attitudes to violence (Huesmann, 2018; Nunes, Hermann, Maimone, and Woods 2015; Tuente, Bogaerts, & Veling, 2019).

3.3. The effect of life stressors on aggressive traits will be mediated by aggressive behaviour (Brown, Fite, DiPierro, & Bortolato, 2017)

3.4. The effect of working memory problems on aggressive behaviour will be mediated by hostile attribution bias (Anderson & Bushman, 2002; Tuente, Bogaerts, & Veling, 2019, Anderson & Bushman, 2002)

3.5. The coping styles that modulate the experienced stress will be associated with working memory problems, as stress has been shown to affect working memory (Shields, Sazma, & Yonelinas, 2016).

The results of Study 3 will inform the proposal for an integrated model of aggressive behaviour that aims to identify and distinguish primary factors directly associated with

aggression and secondary factors that act as amplifiers or buffers. The potential of this model for intervention and application will also be outlined.

CHAPTER FIVE: THE RELATIONSHIP BETWEEN STRESS RESPONSE SYSTEMS AND AGGRESSION: A SYSTEMATIC REVIEW

5.1 Structure of the Chapter

As was discussed in the previous chapters, stress response is an influential factor for aggressive behaviour. However, since there are two types of stress response, and as Chapter 3 suggested that, both hypo- and hyper-responses to a stressor can facilitate aggressive behaviour, it is important to systematically explore the literature addressing the relationship between these two variables. Thus, this chapter presents a systematic literature review aiming to investigate the nature of the relationship between the stress systems response markers and aggression. It has two main parts: a systematic description of the results of included studies and a thematic analysis.

5.2 Review Aim

This review will identify and critically appraise published, peer-reviewed studies to address the relationship between the stress systems' activity and aggressive behaviour whilst noting the influential third variables. Theoretical considerations (discussed in the Chapter 3) and empirical research (Das et al., 2018; Murray-Close et al., 2017; Rausch et al., 2015; Scarpa & Ollendick, 2003; Scarpa et al., 2000; Solanki et al., 2007; Verona & Curtin, 2006; Verona & Kilmer, 2007) suggest that both hyper- and hypo-activity of the stress response systems can facilitate aggression. Thus, it is expected that the markers of stress systems' activity will consistently have both positive and negative relationships with aggression.

5.3 Method

The systematic literature review was conducted following PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analysis) guidelines (Moher et al., 2009). The aim was to identify empirical studies which have investigated the relationship between physiological markers of the stress response systems and aggressive behaviour. The search for

articles that were published online up to November 2018 was conducted using eight databases: PsychINFO, MedlineFULLTEXT, CINHAL, SCOPUS, EMBASE, MEDLINE, Web of Science and Web of Science MEDLINE. The boolean search string was as follows: (Aggress* OR Hostil* OR Anger OR Violen* OR Conflict* OR Physical Confront*) AND (Stress* OR Threat*) AND (HPA* OR Corti* OR Adren* OR Hypothalam* OR Pituitar* OR Amygdal*). Furthermore, the reference lists of articles identified as relevant were hand searched for further studies to be added to the final analysis.

Studies were retained for final analysis if they investigated the effect of the stress systems' activity on aggressive behaviour. Studies reporting a correlation between these variables were also retained if they did not specify the direction of the relationship. Only empirical studies with original data (i.e., no reviews or meta-analyses) that underwent a peer review process and had human participants aged older than 18 years were included[5]. Studies retained after detailed inspection were then subjected to deductive sematic realist thematic analysis (Braun & Clarke, 2006). Although thematic analysis was developed as a method for qualitative data, the current review follows the suggestion of Onwuegbuzie, Leech, and Collins (2012), which indicate that thematic analysis can also be used for literature review in order to add depth to the findings.

5.4 Review Process

Titles of the identified papers from the database search were first screened for relevance. Following this, abstracts of relevant articles were reviewed for exclusion criteria. Studies with abstracts that met the inclusion criteria were further investigated to determine all inclusion

[5] Only studies looking at adult participants were included as they have relatively formed, rather than emerging, personality characteristics and physiology.

criteria were met. Data relevant to the review question was extracted and tabulated. Findings are presented in Table 5.1.

5.5 Results

The database search yielded a total of 21,943 hits. Following removal of duplicates, 10,430 titles were screened for relevance. Of these, 4,945 were identified as relevant and their abstracts were assessed against the exclusion criteria. Papers were removed if they: contained no original data (k =1,150); did not use a sample of adult humans (k = 1,025); did not assess stress response (k = 167); did not have a full-text that was published in a peer-review journal (k = 14); were not published in English (k = 5); and/or was retracted (k = 1).

This left 553 full-text articles, which were subjected to a detailed inspection. Specifically, articles were excluded if they: did not measure aggressive behaviour directed at others (e.g., suicide or only feelings of anger) (k = 219); did not assess physiological markers of stress response (k = 78); did not have a full-text with a detailed methods section (e.g., conference abstract) (k = 33); included participants that were not older than 18 years (k = 15); did not provide original data (k = 10); were not in English (k = 3); did not assess the relationship between stress response and aggression (k = 51); did not undergo peer review (k = 12); reported only one case study (k = 7); used aggressive behaviour as a predictor of the stress response (k = 50); or could not be accessed by the researcher (k = 4). It is important to note that articles reporting correlation coefficients based on regression analysis where stress markers were the outcome variables were excluded. In these cases, stress markers were the variables that were affected by predictors, which means the correlation shows the changes in them when a change in another (predictor) variable occurs. This type of relationship is opposite to the relationship posited in the question guiding this review. Ultimately, 71 full-text articles were considered suitable for the current review. Additionally, six articles were

added after manual inspection of the reference lists, bringing the total number of articles analysed to 77. This selection is shown in the Figure 5.1

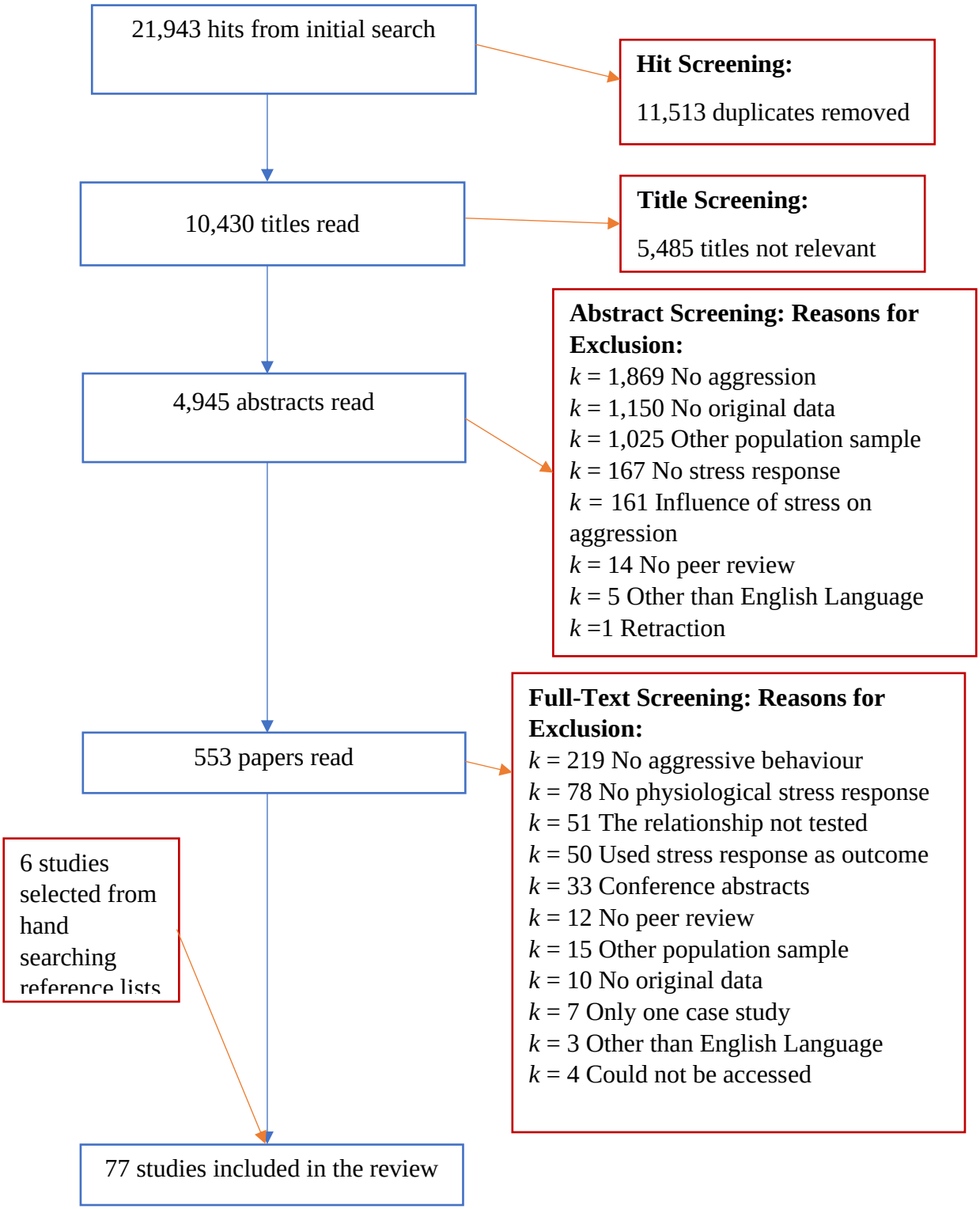

Figure 5.1 Literature search flow chart showing the number of articles included at each step of the screening procedure

Table 5.1 Studies included in the review

Reference	Country	Design	Participant demographics	Stress evoking procedure	Measure of stress reactivity	Measure of aggression	Manipulation checks	Results: relationship between variables
Ai, Kabbaj, & Kathy, (2014)	USA	Cross Sectional	162 patients for cardiopulmonary bypass (57% male), *M*age = 61.2.	Medical procedure.	Blood samples for cortisol were measured at baseline before the procedure.	Hostility Subscale of SCL-90-R.	No information.	Only baseline cortisol was a significant predictor of hostility after the procedure.
Armstrong, & Boutwell, (2012)	USA	Cross Sectional	151 students (58 male), *M*age = 21.43.	None	Resting heart rate measured as average over two timepoints with five-minutes interval.	Intent to commit a crime described in three scenarios: assault, theft and drunk driving.	No information.	Those in low resting HR group reported that they were more likely to commit assault scenario, but not theft or drunk driving. However, when the perceived likelihood of conviction is entered in the model, the association stops being significant, yet the perceived likelihood of conviction also does not have a sign association with intent.
Bergomi, Modenese, Ferretti,	Italy	Cross Sectional	42 bus drivers (5 female), *M*age = 40, only 33	Work	Saliva samples for alpha-amylase and cortisol were taken	Aggression scale of Driver Stress	No association between PSS	Significant positive correlation between cortisol at t5 and

Ferrari, Licitra, Vivoli, ... & Aggazzotti, (2017)			males were used in the analysis.		at: *t*0 = beginning of the shift; *t*1 = middle of the shift; *t*2 = end of the shift; *t*3 = *t*0 time but on day off; *t*4 = *t*1 time, but on day off; *t*5 = *t*2 time, but on day off. Perceived Stress Scale (PSS-10)	Inventory (DSI).	and cortisol or alpha-amylase.	aggression; Significant positive correlation between PSS-10 and aggression. Aggression was a significant predictor of the PSS-10 score.
Berman, Gladue, & Taylor, (1993)	USA	Cross Sectional	202 College students. All male, *M*age = 19.03, no other information reported.	TAP with shocks of 1s duration. Three trials of increasing intensity.	Salivary cortisol was measured at four times: *t*0 = baseline, a *t*1 = pre task, *t*2 = *t*1+35; *t*3 = *t*2+15.	Averaged shock setting in one block of the TAP.	Increase of shock intensity with each block.	No main effect for cortisol. Significant interaction between personality type and cortisol: high-cortisol and Type A as well as low-cortisol and Type B administered more intense average shocks than low-cortisol and Type A; No difference between High-cortisol and Type B and low-cortisol and Type B.
Beyer, Buades-Rotger, Claes, & Krämer, (2017)	UK	Cross Sectional	1st study: 43 females, *M*age = 22. 16 participants were excluded from the analysis.	Provocation in form of high noise levels in CRT.	Fear potentiation (FP) was measured via blink magnitude while presenting threatening and neutral pictures.	Mean punishment score from CRT with aversive sound as	No information.	Significant negative correlation between FP and aggressive responding to provocative, but not to non-provocative opponent. Significant

						punishment. BPAQ.		negative correlation between FP and trait anger subscale of BPAQ.
Bjork, Dougherty, Moeller, & Swann, (2000)	USA	Before and after study	12 males, *M*age = 27.9 with LHA score > 20, 12 males, *M*age = 31.1, with LHA score <20.	Provocation in form of subtracting money in the PSAP. Tryptophan (Trp) was depleted using drink with large neutral amino acids, while Trp loading drink used the same amino acids but with addition of 10.3 g L-tryptophan mixed into the drink	Plasma samples for cortisol were taken within 5 minutes of the last PSAP block on: *d*0=baseline day; *d*1= Trp depletion day; *d*2= Trp loading day; *d*3= food restricted day	3 blocks of PSAP: *t*0 = immediately after the drink; *t*1 = *t*0 + 3h; *t*2 = *t*0 + 5h, *t*3 = *t*0 + 6.75h.	No effect of Trp drinks on cortisol.	Significant interaction between Trp manipulation and history of aggression on the PSAP responding was present only during high provocations: among participants with history of aggression those with depleted Trp were more aggressive than those with increased Trp; However among participants without history of aggression those with increased Trp were more aggressive than those with depleted Trp. Significant negative correlation among participants with history of aggression between plasma Trp after its increase and

								aggressive responding in the PSAP.
Böhnke, Bertsch, Kruk, & Naumann, (2010) Study A	Germany	Randomised Controlled Trial	20 students (10 female and 10 male, *M*age=23;	For provoked group: Increase of noise intensity in the TAP.	Salivary cortisol was taken four times: *t*0 = before the TAP; *t*1 = after the TAP; *t*2 = after non-stressful exercise that followed TAP; *t*3 = 10 minutes after *t*2. Trait HPA activity was measured via cortisol awakening response. Taken on 3 consecutive days, 30, 45, and 60 minutes after awakening scheduled to be between 6.00 am and 8.00 am. The Area Under the curve with respect to the ground (AUCG) was calculated for each participant and day and then averaged over the 3 days to form one reliable indicator of the basal HPA axis activity for each subject. AUCG	Noise intensity setting averaged across one block of the TAP.	Provoked group was significantly more aggressive than the non-provoked control group in blocks 2 and 3. Within provoked group participants were least aggressive in block 1 and most aggressive in block 3. Non-provoked group had significantly high cortisol levels at t0 and t1. Increase in provoked group was not significant.	Significant interaction between cortisol awakening response (CAR) and group: significant negative correlation between CAR and aggressive behaviour in the provoked group; no significant positive correlation in the non-provoked group. Change in cortisol (t2-t0) was not related to the amount of aggressive behaviour: neither total nor per block.

					for 19 subjects were included in the analysis, 9 in the non-provoked control and 10 in the provoked group.		Provoked group had significantly higher cortisol level at *t*2 as compared to non-provoked when baseline values were controlled for.	
Böhnke, Bertsch, Kruk, Richter, & Naumann, (2010) Study B Exogenous cortisol	Germany	Randomised Controlled Trial	48 students (24 females and 24 male, *M*age = 23).	For provoked group: Increase of noise intensity in the TAP. For cortisol treatment: 20 mg of hydrocortisone.	Salivary cortisol was taken several times: *t*0 = before the treatment; *t*1 = before the TAP; *t*2 = after the TAP; *t*3 = *t*2 + 25min, after emotional STROOP; *t*4 = *t*3 + 10min. Trait HPA activity was measured via cortisol awakening response. Taken on 3 consecutive days, 30, 45, and 60 minutes after awakening scheduled to be	Noise intensity setting averaged across one block of the TAP.	There was a significant difference between treatments.	Significant treatment effect on aggression. Significant interaction of treatment and block: cortisol group was more aggressive than the placebo group only in 3rd Block, where more aggression was showed than in previous two blocks. Significant interaction of gender and treatment: only in placebo group males were more aggressive than females; only

					between 6.00 am and 8.00 am. The Area Under the curve with respect to the ground (AUCG) was calculated for each subject and day and then averaged over the 3 days to form one reliable indicator of basal HPA axis activity for each subject.			females in cortisol group were more aggressive than in placebo group. Average acute stress was positively correlated with aggression in placebo group among high provocation. No significant effect of CAR on aggression. Significant interaction of CAR, gender and block. Significant negative correlations between CAR and aggression only among women with increased intensity across blocks, but not for men. Significant interaction of CAR, treatment and block: negative correlations between CAR and aggression were only present in placebo but not in cortisol condition.
Brewer-Smyth,	USA	Case Control	113 female inmates: 27	None	Saliva samples for cortisol were taken	Criminal record was	No information.	Those who were currently sentenced

Burgess, & Shults, (2004)			convicted for violent crime, *M*age= 32.86; 86 convicted for non-violent crime Mage = 33.57.		at: *t*1 = within 30min after awakening, and *t*2 = between 3 and 5pm.	used to establish presence of conviction for violent crime.		for a violent crime had significantly lower average cortisol levels at *t*1 (p<.055) and decrease diurnal variation (p<.05) than those convicted for non-violent crime. Morning cortisol levels were a significant negative predictor of violent conviction.
Brown, McGarvey, Shirtcliff, Keller, Granger, & Flavin, (2008)	USA	Cross Sectional	5 male students	None	Trait HPA activity: Saliva samples for cortisol were collected at three timepoints: 8.00pm, 2.00am and 8.00am during 24 hours on three consecutive weeks.	STAXI 2	No significant variance in cortisol levels between weeks.	Positive cortisol slopes were significantly negatively correlated with trait anger, angry temperament, and angry reaction.
Buades-Rotger, Beyer, & Krämer, (2017)	Germany	Cross Sectional	36 female students, *M*age =2 2. 30 had complete data used in the analysis.	Provocation by loud aversive sound in the FOE version of the TAP	Heart rate (HR) was recorded during the task.	Averaged intensity of sound in the FOE version of the TAP	Loudest blasts were rated as most distressing by participants. Sign effect of provocation for HR: those who confronted	Sign louder blasts were selected for high provoking opponent across all blocks.

							high provocation opponent had significantly higher HR increase as compared to those who confronted low provocation opponent.	
Buades-Rotger, Engelke, Beyer, Keevil, Brabant, & Krämer, (2016)	Germany	Cross Sectional	39 female students, *M*age = 23.22.	No stressor.	Trait HPA activity was measured via cortisol awakening response. Salivary samples were taken on a normal day at the awakening (scheduled between 6am and 8am), half an hour and an hour after and in the evening (6pm to 8pm). AUCG was calculated for the 3 morning measures. The evening and awakening samples were used to check the circadian decay. Also, two samples	Average noise intensity per block of the STAP.	Cortisol followed daily pattern of peak at 20 minutes after awakening and decline in the afternoon.	No significant correlation between morning cortisol and aggression.

					were taken at the 1st session, one before and one after the fMRI scan.			
Buchmann, Zohsel, Blomeyer, Hohm, Hohmann, Jennen-Steinmetz, ... & Esser, (2014)	Germany	Cohort and Cross Sectional	219 participants (43.38% male) underwent TSST at 19.	TSST	Blood samples for cortisol were taken at: *t*0 = baseline; *t*1 = 10min after TSST; *t*2 = *t*1 + 10min; *t*3 = *t*2 + 15min; *t*4 = *t*3 + 25min; *t*5 = *t*4 + 20min. Genomic DNA was isolated from blood sample at 15 and 19 years.	YASR	Sign increase in cortisol at *t*1.	The negative association between cortisol during TSST and aggression was not significant. Significant interaction between DRD4 and prenatal maternal stress: significantly high aggression was reported only in DRD4 r7 allele carries who have been exposed to prenatal maternal stress. Significant interaction between genotype and prenatal maternal stress: only carriers of DRD4 r7 allele who have been exposed to prenatal maternal stress had significantly lower cortisol reaction than those carriers who were not exposed to prenatal stress.

Cima, Smeets, & Jelicic, (2008)	The Netherlands	Case Control / Cross Sectional	74 male participants. 27 university students (*M*age = 24.9) and 47 prison inmates (*M*age = 30.4). No psychiatric illness, Caucasian, and Dutch nationality.	None	Trait HPA activity: salivary cortisol was measured four times during the day. *t*1 = 8.00am; *t*2 = 11.00am; *t*3 = 2.00pm; *t*4 = 4.00pm. Computed variables: AUC; Diurnal cortisol slope; Daily Average Cortisol (DAC).	BPAQ	Similar pattern of decline was present in all groups; Participants diagnosed with psychopathy had lower levels of cortisol than students and inmates without this diagnosis.	No significant correlations between cortisol measures and the BPAQ. Diurnal Cortisol did was not a significant mediator between childhood traumatic experience and aggression.
Cohen, Nisbett, Bowdle, & Schwarz, (1996)	USA	Case Control / Cross Sectional	2nd study: 173 male students, (no information about age). 3rd study 148 male students.	Public or private insult by confederate	2nd study: Saliva samples for cortisol taken at: *t*0 = baseline; *t*1 = after the insult; *t*2 = *t*1 + 12min.	3rd study: Distance at which participants give way to a confederate in a "chicken game" in a corridor.	2nd study: cortisol level at t1 rose significantly only for participants from south US, but not form north US.	3rd Study: There was no significant difference in the behaviour between insult and no insult condition among participants from northern US. Among participants from southern US those who were insulted continued walking significantly further before giving way, than those who were not insulted.

Cote, McCormick Geniole, Renn, & MacAulay, (2013)	Canada	Randomised Controlled Trial	49 participants (25 female) divided into two groups. Sleep deprivation: 11 male *M*age = 20.55; 13 female *M*age = 19.15. Control: 13 male *M*age = 19.23; 12 female *M*age = 19.25.	Sleep deprivation.	Saliva samples for cortisol were taken at: *t*1= evening (22:30–22:45) two days before the PSAP; *t*2 = morning (07:15–07:30) one day before the PSAP, *t*3 = evening (22:30–22:45) one day before the PSAP, *t*4 = morning on the day of PSAP (07:15–07:30), *t*5 = before the PSAP *t*6 = after the PSAP.	Number of selected aggressive responses (i.e. resulting in loss of money for the opponent) in the PSAP.	No significant effect of condition on cortisol among males or females. Circadian rhythm of cortisol was confirmed for both males and females.	No significant effect of time or cortisol levels or interaction for aggression among either males or females. Cortisol change was not a significant predictor of aggression.
Das, Sengupta, Pathak, Sah, Mehta, Avinash,... & Kalita, (2018)	India	Case Control / Cross Sectional	80 participants: 58 diagnosed with psychosis; 22 healthy volunteers.	None	Saliva samples for cortisol were collected in the morning (8.00-10.00am) and evening (2.00-4.00pm)	LHAS	None	Significant lower morning and evening cortisol levels as well as cortisol variability among participants with extensive history of aggression. Significant negative correlation between history of aggression and cortisol in total sample as well as in sample split by diagnosis.
Feinberg, Jones,	USA	Cross Sectional	137 heterosexual	Conflict discussion task	Acute reactivity: Salivary cortisol as	Revised Conflict	The mean cortisol was	Participant's level of cortisol at t0 was a

Granger, & Bontempo, (2011)			couples, female *M*age 28.3; male *M*age=29.7		measured at three time points: *t*0 = baseline, *t*1 = 15 minutes after the end of the conflict discussion; *t*2 = *t*1 + 20min.	Tactics Scales	declining from t0 to t2. Increase in cortisol from t0 to t1 was present in 15.3% of women and 19% of men.	sign predictor of injury perpetration and of physical assault of high and low severity, for both men and women. Cortisol reactivity (*t*1 - *t*0) was not a sign predictor of either form of aggressive behaviour.
Fishbein, Dax, Lozovsky, & Jaffe, (1992)	USA	Cross Sectional	37 males with history of substance abuse, *M*age = 32.	5-hour oral glucose tolerance test.	blood samples for cortisol response to 5-hour oral glucose tolerance test were obtained (no information about timepoints.)	BDHI; Self-reported criminal histories; aggressive behaviour items from EEQ;	No information	Sign positive correlation of peak plasma cortisol with antisocial hostility subscale of BBDHI and with EEQ; Sign negative correlation of cortisol nadir levels with antisocial hostility subscale of BDHI. Sign lower cortisol nadir among those with criminal histories as compared to those without.
Flanagan, Fischer, Nietert, Back, Moran-Santa Maria,	USA	Randomised Controlled Trial	30 heterosexual couples, *M*age = 32.18.	Conflict resolution task, 40 IU of intranasal oxytocin	Saliva samples for cortisol were collected at: *t*0 = baseline, *t*1 = after first conflict resolution task, *t*2 = after treatment	Coding of psychological abuse during the conflict resolution task.	Female participants to whom oxytocin was administered demonstrated sign lower	No sign difference between two groups in the psychological abuse present during the conflict resolution task was present

Snead, & Brady, (2018)					administration and before second conflict resolution task, *t*3 = after the second conflict resolution task, *t*4 = *t*3 + 15min, *t*5 = *t*4 + 15min, an *t*6 = *t*5 + 30min.		stress reactivity as compared to female participants in placebo group. No sign difference for male participants.	among males or females.
Flegr, Hampl, Cernochova, Preiss, & Bicıkova, (2012)	Czech Republic	Cross Sectional	100 male military personnel, *M*age = 27.9; 93 female, *M*age = 29.2.	None	Blood sample for serum cortisol was taken during annual medical examination.	BDHI	None	No sign correlation between cortisol and physical aggression subscale of DBHI either among males or females.
Gerra, Bassignana, Zaimovic, Moi, Bussandri, Caccavari, ... & Molina, (2003)	Italy	Case Control / Cross Sectional	15 males with history of MDMA use, *M*age = 23.8; 15 male healthy controls, *M*age = 22.9	STROOP task, mental arithmetic task, public speaking to unresponsive audience.	Heart rate (HR) and blood pressure (BP) was measured before each of three stressors and afterwards. Blood samples for plasma cortisol and ACTH, were taken at the baseline and after the last stressor.	BDHI	Sign increase in cortisol and ACTH in both groups after the tasks.	Sign negative correlations were present between ACTH and cortisol delta peaks and direct aggressiveness subscale of the BDHI for MDMA users.
Gerra, Zaimovic, Raggi, Giusti,	Italy	Case Control / Cross Sectional	20 males, *M*age = 27.1, with a history of heroine abuse;	Provocation in form of subtracting	Heart rate (HR) and blood pressure (BP) was measured before each of three PSAP	BDHI; Aggressive responses in the PSAP	Sign increase in NE and IPE across blocks	Sign positive correlation between NE and EPI AUC during the PSAP with

Delsignore, Bertacca, & Brambilla, (2001)			20 healthy males, *M*age = 26.4.	money in the PSAP	blocks and afterwards. Blood sample for plasma cortisol, ACTH, norepinephrine (NE), and epinephrine (EPI), were taken at: *t*0 = baseline; *t*1 = *t*0 + 30min before the first PSAP block; *t*2 = after the first PSAP block; *t*3 = after the second PSAP block; *t*4 = after the third PSAP block;		among patients but only after the second block in controls; Sign increase in ACTH and cortisol across blocks was present only in control group.	aggressive responding among both groups. Sign positive correlation between cortisol AUC and aggressive responding was present in control group only. Sign positive correlation between changes in HR and SBP and aggressive responding was present in both groups.
Gerra, Zaimovic, Raggi, Moi, Branchi, Moroni, & Brambilla, (2007)	Italy	Case Control / Cross Sectional	30 heroin-dependent patients, *M*age = 25; 15 healthy male volunteers, recruited from hospital staff, university students and workers, who were matched in age. All male.	Provocation in form of subtracting money in the PSAP	Heart rate (HR) and systolic and diastolic blood pressure (SBP, DBP) were assessed before and after the PSAP sessions. Stress reactivity: cortisol (CORT) plasma concentrations, adrenocorticotropic hormone (ACTH), epinephrine (EPI), and norepinephrine (NE) were measured	Number of selected aggressive responses (i.e. resulting in loss of money for the opponent) in the PSAP.	HR and SBP increased with number of sessions. No sign change in DBP across three groups. Sign increase in mean concentration of NE and EPI over sessions, was sign higher in	Sign positive correlations between NE and EPI AUCs and aggressive responses in PSAP in all three groups. Sing positive correlations between ACTH AUC and aggressive responding was present in control and in buprenorphine-treatment groups, but not in methadone-treatment group. Sing

					based on blood samples taken at: *t*0 = baseline; *t*1 = *t*0 + 30min before 1st PSAP block; *t*2 = *t*1 + 30min, after 1st PSAP block; *t*3 = *t*2 + 60min, after 2nd PSAP Block; *t*4 = *t*3 + 60min, after 3rd PSAP block.		both patient groups over control group. Sign higher baseline CORT and ACTH among both patient groups, as compared to control. Sing increase in ACTH and CORT was present in control and buprenorphine-treatment groups, but not in methadone group.	positive correlation between CORT AUC and aggressive responding was present only in control group.
Gowin, Green, Alcorn, Swann, Moeller, & Lane, (2013)	USA	Randomised Controlled Trial	67 participants, *M*age = 31.5 years, 45 (7 females) on parole or probation, 41 (5 females) had a SUD in	20 mg of hydrocortisone	Saliva samples of cortisol, HR, SBP, DBP were collected at six time points on each of the three separate days: during two days when they received placebo or	BPAQ. Number of selected aggressive responses (i.e. resulting in loss of money for the	Sign main effect of dosing condition on AUCg: higher cortisol in treatment	No sign main effect of treatment or treatment and session interaction on aggressive responding. Baseline cortisol was not a sig predictor of BPAQ score or PSAP

			remission, and 13 (2 females) with antisocial personality disorder diagnosis.		treatment and at the third day. AUCg was calculated. Cortisol reactivity was measured as the difference in cortisol AUCg on the day when placebo was received and the day when cortisol was received. Only 45 participants were used in the analysis with reactivity.	opponent) in the PSAP	condition as compared to placebo and no treatment. No differences in cortisol after the PSAP. No effect of cortisol treatment for HR, SBP, DBP.	responding when accounting for experience of childhood abuse, psychopathy, and impulsivity. HPA reactivity was not a sign predictor of BPAQ or aggressive responding. Negative correlations between HPA axis reactivity and BPAQ and PSAP. HPA axis reactivity was not a significant mediator between experience of abuse and BPAQ or aggressive responding. Nevertheless, 10 % of the total effect of abuse/neglect on BPAQ and on PSAP responding were mediated by HPA axis reactivity.
Hagan, Roubinov, Mistler, & Luecken, (2014)	USA	Cross Sectional	88 students (50% female), *M*age = 18.67	10-minute role-play when participant is trying to prepare for exam but their	Saliva samples for cortisol were taken at: *t*0 = baseline, *t*1 = after the role-play, *t*2	Externalising subscale of ASR.	None reported	No sign correlation between cortisol reactivity and externalising problems. Sign

				neighbour is playing music too loud.	= *t*1 + 20min; *t*3 = *t*2 + 20min.			interaction between childhood maltreatment and cortisol reactivity in predicting externalising problems was present only when average weekly alcohol use, smoking status, current internalizing problems, and current family conflict were controlled for: sign positive association between childhood maltreatment and externalising problems was only present among those with low cortisol reactivity. However, cortisol reactivity was not a sign mediator.
Keltikangas-Järvinen, Räikkönen, Hautanen, & Adlercreutz, (1996)	Not reported	Cross Sectional	90 male participants, *M*age=44.5.	none	Blood samples for cortisol and ACTH were taken on two consecutive days in the morning. DXM test (1mg) was used to assess cortisol response to ACTH	STAXI	None reported	Sign positive association between anger out subscale of STAXI together with vital exhaustion and net increment of cortisol together with high mean basal

					stimulation. Blood pressure (BP) was measured a after 15-minute rest.			cortisol–to–mean basal ACTH ratio. Sign positive correlation between vital exhaustion together with anger out and net increment in cortisol.
Lee, Bechara, Adolphs, Arena, Meador, Loring, & Smith, (1998)	USA	Before and after case studies	2 male patients aged 19 and 21 who underwent amygdalotomy. Both had a history of uncontrollable rage.	Mental arithmetic, Poststress adaptation Stressful imagery, Poststress adaptation, Cold pressor/sound stressor, Poststress adaptation. Habituation was measured by listening several trials of 1 second 60db noise administered through headphones.	Skin conductance response was measured every 60 seconds during stressors and throughout the habituation task.	File trawl.	Patient 1 showed normal habituation response only 5 days after the procedure. Meanwhile patient 2 only showed normal habituation response 8 years after the procedure. Both patients showed lower skin conductance response to stressors after the	Both patients presented with lower number of aggressive outbursts after the amygdalotomy that before. However, both still experienced problems with controlling aggression. While patient 1 showed decline in aggressive incidents within first month after the procedure, patient 2 showed the decline only 4 months after.

							amygdalotomy.	
Lundberg, Hansson, Andersson, Eneroth, Frankenhaeuser, & Hagenfeldt, (1983)	Sweden	Case Control / Cross Sectional	15 hirsute, *M*age = 25.8 and 14 students, *M*age = 14.1, all female.	Mental Arithmetic, STROOP task, reasoning task.	Heart rate (HR) and blood pressure (BP) was recorded four times before, three times during and once after the task. Blood and urine samples for serum cortisol, norepinephrine (NE) (urine sample only), and epinephrine (EPI) (urine sample only) were taken at: *t*0 = baseline; *t*1 = after the stressors; *t*2 = *t*1 + 2hours. Urine samples were also collected; one night after the session; next morning. The session took place during follicular phase of the menstrual cycle	KSP	Stressor tasks lead to sign increase in heart rate, that was highest during STROOP task. Sign variation in EPI excretion during stress. No sign difference in urinary cortisol over time.	No sign correlation between KSP aggression subscales and cortisol or catecholamines.
Madden, & Shaffer, (2019)	USA	Cross Sectional	57 individuals (46 females, 11 males) , *M*age = 19.47, who were in a heterosexual	TSST about current relationships.	Stress reactivity was measured using saliva cortisol as AUC score with two timepoints: *t*0 =	The Conflict Tactics Scale-Revised		No sign correlation between cortisol reactivity and dating conflict. However, cortisol reactivity was

			dating relationship		baseline, *t*1 = 25minutes after the start of TSST.			a significant mediator of the effect of childhood emotional abuse and dating conflict. Only blunted cortisol reactivity had a sign moderating effect.
Margittai, Van Wingerden, Schnitzler, Joëls, & Kalenscher, (2018)	Germany	Randomised Controlled Trial	150 male divided into four conditions: 36 placebo only (*M*age = 24.8), 38 placebo and yohimbine (*M*age = 23.4), 38 placebo and hydrocortisone (*M*age = 26.6), 38 yohimbine and hydrocortisone (*M*age = 26)	20 mg of hydrocortisone	Salivary cortisol, salivary alpha-amylase (sAA), and subjective feelings of stress (VAS) were collected at 5 timepoints: *t*0 = baseline; *t*1 = *t*0 + 10min second baseline; *t*2 = 30 minutes after treatment; *t*3 = *t*2 + 30min; *t*4 = *t*3 + 15min.	Social discounting task via dictator game.	Sign increase in cortisol among those who received hydrocortisone, but not in those who received yohimbine. Sing increase in salivary alpha-amylase levels with time in those who received yohimbine, but not in those who received hydrocortisone.	Sign interaction between yohimbine and hydrocortisone. participants treated with hydrocortisone were more generous to close others, as compared to placebo, and cortisol and yohimbine. No sign effects of cortisol and sAA on the decline in generosity with social distance.

Martorell, & Bugental, (2006)	USA	Cross Sectional	60 mothers of toddlers, *M*age=27.03	The Strange Situation	Saliva samples for cortisol at 3 times: *t*0 = baseline coming to the lab, *t*1 = non stressful activity; *t*2= and stranger situation.	Conflict Tactics Scale (Straus, 1979) for assessment of Harsh Parenting.	No information.	Increase in Mother's cortisol predicted harsh parenting as part of mediation analysis. As a result, increase in cortisol was a mediator between the interaction between the perceived power of the parent and child difficulty) and harsh parenting.
Mehta, DesJardins, van Vugt, & Josephs, (2017)	USA	Cross Sectional	98 students (42 males)	Provocation in form of a decision of the opponent to earn more resources at the expense of the participant in the Hawk-Dove game.	Saliva samples for cortisol were taken before, during, and after the experimental paradigm.	Number of selected decisions resulting in higher earning at the expense of the opponent in Hawk-Dove game.	Sign effect of time on cortisol: only in females the level of cortisol sign decreased over time.	Sign negative correlation between cortisol at the difference in cortisol from after the task to baseline and hawk decisions. Baseline cortisol was not a sign predictor of hawk decisions. Sign effect for cortisol change from before to after the game on decisions: participants with negative slope made more hawk decisions, when controlling for gender.

Melhem, Munroe, Marsland, Gray, Brent, Porta, ... & Driscoll, (2017)	USA	Case Control / Cross Sectional	38 patients with suicide attempts (44.7% females), *M*age=22.8; 40 patients with suicide ideation (27.5% females), *M*age=23.6; 37 controls (54.1% females), *M*age=22.1.	None	Hair cortisol samples	BPAQ	No information.	No sign correlation between aggression and hair cortisol.
Meyerhoff, Norris, Saviolakis, Wollert, Burge, Atkins, & Spielberger, (2004)	USA	Cross Sectional	no information.	Emergency vehicle operation, interactive situations where police instructors engaged in fire exchange using paintballs,	Heart rate, blood pressure, salivary cortisol.	Firing accuracy, shooting judgement.	Sign increase in heart rate, blood pressure, and cortisol levels,	19% shot the hostage. "97% failed to meet the criterion of 70% of their rounds hitting the suspect"
Murray-Close, Holterman, Breslend, & Sullivan, (2017)	USA	Cross Sectional	247 college students (74% female), *M*age = 18.77.	Social stress task: semi structured interview about recent experience of victimisation. Social exclusion in Cyberball. Mental arithmetic task accompanied by standardized minor verbal	Skin conductance levels was assessed before and during each stressor. Mean levels of SCL were calculated for each timepoint. SCL reactivity was calculated by subtracting mean SCL before a	SRASBM. With Proactive and Reactive Relational Aggression.	No information.	No sign association between SCL reactivity during interview and mental arithmetic and either form of aggression. However, there was a sign positive association between SCL-R during Cyberball and reactive

				harassment from the experimenter telling them to work harder and faster, with 30s intervals.	stressor from mean SCL during it.			relational aggression. Moreover, interaction between SCL-R and Respiratory Sinus Arrhythmia Reactivity (RSA-R) was sign for both forms of aggression. Specifically, proactive aggression was highest among participants with high RSA-R but low SCL-R. Meanwhile, there was a sign positive association between RSA-R and reactional aggression only among participants with low SCL-R.
Newman, & McDermott, (2011)	USA	Case Reports	patient 1 a 40-year-old male inpatient of and patient 2 a 20 year old male outpatient. Both had a history of aggressive Behaviour.	Beta-blockers: Pindolol and propranolol	Clinical observation and pulse measured during appointments.	Clinical observation and file trawl.	Patient 1 reported to feel calmer after beginning of the treatment than before. His pulse before administratio n of the pindolol was	Patient 1 was not reported to present aggressive outbursts after beginning of the treatment. Patients 2 reported absence of aggressive outbursts after beginning of the treatment.

							around 80. Patient 2 also reported to feel calmer after beginning of the treatment than before. He was also reported to be visibly less agitated by his social worker.	
Olff, Brosschot, Godaert, Benschop, Ballieux, ... & Ursin, (1995)	The Netherlands	Cross Sectional	86 males, *M*age = 40.5	Impossible to solve puzzle followed by explanation to confederate.	blood samples for cortisol and norepinephrine (NE) were taken at: *t*0 = baseline and *t*1 = after the stressor.	Defensive hostility based on defence mechanism inventory subscales turning against object and projection and on lifestyle index subscales projection and	Sign decrease in cortisol levels, no effect of time on NE.	No sign partial correlation between defensive hostility and cortisol. Sign negative partial correlation between hostility and NE.

						compensation.		
Pesce, La Fratta, Ialenti, Patruno, Ferrone, Franceschelli, ... & Felaco, (2015)	Italy	Cohort / Cross Sectional	25 male kickboxers, *M*age = 28.68	Kick boxing matches	Saliva samples for cortisol were taken: before and after 5 simulated matches occurring once a month, and before and after official match.	STAXI 2	Cortisol concentrations sign decreases across 5 months before the official match. Cortisol concertation was sign higher before and after the official match than before and after last simulation.	No sign correlation between baseline cortisol at the official match and anger score.
Peters, Godaert, Ballieux, & Heijnen, (2003)	The Netherlands	Randomised Controlled Trial	94 male students, *M*age = 22 for cardiovascular measures. Form them only 79 have data for catecholamines levels.	mental tasks performed with continuous noise in the background.	Blood pressure (BP) was measured throughout the session. Blood samples for norepinephrine (NE), epinephrine (EPI) and saliva samples for cortisol were taken at: *t*0 = baseline; *t*1 = after	BDHI	Sign effect of condition on physiological reactivity. Sign effect of stressor intensity: higher intensity stressor evokes	No sign correlations between aggression and baseline measures of cortisol, BP, NE, or EPI. Aggression was a sign moderator of stressor on immunological response.

					the stressor, *t*2 = *t*1 + 15min; *t*3 = *t*2 + 15min		higher increase in BP	
Prasad, Narayanan, Lim, Koh, Koh, & Mehta, (2017)	Singapore	Cross Sectional	39 students (19 females), *M*age=21.69.	TSST.	Saliva samples for cortisol were taken before and after the stressor.	Rejection of unfair offer in Ultimatum game.	Sign effect of condition on cortisol change: there was sign greater increase in cortisol among those who underwent TSST.	Sign interaction of cortisol reactivity and basal testosterone change: marginally sign (p=.077) positive association between basal testosterone and rejection of unfair offers only among participants with decreased cortisol reactivity. However, nether cortisol reactivity nor basal cortisol was not a sign mediator.
Rausch, Gäbel, Nagy, Kleindienst, Herpertz, & Bertsch, (2015)	Germany	Case Control / Cross Sectional	55 patients (35 female; *M*age = 27.4) with diagnosis of borderline personality disorder (BPD) and 47 matched heal controls (26 female, *M*age = 28.0) who had never received a psychiatric	None	Trait HPA activity: Cortisol awakening response assessed using saliva samples on two consecutive days: *t*0 = awakening; *t*1 = *t*0 + 30min; *t*2 = *t*1 + 15min; *t*3 = *t*2 + 15min. AUCg and the mean cortisol increase (MnInc) was computed.	STAXI; BPAQ	Cortisol levels rose after awakening and the declined.	positive associations between AUCG and MnInc and BPQA and STAXI only in female BPD patients. However, neither AUCG nor MnInc, nor mean cortisol were sign in the hierarchical regression with BPAQ as outcome.

			diagnosis or undergone any psychological or psychiatric treatment.					
Ritsner, Maayan, Gibel, Strous, Modai, & Weizman, (2004)	Israel	Case Control / Cross Sectional	40 patients diagnosed with schizophrenia (2 female), *M*age = 38; 15 controls (2 female), *M*age = 35.1.	None	Blood samples for cortisol, Dehydroepiandrosterone (DHEA), and Dehydroepiandrosterone sulphate (DHEA-S) were taken in the morning (8.00-9.00am).	STAXI	None	Sign positive correlation between cortisol/DHEA ration and angry temperament and hostility among patients.
Ritsner, Modai, Gibel, Leschiner, Silver, Tsinovoy, ... & Gavish, (2003)	Israel	Case Control / Cross Sectional	40 patients diagnosed with schizophrenia (2 female), *M*age = 38; 15 controls (2 female), *M*age = 35.1.	None	blood sample for [3H] PK11195 binding were taken in the morning (8.00-9.00am). Equilibrium dissociation constant and maximal number of binding sites for Peripheral-type benzodiazepine receptors (PBR) were determined.	File trawl for aggressive incidents, STAXI	None	Sign negative correlations between number of binding sites ad aggressive Behaviour and hostility were present among patients. However, each correlation lost sign when the other variable was adjusted for. Currently aggressive patients had sign lower binding sites density than homicidal patients, non-

								aggressive patients, and controls.
Romero-Martínez, & Moya-Albiol, (2016)	Spain	Case Control / Cross Sectional	35 parents of a child diagnosed with autism spectrum disorder (ASD): 13 males, *M*age = 45.46, 22 females, *M*age = 45.27; 35 controls: 13 males, *M*age = 39.92; 22 females, *M*age = 45.	TSST	Saliva samples for testosterone/cortisol ratio were taken at: *t*0 = baseline, *t*1 = before TSST, *t*2 = after TSST, *t*3 + *t*2 + 20min, *t*4 = *t*3 + 10min.	state anger measured by STAXI 2 before and after stressor	Sign effect of time for ASD males and females and control males: decrease in cortisol levels from *t*0 to *t*1, but increase from *t*1 to *t*3 followed by decrease from *t*3 to *t*4. No sign was found among control females, but the pattern was similar.	Sign increase in state anger following stress was present among all participants. Sign higher mean state anger among ASD group than controls.
Romero-Martínez & Moya-Albiol, (2017)	Spain	Case Control / Cross Sectional	29 caregivers for an offspring diagnosed with eating disorder (ED), *M*age = 51.34; 36 controls, *M*age = 47.07.	TSST	Salivary samples for cortisol were taken at: *t*0 = baseline, *t*1 = after habituation period; *t*2 = after information about stressor was provided; *t*3 = between second and	state anger measured by STAXI 2 before and after stressor	Sign effect of time for cortisol:	Sign effect of time for state anger was present: in both groups anger sign increased after the stressor.

					third task of the TSST; *t*4 = after the stressor; *t*5 = *t*4 + 10min; *t*6 = *t*5 + 10min; *t*7 = *t*6 + 10; *t*8 = *t*7 + 15; *t*9 =*t*8 + 16min.			
Romero-Martínez, González-Bono, Lila, & Moya-Albiol, (2013)	Spain	Case Control / Cross Sectional	37 male participants: 16 IPV perpetrators, *M*age = 38.31; 21 controls, *M*age = 35.81.	TSST	Saliva samples for testosterone/cortisol ratio were taken at: *t*0 = baseline, *t*1 = before TSST, *t*2 = after TSST, *t*3 = *t*2 + 15min, *t*4 = *t*3 + 15min; *t*5 = *t*4 + 15min.	state anger measured by STAXI 2 before and after stressor	Sign effect of time for testosterone/cortisol ratio: it sign increased from *t*0 to *t*1 and from *t*4 to *t*5, it non-sign decreased from *t*1 to *t*2.	Sign interaction of time and group on state anger: IPV perpetrators had sign higher state anger than controls at *t*0. Decrease in state anger among IPV group following stressor but increase in controls. IPV perpetrators had sign higher testosterone to cortisol ratio than controls at *t*1, *t*4 and *t*5.
Romero-Martínez, Lila, & Moya-Albiol, (2016)	Spain	Case Control / Cross Sectional	36 male volunteers: 16 IPV perpetrators (*M*age = 38.31) and 20 control participants (*M*age = 35.2).	None	Basal saliva cortisol levels were measured once.	STAXI 2	None	Basal cortisol levels predicted 15.8% of the anger expression among IPV perpetrators but not in controls. However basal cortisol was not a sign moderator for any variables.

Romero-Martínez, Lila, Sariñana-González, González-Bono, & Moya-Albiol, (2013)	Spain	Case Control / Cross Sectional	40 males, *M*age = 37.55: 19 IPV perpetrators, 21 controls.	TSST	Saliva samples for cortisol were taken at: *t*0 = baseline; *t*1 = before the stressor; *t*2 = after the stressor; *t*3 = *t*2 + 15min; *t*4 = *t*3 + 15min; *t*5 = *t*4 + 15min.	state anger measured by STAXI 2 before and after stressor	Sign effect of time on cortisol: only in controls there was a sign increase in cortisol between *t*1 and *t*3.	Sign interaction of group and time for anger: sign decrease in IPV but increase in controls.
Rostrup & Ekeberg, (1992)	Norway	Cross Sectional	32 19-year-old males with blood pressure 116 mm Hg or higher.	CPT	blood pressure (BP) was measured throughout the session. Heart rate and blood samples for norepinephrine (NE), epinephrine (EPI) were taken at: *t*0 = baseline, *t*1 = after the stressor, *t*2 = during recovery.	Anger and hostility scales from Karolinska Scale of Personality.	Participants informed about high blood pressure had sign higher increase in NE and HR than those who were not informed. EPI sign increased only in informed group.	No sign correlations between measures of stress and that of aggression.
Roy, (2004)	UK	Cross Sectional	82 male firefighters, *M*age = 25	mental arithmetic, speech task.	Saliva samples for cortisol were taken at: *t*0 = 30 min into adaptation; *t*1 = *t*0 + 15min; *t*2 = after mental arithmetic	STAXI	Sign effect of time on stress measures.	No sign correlation between mean baseline stress measures and STAXI. Sign negative correlation between

					task, *t*3 = between task recovery; *t*4 = after speech task; *t*5 = *t*4 + 10min; *t*6 = t5 + 10min; *t*7 = *t*6 + 10min. Heart rate (HR) and blood pressure (BP) was taken through the session.			cortisol change and anger expression and anger-out scales of STAXI. Sign positive correlation between cortisol change and anger control scale of STAXI.
Ruiz-Robledillo & Moya-Albiol, (2015)	Spain	Case Control / Cross Sectional	64 parents: 16 female, *M*age = 45.62 and 14 male, *M*age = 46.35 caregivers with offspring diagnosed with autism spectrum disorder; 20 female, *M*age = 45.50 and 14 male, *M*age = 41.07 controls.	Cognitive tasks	Skin conductance levels were assessed at: *t*0 = baseline, *t*1 = while participants received instructions about the tasks; *t*2 = during anticipation of the stressor; *t*3 = during the stressor; *t*4 = after the stressor.	STAXI 2	Sign effect of time on SCL for all participants: sign increase from *t*0 to *t*3	Sign effect of time on anger was only present in control group: sign increase of anger following the stressor. No sign association between SCL changes and STAXI 2 scores.
Ruiz-Robledillo, Romero-Martínez, & Moya-Albiol, (2017)	Spain	Cross Sectional	40 caregivers for people with autism spectrum disorder (62% female), *M*age=45.77.	Cognitive tasks	Saliva samples for cortisol were taken at: *t*0 = arrival; *t*1 = adaptation; *t*2 = baseline; *t*3 = between cognitive tasks; *t*4 = after the stressor; *t*5 = *t*4 + 10min;	STAXI	Sign effect of time on cortisol: increase in cortisol after the stressor.	Sign positive correlation between stressor cortisol AUCg (*t*0 - *t*4) and anger expression and angry temperament; Sign negative correlation between

					t6=t5+10min; t7=t6+10min; t8=t7+15min; t9=t+15min.			total AUCi (*t*0 - *t*10) and anger reaction.
Ryff, Love, Urry, Muller, Rosenkranz, Friedman, ... & Singer, (2006)	USA	Cross Sectional	135 women, *M*age = 74.	None	Saliva samples for cortisol were taken three times during the day: morning, midday, and evening. Urinary samples for cortisol, norepinephrine (NE), and epinephrine (EPI) were taken overnight. Blood pressure (BP) was taken after 5 minutes of rest. Blood sample was taken for DHEA-S	10 items from STAI assessed trait anger.		EPI had a sign positive correlation with trait anger. SBP had a sign negative relationship with trait anger.
Salvador, Suay, Martinez–Sanchis, Simon, & Brain, (1999)	Spain	Cross Sectional	28 male judo fighters, *M*age = 18.32	Judo fight encounter.	Cortisol changes**:** Blood samples were taken for cortisol levels before and after encounter. The change values were also calculated.	Videotaped fighting encounters rated by two judo specialists. Attack/counterattack label was given to offensive responses as	Cortisol before the fight was positively correlated to cortisol levels after it. Similarly changes in cortisol were	Basal cortisol was not a sign correlate of fighting or attacking. However, change in cortisol levels sign and positively correlated with both fighting and attacking. Moderation analysis was not performed as cortisol did not

						opposed to defence label that was assigned to avoiding or blocking opponent. Meanwhile, fight label was applied to a struggle during which participant was trying to obtain an advantage.	positively correlated to	interact with testosterone.
Scarpa, & Ollendick, (2003)	USA	Case Control / Cross Sectional	47 Students (*M*age = 20.74): 18 male, 29 female. 25 were victims of community violence, 22 were not.	60 bursts of white noise (100 dB with 2–5) with randomized intervals of 10–40 seconds that could not be prevented despite what was told to the participants. Asking participants to solve 20 five-letter anagrams.	Saliva samples of cortisol; heart interbeat interval (IBI) to assess heart rate (HR) level and HR variability (HRV) were obtained four times: *t*0 = baseline; *t*1 = before the noise; *t*2 = after the noise; *t*3 = after anagrams. Only IBI at *t*0 and cortisol and *t*3 were used for analysis.	BPAQ	No information.	Sign association between low HR and aggression among those who experienced victimisation, but not for those who did not have such experience. Similarly, increased HRV has a sign association with aggression only for those who were victimised. Sign associations association between

								increased aggression and increased cortisol was present only among those who experienced victimisation. However, there were no sign difference in aggression when two groups were split into high and low HR, HRV and cortisol levels.
Scarpa, Fikretoglu, & Luscher, (2000)	USA	Cross Sectional	54 students (35 female), *M*age = 20.5, who varied on exposure to community violence.	Uncontrollable white noise; anagrams.	Salivary samples for cortisol and heart beat interval were assessed at: *t*0 = bassline; *t*1 = before first stressor; *t*2 = after the first stressor; *t*3 = after the second stressor.	BPAQ	No information.	Sign positive correlation between total score on BPAQ and cortisol levels at *t*3 and heart rate variability at t0. Sign negative correlation between total score on BPAQ and heart rate at t0. Sign positive correlation between physical aggression scale and cortisol level at *t*3.
Schneiderman, Kanat-Maymon, Zagoory-Sharon, &	Israel	Case Control / Cross Sectional	60 heterosexual couples, males *M*age = 25.03, females, *M*age = 22.84. 40	Conflict discussion task	Blood samples for serum cortisol and DHEA-S were taken before the conflict	Hostility rating on CIB	No information.	Sign positive correlation was present between cortisol levels before the stressor and

Feldman, (2014)			participants (21 female) who were not involved in a romantic relationship, *M*age = 24.63.		discussion and 6 months afterwards.			hostility only for female participants. Cortisol and DHEA-S levels were found to be sign predictors of hostility
Schwartz & Portnoy, (2017)	USA	Cross Sectional	1255 (46.07% males) *M*age = 55,26. Only 967 completed all cardiovascular measures. Meanwhile completion of covariate ranged from 682 to 742	Mathematical, Verbal, and Physical Challenge.	HR and HRV reactivity were measured twice for baseline and after each step. Meanwhile levels of epinephrine, norepinephrine, and dopamine were assessed based on 12 overnight urine samples.	composite anger score based on STAXI and aggression scale of MPQ.	None reported	Sign and negative association between anger and catecholamines. No sign association between HR, HRV reactivity and anger
Sherwood, Hughes, Kuhn, & Hinderliter, (2004)	USA	Cross Sectional	80 female, *M*age = 50.2	None	Heart rate (HR) and blood pressure (BP). 24-hour Urine sample was collected within 1 week of the session for epinephrine (EPI), and norepinephrine (NE). Beta- and alpha-adrenergic receptors responsiveness were assessed using	CMHS	None	No sign correlation between hostility and arterial measures alpha adrenergic responsiveness. Sign positive correlation between cardiac and vascular measures of beta-adrenergic responsiveness. Sign positive correlation between NE and hostility. No sign

					isoproterenol and phenylephrine sensitivity tests.			correlation between EPI and hostility.
Solanki, Sharma, Tyagi, & Singh, (2017)	India	Case Control / Cross Sectional	30 patients (40% female) diagnosed with first-episode antipsychotic-naïve schizophrenia, *M*age = 24.3, 20 healthy (35% female) controls, *M*age = 27.9.	None	Blood samples for cortisol and dehydroepiandrosterone sulphate (DHEA-S) were taken in the morning (8.30-9.30am)	Modified Overt Aggression Scale (MOAS)	None	Sign negative correlation between DHEA-S and MOAS, No sign correlation between cortisol level and MOAS.
Steptoe, Fieldman, Evans, & Perry, (1996)	UK	Cross Sectional	132: 30 women and 29 men in the younger group (30 - 40 years old), 38 women and 35 men on older group (55 - 65)	Three stress tasks: visual matrix problem-solving task; mirror drawing, speech task, where participants had to explain their innocence following unjust accusation. It was videotaped, and participants were told that it would be rated.	Hear rate during increasing fitness workload was taken prior to stress session. Beat-to beat heart rate, systolic and diastolic blood pressure, skin resistance, were taken at baseline and throughout the stress tasks. Saliva sample for cortisol were collected at the baseline, following each stress task, and 8 minutes after the last task.	STAXI	Sign main effect of task was present for all stress variables apart from cortisol.	Sign negative association between systolic blood pressure responsivity and anger expression in both groups. No other sign effects were present

Sullivan, Procci, DeQuattro, Schoentgen, Levine, Van Der Meulen, & Bornheimer, (1981)	USA	Case Control / Cross Sectional	6 patients with pheochromocytoma, *M*age = 42; 15 patients with primary hypertension, *M*age = 37; 20 healthy controls, *M*age = 36.	Isometric handgrip exercise (IHE). On a different day mental arithmetic.	Blood pressure (BP), heart rate (HR), and blood sample for norepinephrine (NE) and epinephrine (EPI) were taken before and after the IHE. Next day cerebrospinal fluid NE was taken. Following day, the BP, HR, NE, and EPI were taken only after the arithmetic.	BDHI; Anger in and anger out scales of STAXI.	Sign increase in BP and HR after IHE. Sign increase in BP and HR after mental arithmetic was only present in controls. Sign increase of HR and EPI in hypertensive participants following mental arithmetic. Sign increase in NE after IHE.	Sign higher anger score among patients with hypertension as compared to controls.
Van Eck & Nicolson, (1994)	The Netherlands	Cross Sectional	88 males, *M*age = 42.1	None	PSS; Experience Sampling Method (ESM) and saliva samples for cortisol were taken through five consecutive days at approximately 90-minute intervals	STAXI	Sign effect of time for cortisol, which followed normal daily pattern. No sign difference between	No sign correlations between trait anger and average cortisol level during the workdays.

					from 8.00am to 10.00pm		people with high and people with low perceived stress in cortisol levels during the day. No sign difference in cortisol levels on weekdays and on weekends.	
Van Orden, Benoit, & Osga, (1996)	USA	Cross Sectional	24 active duty officers or enlisted personnel males, *M*age = 31.8.	exposure to 4°C cold chamber.	Heart rate (HR), Blood pressure (BP), and blood sample for, norepinephrine (NE), and epinephrine (EPI) were taken at: *t*0 = baseline; *t*1 = before entering the chamber; *t*2 = *t*1 + 40 = the middle of the NAIMES session; *t*3 = 35 minutes after the end of the session.	Navy Advanced Information Management and Evaluation System (NAIMES)	Sign interaction between group and time on NE and HR: NE and HR levels at *t*2 of participants who were in cold chamber were sign higher than at any other time and then levels among	No sign difference in the total number of engagements between stressed and non-stressed group. Participants in the cold chamber authorised sign more unprompted engagements than participants in the mild chamber. Stressed participants show sign more engagements per

							the participants who were in the mild chamber. Sign interaction of time and group for EPI: the EPI levels of participants who were in the cold chamber were sign higher than those of participants in the mild chamber at *t*1, *t*2, and, *t*3.	target than non-stressed ones.
Vaz-Leal, Rodríguez-Santos, Melero, Ramos, Monge, & López-Vinuesa, (2007)	Spain	Cross Sectional	60 female patients seeking treatment for eating disorders, *M*age = 24.7, 25 female controls, *M*age = 24.5.	None	Blood sample for cortisol were taken at: *t*0 = before 1 mg of dexamethasone; *t*1 = next morning. Ability to suppress cortisol was calculated as (*t*1/*t*0)x100.	hostility scale of SCL-90R	None	Sign negative correlation between ability to supress cortisol and hostility.

Verona & Curtin, (2006)	USA	Cross Sectional	117 students (59 female), *M*age = 21	Air blasts of 100psi 50ms duration directed at the throat.	Startle response measured as peak magnitude of eyeblink. They were taken at baseline, and 4 times during each of 4 block of aggression paradigm.	Average shock intensity during one of four blocks of teacher-learner task: providing feedback to scripted "opponent": correct, or shock with varying intensity. Linear increase over block was also computed. subscale of MPQ-BBF.	Aggressive responses during the task correlated with MPQ-BF aggression subscale. Sign main effect of stress on startle sensitisation: greater with the stress then without it.	Sign interaction between gender, stress and block: sign increase in aggressive responding with each block in stress group but not in non-stressed group was present among males. In females the sign increase in aggression with each block was present in non-stressed condition only. Sign interaction between gender and startle sensitisation: sign positive association of startle sensitisation with shock intensity but only among males.
Verona & Kilmer, (2007)	USA	Cross Sectional	122 (66 female) students, *M*age= 20.5.	High stress: air blasts at the throat 100psi 50ms duration; low stress: air blasts 10psi.	Startle response was measured at the baseline and during stress induction.	Average shock intensity during one of four blocks of teacher-learner task: providing feedback to scripted	Aggressive responses during the task correlated with MPQ-BF aggression subscale. Hostile	Sign interaction was present between stress and gender: male participants were more aggressive in a given block than female, and the difference was greater in high stress conditions as

					"opponent": correct, or shock with varying intensity. Linear increase over block was also computed. subscale of MPQ-BBF.	motives accounted for 15% variance of aggressive response, while instrumental motives only added 6%. Sign higher startle sensitisation in high stress group than in low stress. Sign effect of stress across blocks on startle sensitisation: in women this effect dissipated with block, but in males it remained high.	compared to low stress condition. Sign effect of stress was present when sample was split by gender. Female participants in low stress condition were sign more aggressive than those in high stress. The reversed effect for males only approached sign (p=.052). Sign quadratic block effect was present for female participants in both conditions and males in low stress conditions. For males in high stress condition only liner effect was sign. Sign interaction between startle sensitisation, gender, and liner block effect: startle sensitisation was sign associated with liner increase in aggressive responses among

								males, but not females.
Verona, Joiner, Johnson, & Bender, (2006)	USA	Cross Sectional	111 students (56 female), *M*age = 21	Air blasts at the throat.	Startle response was measured at the baseline and during stress induction. Polymerase chain reaction (PCR) amplification of 5HTTLPR was performed on DNA extracted from cells.	Average shock intensity during one of four blocks of teacher-learner task: providing feedback to scripted "opponent": correct, or shock with varying intensity. Linear increase over block was also computed	Aggressive responding was sign correlated with anger expression and self-reported aggression. Main effect of stress on startle sensitisation.	Sign interaction between stress and genotype was present among males only: in high stress condition males with homozygous S genotype were more aggressive than males with one or two L alleles. Exclusion of l/l genotype confirmed the results: Sign interaction between stress, gender, and genotype: Sign effect of genotype was only present in males:
von Dawans, Ditzen, Trueg, Fischbacher, & Heinrichs, (2019)	Germany	Randomised Controlled Trial	94, heterosexual women, Mage=23	TSST split with decision game: public-speaking task (stress), 12 decisions (five min), mental arithmetic task (stress), 12 decisions.	Acute stress measured via salivary cortisol levels, for which AUCG and AUCi were calculated. They were taken at: *t*0 = baseline; *t*1 = start of the TSST; *t*2 = after first half of	The punishment game involving distribution of monetary units.	Main effect of stress for VAS; Sign increase in cortisol, cortisol increases and HR in stress as compared	No sign interactions or main effects.

					TSST; *t*3 = after second half of TSST; *t*4; *t*5; *t*6; *t*7 = during recovery phase with 10; 15; 20 minutes intervals. Hear rate: measured by beat to-beat heart rate data and calculated one-minute mean values: at *t*0, *t*1, *t*2, *t*3, *t*4. Only 84 participants had HR data.		to non-stress condition,	
Von Dawans, Fischbacher, Kirschbaum, Fehr, & Heinrichs, (2012)	Switzerland	Randomised Controlled Trial	67 male students, *M*age=21.31.	TSST split with decision game: public-speaking task (stress), 12 decisions (five min), mental arithmetic task (stress), 12 decisions.	Acute stress measured via salivary cortisol levels, for which AUCG i were calculated and heart rate (HR). Taken with VAS at: *t*0 = baseline; *t*1 = start of the TSST; *t*2 = after first half of TSST; *t*3 = after second half of TSST; *t*4; *t*5; *t*6; *t*7; *t*8 = during recovery phase with 10; 10; 20; 10 minutes intervals. Hear rate: measured by beat to-beat heart	The punishment game involving distribution of monetary units.	Sign effect of condition on cortisol levels and HR. Sign interaction between condition and time for cortisol and HR: increase over time in stress condition.	No sign difference between groups in punishment. No sign correlation between HR or cortisol and punishment.

					rate data and calculated one-minute mean values: at *t*0, *t*1, *t*2, *t*3, *t*4. Only 84 participants had HR data.			
Walther, Waldvogel, Noser, Ruppen, & Ehlert, (2017)	Switzerland	Cross Sectional	Study 1: 271 self-reporting healthy males, *M*age = 57.1; Study 2: 123 males with at least mild score on Maastricht Vital Exhaustion Questionnaire (MVEQ), *M*age = 52.7; Study 3: 384 males who are father to biological or adopted children, *M*age = 43.75	None	Studies 1 & 2: Saliva and hair samples for cortisol; DHEA, cortisone (only hair samples) were taken in the morning. Study 3: saliva samples for cortisol were taken after awakening on two consecutive days.	Studies 1 & 2: BPAQ. Study 3: aggressiveness/hostility scale of the BSI.	None	Study 1: Sign moderation effect of hair cortisol and cortisone on the negative association between age and ager. A trend was observed for salivary cortisol as a moderator of the effect of anger on physical and verbal aggression subscales of BPAQ. A trend was observed for hair cortisol and cortisone as moderators of the effect of age on anger. Study 2: A trend was observed for hair cortisol to moderate the effect of age on verbal aggression. A trend was observed for hair DHEA to moderate the effect of age on anger. Study 3:

								no sign moderation effects of salivary cortisol.
Westrin, Engstöm, Ekman, & Träskman-Bendz, (1998)	Sweden	Case Control / Cross Sectional	38 patients (22 females) with suicide attempts, *M*age = 38; 38 controls (22 females) with suicide attempts, Mage=38;	None	Serum and blood samples for cortisol, CRH-LI, NPY-LI, and DSIP-LI were taken at: *t*0 = before 1 mg dexamethasone; *t*1 = next day between 8.00am and 3.00pm.	Indirect aggression, verbal aggression, inhibition of aggression scales of Karolinska Scales of Personality (KSP)	None	Sign positive correlation between cortisol at t0 and verbal aggression among controls only. Sign negative association between cortisol at t0 and aggression inhibition among controls only. Sign negative correlation between NPY-LI and verbal aggression among controls only.
Woodman, Hinton, & O'Neill, (1978) Study A	UK	Cross-Sectional	58 male patients of high secure hospital aged between 18 and 45.	35-minute session on 2 consecutive days that included: perceptual discrimination tests; criticism; frustration mental tests; looking at pictures of human suffering, and passive noxious stimulation,	Blood samples for epinephrine (EPI) and norepinephrine (NE) were taken several days prior to first stress session and after completion of the session on the second day. NE to EPI ration of 5.5 was used as a cut off for hyperresponsivity.	Nature of crime coded based on case files.	None reported.	Proportion of violent crimes against strangers and proportion of crimes resulting in fatality was sign higher among hyporesponsive patients than among patients with normal stress response.

				following a warning.				
Woodman, & Hinton, (1978) Study B	UK	Cross-Sectional	58 male patients of high secure hospital, 19 hospitalised to neuropsychiatric Royal Air Force males, 18 non-hospitalised males.	35-minute session that included: perceptual discrimination tests; criticism; frustration mental tests; looking at pictures of human suffering, and passive noxious stimulation, following a warning.	Urinary samples for epinephrine (EPI) and norepinephrine (NE) were taken during routine activities to serve as baseline and 2 hours after awakening on the day of the stressor during anticipation of the stressor. NE to EPI ration of 5.5 was used as a cut off for hyperresponsivity.	Case record data.	None reported.	Patient with higher NE to EPI ration had sign more "murderous attacks" on their record that patients with lower NE to EPI ratio.
Yoshihara, Hiramoto, Oka, Kubo, & Sudo, (2014)	Japan	Cohort Study	39 healthy females.	None	urine sample for Cortisol Awakening Response taken between 6.00am and 8.00am was collected before and after 12-week yoga training.	Hostility scale of SCL-90R	No sign change in cortisol levels.	No sign correlation between change in hostility and change in cortisol.
Zhan, Wu, Fan, Guo, Zhou, Ren, ... & Luo, (2017)	China	Cross-Sectional	180 undergraduate and graduate students (66% women), *M*age = 20.76.	CPT, anger induction by negative evaluation of participant's viewpoint on relevant topic.	Stress responses were measured via salivary samples of sAA and cortisol. They were taken at three timepoints: *t*0 = baseline; *t*1 =	TAP	Sign main effect was present for subjective stress and cortisol. No sign effect of	No sign main effect of stress or interaction with regulation. Sign positive correlation between SC level after anger regulation and aggressive behaviour.

				10min after manipulation, *t*2 = after anger regulation. Skin conductance was measured throughout the session.	stress for sAA. Sign increase in SCL after anger regulation among both stressed and non-stressed participants who were subjected to cognitive reappraisal and neutral mood induction.

5.5.1 Characteristics of Included Studies

A detailed tabulation of the included studies is provided in Table 5.1

5.5.1.1 Study Quality Appraisals

Quality appraisals of the studies included in the review were conducted using the Quality Appraisal Checklist – Quantitative Intervention Studies created by the National Institute for Care and Excellence (NICE). It includes 27 questions pertaining to the study design that can be answered as: "++" indicating minimal risk of bias; "+" indicating that not all sources of bias were addressed or it is unclear; "-"reflecting that biases were not addressed; "Not reported" for aspects of the study that were not disclosed; and "Not applicable" for the questions that are irrelevant to a given study. The checklist was developed based on the Graphical Appraisal Tool for Epidemiological Studies (GATE) (Jackson et al., 2006) which allows for the assessment of the validity of studies with designs varying from experimental to observational. All the studies were assessed for quality by the author.

Almost half of the studies (k = 35) were rated as being very good quality, as they had more "++" than "+" ratings, and no "-". Similarly, almost one third of the studies (k = 25) were of good quality, as they had more (or equal number of) "+" than "++" ratings and no "- ". However, there also were several fair quality studies (k = 8) that had one "-" and poor quality studies (k = 9) that had more than one "-".

5.5.1.2 Study Designs, Countries and Participants Demographics

The majority of the studies (k = 36) employed a cross-sectional design, or a cross-sectional/case-controlled design (k = 24), where participants were divided into groups, based on outcome variables, for a study purpose which was not the measurement of aggressive behaviour. There were also randomised controlled trials (k = 10), studies using a before-and-after design (k = 2) and cross-sectional analyses of cohort data (k = 2). Lastly, there were single studies that used case control, before-and-after case studies and case reports.

The included articles were predominantly from Western cultures: USA (*k* = 30), Spain (*k* = 9), Germany (*k* = 8), Italy (*k* = 5), UK (*k* = 5), The Netherlands (*k* = 4), Sweden (*k* = 2), Switzerland (*k* = 2), Canada (*k* = 1), Czechia (*k* = 1) and Norway (*k* = 1). One study did not report the country. The remaining studies were conducted in Israel (*k* = 3), India (*k* = 2), China (*k* = 1), Japan (*k* = 1) and Singapore (*k* = 1).

The total sample consisted of 7,552 participants. This excludes one study that did not report the number of participants. Three studies did not report participant gender. There were a considerable number of studies that included only male (*k* = 31) participants, and a similar number that included participants of both genders (*k* = 32), with a smaller number with only female participants (*k* = 11).

5.5.1.3 Measures of Stress Response

The majority of studies used only one measure of stress response systems activity (*k* = 47) while others used two (*k* = 12), three (*k* = 6), four (*k* = 4), five (*k* = 3) or six (*k* = 5) measures. Assessment of the Hypothalamic-Pituitary-Adrenal (HPA) axis markers was the most popular method among the studies (*k* = 42), indicating interest in the effect of a full stress response. However, there also were studies that inspected only markers of the Sympathomedullary Pathway (SAM system) (*k* = 19), which is also known as the fast stress response. As noted in Chapter 3, this is not necessarily an indicator of the full stress response, as the SAM system reflects the initial reaction to a threat to homeostasis and consequently in some cases reflects only arousal. Nevertheless, there were studies that looked at the markers of both the SAM system and HPA axis response (*k* = 16) showing the relationship of each part of the stress response on aggressive behaviour[6].

Given that cross-sectional and experimental analyses were used in the majority of the studies, it is unsurprising that they employed a paradigm whereby participants experienced

[6] Specific markers of the stress response systems are detailed in Appendix 1.

acute stress (k = 54). This approach allows an examination of how experiencing artificially induced stress at a given point in time is related to aggressive behaviour. Meanwhile, trait stress response (k = 13) and stress system activity at a given point in time (k = 20) were assessed by less than one third of the studies. Both of these measures indicate the typical activity of an individual's stress system; however, while trait response was calculated using more than one assessment of HPA or SAM markers, the activity at a given point in time was calculated using singular assessment.

5.5.1.4 Measures of Aggression

While the study by Fishbein and colleagues (1992) was the only one to use three measures of aggressive behaviour, there were several studies (k = 7) that used two measures. In most cases, this approach allowed for a more precise understanding of aggression as the use of different measures allows for verification as well as to investigation of distinct dimensions of aggression. However, most studies used only one measure of aggressive behaviour (k = 69), which is the standard approach in the field.

There was large variability in instruments used to assess aggressive behaviour. Although over one third employed an interactive paradigm to estimate aggression of participants (k = 20), the paradigms differed between the studies[7]. Nevertheless, use of experimental paradigms where people show behaviour operationalised as indicative of aggression have higher validity than self-report measures. Not only can the latter be influenced by impression management, but they also do not require any behaviour from participants.

Other measures of aggression that were used in more than one study were questionnaires (k = 49), file trawl for aggressive incidents (k = 5), aggressive behaviour during an interaction (k = 5) and judgment in military or police training (k = 2). There were also five measures of

[7] A description of each paradigm used is presented in Appendix 1.

aggression used in only one study (e.g., intent to commit an assault described in a vignette; Armstrong & Boutwell, 2012).

5.5.2 Stress-Aggression Relationship Reported in the Included Studies

Inspection of the included papers showed that studies employed different measures to assess both activity of the stress response systems and aggression. Consequently, in order to systematically present the results of included studies, findings are presented in groups based on the type of stress system activity and type of aggression that was assessed.

Three subgroups were coded based on the nature of stress variables. The first group included studies that assessed acute activation of the stress systems. Their results demonstrated whether increased or blunted responsivity to a particular stressor is positively or negatively related to aggression. The next group represented the studies where the stress systems' activity measures reflected the typical activity of individual's system. Their findings showed whether heightened or lowered activity of stress response systems is related to aggression. Lastly, the studies that assessed activity of the stress systems at one timepoint were also grouped together in order to demonstrate whether increased or decreased activity of a stress system measured at a single time point was associated with aggression.

Similarly, three subgroups for measures of aggression were identified. Anger-based aggression is defined as behaviour captured by questionnaires designed to assess anger, yet including aggressive behaviour. Meanwhile, demonstrated aggression captures behaviour that was exhibited by participants in a laboratory paradigm involving responding to an opponent or a script, in a conflict discussion or interaction with another person. Lastly, past aggression reflects measures that rely on reports about aggressive behaviour in the past, either from participants themselves or via official records.

Moreover, the reported relationships between the HPA axis and aggression were separated from those reporting the relation between the SAM system and aggression. This

distinction is predicated on the full stress response cycle. Upon encountering a stressor, the SAM system is rapidly activated, which in turn facilitates activation of the HPA axis (de Kloet et al., 2005; Everly & Lating, 2019). Table 5.2 shows the number of studies from each of the categories that showed positive, negative or no relationship between markers of stress response systems' activity and types of aggression. Importantly, these categories are not themes, but they reflect methodological differences in the studies included in the review. Also, the numbers in Table 5.2 do not correspond to the studies, but to the times a relationship was mentioned. If a study reported two types of a relationship, it would be marked in two cells. For instance, Berman and colleagues (1993) reported that acute HPA axis activation is associated with increased aggressive behaviour for people with Type A behaviour, but not Type B behaviour. Consequently, this study reports both that acute activation of the HPA increases demonstrated aggression, and at the same time, that acute activation of the HPA axis is not related to demonstrated aggression; this study is therefore marked in two cells.

Within 77 studies, there were 81 reports of a significant relationship between stress response system markers and any form of aggression, and 42 reports of non-significant relationships. From 81 reports of a relationship, 46 were of a positive association and 35 of a negative one. In the following section, papers that were combined in each cell will be discussed in the order of stress response system subgroups (i.e., first acute activation, then trait activity and afterwards routine activation). A short interim summary will be provided for the relationship between each type of stress response system activation and all three forms of aggression.

Table 5.2 Relationships Between Activity of the Stress Response Systems and Aggression

		Anger-Based Aggression		Demonstrated Aggression		Reported Aggression	
		HPA axis	SAM system	HPA axis	SAM system	HPA axis	SAM system
Acute activation of the stress system	Increases	6	2	7	10	4	4
	Decreases	4	1	3	3	5	4
	Is not related	1	2	11	2	7	2
Trait activity of the stress system	Increases	1	1	0	2	1	2
	Decreases	1	1	2	0	1	0
	Is not related	3	0	1	1	3	1
Routine activity of the stress system	Increases	1	0	1	0	4	0
	Decreases	2	1	0	0	4	3
	Is not related	1	0	1	0	4	2

5.5.3 Acute Activation of the Stress Systems

5.5.3.1 Activation of the HPA axis[8] and Anger-based Aggression[9].

Six studies reported that HPA axis activation by an acute stressor is followed by an increase in anger-based aggression (Keltikangas-Järvinen et al., 1996; Romero-Martínez & Moya-Albiol, 2006; 2007; Romero-Martinez et al., 2013a; 2013b; Ruiz-Robledillo et al., 2017). However, studies conducted by Romero-Martinez et al. (2013a; 2013b) specify that the increase in anger is only present among participants who have not perpetrated IPV. Moreover, Ruiz-Robledillo et al. (2017) found this relationship only for the changes in cortisol levels immediately related to an acute stressor. Nevertheless, four studies revealed that there is a negative relationship between the HPA axis response to a stressor and aggression based on anger (Romero-Martinez et al., 2013a; 2013b; Roy, 2004; Ruiz-Robledillo et al., 2017). Romero-Martinez et al. (2013a; 2013b) found this relationship only for participants who have perpetrated IPV. Meanwhile, the results of Ruiz-Robledillo et al. (2017) demonstrated that when cortisol levels after a stressor are assessed for a longer time (80 minutes rather than 20

[8] Activation of the HPA axis is defined as time-limited changes of the HPA axis markers' levels in response to a stressor.
[9] These are the names of subgroups, not themes.

minutes), they negatively correlate with anger. Furthermore, Steptoe et al. (1996) showed that HPA axis activation is not related to anger. The different directions of the relationship reported in these studies highlight the importance of moderator variables (e.g., IPV perpetration). This in turn raises the possibility of an indirect relationship between acute stress and anger-based aggression.

5.5.3.2 Activation of the SAM system[10] and Anger-based Aggression.

Compared to the studies investigating acute activation of the HPA axis, those that focused on the SAM system and anger-based aggression were fewer in number. However, they also did not show uniform results. Ruiz-Robledillo and Moya-Albiol (2015) and Sullivan et al. (1981) showed that a positive increase in anger following an acute stressor was present among carers of normally developing children, and among participants with hypertension. At the same time, Ruiz-Robledillo and Moya-Albiol (2015) reported that SAM system activation did not facilitate anger among carers of children with Autism Spectrum Disorder. The authors suggested that habituation to stressors and different coping strategies might be the reason behind the difference in presentation. Moreover, Schwartz and Portnoy (2017) demonstrated that among adults, anger was not associated with changes in heart rate following a stressful task. Lastly, Steptoe et al. (1996) showed that SAM system activation negatively correlated with anger among both males and females. Taken together, these results support the possibility that the effect of acute stress response on anger-based aggression is not direct, but rather is moderated by other variables.

5.5.3.3 Activation of the HPA Axis and Demonstrated Aggression.

Results of five more studies revealed that the intensity of the stress response was positively associated with aggressive responding in laboratory aggression paradigms (Berman et al., 1993; Böhnke et al., 2010b; Cohen et al., 1996; Gerra et al., 2001; 2007). Nevertheless,

[10] Activation of the SAM system is defined as time-limited changes of the HPA axis markers' levels in response to a stressor.

only the study conducted by Cohen et al. (1996), which included exclusively male participants, found this relationship to be direct. Berman et al. (1993) reported that HPA axis activation facilitates aggression among those with type A behaviour, but not those with type B behaviour. Meanwhile, Böhnke et al. (2010b) demonstrated that when participants' HPA axes were artificially stimulated by exogenous cortisol, its further activation using provocation increased aggressive behaviour, yet only among female participants. However, Gerra et al. (2001; 2007) found that HPA axis activation is positively associated with aggressive responding among male participants without heroin addiction and among those treated with buprenorphine, but not among those treated with methadone.

Adding to the laboratory paradigms assessing aggression, the results of Salvador et al.'s (1999) study showed that the intensity of HPA axis activation during a judo fight positively correlates with attacking, rather than defending, behaviour[11] of a fighter. Likewise, Meyerhoff et al. (2004) reported that, among law enforcement trainees, increased activation of the HPA in response to a simulation of a highly dangerous situation increased the proportion of hostages that were shot by participants. Overall, the studies investigating the effect of acute HPA axis activation on behavioural aggression demonstrated that an increase in the outcome is often moderated by the presence of third variables, such as Type A behaviour.

Three studies reported that low HPA axis activation is associated with aggressive behaviour in laboratory paradigms (Gowin et al., 2013; Margittai et al., 2018; Mehta et al., 2017). However, Margittai and colleagues (2018) only found the effect among males. Although Mehta et al. (2017) found that aggressive behaviour was associated with lower HPA axis activity in both males and females, the provocations from the opponent only affected cortisol levels among female participants. In other words, the findings are more accurate for the male

[11] The type of behaviour was coded by the professionals as they observed judo matches.

participants. Lastly, Gowin and colleagues (2013) reported that the effect of acute HPA axis activation on aggression only *approached* significance. Given these limitations, there appears to be no firm ground to conclude that low HPA axis response to an acute stressor evokes aggressive behaviour.

Furthermore, the majority of studies that measured acute HPA axis activation showed that it is not related to aggression. Nine studies did not find the effect of the HPA axis activation on aggressive behaviour in laboratory paradigms to be significant (Berman et al., 1993; Bjork et al., 2000; Böhnke et al., 2010a; 2010b; Cote et al., 2013; Gowin et al., 2013; Prasad et al., 2017; Von Dawans et al., 2012; 2019; Zhan et al., 2017). Specifically, Berman et al. (1993) reported this relationship was not significant among people with Type B behaviour. Böhnke and colleagues (2010a) also did not find a significant association between change in cortisol levels and behavioural aggression. Similarly, Gowin et al. (2013), Von Dawans et al. (2012) and Zhan et al. (2017) did not find the amount of aggressive behaviour to differ between those whose HPA axis was stimulated and those whose HPA axis was not. Bjork et al. (2000) reported that cortisol levels did not differ between participants whose plasma tryptophan levels were artificially increased or decreased, thereby demonstrating that despite similar cortisol levels, participants differed in aggressive behaviour. In other words, aggression varied independently from HPA axis activation. Moreover, Flanagan et al. (2018) demonstrated that there was no increase in hostile behaviour during a conflict resolution task following stimulation of the HPA axis. Böhnke and colleagues (2010b) showed similar results, but only for males.

Overall, studies investigating the effect of acute HPA axis activation on aggressive behaviour suggest that the nature of this relationship is indirect. The presence of a positive effect was, in most cases, related to the presence or absence of a third variable, indicating an important role played by moderators. Meanwhile, the absence of effect reported by the majority

of the studies hints at the possibility that acute stress is itself a third variable for a relationship between another variable and aggression.

5.5.3.4 Activation of the SAM System and Demonstrated Aggression.

Eight studies demonstrated that the SAM system response to a stressor facilitated aggressive responding in laboratory aggression paradigms (Buades-Rotger et al., 2017; Gerra et al., 2001; 2007; Margittai et al., 2018; Verona & Curtin, 2006; Verona & Kilmer, 2007; Verona et al., 2006; Zhan et al., 2017). Importantly, Verona and Kilmer (2007) and Verona and Curtin (2006) found that the positive relationship between SAM system activation and aggression was present only among males. However, while Buades-Rotger et al. (2017) reported the same effect in a purely female sample, Zhan et al. (2017) showed similar patterns across both sexes.

In addition to the increase in aggressive behaviour in laboratory paradigms following SAM activation among males, two studies demonstrated increase of aggressive behaviour in a navy battle simulator and a police exercise (Meyerhoff et al., 2004; Var Orden et al., 1996). A possible explanation for the controversy between results arises from Verona et al. (2006), where the effect of stress on aggressive behaviour was predicated on the interactions with gender and with genotype, especially homozygous S.

The studies showing a positive association between acute SAM system activation and aggression follow the studies that utilised HPA axis markers in suggesting an indirect relationship between stress and aggressive behaviour. As noted before, one of the possible moderators is gender. Accordingly, Beyer et al. (2017), Verona and Curtin (2006) and Verona and Kilmer (2007) found that SAM system activation was negatively related to aggressive responding among female participants. However, two studies by Von Dawans et al. (2012; 2019) showed that the response of the SAM system to acute stress did not exert a significant effect on aggressive behaviour among either males or females. Instead, the authors note a trend

in an inverted relationship among females, and an association between stress response and pro-social behaviour among both sexes. It appears that acute activation of the SAM system is likely to facilitate aggressive behaviour among males more than among females, but only in certain circumstances. In other words, the stress-aggression relationship is unlikely to be direct.

5.5.3.5 Activation of the HPA Axis and Reported Aggression.

Three studies demonstrated a positive association between intensity of the HPA axis response to a stressor and past aggression, especially among people who were exposed to violence in childhood (Fishbein et al., 1992; Scarpa & Ollendick, 2003; Scarpa et al., 2000). Martorell and Bugental (2006) specified this relationship further. They reported that, among mothers who perceived low power over the children whose temperament was deemed difficult, activation of the HPA axis predicted the use of harsh parenting that included aggressive behaviour. The presence of specific conditions for a positive association between acute HPA axis activation and reported aggression supports the proposition that this relationship is indirect.

Furthermore, five studies reported that HPA axis activation in response to a stressor is negatively associated with aggressive behaviour in the past (Gerra et al., 2003; Gowin et al., 2013; Hagan et al., 2014; Madden & Shaffer, 2019; Vaz-Leal et al., 2007). Similarly to the studies reporting positive associations, some of those reporting negative associations also highlighted the presence of third variables. Hagan et al.'s (2014) study demonstrated that low activation of the HPA axis intensified the association between childhood maltreatment and externalising problems in adulthood, yet only when additional factors were controlled for. Similarly, Madden and Shaffer (2019) showed that among people with low HPA axis activation, those who suffered childhood abuse were more likely to be aggressive during conflict with their partners. Meanwhile, Gerra et al. (2003) reported the inverse relationship to be present among MDMA users.

Nevertheless, there were studies that reported a lack of association between acute HPA axis activation and reported aggressive behaviour. Despite showing a trend towards significance, Gowin et al.'s (2003) mediation analysis did not find HPA axis reaction to an acute stressor to be a mediator for the effect of abuse on reported aggression. Furthermore, the studies conducted by Buchman et al. (2014), Feinberg et al. (2011), Lundberg (1983), Madden and Shaffer (2019) and Westrin et al. (1998) demonstrated that HPA axis activation in response to a stressor is not associated with past aggression. Similarly, Olff et al. (1995) reported no association between defensive hostility and HPA axis activity. However, given that their study was the only one using such a measure of hostility,[12] comparability of the results is difficult to establish.

The comparable number of reports for each direction of the relationship, and the non-significant findings in the community sample studies (Buchman et al., 2014; Feinberg et al., 2011; Madden & Shaffer, 2019), decrease the likelihood that HPA axis reactivity has a direct association with reported aggressive behaviour. Instead, the relationship appears to be indirect and contingent on third variables, such as exposure to violence. Moreover, as Madden and Shaffer (2019) demonstrated, acute activation of the HPA axis has a potential to play the role of a mediator variable. However, as Gowin et al.'s (2003) results showed, the relationship for which acute stress response can be the mediator is not yet identified.

5.5.3.6 Activation of the SAM system and Reported Aggression.

There were two high quality studies that reported a positive association between activation of the SAM system and past aggression (Murray-Close et al., 2017; Sherwood et al., 2004). Additionally, Lee et al. (1998) reported that two patients, whose SAM system activation during stressors decreased after amygdalotomy, engaged in fewer aggressive incidents than

[12] Defensive Hostility Scale using DMI and LSI subscales, both of which assess defence mechanisms.

before the operation. Similarly, Sullivan et al. (1981) demonstrated that activation of the SAM system was associated with increased scores on a questionnaire measuring direct and indirect hostility. However, the study by Lee et al. (1998) used a case study design and Sullivan et al. (1981) utilised a composite measure of reported aggression. Consequently, the evidence for the positive relationship between SAM system reactivity and reported aggressive behaviour can only be considered preliminary.

A similar problem is present for the studies reporting the association between acute activation of the SAM system and past aggression. Two studies demonstrated that patients with a less responsive SAM system have more aggressive histories than those whose SAM system activation by stressors is higher (Woodman & Hinton, 1978; Woodman et al., 1978). However, the studies employed case files to measure aggressive behaviour, which only reflect known instances. Likewise, although the results of Olff et al. (1995) also show a negative relationship between hostility and changes in SAM system activity levels during a stressor, due to the unique instrument used to measure hostility, these results are doubtful. Meanwhile, the study by Beyer et al. (2017) employed a more robust method of estimating reported aggression (i.e., BPAQ) and its results support an inverse relationship. Nevertheless, a small sample size (n = 27) indicates that the inverse relationship is suggested rather than confirmed.

Moreover, Lundberg et al. (1983) did not find a relationship between the SAM system response to a stressor and past aggression among female participants. Meanwhile, Rostrup et al. (1992) did not find a significant correlation between these variables among males. Consequently, there needs to be a more robust investigation of the association between SAM system responsivity and reported aggression. Currently available studies do not provide robust evidence for it.

5.5.3.7 Summary of the Relationship Between Acute Activation of Stress Responses Systems and Aggression.

Although the results of the studies investigating the relationship between aggression and acute activation of the SAM system and HPA axis are conflicting, there was a common pattern among the reviewed studies. Specifically, the effect that the magnitude of the stress systems' response has on aggressive behaviour appears predicated on other variables from different domains (e.g., gender or exposure to violence). This, in turn, suggests that the stress-aggression relationship is indirect and requires a moderator. Further, it raises the possibility that reactivity to acute stress might act as an amplifier or inhibitor for the influence that other characteristics have on aggression. However, it is unclear whether the direction (increasing or decreasing) that the effect of stress reactivity has is uniform, or if it depends on a primary variable. For example, Hagan et al. (2014) highlighted that low stress responsivity amplifies the association between childhood maltreatment and aggressive behaviour. Meanwhile, Verona et al. (2006) demonstrated that activation of the SAM system only facilities aggression among males with a homozygous S genotype.

5.5.4 Trait Activity of the Stress Systems

5.5.4.1 Trait Activity of the HPA Axis[13] and Anger-based Aggression.

Rausch et al. (2015) demonstrated that HPA axis activity after waking up correlated with trait anger among female patients diagnosed with Borderline Personality Disorder (BPD). Meanwhile Brown et al. (2008) showed that among males, the activity of the HPA axis is negatively associated with anger-based aggression. However, as the study by Brown et al. (2008) was a pilot with only five participants, its results are only indicative of a trend. Moreover, Rausch et al.'s (2015) results indicate that trait activity of the HPA axis is not related to anger among healthy females, healthy males or those diagnosed with BPD. Likewise, a study by Van Eck and Nicolson (1994) supported the absence of a significant association for healthy

[13] Trait activity of the HPA axis is defined as the activity level of the HPA axis' markers, measured over a time period without an acute stressor.

males, while Ryff et al.'s study (2006) supported the absence of a significant association among females. Thus, it appears that the relationship between trait activity of the HPA axis and anger is only present among females diagnosed with BPD. This in turn indicates that the noted relationship is dependent on specific circumstances.

5.5.4.2 Trait Activity of the SAM System[14] and Anger-based Aggression.

The studies assessing trait activity of the SAM system also did not agree regarding its effect on anger-based aggression. Ryff et al. (2006) showed that overnight SAM system activity positively correlated with trait anger among females. However, Schwartz and Portnoy (2017) reported that overnight SAM system activity was negatively related to anger among both males and females. It is possible that the difference in the results can be explained by the higher average age of the participants in Ryff et al.'s (2006) study (74 years) compared to that of Schwartz and Portnoy's (2017) participants (55.26 years). Nevertheless, as Schwartz and Portnoy (2017) found a negative association between age and anger and their study had larger sample, their results appear more reliable. In other words, trait activity of the SAM system is likely to be negatively related to anger-based aggression.

5.5.4.3 Trait Activity of the HPA Axis and Demonstrated Aggression.

While no studies showed a positive association between trait activity of the HPA axis and behavioural aggression, two studies reported this relationship to be negative (Böhnke et al., 2010a; 2010b). However, in both studies there were specific circumstances on which the effect was predicated. Böhnke et al., (2010a) demonstrated that low trait HPA axis activity facilitated aggressive behaviour only for those who were provoked in the laboratory paradigm[15]. Meanwhile, Böhnke et al. (2010b) showed that a negative relationship was present only for females. However, Buades-Rotger et al. (2016) did not find such an association to be

[14] Trait activity of the SAM system is defined as the activity of the SAM system during a period of time without an acute stressor.
[15] For more details on the paradigm, please see Appendix entry for TAP

significant among female participants. Overall, these studies highlight the relevance of extraneous variables to the relationship between trait activation of the HPA axis and behavioural aggression.

5.5.4.4 Trait Activity of the HPA Axis and Reported Aggression.

In addition to reporting a positive association for anger, Rausch et al. (2015) demonstrated that trait HPA axis activity correlated with trait aggressiveness among female patients diagnosed with BPD. However, Das et al. (2018) showed a negative relationship between trait HPA axis activity and past aggression among healthy participants and those diagnosed with schizophrenia. It is possible that the different direction of the relationship among the patients is related to their distinct diagnoses. Nevertheless, Rausch et al.'s (2015) analysis yielded a non-significant relationship between trait HPA axis activity and reported aggression for healthy participants. Moreover, Cima et al. (2008) reported that HPA axis daily activity is not related to aggression amongst either male university students or inmates. Taken together, these studies indicate the possibility of an effect of trait HPA axis activity on reported aggression in specific cases, such as the diagnosis of a psychological disorder. However, the direction of this effect appears to be dependent on the type of the disorder.

5.5.4.5 Trait Activity of the SAM system and Reported Aggression.

Two studies found that trait activity of the SAM system is positively associated with reported aggression. Using female participants, Sherwood et al. (2004) demonstrated a positive correlation between hostility and a 24-hour sample of norepinephrine (NE), but not epinephrine (EPI). Likewise, Newman et al. (2011) reported that following a course of beta blockers, two male patients with histories of aggressive behaviour were reported to have an absence of aggressive outbursts and to have a calmer presentation. However, as Newman et al. (2011) utilised a case study design, their conclusions can only be deemed as indicative of a possible trend rather than robust evidence. Consequently, the relationship between trait SAM system

activity and reported aggression appears to be dependent on the specific SAM system marker measured (Sherwood et al., 2004).

5.5.4.6 Summary of the Relationship Between Trait Activity of Stress Responses Systems and Aggression.

Similarly to the studies investigating the effect of the acute stress systems response on aggression, those that focused on trait activity of both the SAM system and HPA axis highlighted the relevance of extraneous variables. This pattern was especially prominent for trait HPA axis activity, the effect of which on different forms of aggression appeared contingent on the participants' sex, psychiatric diagnoses and specific conditions in the aggression assessment paradigms. Meanwhile, trait activity of the SAM system appeared to be negatively related to anger, but positively related to reported aggressive behaviour, though only when it was measured via NE levels. Overall, these studies suggest that, similar to acute activation of the stress systems, their trait levels' effects on aggression are indirect.

5.5.5 Routine Activation of the Stress Systems

5.5.5.1 Routine Activation of the HPA Axis[16] and Anger-based Aggression.

Although Ritsner et al. (2004) reported a positive association between routine HPA axis activity and anger-based aggression, Ritsner et al. (2003) reported this relationship to be negative. Moreover, Romero-Martínez et al. (2016) specified that activity of the HPA axis has a negative association with anger expression only among male IPV perpetrators. However, Pesce et al. (2015) found no correlation between HPA axis activity before a boxing match and anger among males. Both studies conducted by Ritsner and colleagues (2003; 2004) also used predominantly male samples. However, as they differed in the assessed stress system markers, it appears that the relationship between routine activity of the HPA axis and aggression is affected by the measurement methods.

[16] Routine activation of the HPA axis is defined as the measurement of HPA axis activity taken at one timepoint.

5.5.5.2 Routine Activation of the SAM system[17] and Anger-based Aggression.

There was only one study investigating the relationship between routine activation of the SAM system and anger-based aggression. Ryff et al. (2006) reported this association to be positive, but only for EPI rather than NE. As this study's participants were females aged over 75, these results cannot be held as representative of the general population.

5.5.5.3 Routine Activation of the HPA Axis and Observed Aggression.

Although there were only two studies investigating the association between routine activation of the HPA axis and aggressive behaviour (Schneiderman et al., 2014; Salvador et al., 1999), both of them utilised observation to assess aggression. Specifically, Schneiderman et al. (2014) demonstrated that routine HPA axis activity in male and female participants is predictive of hostile behaviour coded as such from a conflict interaction paradigm. Meanwhile, Salvador et al. (1999) reported that HPA axis activity before a judo fight does not correlate with aggressive behaviour during the fight. In this case, aggressive behaviour was coded by judo specialist observers. It is likely that the differences in the situations where participants were showing aggression (conflict discussion with one's partner vs. martial arts competition) can explain the differences in the results. Adoption of such an explanation would in turn suggest the importance of extraneous variables for the relationship between routine HPA axis activation and aggressive behaviour.

5.5.5.4 Routine Activation of the HPA Axis and Reported Aggression.

Four studies reported a positive relationship between the HPA axis activity marker and reported aggression among both males and females (Ai et al., 2014; Bergomi et al., 2017; Feinberg et al., 2011; Westrin et al., 1998). On the contrary, four other studies demonstrated this relationship to be negative (Brewer-Smyth et al., 2004; Ritsner et al., 2003; Solanki et al., 2007; Walther et al., 2017). Importantly, while the study of Brewer-Smyth et al. (2004)

[17] Routine activation of the SAM system is defined as the activity of the SAM system at a given timepoint.

included only female participants, Ritsner et al. (2003) used a predominantly male sample. Meanwhile, Walther et al. (2017) found that HPA axis activity markers based on hair samples moderated the negative effect of age on anger among self-reporting healthy males. Moreover, Solanki et al.'s (2007) participants were patients diagnosed with first-episode schizophrenia. The discrepancy between the results is further complicated by four studies reporting no significant relationship between routine activation of the stress system and aggression. Three studies (Flegr et al., 2012; Melhem et al., 2017; Peters et al., 2003) showed that routine HPA axis activation was not associated with past aggression among either males or females. Additionally, Walther et al. (2017) reported that salivary markers of routine HPA axis activation were not a significant moderator of the effect of age on physical and verbal aggression among fathers and males with vital exhaustion[18]. Taken together, the contradictions between the results suggest that the relationship between routine HPA axis activation and reported aggression is predicated on extraneous variables. Although these variables have not been identified, participant sex is unlikely to be among them.

5.5.5.5 Routine Activation of the SAM System and Reported Aggression.

No studies reported a positive association between routine SAM system activity and aggression, but three studies found a negative association. Results of Scarpa and Ollendick (2003) and Scarpa et al. (2003) demonstrated that low SAM system activity was associated with past aggression among participants who experienced victimisation. Likewise, Armstrong and Boutwell (2012) showed that those with low routine SAM system activation were more likely to report willingness to engage in assault than were individuals with higher SAM system activation. However, this relationship lost significance when perceived likelihood of conviction was added to the analysis. This indicates that perceived consequences of the behaviour might act as a mediator between SAM system activity and aggression. In addition to these variables,

[18] Defined as a state encompassing tiredness, irritability, loss of motivation and lack of energy.

Armstrong and Boutwell (2012) demonstrated that deeming guilt resulting from aggressive behaviour a problem also mediated the relationship between routine SAM system activity and aggression.

Although two studies did not find a relationship between SAM system activation and aggression (Bergomi et al., 2017; Peters et al., 2003), their results also highlight the possibility of an indirect relationship. Peters et al. (2003) found that aggression acts as a moderator to the stress response in demanding situations. Specifically, they showed that participants with high trait aggressiveness demonstrated a pronounced reaction to stressors even when situational demands were low. Meanwhile, Bergomi et al. (2017) demonstrated that although routine activity of the SAM system was not related to reported aggression among males, the measure of aggression they used was specific to driving. Given that their sample consisted of professional bus drivers, it is unlikely that their results are generalisable to the general population. Consequently, despite the differences in these results, all the studies investigating the association of routine SAM system activity with reported aggression highlight the importance of third variables.

5.5.5.6 Summary of the Relationship Between Routine Activation of Stress Responses Systems and Aggression.

Following the studies measuring acute and trait activation of the stress systems, those investigating routine activity suggest an indirect relationship between stress markers and aggression. This appears to be especially likely for reported and observed aggression. Importantly, the study of Armstrong and Boutwell (2012) demonstrated that the third variable can be from the cognitive domain—specifically, expectations about the consequences of behaviour. In other words, the effect of increased or decreased activity of a stress system at a given time point is likely to be influenced by additional variables, which can include expectations about consequences, past victimisation or the type of the interaction itself. Thus, in order to uncover such influential third variable, a thematic analysis was conducted.

5.6 Thematic Analysis

Lack of agreement between study results highlighted the need to identify common features explaining it. For these purposes, a deductive sematic realist thematic analysis was performed via inspection of the included studies for patterns and following the guidelines outlined by Braun and Clarke (2006). It included six phases: gaining knowledge about the data; creating initial codes; identifying common themes; reviewing them; defining and naming them and reporting the results. The theoretical or deductive framework was chosen in order to keep the focus on the second aim of the systematic literature review, namely, to identify potentially third variables that affect the stress-aggression relationship. Meanwhile, the sematic realist approach was chosen as the unit of analysis were published peer reviewed papers, where the meaning is explicit.

Consequently, papers included in the review were read in full and relevant information from them was coded. Consistent with the stated aim the codes primarily reflected variables included in the studies that were extraneous to the stress marker – aggression relationship and how they affected it. They were mostly found in methodology, results and discussion sections. For example, methods section of Schneiderman et al (2014) stated "To determine hormones concentrations, serum samples were defrosted to room temperature before analysis. T, PRL, and CT determinations were done by microparticle enzyme immunoassay (MEIA) technology…" (p. 341) and this was coded as "Testosterone". Meanwhile, discussion section of Verona and Kilmer (2007) reported "The current study established the dimorphic effects of acute stress exposure on subsequent aggressive behavior in men and women, with women displaying less aggression and men displaying increases in aggression following high versus low stress." (p.416) and was coded "Sex Differences". Likewise, Gerra and colleagues (2007) wrote in the results section "Areas under the curve for NE and EPI during aggressive sessions correlated positively with PSAP aggressive responding measures in heroin-dependent patients,

independently of METH and BUP treatment, and in CONT subjects…" (p.209). This was coded as "Effect of Substance Use".

The codes that consistently emerged were used to establish the following themes: 1) the impact of testosterone on the relationship between the stress systems' activity and aggression is undetermined; 2) the presence of sex differences in the relationship between stress response and aggression depends on the stress systems and type of aggression; 3) specific disorders do not influence the relationship between stress and aggression; 4) experience of victimisation does not have a clear influence on the relationship between stress response systems and aggression; 5) the relationship between the stress systems' activity and aggression differs between people with high stress exposure; and 6) history of aggression affects the relationship between stress response markers and anger-based aggression. These themes are described in the following sections.

5.6.1 Theme One: The Impact of Testosterone on the Relationship Between the Activity of Stress Response Systems and Aggression is Undetermined

Fifteen studies (19%) highlighted that testosterone might impact the relationship between activity of stress systems and aggression. Studies showing that the testosterone-to-cortisol ratio was associated with anger-based aggression suggested a joint influence on behaviour (Romero-Martínez & Moya-Albiol, 2016; Romero-Martínez et al., 2013b; 2016). Those with high testosterone were more aggressive than those with low, but only when there was no stressor (Prasad et al., 2017). However, despite individual associations of these hormones with aggression, their interaction was not consistently confirmed (Buades-Rotger et al., 2016; Cote et al., 2013; Mehta et al., 2017; Romero-Martínez et al., 2013a; Salvador et al., 1999). Moreover, there was disagreement about whether the association with aggression is similar (Berman et al., 1993; Cohen et al., 1996; Lundberg et al., 1983; Schneiderman et al., 2014) or different (Brown et al., 2008; Flegr et al., 2012).

5.6.2 Theme Two: Sex Differences in the Relationship Between Stress Response and Aggression Depends on the Stress Systems and Type of Aggression

Eighteen percent of the included studies investigated whether the association between activity of the stress response systems and aggression is different between sexes, as they accounted for this variable. Literature consistently showed an effect of the SAM system, specifically that its acute activation is likely to promote aggressive conduct among males, but diminish aggressive conduct among females (Verona & Curtin, 2006; Verona & Kilmer, 2007; Verona et al., 2006). However, there was a discrepancy regarding the influence of HPA axis activation, which was most likely related to the method used to stimulate it (Böhnke et al., 2010b; Prasad et al., 2017). Although one study did not find a sex difference, it did not achieve a comparable response of the HPA axis to the competitive task among men and women, suggesting that stress induction was not successful across participants (Mehta et al., 2017).

However, eight studies demonstrated that the association between past aggression and stress response markers was similar among men and women, as inclusion of sex in the analysis did not impact the results (Armstrong & Boutwell, 2012; Feinberg et al., 2011; Hagan et al., 2014; Murray-Close et al., 2017; Rausch et al., 2015; Ruiz-Robledillo & Moya-Albiol, 2015; Schwartz & Portnoy; 2017; Steptoe et al., 1996). However, there was no consistency in the association between these two variables for men and women, as the studies disagreed about its direction and existence. This inconsistency undermines any conclusions about the presence of sex differences.

5.6.3 Theme Three: Specific Disorders do not Influence the Relationship Between Stress and Aggression

Fourteen percent of the reviewed studies explored whether the association between activity of the stress systems and aggression was influenced by specific disorders. These disorders are best reflected by two subthemes: psychotic symptoms and substance use.

Subtheme 1: Psychotic Symptoms. Seven studies highlighted that the presence or severity of psychotic symptoms was unlikely to affect the relationship between stress response systems and aggressive behaviour (Das et al., 2018; Newman & McDermott, 2011; Solanki et al., 2017; Ritsner et al., 2003; 2004; Woodman & Hinton, 1978; Woodman et al., 1978). However, Ritsner and colleagues (2004) suggested that psychotic symptoms might exert an indirect influence, as they create considerable and potential chronic stress for the patients. Nevertheless, this presumption was challenged by Solanki and colleagues' (2017) findings that the duration of untreated psychosis was not associated with stress, suggesting that symptoms of psychosis do not necessarily facilitate stress.

Subtheme 2: Substance Use. Four studies noted that the relationship between stress response and aggressive behaviour was similar among drug users and non-users (Fishbein et al, 1992; Gerra et al., 2001; 2003; 2007). Specifically, Gerra and colleagues (2001; 2007) demonstrated that SAM system activation was associated with aggressive responses to provocation among those who were heroin users and among "healthy" participants. This relationship corresponds to the one found among patients of an addiction research centre (Fishbein et al., 1992), suggesting that hyperactivity of stress systems facilitates aggression among those who use illicit substances and those who do not. Although an inverse association was reported among MDMA users (Gerra et al., 2003), the lack of results from those who did not use MDMA in this study precludes conclusions about the role of MDMA use in the relationship between stress and aggression.

5.6.4 Theme Four: Experience of Victimisation Does Not Have a Clear Influence on the Relationship Between Stress Response Systems and Aggression

Nine percent of included papers compared the association between activity of stress response systems and aggression among those who had experienced victimisation and those who had not. Of these, four studies showed that these groups differed. Low trait SAM system

activity and stronger HPA axis activation in response to an acute stressor facilitated aggressive behaviour among those who had experienced victimisation, regardless of the intensity, but not among those who had not (Scarpa & Ollendick, 2002; Scarpa et al., 2000). Moreover, experiencing abuse was predictive of aggressive behaviour only among those with blunted HPA axis response (Hagan et al., 2014; Madden & Shaffer, 2019). Nevertheless, the interaction between victimisation and stress system response was not consistently confirmed (Brewer-Smyth et al., 2004; Cima et al., 2008; Gowin et al., 2017).

5.6.5 Theme Five: The Relationship Between Stress Response Markers and Aggression Differs Between Individuals with High Stress Exposure

Six percent of the included studies looked at the association between activity of stress response systems and aggression among people who are likely to be frequently exposed to stressful situations. Despite sharing frequent exposure to stress, this relationship varied between as well as within occupations such as firefighters, military personnel, and martial arts practitioners (Meyerhoff et al., 2004; Pesce et al., 2015; Roy et al., 2004; Salvador et al., 1999; Van Orden et al., 1996). For example, in judo fighters, HPA axis activation during the fight was positively associated with attacking behaviour (Salvador et al., 1999); however, in boxers it was not associated with anger-based aggression (Pesce et al., 2015).

5.6.6 Theme Six: History of Aggression Affects the Relationship Between Stress Response Markers and Anger-based Aggression

Three per cent of papers investigated the impact of history of aggressive behaviour on the relationship between activity of the stress response systems and aggression (Romero-Martínez et al., 2013a; 2013b). Specifically, results indicate that acute HPA axis response is unlikely to facilitate anger-based aggression among those who have a history of IPV perpetration. However, it was also shown that there is no association between trait HPA axis activity and aggression among prisoners or university students (Cima et al., 2008).

5.7 Discussion

The systematic review has demonstrated that the relationship between physiological markers of stress response systems and aggression is not straightforward. This association is likely present, as it was reported as such in the majority of studies reviewed (n = 81). However, there were also several reports (n = 42) indicating a lack of relationship between these two variables. Moreover, the studies reported both positive (n = 46) and negative (n = 35) associations between stress systems' markers and aggression. Consequently, as predicted in Chapter 4, both hypo- and hyperactivity of the stress systems are associated with aggressive behaviour.

Increased activity of the stress response systems was most consistently related to aggressive behaviour among the studies assessing the SAM system responses to acute stressors, especially among men (Verona & Curtin, 2006; Verona & Kilmer, 2007). This finding supports the definition of reactive aggression as a response to a stress-evoking environment (Fabian, 2020); it also supports the inclusion of arousal as a contributor to aggression, as argued by the GAM (Anderson & Bushman, 2002). This is also consistent with studies demonstrating an association between heightened threat sensitivity and reactive aggression (e.g., Farah et al., 2018). However, as Theme Two (sex differences in the relationship between stress response and aggression depend on the stress systems and type of aggression) has indicated, although men and women showed different aggressive behaviour following acute stressors, the aggressive response pattern was only consistent when the SAM system was measured (Verona & Curtin, 2006; Verona & Kilmer, 2007), but not the HPA axis (Böhnke et al., 2010; Mehta et al., 2017; Prasad et al., 2017). When the activity of the stress system was used as the predictor for past aggressive conduct, there were no sex differences (Feinberg et al., 2011; Schwartz & Portnoy, 2017). However, the studies could not confirm whether hypo- or hyperactivity of the stress systems was indicative of aggression.

Meanwhile, negative associations were present between trait stress system activity and aggression (Böhnke et al., 2010a; 2010b), but it was present only in specific populations, especially those who exhibited aggressive behaviour in the past or who do so currently (Brewer-Smyth et al., 2004; Ritsner et al., 2003; Solanki et al., 2007). Although these results provide support for characterising proactive aggression as resulting from decreased threat sensitivity (Blair, 2018), studies involving participants with psychopathy (Cima et al., 2008; Gowin et al., 2017) did not confirm this. Furthermore, as Theme Six (history of aggression affects the relationship between stress response markers and anger-based aggression) indicted, past history of aggression only affects the relationship between stress responsivity and anger-based aggression. This highlights that while blunted stress responsivity may contribute to aggressive behaviour, it is unlikely to be the root cause.

The probability of the indirect relationship between stress systems activity and aggression increased due to the disagreement between studies assessing similar markers of stress and similar types of aggressive behaviour. The relationship between the SAM system response to a stressor has been shown in this review to be positive, negative and absent (Ruiz-Robledillo & Moya-Albiol, 2015; Sullivan et al., 1981; Schwartz & Portnoy, 2017). Similarly, investigations of the impact of the intensity of HPA axis response on aggressive behaviour showed that it can be direct (Gerra et al., 2001; 2007), inverted (Margittai et al., 2018; Mehta et al., 2017) or non-existent (Flanagan et al., 2018; Von Dawans et al., 2012), even when similar paradigms were used. Together, these discrepancies highlight that stress response is unlikely to have a direct effect on aggression, suggesting a need for consideration of what other factors contribute.

However, the reported influences of third variables identified by the thematic analysis were also inconsistent. Neither the level of testosterone (Theme One), sex (Theme Two), victimisation (Theme Four) or exposure to stressful environments (Theme Five) showed a clear

impact on the relationship between stress response markers and aggression. Theme One (impact of testosterone on the relationship between the activity of stress response systems and aggression is undetermined) demonstrated that cortisol and testosterone interact (Prasad et al., 2017) and have only an independent (Salvador et al., 1999) effect on aggression. Theme Five (the relationship between stress response markers and aggression differs between individuals with high stress exposure) reflected that past experience of abuse or victimisation did not exert a stable impact on the association between activity of stress response systems and aggression (Madden & Shaffer, 2019; Gowin et al., 2017). Overall, the inconsistent effect of the described variables suggests the activity of the stress response systems to be a mediator or moderator.

Another possible explanation for variability in the results is that the extraneous variable(s) have not yet been accurately identified. The most consistent theme (Theme Three: specific disorders do not influence the relationship between stress and aggression) identified that specific disorders, such as psychotic symptoms and substance use, do not influence the relationship between stress and aggression. (Theme three: Specific disorders do not influence the relationship between stress and aggression.). Meanwhile, as shown in Chapter 2, socio-cognitive (Anderson & Bushman, 2002; Huesmann, 2016) and neurocognitive models (Blair, 2016) of aggressive behaviour suggest that cognitions or decision-making capacities can be variables on which this association is dependent. Interestingly, two studies that took into account participants' cognitive processes showed that the stress response does not have a direct relationship with aggression. Instead, it either acts as a mediator (Martorell & Bugental, 2006), or its effect disappears once an individual's perception of the situation is accounted for (Armstrong & Boutwell, 2012).

5.8 Limitations

The current review is not without its limitations. One of the main limitations is the variability in the markers of stress response systems (n = 20), as it diminished the comparability

of the results. However, as inclusion of distinct measures of both the HPA axis and SAM system afforded an in-depth profile of stress-related activity, it was thought warranted. The variation in the measures of aggressive behaviour (n = 32) was another limitation that unavoidably decreases the precision of this variable. Nevertheless, in order to account for the multi-faceted nature of aggression, it was deemed necessary to include studies that employed questionnaires with only a few items reflecting such behaviour. Finally, a further limitation of this review is its restriction to studies published in English. This led to the exclusion of three papers written in other languages and left only eight studies with non-Western samples, arguably limiting the generalisability of the results.

5.9 Concluding Comments

The results of this systematic review demonstrate that although the relationship between aggression and stress system activity markers appears to be present, it is unlikely to be direct. Rather, the relationship between stress response and aggression might be affected by a third variable. However, there appears to be no consistency in the effects of sex, testosterone, past experiences of victimisation, history of aggression or exposure to stress. Meanwhile, as discussed in Chapter 2, socio-cognitive and neurocognitive models suggest that these variables might be aggression-related cognitions and decision-making processes (Blair, 2016; Huesmann, 2016). Moreover, the lack of agreement between the studies lends support to the suggestion from Chapter 3 that experiencing stress does not immediately lead to aggressive behaviour, but rather serves as the impellance process from the I^3 model, which is associated with aggression only by amplifying an independently-originated proclivity to aggress (Finkel & Hall, 2018). The HPA axis and SAM system could modify aggressive behaviour resulting from different factors, such as the presence of aggression-supportive cognitive structures, habitual use of aggression and poor decision-making capacities. Consequently, Study 1

(presented in the next chapter) investigates the effect of aggression supportive cognitions and acute stress response markers on aggressive behaviour.

CHAPTER SIX: STUDY ONE: INVESTIGATING EFFECTS OF STRESS AND IMPLICIT THEORIES ON AGGRESSIVE BEHAVIOUR

6.1 Structure of the Chapter

This is a small scale study assessing the relationships between cognitions, stress, and aggression. Based on the sociocognitive models, it tests the presence of a positive association between implicit theories, which represent cognitive structures, and aggressive behaviour. Additionally, based on the findings from the systematic literature review, it assesses whether acute stress facilitates aggressive behaviour and whether this effect differs between students and patients of high secure forensic hospital.

6.2 Current Study

The current study used an aggression paradigm with a competitive reaction time task to operationalise aggressive behaviour. It constituted the Taylor Aggression Paradigm (TAP) (Taylor, 1967), which is an established method of measuring aggressive behaviour (Chester & Lasko, 2019). Before engaging in it, participants were interviewed by the researcher to establish their history of aggression and presence of implicit theories. Then, to evoke minor stress participants were asked to complete a STROOP task, where they were presented with words describing colours but written in an ink of different colour and asked to respond only to the latter. Each word was presented for 600ms per word. While previous studies showed STROOP task with 500ms per word to increase skin conductance response (Fechir et al., 2010), the current study slightly increased the duration to take into account the vulnerable nature of forensic participants. Furthermore, as the STROOP task represents a taxation of resources similar to that of other neurocognitive tests, its use meant that participants were exposed to a stressor that they might encounter during their treatment.

6.3 Method

6.3.1 Participants

Student participants were recruited using an online research platform SONA of the University of Central Lancashire. Meanwhile, patient participants were recruited by the researcher. Before approaching a patient, permission to approach was sought from their responsible clinician. Then the ward staff was consulted and together with a nurse the researcher approached a patient to advertise the study and provide information about it. Only those with good command of English language and without tinnitus were invited to participate. The total sample for this pilot (*N*=31), consisted of male students (*N*=20) and patients of high secure hospital[19] (*N*=11) in the United Kingdom. It is important to note that not all participants from forensic hospitals have been charged with or have committed a violent offence. Unfortunately, participant recruitment had to be prematurely terminated due to the COVID-19 pandemic and the close contact nature of the study. The student sample comprised 17 people with ages between 18 and 25 and three people aged 26 to 35. Meanwhile, the patient sample consisted of one person aged 18 to 25, four people aged 26 to 35, five people aged 36 to 45, and one person aged between 46 and 55. The ethical approval that was granted for the study allowed only age as the socio-demographic descriptor.

The ethical approval for the study was acquired from the University of Central Lancashire and the NHS ethics boards[20]. Before approaching patients of high secure hospital their responsible clinicians (RC) were consulted regarding the competency of their clients to make informed decisions. Those patients who were allowed to be approached by their RC, were approached by the researcher and a ward nurse to present participants information sheet

[19] High secure psychiatric hospitals are those that provide multidisciplinary psychological and psychiatric treatment and care to the clients that have been charged with an offence, demonstrate high risk of (re)offending, and exhibit mental health or personality disorders.
[20] IRAS project ID: 263017, REC reference: 19/YH/0227

and answer any questions. Meanwhile, for the students the research was advertised using the SONA student portal. The consent form was signed at the date of the experimental session.

6.3.2 Materials:

The current study utilised a combination of self-report, physiological, and experimental measures.

6.3.2.1 Aggressive behaviour

Life history of aggression (LHA) (Coccaro et al., 1997) was used to estimate past aggressive behaviour. In the current study, the LHA was used as a semi-structured interview conducted by the researcher with the participants. They were asked to indicate how often they have engaged in different forms of aggressive or anti-social behaviour. The researcher clarified what constituted specific behaviours (e.g. tantrums) when needed and helped participants determine whether a specific instance falls into a given category. Frequency of the target behaviour was rated on a scale ranging from 0 (no occurrence) to 5 (more than can be counted). The questionnaire has three subscales: Aggression (e.g. "Physical assault against people") consisting of five items; Antisocial Behaviour (e.g. "Antisocial behaviour not resulting in police involvement") consisting of four items; and Self-directed Aggression (e.g. "Self-Injurious behaviour") consisting of two items. The LHA has a good reliability (Cronbach's α for total score .88; for Aggression .87; for Antisocial Behaviour .74, for Self-Directed Aggression .48) and validity indicators (Coccaro et al., 1997). With the current sample, the Cronbach's α for aggression subscale, was .66 indicating acceptable, but not great internal consistency (Vaske, Beaman, & Sponarski, 2017; Ursachi, Horodnic, & Zait, 2015).

6.3.2.2 Implicit Theories Questionnaire

The presence of six Implicit Theories (ITs), identified by Polaschek, Calvet and Gannon, (2009) was explored using a semi-structured interview. This approach was chosen over a Likert scale questionnaire due to the latent nature of the ITs. Participants might not necessarily be

aware that they hold an IT, and an interview with them allowed the researcher to ask personalised follow up or clarifying questions in order to establish whether a given theory is present or absent. During the interview participants were asked questions related to each of the ITs and encouraged to provide open ended answers. The four ITs were: "Violence is normal", "Beat or be beaten: general", "Beat or be beaten: self-enhancement", "Beat or be beaten: self-preservation", "I am the law", and "I get out of control". Based on the participants' answers, each IT was rated as absent, partially present, or fully present. An example, one of the questions assessing self-enhancement subtype of beat or be beaten was: "Do you think a person can prove himself worthy by being aggressive towards others?" followed by asking participants to explain the reasoning behind their answer. In the current sample, total score on Implicit theories questionnaire showed borderline acceptable reliability index, Cronbach's α = .63. Furthermore, inspection of the results showed that while removal of IT item "violence is normal" would increase the reliability, the removal of the "I am the law" IT item would considerably decrease it. Consequently, it was decided to use questions assessing presence or absence of the IT items individually.

6.3.2.3 Taylor Aggression Paradigm

Aggressive behaviour was assessed using competitive reaction time[21] (CRT) measure of laboratory aggression (Bushman & Baumeister, 1998), which represents the Taylor Aggression Paradigm (TAP) (Taylor, 1967). Previous research has demonstrated that the CRT task can be used as a measure of aggression with good validity (Anderson & Bushman, 1997; Bernstein, Richardson, & Hammock, 1987; Chester & Lasko, 2019; Giancola & Parrott, 2008; Giancola & Zeichner, 1995). Participants were told that they would play 25 trials against a real life player, while in fact the outcome of each session was scripted. Before each trial, a participant

[21] Copyright 2006 by Bushman & Saults. The computer program utilised in this study was shared by Dr Bushman and Dr Saults.

was asked to set intensity using 1 to 10 slider (from 65 to 110dB with 5 dB difference) and duration, using also 1 to 10 slider (0.5 to 5.0 seconds) of an unpleasant noise. Then, the participant pressed the button indicating that he is ready, and a countdown started. At the end of the countdown, the field in the screen centre turned red, and the participant needed to click on it as fast as possible. Participants were told that the player who clicks on the designated field faster than the "opponent" was the winner of the trial and they saw what parameters of unpleasant noise the "opponent" had set for them but does not hear the noise. Meanwhile, the loser both sees the parameters of the unpleasant noise that the winner has set and hears it. To ensure that the participants were familiar with the noise, before the first trials they heard 65-, 85-, and 110-dB noise for 2.5 seconds.

Furthermore, following the provocation vs no-provocation paradigm utilised in Böhnke, Bertsch, Kruk, and Naumann (2010), participants were randomly assigned to one of two conditions. The 25 trials were split into three blocks, each consisting of seven trials. The first trial constituted a separate category. For the provocation condition, the task was scripted so that in the first block the "opponent" randomly assigned noise duration and intensities between 1 and 4 (intensity of 65 to 80 dB and duration of 0.5 to 2 seconds), for the second – between 5 and 7 (intensity of 85 to 95 dB and duration of 2.5 to 3.5 second), and for the last block – between 8 and 10 (intensity of 100 to 110 dB and duration of 4 to 5 seconds, respectively). Meanwhile, in the no-provocation condition the task was scripted so that when participants lost, they only heard noises of duration and intensity between 1 and 5 (intensity of 65 to 85 dB and duration of 0.5 to 2.5 seconds)[22]. For each block, noise duration and intensity for each participant were averaged and summed to represent the aggressive behaviour in a given part of the task. Due to the restrictions placed on the number of statistical analysis by the limited

[22] The first two participants from the student sample were exceptions to this approach as their trials were scripted to range between 1 and 10 for both intensity and duration across all three blocks.

sample size, only the intensity and duration selected for the first trial was used as the outcome variable. Given that it was the first trial against entirely unknown "opponent", it represented aggressive behaviour towards a "stranger".

6.3.2.4 Physiological Measures

Participants' heart rate (HR) (measured in beat per minute (BPM)) and skin conductance level (SCL) (measured in microsiemens (μS) were obtained using Edu Loggers Heart Rate and Pulse Logger sensor and Galvanic Skin Response logger sensor. The data was recorded using Edu Logger Software and stored in individual .csv files. The heart rate sensor consists of a plastic clip with an infrared LED transmitter and matched infrared phototransistor receiver. The clip was attached to participant's little finger, as per recommendations in the manual. The SCL sensor consists of two probes with Velcro finger connectors that were attached to the middle and ring fingers. Both variables were sampled at the rate of 10 per second. As discussed in the Chapters 3 and 5 increased heart rate and skin conductance are established markers of the activity of Sympathomedullary (SAM) system, which is the fast stress response system (Chrousos, 2009; Murray-Close et al., 2017; Schwartz & Portnoy, 2017).

6.3.3 Procedure

First, the Edu-logger hardware was calibrated and the baseline HR and SCL were measured for each participant. Afterwards, participants were interviewed to complete the LHA and ITQ questionnaires. A second baseline measure was taken following the interview and before the STROOP task. Participants were informed that they would engage in the normal STROOP task, with the rules explained to them. However, in reality, participants were completing a STROOP task with increased speed to provoke stress response in the participants (Mejía-Mejía, Torres, & Restrepo, 2018; Prinsloo, Derman, Lambert, & Rauch, 2013). The high speed of the word presentation was specifically designed to elicit stress-like response in the participants (Fechir et al., 2010). The task was created using PsyToolkit platform (Stoet,

2010; 2017). During the STROOP task a single word for a colour was presented on the screen, written in an ink of a different colour. There were four word and ink colours: red, yellow, blue, and green. Participants were instructed to respond to the colour of the ink rather than the word, by pressing a predetermined button. For example, if the word Red was presented in yellow ink, participants needed to press button "Y". The STROOP task consisted of 50 trials, where a fixation point was presented for 200 ms, followed by the 600ms presentation of the colour word allowing participant to press corresponding button, and followed by the 500ms presentation of the feedback (right or wrong). During this task, the third measure of the HR and SCL were taken, and afterwards the fourth measure commenced. Participants were then introduced to the CRT. They were told that they would be playing against a real life opponent. Fifth, sixth, and seventh physiological measurements were taken after first, second, and third block of the CRT, respectively. At the end of the session participants were thanked, debriefed and were told that there was no real person playing against them. During the debrief a special attention was paid to the explanation that deception was required in order to maintain the validity of aggression assessment.

6.3.4 Data Analysis

All data was analysed using R software version 4.0.3 (R Core Team, 2020). Before the main analysis, scores on the IT questionnaire were subject to the reliability analysis, in order to establish whether they should be treated as single total score or as separate. Afterwards manipulation checks were performed to assess whether stress induction was successful. Lastly, the proposed hypotheses were tested using linear regressions with confidence intervals obtained via bootstrapping using 1000 samples.

Due to data corruption only a fraction of HR and SCL recordings from the patient sample were recovered ($N = 7$). Consequently, there were two datasets. A full one ($n = 31$) that included LHA and IT measures and a subset ($n = 17$) that comprised HR and SCL measures and

aggressive responses in the TAP. Moreover, premature end of the data collection that resulted in small sample size restricted both the number and the complexity of the statistical tests that can be performed. The intended sample size was 70 participants with 35 per group and was based on the power analysis for linear regression with medium effect size using G power (Faul, Erdfelder, Buchner, & Lang, 2009). Consequently, the existent dataset was underpowered to the extent that only large effect sizes were detectable.

6.4 Results

Table 6.1 Descriptive statistics

Variable name	Students M (SD)	Patients M (SD)	Total M (SD)
Past Aggression (LHA) [f]	7.9 (4.59)	11.18 (7.37)	9.06 (5.83)
Past Antisocial behaviour (LHA) [f]	3.4 (3.59)	10.64 (4.13)	5.97 (5.12)
Violence is normal (ITQ) [f]	0.05 (0.22)	3.18 (2.64)	1.16 (2.16)
Beat or be beaten (ITQ) [f]	1.25 (0.85)	1.27 (0.65)	1.26 (0.77)
Beat or be beaten self-enhancement (ITQ) [f]	0.25 (0.44)	0.82 (0.87)	0.45 (0.68)
Beat or be beaten self-preservation (ITQ) [f]	0.6 (0.6)	1.36 (0.81)	0.87 (0.76)
I am the law (ITQ) [f]	1.4 (0.5)	1.36 (0.81)	1.39 (0.62)
Aggressive Response (TAP) [s]	0.85 (0.67)	1.27 (0.9)	1 (0.77)
Change in Heart Rate [s]	1.5 (0.69)	1.73 (0.47)	1.58 (0.62)
Change in Skin Conductance Level [s]	5.85 (2.21)	7.82 (2.64)	6.55 (2.51)

f – full sample (n=31), subset of sample (n=17)

6.4.1 Manipulation Check

To assess the effect of the STROOP task on the physiological markers, paired samples t-test was used for the second baseline measurement (T2) and post-STROOP measurement(T4). Due to the heavy skewness of the SCL values and their small amount, log transformation was used to adjust the distribution's form, assessed via histograms. There was no significant difference between average HR before (M = 78.84) and after (M = 81.50) the STROOP task,

t(16) = -1.15, p = 0.27. However, there was a significant difference between the average SCL at T4 (M = 2.99) and T2 (M = 2.60), t(16) = -3.13, p<.01. This suggests that the STROOP task was a mild stressor and was only partially successful as it only elicited the expected increase in the SCL.

6.4.2 Testing Prediction 1.1. Aggression supportive cognitions will be positively associated with aggressive behaviour

To test the relationship between LHA aggression and antisocial subscales and the assessed ITs, a Spearman correlation analysis was performed for the whole sample (N = 31) (Table 6.2). They indicate that the only ITs to have significant covariation with two LHA subscales was "Beat or be beaten: self-enhancement type". Meanwhile, "I am the law" and "I get out of control" had a significant moderate correlation with aggressive subscale of the LHA.

Table 6.2 Correlations between past behaviour and Implicit Theories (n = 31)

	Aggressive behaviour	Past Aggression	Past Antisocial behaviour	Violence is normal	Beat or be beaten	Beat or be beaten self-enhancement	Beat or be beaten self-preservation	I am the law	I get out of control
Aggressive behaviour	1								
Past Aggression	0.24	1							
Past Antisocial behaviour	0.32	0.63***	1						
Violence is normal	0.31	0.17	0.18	1					
Beat or be beaten	-0.03	0.15	0.34	0.09	1				
Beat or be beaten self-enhancement	0.03	0.46**	0.53**	0.28	0.25	1			
Beat or be beaten self-preservation	-0.11	0.16	-0.04	-0.15	0.13	0.11	1		
I am the law	0.32	0.39*	0.24	0.17	0.13	0.34	0.56**	1	
I get out of control	0.26	0.39*	0.27	0.09	0.23	0.16	0.53**	0.49**	1

*<0.05, **<0.01, ***<0.001

Multiple linear regression was used to establish the association between the separate ITs and aggressive behaviour. To test the prediction 1.1. all six ITs were entered as predictors of aggression in the first model. The resulting model did not have a significant overall fit, $F(6,24) = 2.04$, $p = .1$. However, "beat or be beaten self-enhancement" and "I am the law" ITs were shown to be positively associated with the aggressive behaviour. Consequently, a model with only these two ITs was fitted for the same outcome variables. The second model was a good fit, $F(2,28) = 3.95$, $p < .05$. However, despite indicated significance for the self-enhancement type of "beat or be beaten" IT, the corresponding CIs included zero suggesting that the effect is spurious (Table 6.3). Meanwhile, the "I am the law" IT was significantly and positively associated with the aggressive behaviour in the first session of the TAP.

Table 6.3 Summary of regression analysis for implicit theories predicting aggressive behaviour (n = 31)

$F(6,24) = 2.04$, $p = .1$, $R^2 = .34$, adjusted $R^2 = .17$				
	Estimate [95% CI]	SE	t	*p*
Intercept	7.91 [0.03,13.89]	3.33	2.37	0.03
Violence is normal	1.39 [-1.74,4.16]	1.33	1.04	0.31
Beat or be beaten	-0.63 [-3.35,2.68]	1.47	-0.43	0.67
Beat or be beaten self-enhancement	-1.24 [-4.68,2.46]	1.39	-0.89	0.38
Beat or be beaten self-preservation	-4.38 [-9.41,1.4][1]	2.09	-2.10	0.046
I am the law	3.44 [-0.20,6.79]*	1.64	2.10	0.047
I get out of control	2.81 [-0.37,6.98]	1.90	1.48	0.15
$F(2,28) = 3.95$, $p < .05$, $R^2 = .22$, adjusted $R^2 = .16$				
Intercept	11.37 [7.04,16.19]	2.33	4.88	<0.001
Beat or be beaten self-preservation	-3.82 [-7.37, 0.37]*	1.86	-2.06	0.049
I am the law	4.03 [0.85, 6.48]*	1.47	2.74	0.01

*<0.05, **<0.01, ***<0.001

[1]Although this estimate has *p* value below .05, the inclusion of 0 in the 95% confidence interval suggest that the association is spurious.

6.4.3 Testing Prediction 1.2. An increase in the heart rate and skin conductance level will be positively associated with aggression

Multiple linear regression was used to assess the prediction 1.2. stating that an increase in the heart rate and skin conductance level would be positively associated with aggression. The overall model was significant, $F(2,14)=5.4$, $p < 0.05$. Moreover, while the change in the SCL was significantly positively associated with aggressive behaviour, the change in the HR did not have significant association with the outcome (Table 6.4). Given that the stress task evoked change only in the SCL, its association with the noise intensity and duration at the first session of the TAP suggests that a higher stress response, indicated by SCL, is positively associated with aggressive behaviour. To establish whether there was a difference between students and patients in the relationship between SCL and aggression, an interaction model was run (Table 6.4). Although the overall model was not significant, $F(3,13) = 2.99$, $p = 0.07$, the individual estimates showed that SCL is positively associated with aggression only among patients but not the students. From the Table 6.1 it is evident that patients have more extensive histories of antisocial behaviour and have more entrenched aggression supportive ITs: "Violence is normal"; "Beat or be beaten: Self-Preservation".

Table 6.4 Results of regression analysis for physiological changes predicting aggressive behaviour (n = 17)

	Estimate [95% CI]	SE	t	*p*
$F(2,14) = 5.4$, $p < 0.05$, $R^2 = .44$, adjusted $R^2 = .36$				
Intercept	7.76 [5.11, 11.11]	1.41	5.51	<.001
HR change	0.14 [-0.22, 0.31]	0.15	0.91	0.38
SCL change	5.30 [2.52, 8.91]*	2.31	2.30	0.04
$F(3,13) = 2.99$, $p = 0.07$, $R^2 = .41$, adjusted $R^2 = .27$				
Intercept	7.13 [3.27, 11.24]	2.31	3.09	0.009
Student	0.96 [-5.31, 19.26]	3.04	0.32	0.76
SCL among patients	6.90 [3.22, 10.34]*	2.66	2.59	0.02
SCL among student	-1.61 [-84.64, 28.49]	6.26	-0.26	0.80

*<0.05, **<0.01, ***<0.001

6.5 Discussion

The findings have demonstrated that both aggressive supportive cognitions and stress response are associated with aggressive behaviour towards a stranger. However, while one implicit theory was directly associated with aggression in both samples, elevated skin conductance response was related to aggression only among forensic patients.

The results partially supported the first hypothesis (prediction 1.1), which stated the positive associations between aggressive implicit theories and aggressive behaviour. However, from six aggression supportive cognitions only one was shown to be related to aggressive behaviour. Specifically, the belief that a person has the right to decide whether others deserve aggression and whether to deliver it, was associated with aggressive behaviour towards a stranger. The positive relationship between cognitions condoning use of aggression, supports the proposition of the sociocognitive models of aggression that aggression results from enactment of aggression supportive cognitive structures (Anderson & Bushman 2002, Huesmann, 1988; 1998; 2016). However, given that not all forensic participants' index offence

was violent, it should be noted that the ITs discussed in this study might not be of a core importance to their index offence. Furthermore, contrary to the prediction 1.1 not all of the aggression supportive cognitions were related to aggressive behaviour. A possible reason behind this, is the nature of "I am the law" Implicit Theory, which describes both the norm and responsibility. In addition to justifying the use of violence, it also appears more fundamental as it gives the control over the situation to the aggressor as it awards the role of "*norm enforcer*" (Polaschek, Calvert, & Gannon, 2009). In terms of Theory of Planned Behaviour (Ajzen, 1991) this means that the "I am the law" IT includes both subjective norm and perceived behavioural control, while other ITs only include the former. The cognition that portrays oneself as judge, jury and 'executioner' might be more fundamental to aggression behaviour and consequently account for their effect on aggression. This result extends previous research by adding the fundamental Implicit theory, condoning and arguably empowering the use of violence, to the aggression supportive cognitive structures that have been shown to associate with aggressive behaviours (Bowes & McMurran; 2013; Gilbert, et al., 2013; Hoise, et al., 2014; Ireland et al, 2019).

The results of the study also partially confirmed the second hypothesis (prediction 1.2) proposing that an increase in stress evidenced by the change in stress response markers would be associated with aggressive behaviour. Only the changes in the SCL during the acute stressor were associated with aggressive behaviour. This is in line with previous research showing the positive association between increased SCL and anger (Zhan et al., 2017) or relational aggression (Murray-Close et al., 2017). Although contrary to some of the existent studies (Gerra et al., 2001) and in line with others (Schwarz & Portnoy, 2017) the changes in the HR were not associated with aggressive behaviour, the current results should be interpreted with caution. The lack of the effect for the change in the HR might not be informative, as the manipulation check showed that the stressor did not evoke a change in it, indicating that it was

a mild stressor. Given that both HR and SCL reflect activation of the SAM system, the current finding falls in line with the discussion in the Chapter 3 and 5 suggesting that there is third variable in stress – aggression relationship.

Indeed, further analysis specified that an acute stress response was, in fact positively associated with aggressive behaviour only among the patients. These findings partially support previous studies showing the same relationship between markers of SAM system activation and aggression among male students (Verona & Curtin, 2006; Verona & Kilmer, 2007). Although the obtained results did not show the stress – aggression association to be present among students, it might arguably be due to the measure of aggression. The current study used only the first "unprovoked" response in the TAP representing behaviour towards a stranger, rather than the average responses from the paradigm reflecting behaviour in an interaction. Hence, it is possible that exposure to stress on its own is not enough to provoke aggressive behaviour among students, as they require further provoking or competitive interaction with others. However, for the patients it appeared that it could be sufficient. This assumption is based on the descriptive statistics of the sample that demonstrate higher prevalence of past aggression, antisocial behaviour and implicit theories condoning use of aggression among patients as compared to the students.

6.6 Limitations

Other explanations behind the partial agreement with previous research reflect the limitations of the current study. The small sample size impairs detection of minor effects. The interaction model for aggressive behaviour (Table 6.4) was not significant, while the model for the whole sample was. Consequently, it is possible that the positive stress – aggression association was significant only for the patients, due to its effect size being larger than that for the students. Similarly, the results of the present study should only be considered as preliminary

due to the sample size, which restricts inferences from the results to the wider population. Although the sample size for the Study 1 was small, which precluded a more complex and detailed analysis of the data[23], the results showed that forensic patients represent a valuable research group, especially for aggression studies, as they had a distinct stress - aggression relationship. Given the need to establish the influential third variables for this relationship, further research into this population would be beneficial. Moreover, in order to maintain the confidentiality of the IT interview, this assessment was not subjected to inter-rater reliability check. This increases the possibility that identification of the ITs was influenced by the interviewer bias, which is a drawback. Another limitation was the inability of the chosen stress task to elicit a HR response, which in turn suggests overall low activation of the stress response system. Given that part of the sample in the current study was represented by a vulnerable population (patients), this drawback highlights the need to calibrate stress tasks more accurately to the sample. Consequently, in the next studies life stress experiences were used as they correspond to individual idiosyncrasies.

6.7 Concluding comments

Despite the noted limitations, the current study supports the principles governing aggression suggested in previous chapters. A fundamental cognitive structure that not only condones the use of aggression, but also entitles the person to decide when it is suitable and who deserves it, was shown to be positively associated with aggression towards a stranger among both student and patients. This, therefore, captures the link between cognitive structure and conduct proposed in Chapter 2, according to which aggressive behaviour is an enactment of aggression supportive cognitive structures. However, given the limited sample size and small power of the analysis, this result is only preliminary and need to be replicated. Moreover,

[23] This was due to the COVID-19 pandemic

further analysis showed that this relationship is likely to be predicated on another variable. In this case it was the population of the participant that is likely reflective of closer familiarity with, and habitual use of, aggression. However, this proposition was based on observation of descriptive only, meaning that it needs to be specifically tested in following studies. To extend these findings, the next chapter investigates the effect of history of aggression and aggressive traits have on such behaviour in a given situation. This in turn supported the proposition from the Chapter 3, that the stress response is an important impellent, using I^3 terms (Finkel & Hall, 2018), for aggressive behaviour, but is not a direct determinant. Further possible influential third variables for the stress – aggression relationship are discussed in next two chapters. While the next chapter addresses the relationship between positive and negative affect, which often accompany stress, Chapter 8 takes into account the coping styles, which can negate exposure to stressors with different effectiveness. As this chapter addressed aggression supportive cognitive structures and stress response, the following chapter focuses on the second proposition derived in the Chapter 2, stating that aggression results from decision-making processes.

CHAPTER SEVEN: STUDY TWO: THE ROLE OF NEUTRAL AND EMOTIONAL INFORMATION PROCESSING AND AFFECTIVE STATES IN FACILITATING AGGRESSIVE BEHAVIOUR

7.1 Structure of the chapter

This study explores the relationship between history of aggressive behaviour, affective states, and neutral and emotional information processing. In the Chapter 2, based on the socio-cognitive and neurocognitive models it was suggested that the three aforenoted variables are direct contributors to aggressive behaviour. Information processing affect selection of behavioural scripts that are partially formed by past behaviour. It is followed by the addition of variables representing arousal, as the literature review suggested this will have an indirect relationship with aggressive behaviour by favouring habitual behaviour.

7.2 Current Study

This study employs a series of questionnaires to assess trait aggressiveness, history of aggressive behaviour, and trait worry. Through the experimental session participants were also asked to rate their affective states several times. To investigate the effect of neutral and emotional information processing on aggressive behaviour the present study utilised the P3 difference amplitude during a Go/No-Go task. During it participants were asked to respond as fast as possible to words presented in normal front (Go), but not to respond to words presented in italics font (No-Go), regardless of the words (negative or positive or neutral). The task, and specifically the P3 Event Related Potential (ERP) component during the linguistic Go/No-Go task was chosen as the operationalisation for information processing, as the Go/No-Go task facilitates increase in the P3 amplitude as the effort for inhibitory control rises (Verona, Sprague, & Sadeh, 2012). ERPs reflect voltage produced by neurotransmission-mediated activity associated to specific events (Luck, 2014). The P3 component refers to the third

positive peak that occurs around 300 ms after onset of the stimulus. Functionally, the P3 amplitude is argued to represent the allocation of cognitive resources necessary for task completion and to involve attention and working memory processing. Furthermore, P3 has been used to index the change to subjective expectations (the more unexpected the occurrence, the higher the peak) and to reflect inhibitory processing (Dierolf, Fechtner, Böhnke, Wolf, & Naumann, 2017; Kropotov, Ponomarev, Hollup, & Mueller, 2011; Polich, 2007). Consequently, in previous research P3 amplitude has been used to reflect cognitive processing of the situation that precedes the aforementioned "choice" of a given behavioural script and has been shown to be negatively related to aggression (Fanning, Berman, & Long, 2014; Jabr, Denke, Rawls, & Lamm, 2018; Verona, E., & Bresin, 2015). In the present study the P3 difference amplitude was used as an index of the cognitive resources engaged before making a decision. After completion of the Go/No-Go task participants were asked to participate in a "supervisor – employee" laboratory paradigm which has been used to assess aggressive behaviour via seeming delivery of electric shocks in previous studies (Buss, 1961; Verona, Sadeh, & Curtin, 2009). The effect of feeling hostile, as measured by the Positive and Negative Affect Schedule item was analysed separately from general negative and positive affect subscales, partially due to the nature of the aggression paradigm, which "allows" aggressive behaviour. Although both negative affect and hostile perception of others has been shown to correlate with aggression proneness (Burt et al., 2009), hostile feelings and thoughts have been specifically highlighted by the GAM as potential route for aggressive behaviour (Anderson & Bushman, 2002). Consequently, the present study looks at hostility individually.

7.3 Method

7.3.1 Participants[24]

The current study analyses a dataset based on a subset of community participants (n = 100) from a larger study, recruitment for which took place in Hillsborough County, Florida and utilised advertisement via print and social media. One hundred and forty one participants originally completed the study session, with 40 excluded from Event Related Potentials (ERPs) analysis due to technical issues with the obtained data. In addition, 13 participants were excluded from all analyses as they did not adhere to the rules of the aggressive behaviour paradigm (e.g. not reading the provocative feedback on their work or understanding the deception). Furthermore, one of the participants had missing data for the aggression paradigm. Given that it was the only missing case, it was decided to exclude it from the analysis. Thus, since exclusion categories were not mutually exclusive, the study comprised 49 men and 48 women, with three participants who did not specify their sex. The average age was 29.06 (SD = 6.68). Most participants reported themselves as White Americans (54%), followed by the Black or African American (31%), Asian American (5%), American Indian, or Alaska Native (3%). Six per cent of participants selected their race as "other" and one per cent did not respond. The study was approved by the Institutional Review Board (IRB) in University of South Florida (IRB#: Pro00030534).

7.3.2 Go/No-Go task

Event Related Potentials (ERPs) were recorded as participants completed the Emotional-Linguistic Go/No-Go task (Goldstein et al., 2007; Verona, Sprague, & Sadeh, 2012), which included positively (e.g. mighty) and negatively (e.g. hate) valenced and neutral words (e.g.

[24] The US sample was obtained via collaboration with a US research group due to Covid-19 preventing UK data collection. The US research team collected the data in their laboratory and the current PhD fully processed and analysed this data in accordance with the current predictions; all analyses were unique. Acknowledgment and thanks are given to Professor Verona for facilitating access to this dataset.

lamp). Each category included 32 words selected from the Affective Norms for English Words (Bradley & Lang ,1999). Moreover, the selection of negative words was aimed to ensure salience for those with histories of aggression (Sprague & Verona, 2010).

During the task, participants were asked to respond to the font of the word (normal or italicized) rather than its valence (i.e. positive, negative, neutral). Participants were required to press a button for words written in a normal font (Go trial), and not to press a button for words in an italicized font. Before the task started participants were provided with 20 practice trials, with neutral words. The task itself comprised six randomised blocks, for each of the three valences. Across participants, the sequence of the emotional categories was counterbalanced. To ensure that No-Go trials were "unexpected", each block contained fewer of them (nine per block) than of Go trials (23 per block). Each word was presented for 1400ms and followed by a 750-1000ms interval between trials.

7.3.3 ERP components

The ERPs were recorded using Electrical Geodesics system hydrocel 64-channel sensor nets and amplifiers (EGI, Eugene, OR) while the participants engaged in the Go/No-Go task. Frontocentral and Parietal electrodes were selected to index inhibitory control and emotional processing (Verona et al., 2012). Analog signals were digitized online at 250 Hz and bandpass filtered (.15-200 Hz) and amplified using Net Amps amplifiers. Electrodes underneath the eyes imbedded in the nets were used to record eye movements. Impedances were kept below 50 kΩ. Stimuli were presented on a flat-panel display using E-Prime software (PST Inc., Pittsburgh, PA), and behavioural responses were collected with a four-key keypad that interfaces to E-Prime.

The processing of the obtained data was completed in Netstation software. Average head model[25] was used as re-referenced point. The data was epoched 200ms before and 800ms after the stimulus onset, with a .10 to 30 Hz filter utilised for correction. Trials with deflections greater than 140 mV in absolute value or with eye movements greater than 55 mV were discarded. For those channels where more than 20% of trials were discarded, channel replacement was performed. In the course of data processing, an average of 80% Go trials and 77% of No-Go trials were retained. Participants with more than 50% of unusable trials were excluded to maintain the minimum number of trails required for statistical stability. Within each condition, average ERP waveforms were calculated, and a 30 Hz Butterworth filter was applied with baseline correction. The P3 component[26] was defined as adaptive mean peak amplitude (+/-) within 400 to 600 ms post-stimuli at frontal and parietal sites.

Since the correct completion of the Go/No-Go task required responding to the form rather than content of the presented word, in the current study P3 difference amplitude to used primarily to reflect inhibitory processing.

7.3.4 Self-report questionnaires

Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988) scale consists of 20 Likert scale items, asking participants to rate how they are feeling at a given moment (Crawford & Henry, 2004) ranging from 1 (Very slightly or not at all) to 5 (Extremely). The positive affect scale measures generally positive emotional states and includes items such as "excited". The negative affect scale assesses negative emotions, including items such as "scared". The PANAS has been reported to have good aggregate reliability (For NA Cronbach's $\alpha = .95$, for PA Cronbach's $\alpha = .95$) (Leue & Lange, 2011).

[25] Technique describing use of average of channels as reference point.
[26] ERP components is defined as "scalp-recorded neural signal that is generated in a specific neuroanatomical module when a specific computational operation is performed" (Luck, 2014, p.66). P3 component is specifically defined as third positive peak that occurs around 300 ms after onset of the stimulus (Luck, 2014).

Participants were asked to complete PANAS at four time points: 1) at the beginning of a session; 2) after completion of the Go/No-Go task; 3) after the provocation in form of feedback from the confederate; 4) after the aggression phase where they believed they delivered shocks to confederate.

The Aggression Questionnaire (AQ) (Buss & Warren, 2000) was used to assess trait aggression proneness. It is a self-report questionnaire with 34 Likert scale items asking participants how much an item describes them. The responses range from 1 (Extremely uncharacteristic) to 5 (Extremely characteristic). The AQ consists of five scales: Physical Aggression ("I may hit someone if he or she provokes me"); Verbal Aggression ("I tell friends openly when I disagree with them"), Anger ("I let my anger show when I do not get what I want"), Indirect Aggression ("I sometimes spread gossip about people I don't like"), and Hostility ("I wonder why sometimes I feel so bitter about things"). The subscales have shown good reliability: for physical aggression Cronbach's $\alpha = .88$; for verbal aggression Cronbach's $\alpha = .76$; for anger Cronbach's $\alpha = .78$; for indirect aggression Cronbach's $\alpha = .71$ and for hostility Cronbach's $\alpha = .82$ (Buss & Warren, 2000).

Life History of Aggression (LHA) (Coccaro, Berman, & Kavoussi, 1997) interview was used assess the past number of aggressive acts. Participants were asked about the frequency with which they had engaged in 11 types of aggressive and antisocial behaviour after the age of 13. Rather than providing a specific number for each question, participants were asked to select a category ranging from "never" (0) to "so many events they can't be counted" (5). While the LHA has three distinct subscales: aggressive behaviour ("Physical fighting (e.g. history of physical fights with other people whether or not the subject started the fight or not)"), antisocial behaviour ("Antisocial behaviour involving the police (e.g. warnings, arrests and/or convictions for misdemeanour or felony offenses"), self-directed aggression ("Suicide attempts"). Only the subscale including past aggressive acts was used in the current analysis

(Coccaro et al., 1997). The LHA aggression subscale has shown good reliability (Cronbach's α = .87) in the prior studies (Coccaro et al., 1997).

The Penn State Worry Questionnaire (PSWQ) (Meyer, Miller, Metzger, & Borkovec, 1990), was used to assess trait cognitive symptoms of anxiety. It is a 16 item Likert scale questionnaire with good validity (Cronbach's α = .95). An example of an item is: "My worries overwhelm me", which needs to be rated from "not at all typical of me" (1) to "very typical of me" (5). Participants are asked to rate how the items reflect them, from 1 (Very typical of me) to 5 (Not at all typical of me).

7.3.5 Procedure

Aggressive behaviour was measured via the "supervisor – employee" laboratory paradigm, developed by Buss (1961) and modified by Verona, Sadeh, and Curtin (2009). At the start of the experimental session, participants were introduced to a confederate with whom they were matched with respect to sex and minority status. Participants believed that they then "drew lots" to determine which role they would take up in the session; in reality, they were always assigned the role of the "supervisor" in the recall task, and the role of the "feedback receiver" in the provocation task. In the course of the introduction, the confederate acted according to the script to portray them as rude and annoying. This provided the foundation for the confederate's behaviour in the provocation task that followed the completion of a Go/No-Go task. Specifically, participants were asked to write an essay about their personality qualities in five to seven minutes, which then was judged as "poor" by the confederate regardless of its content. Moreover, the confederate rated the participant negatively with respect to participant attractiveness, friendliness, and likability as well as remarking that the essay was "defensive" and "uninteresting". Despite being asked to be considerate in rating, the confederate completed the evaluation in approximately two minutes, and the participant "overheard" researchers remarking how fast the confederate completed his or her task. To ensure the provocation, an

apparently random member of research team "accidentally" left the feedback form with the confederate.

Afterwards, participants were told that the confederate, as an employee, would perform a recall task in a separate room and the participant, as a supervisor, would have to assess how correct the responses were. The feedback to the employee consisted of one option for correct responses and several for incorrect responses. For incorrect responses, participants could choose to deliver no electric shock (represented by the button 0) or chose an intensity of electric shock from 1 to 7. However, following what they believed to be shock administration, no visual or auditory "response from the confederate" was shown to the participants. In the previous session, participants already experienced the shock levels themselves in previous sessions (Fan, McCandliss, Sommer, Raz, & Posner, 2002). In reality, no shocks were delivered to the confederate.

The supervisor-employee task had four blocks, each of which included 10 trials. By design, approximately 40% of all trials in all blocks were incorrect responses, which afforded the participant an opportunity to select shock intensity. To index aggressive responding in each block, a mean shock intensity from trials when shock was delivered was calculated. In cases where no shock was selected despite the opportunity across all four blocks, the average was recorded as zero.

The previous research using similar paradigms, have shown that aggressive responses across blocks do not always increase with time (i.e. show linear growth) (Verona & Kilmer, 2007). Consequently, before using the index of aggressive responding as the outcome variable in the analyses, growth models were applied to determine the pattern of changes in the intensity of aggressive responses across all four blocks.

Although participants were aware of the inclusion of electric shocks in the paradigm when they were given information about the study, they belief in the paradigm was verified during the debrief. Participants were asked to fill seven Likert scale items about the employee – supervisor task. Afterwards a structured interview with 7 open-ended questions was conducted to assess their experience during all paradigms, followed by deception check question ("What did you think we were trying to investigate in this study?"). As a result, 13 participants were excluded from the study as either understood the employee supervisor paradigm or did not read the provoking judgement on their essay by the confederate.

7.3.6 Data analysis

All statistical analyses were conducted using SPSS software v23 (IBM Corp., 2015). For moderation and mediation analysis the PROCESS tool (Hayes, 2017) was utilised. All variables expressing product values were mean centred, in order to carry out simple slopes effects at mean, one standard deviation lower than mean and one standard deviation above the mean. For moderation analysis Davidson-McKinnon heteroscedastic-consistent interference was used to ensure that heteroscedasticity was not a concern. Given that 23% of participants who responded with "0" in the aggression paradigm, the distribution of average aggressive responses was positively skewed. However, in light of central limit theorem[27] no transformation was performed (Field, 2018). All the analysis utilised bootstrapped 95% confidence intervals, based on 5000 samples, to provide a robust assessment of the effects highlighted by the analyses. Consequently, the "significance" of the effects was further tested by looking at 95% confidence intervals. If they included 0, then the effect was deemed to be unreliable. However, if they did not include 0, the effect was considered valid.

[27] Refers to the assumption that estimated parameters for the population approach normal distribution when sample size is large (Field, 2018).

7.4 Results

The descriptive statistics and reliability indices for the sample are presented in the Table 7.1. Before presenting the results of the main analyses, the growth type of the aggressive behaviour in the paradigm is assessed.

Table 7.1 Descriptive statistics (n = 100)

	Mean	Std. Deviation	Reliability[1]
Average shock level	2.34	2.04	
Quadratic change in aggression	.44	2.21	
Feeling hostile at the baseline	1.18	.66	
Trait Aggressiveness (AQ total score)	75.50	20.62	.91
Past aggressive behaviour (LHA)	9.14	5.21	.72
Cognitive Symptoms of Anxiety	48.15	15.05	.94
Average shock level among males[2]	2.63	2.01	
Average shock level among females[3]	1.39	1.91	
Quadratic shock level among males[2]	.71	2.83	
Quadratic shock level among males[3]	.24	1.37	

[1]Cronbach's Alpha, [2] $N = 49$, [3]$N = 48$.

7.4.1 Aggressive behaviour in supervisor-employee paradigm

To establish the pattern of changes in the intensity of aggressive responding across blocks growth models were utilised. The initial model (variance components) showed that the linear trend was significant, F(1, 186.88) = 7.3, $p = .008$. Similarly, the quadratic change was significant, $F(1,325.53) = 5.4$, $p = .021$. Moreover, addition of the quadratic term significantly improved the model fit, as evidenced by the $\chi^2 = 5.53$, $df = 1$, $p < 0.05$. However, cubic change was not significant, $F(1,324.99) = 1.94$, $p = .17$ and, consequently, its addition did not significantly improve model fit, the $\chi^2 = 1.93$, $df = 1$, $p > 0.05$. These results demonstrated that aggressive behaviour followed a quadratic trend, meaning that it increased across blocks two

and three but decreased afterwards (Figure 7.1). Consequently, both average aggressive behaviour across blocks and quadratic change in aggressive behaviour across blocks[28] were used as outcome variables in the analysis testing the hypotheses outlined in the Chapter 4. To reflect the differences in the change of aggressive behaviour across blocks, the linear growth is also referred to as continuous aggression and quadratic change is also referred to as short-lived aggression further in the thesis.

To compare male and female participants' level of aggression, two linear regressions were utilised with each type of change in aggression as an outcome (N = 97). The model for average aggressive behaviour did not fit the data well, $F(1, 96) = 2.92$, $p = .09$, and the was no significant differences between male and female participants, $B = -.68$, 95% CI [-1.48, .13], $p = .10$. Similarly, neither the model for quadratic change in the behaviour, $F(1, 96) = 1.08$, $p = .30$, nor difference between sexes was significant, $B = -.47$, 95% CI [-1.37, .35], $p = .08$. This

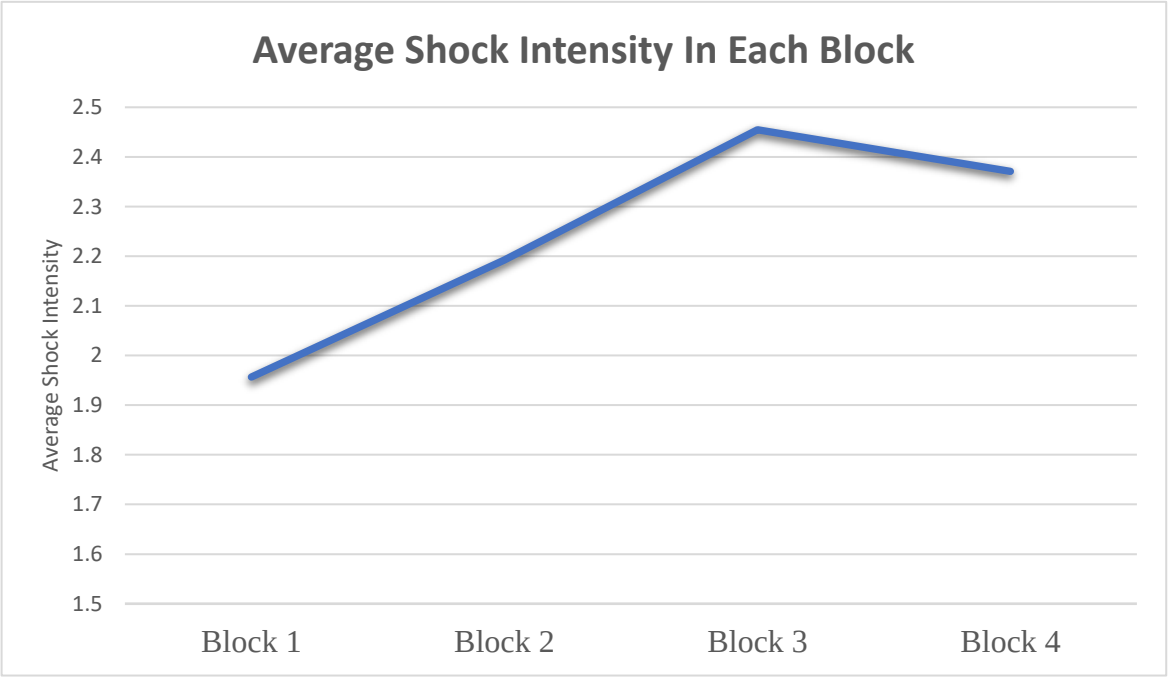

Figure 7.1 Average shock intensity across blocks

[28] Computed as sum of average shock levels at the timepoints 2 and 3 minus sum of average shock levels at the timepoint 1 and 4

shows that there was no difference in aggressive behaviour between male and female participants, allowing them to be analysed together in one sample.

7.4.2 Testing Prediction 2.1. An allocation of cognitive resources moderates the relationship between trait aggressiveness and aggressive behaviour

Moderation analysis was applied to investigate whether the relationship between trait aggressiveness (AQ) and aggressive behaviour changes depending on allocation of cognitive resources. There were significant interaction effects between the total score on AQ and frontocentral P3 difference amplitude in predicting average aggressive responding (Table 7.2). However, as the bootstrapped confidence intervals, [-.0007, .03] included 0, it is likely that the association between trait aggressiveness did not depend on the response inhibition. Simple slopes analysis also showed that trait aggressiveness was not significantly associated with average aggressive responding at lower than mean level of frontocentral P3 difference amplitude, B = .01, 95% CI [-.02, .04], t = .75, p = 45. Nevertheless, at the mean and higher values of the frontocentral P3 amplitude, trait aggressiveness was positively associated with average shock delivered by the participants, B = .03, 95% CI [.01, .05], t = 3.09, p = .002, B =.05, 95% CI [.02, .08], t = 3.56, p = .001. In other words, trait aggressiveness was only associated with increasing or continuous aggressive behaviour only among those showing medium or higher cognitive resource allocation, thereby indicating that P3 difference amplitude acts as a moderator.

A similar pattern of results was demonstrated when the parietal P3 amplitude was used as a moderator in the model. Although the interaction term between trait aggressiveness and average aggressive responding was significant, the bootstrapped confidence intervals included 0. Moreover, the simple slopes analysis also showed that at the lower than mean level of the parietal P3 difference amplitude, trait aggressiveness was not associated with average aggressive responding, B = .007, 95% CI [-.02, .04], t = .46, p = .65. However, at the mean and

higher levels of parietal P3 amplitude, trait aggressiveness was positively associated with average aggressive responding, B = .03, 95% CI [.01, .05], t = 2.97, p = .004, B = .06, 95% CI [.02, .10], t = 2.84, p = .006. Thus, parietal P3 amplitude also acts as a moderator between trait aggressiveness and continuous aggressive behaviour.

However, moderation analysis of the interaction between trait aggressiveness and frontocentral P3 difference amplitude in predicting quadratic change in aggressive behaviour did not yield a significant interaction. Likewise, there was no significant interaction between parietal P3 amplitude or significant simple slope results. These results demonstrate that P3 difference amplitude does not act as a moderator between trait aggressiveness and short lived aggressive behaviour (See Table 7.2).

Table 7.2 Moderation models looking at the interaction between response inhibition and trait aggressiveness (AQ) predicting aggressive behaviour (n = 100)

	B [95% CI]	S.E.	*t*	*p*
Average shock across blocks, R^2 = .12, *p* = .001				
Constant	2.41 [1.999, 2.81]	.21	11.62	<.001
AQ total score	.03 [.01, .05]**	.01	3.09	.003
Frontocentral P3 GNG	-.03 [-.35, .20]	.15	-.21	.83
AQ total score X P3 GNG	.01 [-.0007, .03]*	.01	2.05	.043
Average shock across blocks, R^2 = .12, *p* = .009				
Constant	2.42 [2.01, 2.83]	.22	11.16	<.001
AQ total score	.03 [.01, .05]**	.01	2.97	.004
Parietal P3 GNG	.09 [-.18, .33]	.14	.62	.53
AQ total score X P3 GNG	.01 [-.003, .03]*	.01	1.67	.10
Quadratic shock across blocks, R^2 = .11, *p* = .18				
Constant	.37 [-.04, .77]	.22	1.71	.09
AQ total score	.02 [-.01, .04]	.01	1.42	.16
Frontocentral P3 GNG	-.27 [-.66, .01]	.17	-1.61	.11
AQ total score X P3 GNG	-.01 [-.04, .001]	.01	-1.42	.16
Quadratic shock across blocks, R^2 = .06, *p* = .22				
Constant	.45 [.05, .88]	.22	2.05	.04
AQ total score	.03 [.003, .05]	.01	1.92	.06
Parietal P3 GNG	.03 [-.20, .25]	.11	.26	.79
AQ total score X P3 GNG	.002 [-.01, .02]	.01	.45	.65

*<0.05, **<0.01, ***<0.001

7.4.3 Testing Prediction 2.2. An Allocation of cognitive resources moderates the relationship between past history of aggression and aggressive behaviour

Regression models similar to the outlined for the prediction 2.1 but using history of aggression rather (LHA) than trait aggressiveness (AQ), were performed. However, neither frontocentral nor parietal P3 amplitude interacted with past aggressive conduct in predicting either average or quadratic aggressive responding (see Table 7.3).

Table 7.3 Moderation models looking at the interaction between response inhibition and past aggression (LHA) predicting aggressive behaviour (n = 100)

	B [95% CI]	S.E.	*t*	*p*
Average shock across blocks, R^2 = .02, p = .65				
Constant	2.34 [1.94, 2.75]	.21	11.1	<.001
LHA aggression score	.03 [-.05, .11]	.04	.83	.40
Frontocentral P3 GNG	-.14 [-.47, .15]	.17	-.81	.42
LHA aggression score X P3 GNG	.01 [-.06, .10]	.04	.25	.80
Average shock across blocks, R^2 = .01, p = .81				
Constant	2.35 [1.94, 2.77]	.21	10.93	< .001
LHA aggression score	.03 [-.05, .11]	.04	.76	.45
Parietal P3 GNG	-.06 [-.33, .22]	.14	-.42	.68
LHA aggression score X P3 GNG	.002 [-.06, .06]	.03	.06	.95
Quadratic shock across blocks, R^2 = .06, p = .29				
Constant	.43 [.03, .85]	.23	1.90	.06
LHA aggression score	.05 [-.06, .16]	.07	.74	.46
Frontocentral P3 GNG	-.27 [-.60, -.04]	.14	-1.87	.06
LHA aggression score X P3 GNG	-.04 [-.16, .08]	.07	-.52	.60
Quadratic shock across blocks, R^2 = .02, p = .63				
Constant	.44 [.06, .85]	.21	2.09	.04
LHA aggression score	.06 [-.06, .17]	.06	.87	.39
Parietal P3 GNG	-.004 [-.26, .22]	.12	-.03	.98
LHA aggression score X P3 GNG	.008 [-.06, .07]	.03	.24	.81

7.4.4 Testing Prediction 2.3. Participants who respond with increases in negative affect during provocation will show more aggressive behaviour than participants who responded with increases in positive affect during provocation

Principal Component Analysis was conducted to establish the composition of the PANAS subscales, due to their lack of accuracy in noting transient emotional states (Harmon-Jones, Bastian, & Harmon-Jones, 2016). The two factor solution based on eigenvalues and scree plot inspection showed that three items had a loading less than .5. Specifically, item 17 (Attentive), item 6 (Guilty), item 11 (Irritable), and item 8 (Hostile) did not load on either positive (PA) or negative affect (NA) factor (See the Appendix 2 Table A3.1, Figure A3.1). Consequently, these variables were not included in the calculation of PA and NA scores for each time point. Newly computed PA had the M = 26.20, SD = 8.96, and reliability Cronbach's alpha = .79, while the NA had the M = 8.51, SD = 2.85, and reliability Cronbach's α =.55[29].

Multiple linear regression of change in PA and NA during provocation on average aggressive responding across the tasks showed the model was not significant, $F(2,121) = 1.8$, $p = .17$. Similarly, neither PA nor NA was associated with average aggressive responding, $B = .09$, 95% CI [-.006, .18], $t = 1.86$, $p = .07$, $B = -.009$, 95% CI [-.15, .11], $t = -.14$, $p = .89$. When quadratic change in aggression was used as the outcome variable, the model was also not significant, $F(2,121) = 1.34$, $p = .27$. Similarly, the changes in PA and NA were not associated with quadratic change in aggression, $B = .04$, 95% CI [-.06, .14], $t = 79$, $p = .34$, $B = -.09$, 95% CI [-.26, .02], $t = -1.34$, $p = .19$. This indicates that changes in affect during provocation are not associated with aggression.

[29] Although this is quite low value for the Cronbach's α, it is reflective of the internal consistency of this subscale in this specific sample.

7.4.5 Testing Prediction 3.4 Participants who reported higher hostility following provocation will show more aggression than those who reported other forms of negative affect.

Since hostility did not load onto the negative affect subscale of the PANAS, its association with aggressive behaviour as an individual item was tested separately. A hierarchical regression with forced entry method was employed, with the first model including hostility score at the baseline, and with addition of the change in hostility score resulting from provocation (baseline subtracted from post-provocation score, See Table 7.4 & 7.5. For both average and quadratic change in aggressive response, the addition of change in hostility did not improve the model fit. The coefficient for change in hostility was also not significant. Only baseline hostility was used in further analysis, as the model including it showed significant fit for predicting both average and quadratic change in aggressive responding. Although the coefficient for baseline hostility had a borderline significant association with average aggressive responding, inspection of the confidence intervals confirmed that the association was positive and valid only with continuous aggression (See Table 7.4 & 7.5).

Table 7.4 Stepwise regression models testing baseline hostility, change in hostility, for average aggressive responding (n = 100)

	B [95% CI]	S.E.	*t*	*p*
Model 1, R^2= .034, *p* = .07				
Constant	1.67 [.63, .2.40]	.44	4.01	<.001
Average baseline hostility	.57 [.08, 1.44] *	.34	1.86	.045
Model 2, R^2=.039, *p* = .1.5; R^2 change = .005, *p*=.49				
Constant	1.64 [.57, 2.39]	.46	3.93	.001
Average baseline hostility	.54 [.02, 1.39]	.36	1.74	.08
Hostility change during provocation	.18 [-.30, .79]	.28	.70	.47

estimation based on 4999 samples

*<0.05, **<0.01, ***<0.001

Table 7.5 Stepwise regression models testing baseline hostility, change in hostility, for quadratic aggressive responding (n = 100).

	B [95% CI]	S.E.	*t*	*p*
Model 1, R^2 = .06, *p* = .01				
Constant	-.57 [-2.03, .60]	.72	-1.29	.41
Average baseline hostility	.85 [-.20, 2.11]	.66	2.59	.15
Model 2, R^2 =.07, *p* =.03 R^2 change = .004, *p* = .52				
Constant	-.55 [-2.09, .58]	.75	-1.23	.42
Average baseline hostility	.88 [-.18, 2.12]	.68	2.65	.12
Hostility change during provocation	-.18 [-76, .44]	.30	-.65	.57

estimation based on 4999 samples

*<0.05, **<0.01, ***<0.001

7.4.6 Testing Prediction 2.5. Negative affect measured during provocation (pre to post mood induction) will be predictive of aggressive behaviour only among participants with high trait aggressiveness and a history of aggression

Moderation analysis was utilised to establish whether the relationship between aggressive responding change in negative affect during provocation was moderated by trait aggressiveness. This interaction term was not significant (Table 7.6). Similarly, the interaction between change in negative affect during provocation and trait aggressiveness (AQ) was not significant when quadratic change in aggressive was used as the outcome variable. Moderation analysis was also used to explore whether the relationship between aggressive responding and change in negative affect during provocation was moderated by history of aggressive behaviour (LHA). When average aggressive responding across blocks was used as the outcome, the interaction between history of aggressive conduct and change in negative affect during provocation was not significant (see Table 7.6).

Table 7.6 Moderation models assessing the interaction between trait aggressiveness/past aggression with change in negative affect for aggressive behaviour (n = 100)

	B [95% CI]	S.E.	*t*	*p*
Average shock across blocks, R^2 = .09, *p* = .02				
Constant	2.35[1.97, 2.74]	.20	11.79	< .001
Change in negative affect	-.02 [-.18, .13]	.08	-.26	.8
Total score on AQ	.03 [.01, .05]**	.01	2.74	.007
Change in negative affect X Total score on AQ	-.003[-.01, .005]	.004	-.88	.38
Quadratic shock across blocks, R^2 = .11, *p* = .11				
Constant	.46 [.03, .87]	.22	2.04	.04
Change in negative affect	-.05 [-.28, 11]	.11	-.47	.64
Total score on AQ	.02 [.002, .05]	.01	1.93	.06
Change in negative affect X Total score on AQ	-.008 [-.02, .005]	.006	-1.21	.23
Average shock across blocks, R^2 = .06, *p* = .25				
Constant	2.29 [1.90, 2.69]	.20	11.39	< .001
Change in negative affect	-.09 [-.24, .04]	.08	-1.19	.24
Aggression subscale of LHA	.01 [-.06, .09]	.04	.33	.74
Change in negative affect X Aggression subscale of LHA	-.04 [-.08, -.01]	.02	-1.88	.06
Quadratic shock across blocks, R^2 = .08, *p* = .12				
Constant	.38 [-.02, .79]	.22	1.77	.08
Change in negative affect	-.16 [-.32, -.02]*	.08	-2.14	.04
Aggression subscale of LHA	.03 [-.09, .14]	.06	.47	.64
Change in negative affect X Aggression subscale of LHA	-.04 [-.07, .02]	.02	-1.71	.09

*<0.05, **<0.01, ***<0.001

However, when the average aggressive response across blocks was replaced by the quadratic change in aggressive responses, the results were different. Although the interaction term was still not significant, the simple slopes analysis showed that only at the lower than mean levels of past aggressive conduct (LHA), the association between change in negative affect and aggressive responding was non-significant, B = .04, 95% CI [-.20, .29], t = .36, p = 72. However, for mean values of history of aggressive behaviour and for values above the mean the change in negative affect had a significant negative association with quadratic change in aggressive responding, B = -.16, 95% CI [-.31, -.01], t = -2.14, p = .04, B = -.37 , 95% CI [-.69, -.05] , t = -2.30, p = .02. This suggests that among those with average or higher than average history of aggressive behaviour, an increase in negative affect was followed by decrease of aggressive behaviour initially but with a rise at the last block.

7.4.7 Testing Prediction 2.6. A disposition toward worry will have an indirect effect on aggressive responding that will be mediated by allocation of cognitive resources

Moderation analysis demonstrated that predisposition towards worry is associated with neither average aggressive responding nor with quadratic change in aggressive responding, consequently P3 difference amplitude was not a significant moderator (see Table A3.2 in the Appendix 2)

7.4.8 Testing Prediction 2.7 Strength of the association between hostility and aggressive responding will increase as the allocation of cognitive resources decreases

Baseline hostility was associated with average change in aggression, while the change in aggression was not. Consequently, this measure of hostility was used in subsequent moderation models. The first model looking at frontocentral P3 difference amplitude moderation of the relationship between hostility and average aggressive responding did not include a significant interaction. However, in the similar model with quadratic change in aggression as the outcome, the interaction between hostility and frontocentral P3 difference amplitude was significant. A

simple slopes test specified that hostility was positively associated with a quadratic change in aggressive responding only at lower than mean levels of P3 difference amplitude, *B* = 1.88 , 95% CI [.64, 3.12], *t* = 3.01, *p* = .003.At mean and higher than mean levels of P3 difference amplitude, hostility was not associated with a quadratic change in aggression, *B* = .43 , 95% CI [-.87, 1.72], *t* = .65, *p* = .51, *B* = -1.02 , 95% CI [-3.26, 1.22], *t* = -.91, *p* = .37.

The regression model predicting average aggressive responding contained a non-significant interaction between hostility and parietal P3 difference amplitude. Likewise, when quadratic change in aggressive responding was used as the outcome variable, the interaction between hostility and parietal P3 difference amplitude was not significant (Table 7.7).

7.4.9 Testing Prediction 2.8. Change in negative affect will have an indirect effect on aggressive behaviour mediated by allocation of cognitive resources

Although P3 difference amplitude was found not to be directly associated with either continuous or quadratic change in aggressive behaviour, to account for possibility of full mediation, initial associations between the variables were tested. However, neither of them yielded significant results. Specifically, change in negative affect during provocation was not associated with frontocentral P3 amplitude, *B* = -.06, 95% CI [-.14, .06], *t* = 9.71, *p* < .0001. Similarly, neither change in negative affect nor frontocentral P3 amplitude were associated with average aggressive responding, *B* = -.5, 95% CI [-.20, .09], *t* = -.63, *p* = .53, β = -.15, 95% CI [-.49, .14], *t* = -.87, *p* = .39. Likewise, change in negative affect during provocation was not associated with parietal change in P3 amplitude, *B* = .004, 95% CI [-.18, .14], *t* = .04, *p* = .97. Change in negative affect and parietal P3 amplitude also were not associated with average aggressive responding, *B* = -.04, 95% CI [-.20, .09], *t* = -.48, *p* = .63, *B* = -.07, 95% CI [-.29, .18], *t*= -.57, *p* = .57. This shows that allocation of cognitive resources did not mediate the relationship between change in negative affect and aggressive behaviour.

Given that change in negative affect was associated with neither frontocentral nor parietal P3 amplitude, the further models using quadratic change in aggression were not carried out.

Table 7.7 Moderation analysis predicting aggressive behaviour from hostility and P3 difference amplitude. (n = 100)

	B [95% CI]	S.E.	*t*	*p*
Average shock across blocks, R^2 = .04, *p* = .33				
Constant	2.35 [1.94, 2.79]	.21	11.10	<.001
Hostility at baseline	.83 [-.24, 2.75]	.63	1.32	.19
Frontocentral P3 GNG	-.11 [-.50, .26]	.17	-.63	.53
Hostility at baseline X Frontocentral P3 GNG	.04 [-2.10, 1.89]	.46	.09	.93
Quadratic shock across blocks, R^2 = .17, *p* = .004				
Constant	.37 [-.01, .81]	.21	1.76	.08
Hostility at baseline	.43 [-.62, 2.26]	.65	.65	.51
Frontocentral P3 GNG	-.33 [-.91, -.08]*	.15	-2.25	.03
Hostility at baseline X Frontocentral P3 GNG	-1.03 [-5.11, -.12]*	.45	-2.28	.03
Average shock across blocks, R^2 = .05, *p* = .21				
Constant	2.43 [1.95, 2.91]	.25	9.78	<.001
Hostility at baseline	1.38 [-.47, 3.84]	.93	1.48	.14
Parietal P3 GNG	.08 [-.40, .44]	.21	.37	.71
Hostility at baseline X Parietal P3 GNG	1.07 [-2.00, 3.23]	1.35	.79	.43
Quadratic shock across blocks, R^2 = .09, *p* = .63				
Constant	.39 [-.09, 1.02]	.32	1.20	.23
Hostility at baseline	1.00 [-1.45, 3.74]	1.71	.58	.56
Parietal P3 GNG	-.08 [-.60, .56]	.39	-.21	.83
Hostility at baseline X Parietal P3 GNG	-.60 [-4.30, 3.90]	2.79	-.21	.83

*<0.05, **<0.01, ***<0.001

7.4.10 Testing Prediction 2.9. Emotional vs Neutral P3 differences will mediate the relationship between lower P3 difference Go/No-Go and aggressive behaviour

The mediation analyses investigating whether emotional processing mediated the relationship between P3 difference amplitudes (FC for frontocentral and Par for parietal) and aggressive behaviour (indexed by average shock intensity), yielded non-significant results (see Figure 7.2 and Figure 7.3).

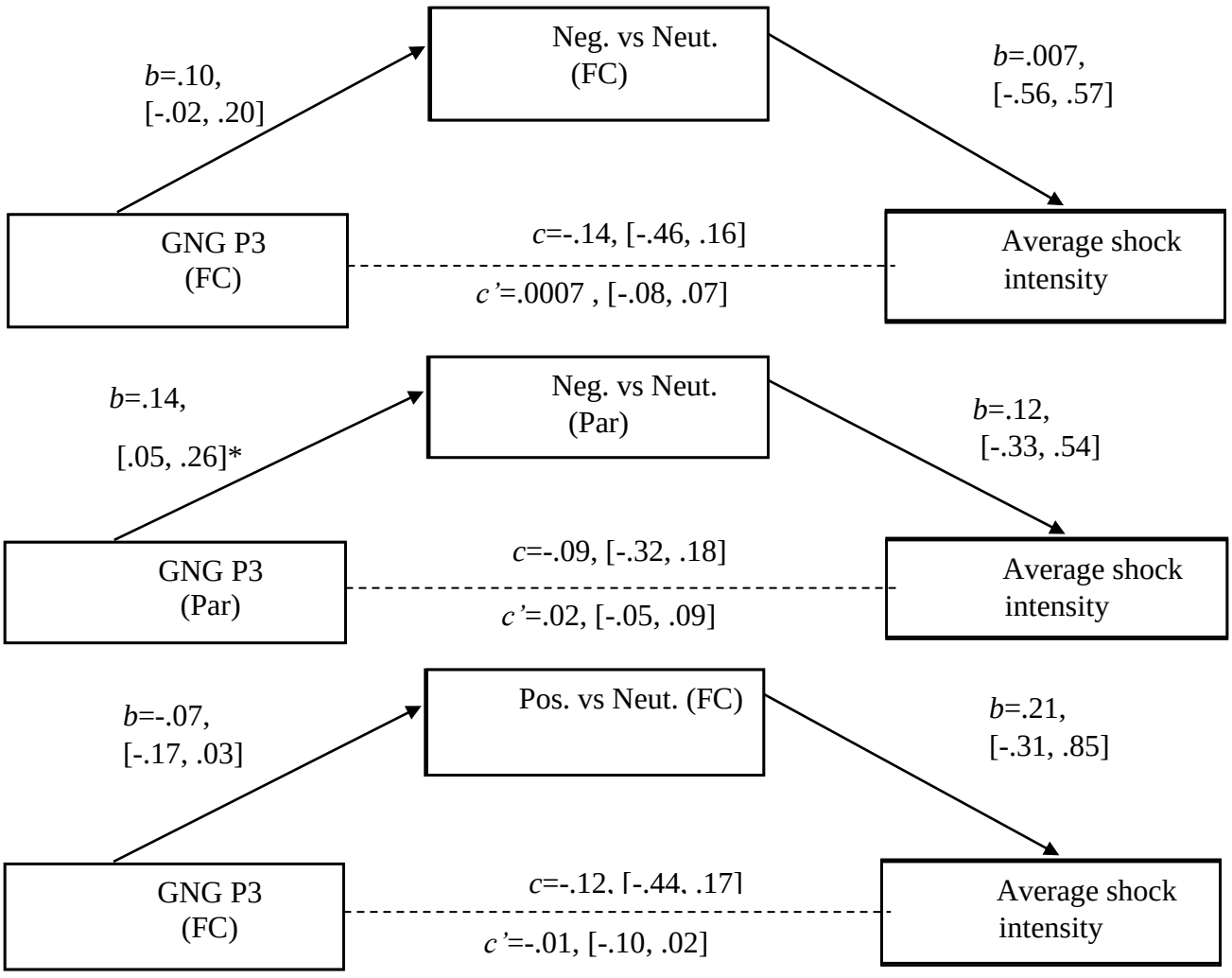

Figure 7.2 Mediation model for average aggressive behaviour from P3 difference amplitude and emotional information processing

However, it is noteworthy that both frontocentral P3 difference amplitudes were negatively associated with quadratic change in aggressive responding as the bootstrapped confidence intervals did not include zero (See Figure 7.4). This shows that although emotional processing does not mediate the relationship between allocation of cognitive resources and aggression, allocation of a lower amount of cognitive resource is inversely related to short lived aggressive behaviour.

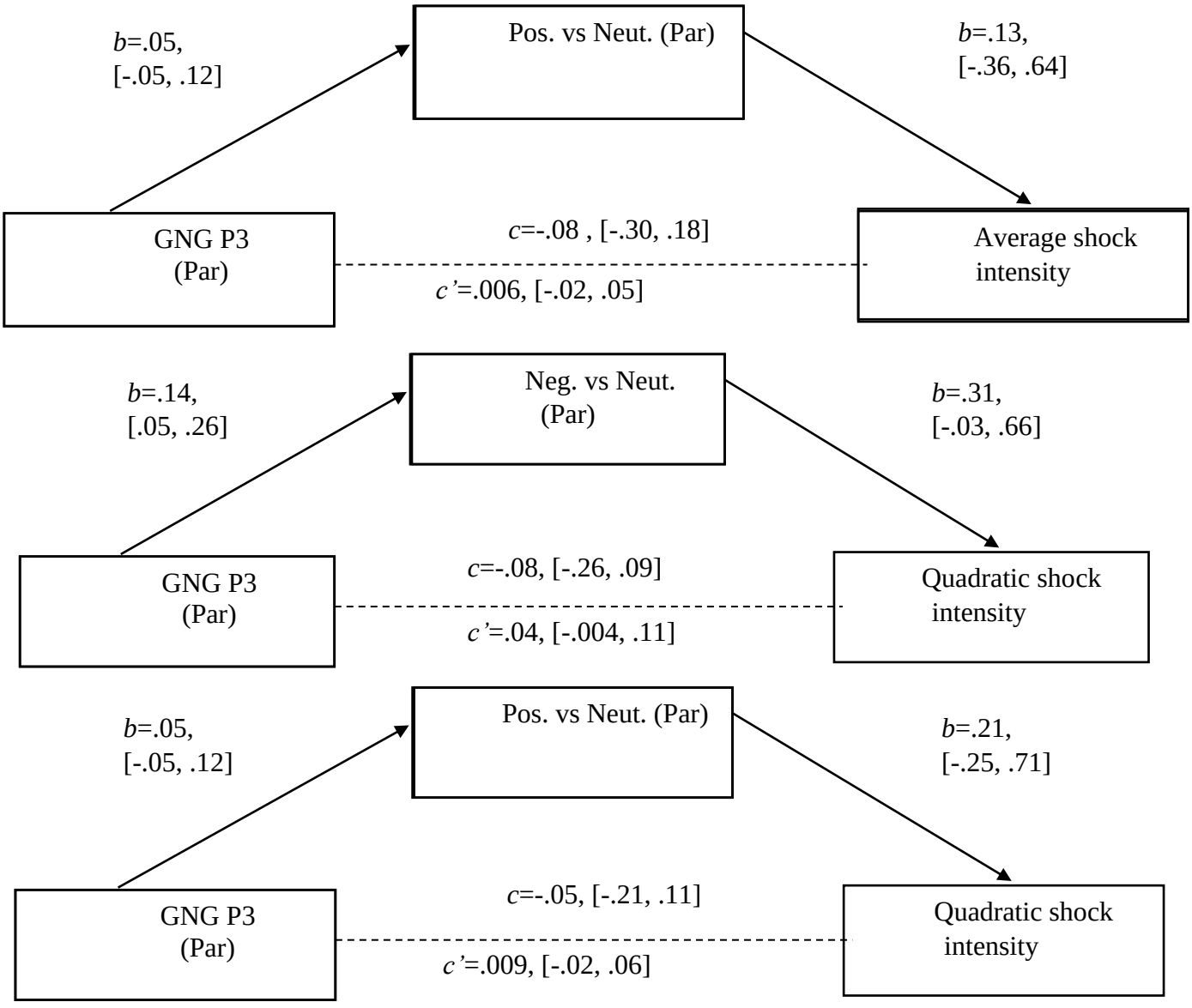

Figure 7.3 Mediation model for quadratic aggressive behaviour from P3 difference amplitude and emotional information processing

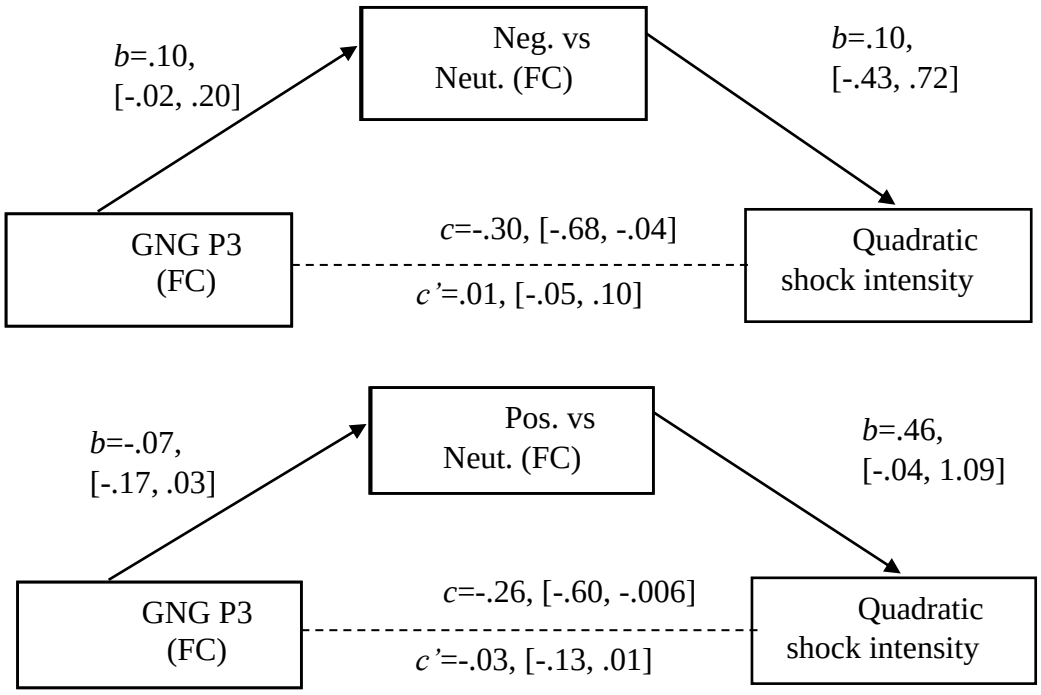

Figure 7.4 Mediation model for aggressive behaviour from P3 difference amplitude and emotional information processing

7.5 Discussion

The current study had nine primary hypotheses that were as follows:

2.1. An allocation of cognitive resources moderates the relationship between trait aggressiveness and aggressive behaviour

2.2. An allocation of cognitive resources moderates the relationship between past history of aggression and aggressive behaviour.

2.3. Participants who respond with increases in negative affect during provocation will show more aggressive behaviour than participants who responded with increases in positive affect during provocation.

2.4. Participants who reported higher hostility following provocation will show more aggression than those who reported other forms of negative affect.

2.5. Negative affect measured during provocation (pre to post mood induction) will be predictive of aggressive behaviour only among participants with high trait aggressiveness and a history of aggression.

2.6. A disposition toward worry will have an indirect effect on aggressive responding that will be mediated by allocation of cognitive resources.

2.7. Strength of the association between hostility and aggressive responding will increase as the allocation of cognitive resources decreases.

2.8. Change in negative affect will have an indirect effect on aggressive behaviour mediated by allocation of cognitive resources.

2.9. Emotional vs Neutral P3 differences will mediate the relationship between lower P3 difference Go/No-Go and aggressive behaviour.

Five of them, specifically 2.2, 2.3, 2.6, 2.8, 2.9 were not supported. Allocation of cognitive resources did not moderate the relationship between history of aggression and aggressive behaviour (2.2). Likewise, allocation of cognitive resources did not mediate the effect of disposition to worry or change in negative affect on aggression (2.6 & 2.8). Furthermore, index of emotional processing did not mediate the association between response inhibition and aggressive responding (2.9). Meanwhile, change is negative affect was not directly related to aggressive behaviour (2.3). However, other predictions were either fully or partially confirmed. The yielded results matched hypotheses 2.1 and 2.7, showing that allocation of cognitive resources moderates the relationship of trait aggressiveness and hostility with aggressive behaviour. Also, partially supporting hypothesis 2.4, baseline hostility rather than change in it was associated with aggression. Similarly, although trait aggressiveness did

not moderate the relationship between change in negative affect and aggressive behaviour, history of past aggression did.

Additionally, male and female participants did not differ significantly in the average or quadratic increase in aggressive behaviour. Consequently, the identified relationships between variables are likely to be present among both sexes, as all participants were analysed in a single sample. Although, this lack of differences was not in line with previous research showing sex differences in aggressive behaviour following acute stress (Verona & Curtin, 2006; Verona & Kilmer, 2007; Verona et al., 2006), it might be explained by the study's design. It did not incorporate specific variables that could explain potential differences between genders, such as motivation (Arriaga & Aguiar 2019) or personality traits (e.g., psychopathy (Hecht, Berg, Lilienfeld, & Latzman, 2016).

This study showed that while inhibitory processing during neutral task is associated with aggressive behaviour, the inhibitory processing during emotionally charged task is not. The findings supported the prediction 2.1 positing that an allocation of cognitive resources moderates the relationship between trait aggressiveness and aggressive behaviour. Specifically, the results demonstrated that trait aggressiveness was positively associated with aggressive behaviour at mean and higher than mean levels of P3 difference amplitude, which reflects inhibitory processing (Kropotov et al., 2011; Polich 2007; Verona & Bresin, 2015). This suggests that trait aggression, which can be seen as an index of aggressive behavioural scripts, facilitates aggressive behaviour only among those with average or higher cognitive abilities involved in inhibition of impulses. Interestingly, this result was inconsistent with previous research, that showed the inverse relationship between P3 amplitude and aggressive behaviour (Fanning et al., 2014; Jabr et al., 2018). Specifically, it was expected that the development of aggression supportive cognitions is associated with such behaviour at low levels of P3 amplitude (Jabr et al., 2018). However, the obtained results can be interpreted with reference

to the employee-supervisor task that was employed in the present study, where an aggressive response is predicated on the "incorrect" response of the "employee". In this setting, average and higher P3 difference amplitude, which is indicative of more cognitive resources being allocated to tasks, might reflect that the participant attended to and processed more cues (Hajcak, MacNamara, & Olvet, 2010). This in turn might have translated to perceiving more reasons to behave aggressively, as compared to those who showed low P3 difference amplitude. Nevertheless, as the moderating role of inhibitory processing was only present for average shock level, the data showed that those with trait aggressiveness consistently behave aggressively, rather than engage in aggressive conduct at first but then stop. Opposingly to the prediction 2.1, prediction 2.2 stating that an allocation of cognitive resources moderates the relationship between past history of aggression and aggressive behaviour was not supported. There was no relationship between history of aggression and aggressive responding within the paradigm. The most likely explanation behind this result is generally low level of history of aggressive behaviour among participants.

Prediction 2.3 stated that participants who respond with increases in negative affect during provocation will show more aggressive behaviour than participants who responded with increases in positive affect after provocation. It was not confirmed as changes in neither form of affect were associated with aggressive behaviour. However, prediction 2.4, which stated that participants who reported higher hostility following provocation will show more aggression than those who reported other forms of negative affect was partially supported. Namely, trait hostility was positively associated with a continuous increase in aggressive behaviour. This partial support suggests that self-reported change in an emotional state does not have an effect on aggressive behaviour. This is consistent with previous research showing a lack of direct relationship between affect and aggression among university students (Wyckoff, 2016). However, these results are only partially consistent with the study of Burt and colleagues

(2009), since while hostility was positively related to aggressive behaviour, the change in negative affect was not. Nevertheless, the association of baseline hostility with aggressive behaviour appears to confirm the General Aggression Model's (GAM) (Anderson & Bushman, 2002) proposition that behaviour in interactions is partially affected by the inherent characteristics an individual possesses before entering the interaction. Since trait hostility rather than its change during provocation was associated with aggressive behaviour, it highlights the importance of the stable characteristics of individuals that they bring to the situation rather than the state they experience in that situation. Furthermore, the lack of direct association between a change in negative affect and aggression found in the current study can also be explained by the GAM. The GAM suggests that negative affect is a route through which aggression-supportive cognitions, stored in memory, increase in salience and are then chosen as behaviour. To investigate this proposition further, the current study considered the possible role of change in negative affect as a moderator. The results partially diverged from the prediction 2.5 stating that negative affect measured during provocation will be predictive of aggressive behaviour only among participants with high trait aggressiveness and a history of aggression. This association was found non-significant at any level of trait aggressiveness. Nevertheless, a history of aggressive behaviour was shown to be a significant moderator. Specifically, for those with an average or higher number of past aggressive acts, a decrease in negative affect during provocation was associated with an increase in aggressive behaviour.

This was unexpected, as previous research showed that when negative affect is included in models predicting aggressive behaviour (e.g., with emotional intelligence), it has a positive rather than a negative relationship (Megías et al, 2018). This is expected as experiencing negative emotional states has been suggested to precipitate aggression (Anderson & Bushman, 2002). However, the current study demonstrated that those who experienced less negative affect were more aggressive, regardless of the positive affect they reported. Similar to the

findings pertaining to aggression proneness and response inhibition, it is possible that the unexpected direction of the relationship was related to the aggression paradigm where participants perceived their aggression *as necessary due to conditions* rather than due to their internal state. Within the current paradigm aggressive behaviour was a 'punishment' for the opponent and as such resembles proactive rather than reactive aggression (Fabian, 2010). In this light, the inverse relationship between negative affect, which corresponds to low levels of emotional activation, resembles the reported association between low activation of the threat system and proactive aggression (Blair et al., 2018; Lozier et al.,2014; Siep, et al 2019).

Further exploration of the potential *indirect* effect of the change in negative affect on aggressive behaviour also did not yield significant results. Contrary to the prediction 2.8 stating that change in negative affect would be associated with aggression and its influence would be mediated by the P3 difference amplitude, no significant associations between the variables was established. Thus, a moderation analysis was not performed. However, the results followed the prediction 2.7 stating that strength of the association between hostility and aggressive responding will increase as the allocation of cognitive resources decreases. When self-reported hostility, treated as a *trait*, was entered into the analysis instead of 'general negative state' it was associated with aggression. Specifically, hostility was positively associated with the quadratic increase in aggressive behaviour only among participants with low response inhibition. This suggests that among participants who reported feeling hostile before the experimental provocation took place, only those who showed poor response inhibition were likely to respond with increased aggression. This result is consistent with previous research showing a positive association between feeling hostile and aggression (Ramirez & Andreu, 2006; Rubio-Garay Carrasco, & Amor, 2016), which is arguably an extension of hostile expectancy bias predisposing to aggressive behaviour (De Castro et al., 2002; Tuente et al., 2019).

Although prediction 2.9 positing that Emotional vs Neutral P3 differences will mediate the relationship between lower P3 difference Go/No-Go and aggressive behaviour, was not confirmed, the results are still noteworthy. Specifically, the performed analyses showed that emotional processing that can serve as an index of arousal is not associated with aggressive behaviour, whilst response inhibition, which reflects the allocation of cognitive resources to a given task, *is* negatively associated with quadratic change in aggressive behaviour. Since this suggests that participants with lower cognitive functioning demonstrated more aggressive responding, it is consistent with Verona and Bresin, (2015) study showing that those with low response inhibition have higher trait aggressiveness. This result is also consistent with the proposition that use of lower cognitive resources among those who have aggression supportive cognitive structures would facilitate aggressive behaviour (Anderson & Bushman, 2002; Crick, & Dodge, 1996; Huesmann, 2018). Using the terms of the I^3 model lower allocation of the cognitive resources appears as a reversed inhibitor for aggression (Finkel & Hall, 2018).

Continuing with the I^3 terminology, an expected impeller for aggressive behaviour was state worry. Prediction 2.6 stated that a disposition toward worry will have an indirect effect on aggressive responding that will be mediated by allocation of cognitive resources. Due to the lack of the association between state worry and either continuous or short lived aggression it was rejected. This indicates self-reported worry has no effect on aggressive conduct.

However, the association between lower P3 difference amplitude and aggressive behaviour appears to be at odds with the results of the earlier analyses, showing that trait aggressiveness was associated with aggressive behaviour only among participants with average or higher P3 difference amplitude. The apparent resolution of this contradiction can be derived from the difference in aggressive behaviour measures, which served as the outcome. Aggressive behaviour, which was associated with trait aggressiveness at average and higher than average levels of inhibitory processing, was the average aggressive responding that shows

continuous increase. Meanwhile, the type of aggressive behaviour that was associated with lower inhibitory processing was quadratic change in shock levels, which reflects an initial increase in aggressive conduct followed by the decrease in it. So different patterns of aggressive behaviour had different associations with the same index of response inhibition.

Since the trend analysis showed quadratic growth to be a significant improvement of linear growth model, the results showing trait aggressiveness association with aggressive behaviour only at average or higher level should be discarded, as the outcome variable did not accurately reflect the data. Nevertheless, as the linear growth model was still significant, and as the improvement in model fit was relatively small, the linear growth model did partially reflect the data. Consequently, discarding results for average aggressive behaviour is inappropriate, which means that both associations need to be reconciled. This can be achieved by deeming response inhibition as the possible "brake" for aggressive behaviour, which is what Brennan & Baskin-Sommers (2018) demonstrated in relation to externalising. They have shown that externalising is underpinned by failure to control responses to external cues. In the current study, however, it appears that the average or higher P3 difference amplitude may be "balancing out" intended aggressive behaviour. Consequently, lower P3 amplitude was associated with quadratic change in aggression, which reflects an increase in initial aggression that then falls across time. However, response inhibition cannot balance out aggression proneness, which leads to a linear increase in aggressive behaviour when the opportunity arises. This suggests that response inhibition is unable to *inhibit* aggressive behaviours stemming from a stable trait such as aggressiveness. However, there does appear to be a capacity to inhibit aggressive behaviour that stems from more transient emotions, such as hostility, which was only associated with quadratic increases in aggressive behaviour at lower levels of P3 amplitude.

7.6 Limitations

The current study has a number of limitations that can affect the produced results. Firstly, the chances of type I error are increased owing to the number of statistical analyses. However, to counterbalance this, bootstrapped confidence intervals were calculated to increase the robustness of effects highlighted as significant. Moreover, similar to other studies utilising ERP components as indices of cognitive processes, the current study falls to the inherently correlational nature of the link between a given ERP component and cognitive process (Luck, 2014). In this case, P3 difference amplitude was used to reflect inhibitory processing. However, it is possible that it was affected by other processes. Recent meta-analysis indicated that the P3 amplitude, which was used in the current study might lack specificity (Huster, Messel, Thunberg, & Raud, 2020). Namely, there are concerns the amplitude of this ERP component does not reflect inhibitory control, but rather represents broader cognitive processes such as resource allocation to a task, or amendment of expectations. Although concerning, this lack of specificity does not necessarily undermine the conclusions of the presented study. In this research, P3 amplitude was used as an operationalisation for broader information processing. Consequently, its relation to several cognitive process is beneficial in terms of capturing the intended construct but is harmful to the accuracy of mechanism identification. Additionally, the use of a specific interaction in the aggression paradigm (employer - employee) might have shaped the pattern of aggressive behaviour to fit the given interaction. This in turn limits the generalisation of findings pertaining to aggression to the types of social situation where similar roles are present, but not to others (e.g., strangers). Importantly it limits the inferences of the patterns to the situations where aggression is externally approved (in the current paradigm participants were given instruction that they *can* administer shock). Furthermore, the use of self-report measures raises the inherent problems of validity. The constructs such as negative

and positive affect require participants to have good introspection and ability to accurately identify the emotions they are experiencing.

Lastly, the given sample included students with lower levels of aggressive behaviour, which further limits the applicability of the results to the population where aggressive behaviour is more extreme or is exhibited more often. Consequently, in the next study the participants were selected in a way to create a wider variability of aggressive behaviour in the past.

7.7 Conclusion

The main findings of the current study are threefold. First, inhibitory processing indexed by the P3 difference amplitude during emotional Go/No-Go was shown to be related to aggressive behaviour. Specifically, it is likely to serve as an inhibitor of aggressive tendencies, but only for internal state like hostility, rather than for personality traits, such as aggressiveness. Consequently, to confirm that executive functioning overall is associated with aggressive behaviour in a larger sample, working memory, as its broader index, is included in the model built in the next study. Second, the *inverse* association between general negative states and aggressive behaviour among those with a past history of aggression highlights the relevance of what a person 'brings with them' to the situation where aggressive conduct is enacted. However, in the current study not all items from the PANAS were included in the analysis, undermining the validity of the assessed concepts. Thus, the next study shifts the focus to a more enduring individual characteristics such as aggression supportive cognitions, assessed using reliable instruments. Lastly, the results showed emotional processing, which reflects arousal, does not mediate the relationship between inhibitory processing and aggressive behaviour. This suggests that the physiological state a person experiences when they enter an interpersonal situation does not exert direct influence on their behaviour. It is important to

mention, however, that emotional processing is not as accurate index of physiological state as direct measures such as hear rate. Nevertheless, taken together these findings lend grounds to proposing that the relative dominance of enduring (i.e., acquired before a situation) characteristics of a person in facilitating aggressive conduct, over transient states that precede such conduct. However, this pattern appears predicated on a specific situation where aggressive behaviour is situationally allowed. Additionally, the obtained results suggest the potential relevance of the context in which a person has an *option* of behaving aggressively. Namely, when people with aggressive tendencies are in the social context that allows for aggressive behaviour, their cognitive processes do not inhibit aggressive tendencies. This in turn raises the question about the role of a predisposition to interpret social interactions in a particular way in facilitating aggressive behaviour. To addresses this question and based on the theory discussed in the Chapter 2, the next study will investigate the effect of hostile attribution bias on aggressive behaviour. To identify the influential factors for aggression in daily life, the next study utilises a cross sectional rather than experimental design, which also corresponds to assessing a more enduring characteristics rather than transient states in a particular situation.

CHAPTER EIGHT: STUDY THREE: ESTABLISHING PATHWAYS FROM COGNITIONS AND STRESS TO AGGRESSIVE ACTS AND TRAITS

8.1 Structure of the Chapter

This study aims to identify the relationships between aggression, aggression supportive cognitive structures, working memory problems, stress, and coping styles. In the Chapter 2 it was suggested that aggression results from enactment of aggression supportive cognitive structures and the results discussed in the Chapter 6 supported this suggestion for situational aggression. Consequently, the current study investigates the direct effect of aggression supportive cognitive structures on aggressive behaviour. Similarly, in the Chapter 3 it was suggested that stress affects aggression indirectly, and Chapters 5 and 6 supported this. Thus, the present study assesses the coping styles as potential mediators of this relationship. The results discussed in the Chapter 7 highlighted the role of pre-existent feelings of hostility and poor response inhibition to aggressive behaviour. To further this inquiry the current study also investigates the effect of hostile expectancy bias and working memory problems on aggression. Importantly, this chapter tests all of these relationships direct and indirect (i.e. mediation) in one model.

8.2 Current Study

The present study extends the findings of the research described in the Chapters 6 and 7 by testing the pathways to aggression identified with an experimental method in a wider population. Consequently, only online self-report questionnaires were used to assess the variables of interest: aggressive behaviour and traits, coping styles, experienced stress, working memory problems, and hostile attribution bias. Importantly, inclusion of hostile attribution bias is based on the study 2, highlighting the relevance of hostility. Meanwhile, the addition of coping styles reflects an attempt to find the influential third variables for stress – aggression

relationship. Since maladaptive coping has been linked with aggression (Whitman & Gottdiener, 2015), the current study takes a step further and investigates its role as a mediator for stress – aggression relationship. In order to accurately model the relationship between these constructs while taking into account the possible mediation effect, Structural Equation Modelling was used. Furthermore, after adjusting the model for the best fit to an adult sample, the final version of the model was also fit to the sample of Transitional Aged Youth (TAY). Aggression is behaviour that differs between ages (Petersen, Bates, Dodge, Lansford, & Pettit, 2015). Since the TAY represent an emerging subset of population with distinct psychological characteristics (Wilens & Rosenbaum, 2013) the attempt to confirm pathways identified with adult in the TAY sample will demonstrate whether this subset is different with regards to aggression as well.

8.3 Method

8.3.1 Participants

The total sample (n = 462) recruited online included 172 male participants and 290 female participants. Seventy five percent identified as white British, 8% identified as Asian British, 2% identified as Black British and 15% identified as "other ethnicity. They comprised two groups; adults (n = 300), who were age 26[30] and above (Mage = 36.62), recruited through the Prolific platform[31]; and the transitional age youth (TAY) group[32] (n= 162), who were between the ages of 18 and 25 (Mage = 20.48), recruited using online advertising through the University of Central Lancashire psychological research participant system and Facebook

[30] Two participants did not give correct age.

[31] Prolific is an online platform where potential participants are registered and have filled out questionnaires with socio-demographics or other basic information. Research can post an advertisement for their project and select the audience that will see it based on this information. However, the participation itself is still voluntary,

[32] TAY refers to the population merging late adolescence (ages 15 or 16) and young adulthood (ages 24 or 26) (Wilens, & Rosenbaum, 2013) .

social media platform. The resulting adult sample included 151 males and 141 females, while the TAY sample included 21 males and 141 females.

8.3.2 Materials

Short Form Buss Perry Aggression Questionnaire (BPAQ-SF) (Bryant & Smith, 2001) was used as a measure of trait aggression. The BPAQ-SF had 12 Likert scale items, with reliability (Cronbach's a = .80 for total score) and validity (Webster et al., 2014). It assesses four dimensions of aggression. Physical Aggression (Cronbach's α = .80), using items such as "Given enough provocation, I may hit another person"; Verbal Aggression (Cronbach's α = .63) – "I can't help getting into arguments when people disagree with me"; Anger (Cronbach's α = .78) – "I sometimes feel like a powder keg ready to explode"; and Hostility (Cronbach's α = .57) – "Other people always seem to get the breaks". Participants are asked to rate the extent to which the statements describe them, ranging from 1 "Very unlike me" to 5 "Very like me". In the current sample, the Cronbach's αs for subscales were similar or higher: 76 for Physical Aggression, .79 for Verbal Aggression, .76 for Anger, and .77 for Hostility.

Reactive Proactive Aggression Questionnaire (RPQ) (Raine et al., 2006) assesses aggressive behaviour. The RPQ consists of 23 items that ask participants to rate how often that have done something on a scale of 0(never) to 2 (often). The scale is reported to have good reliability (Cronbach's α = .90 for total score) and construct validity (Raine et al., 2006). It divides aggressive behaviour into: Reactive (Cronbach's α = .84) assessed by items such as "Reacted angrily when provoked by others" and Proactive (Cronbach's α = .86) assessed by items such as "Had a gang fight to be cool". In this study, the Cronbach's αs were comparable, with .88 for Proactive and .85 for reactive.

Life History of Aggression (LHA) (Coccaro et al., 1997) establishes the frequency of engaging in aggressive behaviour. It is the same scale as outlined in the Chapter 6. However,

while the LHA was developed as a semi-structured interview, in the current study it was used as a self-report questionnaire for the screening. Participants were asked to rate how often they have engaged in different types of behaviour using a scale ranging from 0 (no occurrence) to 5 (more than can be counted). The LHA aggression subscale has also been utilised as self-report questionnaire before (Coccaro et al., 2018). In the present study it also showed good internal consistency, Cronbach's $\alpha = .75$.

Criminal Attitudes to Violence Scale (CAV) (Polaschek, Collie, & Walkey, 2004) is a questionnaire designed to estimate general attitudes to crime. The CAV includes 20 Likert scale items asking participants to indicate their agreement with statements describing criminal behaviour. The answers ranged from 1(Strongly disagree) to 5 (Strongly agree). The questionnaire is reported to have good reliability (Cronbach's $\alpha = .95$) and validity (Polaschek et al., 2004). Despite the intent of the CAV's authors, Nunes, Hermann, Maimone, and Woods (2015) suggest that it is likely to measure normative beliefs about the use of aggression. Consequently, in the current study the CAV scores are treated as reflecting aggression supportive cognitive structures rather than purely attitudes. The total score on the CAV showed very high internal consistency with Cronbach's $\alpha = .93$.

Social Information Processing-Attribution and Emotional Response Questionnaire (SIP-AEQ) (Coccaro, Noblett, & McCloskey, 2009) assesses emotional and attributive responses to socially ambiguous situations with generally negative connotations. The SIP-AEQ consists of eight vignettes describing situations followed by four Likert scale questions about another possible motives for the behaviour described, and two questions about possibility of angry and aggressive reactions to such situations. For instance, vignette four is:

Imagine that you and a group of your co-workers went on a business trip. While at the hotel, waiting to meet a customer, you stop to buy a cup of coffee. Suddenly, one of your co-

workers bumps your arm and spills your coffee over your shirt. The coffee is hot and your shirt is wet

Participants are then asked to rate on a scale of 0 (not at all likely) to 3 (very likely) the likelihood of answers to this question: "Why do you think your co-worker bumped your arm making you spill your coffee?". A direct hostile intent is exemplified by item 4.1A "A1. My co-worker wanted to burn me with the hot coffee". In the current study only direct hostility items were included in the analysis as they are closer to direct aggression assessed by the LHA, BPAQ-SF, and RPQ. The subscale comprising both direct and indirect hostile attribution is reported to have good reliability (Cronbach's $\alpha = .90$) (Coccaro, Noblett, & McCloskey, 2009). Similarly, the subscale of only direct hostile intent attribution had good reliability of Cronbach's $\alpha = .73$ in the current sample.

Perceived Stress Scale 10 (PSS) (Cohen, Kamarck, & Mermelstein, 1983), comprises 10 Likert scale items, asking participants to indicate how often they have felt or were able to do something, ranging from 0 (Never) to 4 (Very often). The scale is reported to have acceptable reliability and validity with Cronbach's $\alpha > .74$ across 12 studies reviewed by Lee, 2012. In the current research participants were asked about the past month, for instance: "In the last month, how often have you felt nervous and "stressed"?". As such the total score on this scale was used to represent average perceived stress. It had acceptable Cronbach's α of .77.

List of Threatening Experiences (LTE) (Brugha & Craig, 1990; Motrico et al., 2013) identifies whether or not participants have experienced life stress events. The LTE consists of 12 yes or no items. Since the scale has been reported to have low and varying reliability with Cronbach's α ranging from .44 (Motrico et al., 2013) to .56 (Veenstra et al., 2007) the internal consistency with the obtained sample was tested and an acceptable level was shown with Cronbach's $\alpha = .69$. It is also important to note that as the LTE is a check list for life events,

the internal consistency index is not as informative as for typical scales (Motrico et al., 2013). In the current study the total score was used to estimate the level of stressful experiences in participant's life.

Brief COPE inventory (COPE-B) (Carver, 1997) identified coping styles that participants use when they encounter stressors. The COPE-B includes 28 items, measured on a Likert scale asking whether participants engage in particular responses to stressors in their life. The frequencies ranged from 0 "I have not been doing this at all" to 3 "I have been doing this a lot". The inventory assesses 14 styles of coping: Self-Distraction (e.g. "I've been turning to work or other activities to take my mind off things"); Active Coping (e.g. "I've been taking action to try to make the situation better"); Denial (e.g. "I've been saying to myself "this isn't real""); Substance Use (e.g. "I've been using alcohol or other drugs to help me get through it"); Use of Emotional Support (e.g. "I've been using alcohol or other drugs to help me get through it"); Use of Instrumental Support (e.g. "I've been trying to get advice or help from other people about what to do"); Behavioural Disengagement (e.g. "I've been giving up the attempt to cope"); Venting (e.g. "I've been expressing my negative feelings"); Positive Reframing (e.g. "I've been looking for something good in what is happening"); Planning (e.g. "I've been thinking hard about what steps to take"); Humour (e.g. "I've been making fun of the situation"); Acceptance (e.g. "I've been learning to live with it"); Religion (e.g. "I've been praying or meditating"); and Self-Blame (e.g. ".I've been blaming myself for things that happened"). COPE-B has been shown to have acceptable reliability and validity (Monzani, Steca, Greco, D'Addario, Cappelletti, & Pancani, 2015). Each scale consisted of only two items which precluded computation of reliability coefficient. However, measurement models were used to establish consistent broader subscales for the current sample.

Working Memory Questionnaire (WMQ) (Vallat-Azouvi, Pradat-Diehl, & Philippe Azouvi, 2012) assesses possible problems in the functioning of the working memory in daily

life. WMQ is comprised of 30 Likert scale items that address three components: Short-Term Storage (e.g. “Do you find it difficult to participate in a conversation with several people at once?”), Attention (e.g. “Do you feel that you are very slow to carry out your usual activities?”), and Executive Control (e.g. “Do you find it difficult to carry out a project such as choosing and organising your holidays?”). Participants could rate each item from 1 “Not at all” to 5, with a possible option of “Not Relevant” counted as 0. “Extremely” The WMQ has been shows to have good validity and reliability in both patients (Cronbach’s α = .94) and healthy participants (Cronbach’s α = .89) (Vallat-Azouvi et al., 2012). In the present sample the Cronbach’s α of Sort Term Storage subscale was .86, of Attention - .86, and of Executive Control - .83.

8.3.3 Procedure

To address the limitation of the study 2 described in Chapter 7, where most participants had low scores on LHA, the current study used a screening procedure for the adult group. First a Life History of Aggression questionnaire (LHA) (Coccaro et al., 1997) was administered to 1000 participants. From those who participated in the screening, 300 participants were invited to participate in the full study. The selection of participants to be invited was pseudo random and based on the following procedure. The participants of the screening were divided into males and females who were then ordered by their score on the LHA[33] subscale of aggressive behaviour. First participants with the upper quartile possible score on the LHA aggression subscale (19 and above, from 0 to 25 range) were identified. There was 20 men and 18 women fitting this criterion. To balance the groups 19 men with score of 19 and above on LHA aggression subscale and 19 women with score 18 and above were invited for the full study. Then to maintain the possible median score on the LHA aggression subscale (12.5, i.e. 12 is below and 13 is above) in the invited sample, 56 participants with scores 13 and above but

[33]Ranging from 0 to 25

below 19 were randomly selected. Then, to mirror the selection process but accounting for the high number of pilot participants with lower scores, 19 participants from each group with scores within the 25th percentile of the 12 (as noted it was the "median" score), i.e. 3 or below were randomly selected. Afterwards, 56 participants with scores between 4 and 12 were randomly selected from each group. As a result, 300 participants were identified for invitation to participate in the full study. In those cases, when a participant declined the invitation to participate in the full study, a different participant with matching or next closest score was invited. This sampling technique was used to recruit participants with a wide variation of scores on aggressive behaviour to avoid the overrepresentation of participants with little history of aggression. As a result, the obtained sample for adults had LHA aggression score of 6 as the 25th percentile, score of 10 as the median, score of 14 as 75th percentile, and the mean of 9.91 representing differing ranges of past history of aggression.

Participants from the adult group were paid for their time and participants in the TAY group who were Psychology students at University Central Lancashire were awarded partial course credit. The other participants of the TAY groups were volunteers whose participation was not rewarded.

8.3.4 Analysis

All statistical analyses were performed using R software version 4.0.3 (R Core Team, 2020). Before building the main model proposed in the study, measurement models including all latent variables were constructed. Given the large number of the COPE subscales, the measurement models were also used to create constructs reflecting general coping styles. Since the latent variables would be used in both adult and TAY samples the measurement models were fitted on the total sample.

Following this a model including direct and indirect pathways described in the hypotheses was tested. The total effect for each of the proposed mediation pathways was estimated as a sum of the estimated direct and indirect effect. To refine the model based on the data, the second model was built where all components of the indirect effects that were non-significant were removed. In order to determine whether indirect effects represented full or partial mediation, the direct effects from the remaining pathways that were non-significant were removed to build a comparison model. Lastly, to test applicability of the pathways identified in the adult sample to the TAY sample, the model based on the proposed hypotheses was fitted to the second sample.

The models were build using Structural Equation Modelling (SEM) via the Lavaan package (Rosseel, 2012). SEM is a statistical analysis technique allowing for a better control over direct and indirect effects than traditional regression approaches. Maximum likelihood estimator was used as the data was continuous. Moreover, the analysis utilised bootstrapping with 1000 samples to obtain standard errors and establish the Bias Corrected and Accelerated (BCA) confidence intervals for the effects shown in the models. This allowed verification that the effects, specifically the indirect effects shown by the mediation are genuinely present.

Chi-Square tests are recommended to report the goodness of fit of the SEM models. However, the value of this test is very dependent on the sample size (Shi, Lee, & Maydeu-Olivares, 2019), thus different indices were used in the current study in combination to assess the fit of the models, while the Chi-Square differences test was only utilised for model comparisons. Specifically, Comparative Fit Index (CFI) with values higher than .95, Root Mean Square Approximation (RMSEA) with values lower than .06 and the upper confidence interval lower than .08, and Standardised Root Mean Square Residual (SRMR) lower than .08 were used as indicators of good fit (Kenny, 2015; Schreiber, Nora, Stage, Barlow, & King, 2006).

There were eight missing values for the total score on the List of Threatening Experience scale. To establish whether the missing values had a significant effect, a t-test was performed. This showed no significant difference between the mean LTE total score computed by treating missing values in individual items as 0 (M=3.86) and the mean LTE total score where total missing values for all participants who has missed at least one item were removed (M = 3.89), t(913.84) = -0.16, p = .87. The difference was also non-significant within the adult sample, t(591.82) = -0.11, p = .91 and within the TAY sample, t(319.98) = -0.15, p = .88. Consequently, the LTE total score based on treating missing values as 0 was used in the analysis.

8.4 Results

Means and standard deviations for both samples are presented in the Table 8.1. Bivariate correlations for the variables included in the SEM models for adult and TAY samples are presented in the Tables 8.2 and 8.3.

Table 8.1 Means and Standard Deviation of the sample

	Adult (n = 300)		TAY (n = 162)	
Variable	*M*	*SD*	*M*	*SD*
1. Past Aggression (LHA)	9.91	5.46	8.6	5.12
2. Storage domain of WM (WMQ)	18.99	6.79	21.51	8.09
3. Attention domain of WM (WMQ)	21.11	7.15	24.06	7.68
4. Executive domain of WM (WMQ)	18.27	6.3	20.61	7.3
5. Perceived Stress (PSS-10)	18.13	7.31	22.44	6.97
6. Self distraction (COPE)	3.15	1.52	3.67	1.46
7. Active coping (COPE)	3.53	1.46	3.14	1.64
8. Denial (COPE)	0.85	1.29	1.09	1.46
9. Substance use (COPE)	1.25	1.83	1.02	1.65

10. Use of emotional support (COPE)	2.8	1.79	2.93	1.75
11. Use of instrumental support (COPE)	2.46	1.78	2.77	1.74
12.Behavioural disengagement (COPE)	1.08	1.39	1.83	1.76
13. Venting (COPE)	2.23	1.45	2.44	1.56
14. Positive reframing (COPE)	2.98	1.53	2.87	1.65
15. Planning (COPE)	3.53	1.5	3.11	1.57
16. Humour (COPE)	2.77	1.84	3.22	2.05
17. Acceptance (COPE)	3.59	1.38	3.57	1.4
18. Religion (COPE)	0.84	1.52	1.22	1.81
19. Self-blame (COPE)	2.83	1.84	3.57	1.92
20. Criminal Attitudes to Violence (CAV)	35.03	14.35	35.2	13.26
21. Physical Aggression (BPAQ)	8.19	3.64	8.22	3.62
22. Verbal Aggression (BPAQ)	7.79	2.9	8.17	3.04
23. Anger (BPAQ)	4.37	2.27	4.49	2.35
24. Hostility (BPAQ)	8.39	3.32	8.35	2.88
25.Proactive Aggression (RPQ)	1.78	3.04	1.39	2.86
26. Reactive Aggression (RPQ)	7.38	4.22	7.58	3.94
27. List of Threatening Experiences (LTE)	4.16	2.56	3.3	2.09
28. Hostile Attribution Bias (SIP-AEQ)	9.83	3.52	9.27	3.78

Table 8.2. Index of variables included in bivariate correlation analysis

1. Past Aggression	15. Planning (COPE)
2. Storage domain of WM (WMQ)	16. Humour (COPE)
3. Attention domain of WM (WMQ)	17. Acceptance (COPE)
4. Executive domain of WM (WMQ)	18. Religion (COPE)
5. Perceived Stress (PSS-10)	19. Self-blame (COPE)
6. Self distraction (COPE)	20. Criminal Attitudes to Violence (CAV)
7. Active coping (COPE)	21. Physical Aggression (BPAQ)
8. Denial (COPE)	22. Verbal Aggression (BPAQ)
9. Substance use (COPE)	23. Anger (BPAQ)
10. Use of emotional support (COPE)	24. Hostility (BPAQ)
11. Use of instrumental support (COPE)	25. Proactive Aggression (RPQ)
12. Behavioural disengagement (COPE)	26. Reactive Aggression (RPQ)
13. Venting (COPE)	27. Life Traumatic Experiences (LTE)
14. Positive reframing (COPE)	28. Hostile Attribution Bias (SIP-AEQ

Table 8.3 Bivariate Correlations between variables included in the model for adults (n=300)

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	27
2	.23**																										
3	.21**	.81**																									
4	.23**	.74**	.73**																								
5	.17**	.49**	.53**	.51**																							
6	.23**	.26**	.32**	.26**	.37**																						
7	0.07	-.18**	-.21**	-.22**	-.33**	-0.05																					
8	.18**	.30**	.29**	.32**	.29**	.21**	-0.08																				
9	.15**	.19**	.22**	.26**	.21**	.15**	-.18**	.19**																			
10	-0.04	-0.07	-0.04	-0.02	-0.07	0.02	.20**	-0.09	-0.02																		
11	0.01	-0.05	-0.01	-0.03	-0.01	0.06	.23**	-0.05	-0.06	.79**																	
12	.15*	.37**	.41**	.43**	.58**	.20**	-.30**	.40**	.20**	-.12*	-0.05																
13	.22**	.26**	.30**	.25**	.39**	.27**	0.07	.13*	0	.26**	.32**	.18**															
14	-.13*	-0.09	-.16**	-.12*	-.32**	0	.43**	0	-0.1	.29**	.30**	-.23**	0.01														
15	0.02	-0.11	-.13*	-.17**	-.21**	0.05	.67**	0	-.17**	.17**	.26**	-.16**	0.07	.46**													
16	0.03	-0.01	-0.02	0.03	-.11*	.11*	.16**	-0.04	0.05	.12*	.17**	-0.07	.12*	.26**	.13*												
17	0	-.18**	-.25**	-.24**	-.29**	0.01	.43**	-.14*	-0.05	.14*	.15**	-.17**	-0.06	.44**	.46**	.28**											

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
18	0.03	-0.02	-0.04	-0.01	-0.03	0.01	.14**	0.1	-0.09	0.09	.13*	0.02	0.11	.17**	.14*	-0.02	0.08										
19	.20**	.35**	.43**	.36**	.54**	.29**	-.17**	.25**	.14*	-0.06	0.02	.43**	.31**	-.11*	-0.04	0.01	-0.08	-0.08									
20	.43**	.18**	.17**	.14*	.15**	.16**	-0.04	.22**	.17**	-.14*	-0.11	.15**	0.11	-.21**	0.04	-0.06	-0.05	.12*	0.09								
21	.56**	.30**	.30**	.31**	.27**	.25**	-0.07	.30**	.20**	-0.08	-0.08	.22**	.28**	-.18**	0.03	-0.11	-0.07	0.05	.25**	.53**							
22	.39**	.28**	.30**	.35**	.34**	.24**	-0.03	.17**	.18**	0.02	0.02	.20**	.29**	-.17**	-0.01	0.05	-0.08	0.05	.25**	.27**	.50**						
23	.46**	.42**	.41**	.41**	.48**	.30**	-.13*	.32**	.17**	-.13*	-0.08	.36**	.38**	-.24**	-0.06	-0.08	-.15**	-0.01	.38**	.32**	.61**	.60**					
24	.36**	.47**	.43**	.42**	.57**	.33**	-.22**	.28**	.18**	-.17**	-.12*	.41**	.25**	-.21**	-0.09	-0.08	-.15**	-0.05	.44**	.29**	.48**	.49**	.63**				
25	.49**	.33**	.28**	.39**	.22**	.17**	-0.11	.24**	.27**	-0.03	-0.02	.22**	.19**	-.14*	-0.05	-0.02	-0.07	0.05	.16**	.48**	.57**	.38**	.36**	.33**			
26	.66**	.36**	.37**	.39**	.35**	.32**	-0.08	.23**	.20**	-0.07	0.01	.24**	.32**	-.22**	0.03	-0.03	-.12*	0	.32**	.51**	.69**	.54**	.65**	.51**	.65**		
27	.25**	.17**	.13*	.18**	0.11	.21**	-0.01	0.03	.20**	-0.03	-0.06	0.01	0.01	-0.06	0.06	0.03	0.08	0	0.11	.16**	.27**	.23**	.14*	.23**	.23**	.32**	
28	.26**	.26**	.26**	.25**	.28**	.17**	-0.07	0.11	0.03	-0.11	-0.06	0.11	.16**	-.12*	-0.03	-0.08	-.15**	0.03	.15**	.18**	.28**	.25**	.36**	.29**	.33**	.33**	0.11

*<0.05, **<0.01, ***<0.001

Table 8.4 Bivariate Correlations between variables included in the model for TAY (n=162)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
2	-0.02																										
3	0.04	.80**																									
4	0.1	.73**	.79**																								
5	0.06	.51**	.52**	.58**																							
6	0.08	.25**	.17*	0.14	.21**																						
7	0.15	-.31**	-.36**	-.38**	-.51**	0.01																					
8	-0.01	.23**	.26**	.30**	.20**	-0.07	-.20*																				
9	.23**	0.12	.17*	.30**	.26**	0.07	-.16*	.27**																			
10	-0.03	-0.11	-.17*	-.20**	-0.11	.16*	.17*	-0.09	-0.06																		
11	-0.07	-0.11	-.19*	-.19*	-.19*	.19*	.24**	-0.08	-0.11	.81**																	
12	0.07	.36**	.40**	.43**	.57**	0.14	-.47**	.41**	.23**	-0.13	-0.15																
13	0.07	0.09	0.09	0.13	.15*	.25**	0.02	0.15	0.11	.38**	.42**	.18*															
14	0.06	-0.07	-0.07	-0.11	-.28**	-0.01	.42**	0.01	-0.08	0.13	0.14	-0.13	-0.05														
15	0.07	-.23**	-.26**	-.29**	-.34**	0.14	.61**	-.20**	-.20*	.26**	.28**	-.34**	0.15	.38**													

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27

16	0.03	.22**	.21**	.21**	0.15	.16*	-.19*	0.14	0.14	-0.04	0.01	.25**	0.13	.20*	-0.11														
17	.22**	-0.03	0	-0.07	-0.11	.28**	.32**	-.24**	-0.1	.16*	.20*	-0.09	0.11	.16*	.37**	0.14													
18	0.06	-0.1	-0.13	-0.15	-0.04	-0.08	.21**	0.11	-0.1	-0.01	0.1	-0.06	-0.01	.21**	.20*	-0.12	0.1												
19	0.09	.31**	.34**	.35**	.53**	.27**	-.22**	.27**	.20*	0.02	-0.02	.53**	.22**	-.20**	-0.15	.21**	0.01	-0.07											
20	.28**	-0.14	-0.08	-0.04	-.16*	0.02	0.02	.25**	.21**	-0.01	0.05	0.12	0.13	0.07	0.01	0.03	.19*	.21**	-0.07										
21	.50**	0.08	0.12	0.14	0.09	0.02	0	.28**	.37**	-0.09	-0.06	.28**	0.14	-0.04	-0.05	0.11	0.14	0.09	0.12	.47**									
22	.27**	0.03	0.14	0.11	0.07	0.11	-0.14	0.1	.19*	-0.01	-0.04	0.15	.17*	-0.13	-0.09	0.14	.17*	-0.08	0.12	.32**	.44**								
23	.37**	.18*	.32**	.34**	.34**	0.03	-.17*	.29**	.42**	-0.1	-0.15	.39**	0.14	-0.06	-.26**	0.13	0.01	0.06	.26**	.36**	.70**	.43**							
24	.21**	.23**	.29**	.34**	.42**	0.14	-.31**	.24**	.32**	-0.14	-.19*	.38**	0.15	-.20**	-.24**	0.15	-0.06	-0.09	.37**	.23**	.49**	.33**	.63**						
25	.27**	0.03	0	0.05	-0.09	0.04	0	.18*	.28**	-0.01	-0.01	0.05	0.02	0.1	-0.09	0.04	0.01	0.14	-0.01	.52**	.36**	.16*	.30**	.18*					
26	.56**	.16*	.23**	.24**	.22**	0.12	-0.13	0.12	.32**	-0.06	-0.12	.22**	0.15	-0.06	-0.14	0.04	0.13	-0.06	.23**	.34**	.56**	.40**	.56**	.38**	.44**				
27	.33**	0.09	0.12	.23**	.15*	0.06	-0.12	0.1	.32**	-0.1	-.16*	0.14	0.1	-0.05	-0.13	.16*	0.01	-.27**	0.11	0.03	.28**	.23**	.33**	.32**	.26**	.35**			
28	-0.1	.31**	.31**	.25**	.31**	0.1	-.34**	0.11	0.14	0.01	-0.04	.20*	0	-.21**	-.26**	0.12	-0.03	-0.12	0.15	0	0	0.02	0.13	.18*	0.04	0.13	0.07		

*<0.05, **<0.01, ***<0.001

8.4.1 Measurement Models

The first step of the analysis was construction of the measurement model with the latent variables. The first variable was named Trait Aggression and comprised three subscales of the BPAQ-SF: anger, hostility, and verbal aggression. Meanwhile, the second construct was titled Behavioural Aggression and included the both subscales of the RPQ, aggression subscale of the LHA, and physical aggression subscale of the BPAQ-SF. The aggression construct was separated into two, to reflect the different wording in the (sub)scales; while the BPAQ-SF asks participants to indicate the extent to which the items describe them (e.g. item 12 "I sometimes feel like a powder keg ready to explode"), only the physical aggression subscale included items describing acts of aggression (e.g. item 1 "Given enough provocation, I may hit another person"). Such wording made it closer to the items from the RPQ and LHA aggression subscale that asks participants to judge how often they engage in a particular behaviour. Moreover, previous research indicated that behavioural acts and trait aggression measures are not identical (Archer & Webb, 2006).

The third latent variable reflected three subscales of the WMQ and was titled working memory problems. The last latent variable titled coping styles was used to represent the Brief COPE questionnaire. This model did not fit the data well, CFI = .7, RMSEA = .11 [.10, 11], SRMR = .12. However, this was expected as the COPE has 14 subscales that assess specific strategies people engage in when confronted with problems. It was hypothesised that these strategies should be unified by two broad categories: Adaptive and Maladaptive coping. The first measurement model was used to as guide to establish the possible components of maladaptive coping. Inspection of the individual loadings showed there were five items with negative loadings (see Figure 8.1). Specifically, items: Self Distraction, Denial, Substance use, Self-Blame, and Behavioural Distraction. Given that they share the negative connotation, a second measurement model with these variables separated to represent maladaptive coping and

the other coping variables to represent an adaptive approach. No other alterations were made as the latent variables representing two types of aggression and problems with working memory showed good loadings with similar direction across individual subscales.

The new model had significantly improved fit, ($\chi 2$ (4) = 365.80, $p < 0.001$. However, the total fit was still poor, CFI =. 79, RMSEA =. 09 [.09, .10], SRMR =. 09. Inspection of the individual loading on the variable titled Adaptive Coping showed there were five items with standardised loadings below .5 (see Figure 8.2). These items were: Venting, Instrumental Support, Emotional Support, Humour and Religion. Given that the loadings of first three of these items were also non-significant, it was decided to explore the possibility of a third coping construct reflecting use of support. It is important to note that the in brief COPE venting is measured by two items: "(9). I've been saying things to let my unpleasant feelings escape" and "(21). I've been expressing my negative feelings". Both suggest interaction with others, which allows them to be seen as a use of support, albeit a negative use of a support network. Similarly, the Humour coping is described by two items: "(18). I've been making jokes about it" and "(28). I've been making fun of the situation", both of which imply interaction with others. Although religious coping had low standardised loading (2, $p = .001$), the items assessing it do not suggest engagement with religious community or seeking support ("22. I've been trying to find comfort in my religion or spiritual beliefs", "27. I've been praying or meditating"). Consequently, the new latent variable representing coping through support did not include it and was restricted to coping approaches that engage with others. The individual standardised loadings on other factors were also assessed, however there was no indication that alterations were needed.

The resulting measurement model with three coping styles was a significant improvement over the second model, $\chi 2$ (5) = 499.51, $p < 0.001$. However, the overall fit was only borderline acceptable, CFI = .90, RMSEA = 06 [.06, .07], SRMR = .08. Using

modification indices to inspect the possible modifications to the model (see Figure 8.3)., two alterations were made. First, venting as a coping strategy was allowed to load on maladaptive coping as well as support coping. The reasoning for this was that expression of negative feelings can have different connotations. In the context of aggressive behaviour, venting negative emotions through threats or using anger would represent an example of maladaptive coping, while expression of negative emotions within support networks is likely to have different consequences. The second alteration to the third measurement model was the addition of covariance between humour as coping and positive reframing. This was accounted for by the similarity between the items assessing these two approaches. Specifically, item (12) "I've been trying to see it in a different light, to make it seem more positive", representing positive reframing, also resembles items 18 and 28 that reflect Humour. The specified fourth measurement model was a significant improvement over the previous one, $\chi 2$ (2) = 96.80, p < 0.001, although the fit was still borderline acceptable, CFI = .92, RMSEA = .06 [.05, .06], SRMR = .06, this model was retained. Furthermore, the alterations made were shown to be worthwhile, as venting showed almost comparable standardised loadings on both maladaptive coping (.40, $p < .001$) and support coping (.39, $p < .001$). Similarly, the covariance between positive reframing and humour was significant (.25, $p = < .001$) (see Figure 8.4).

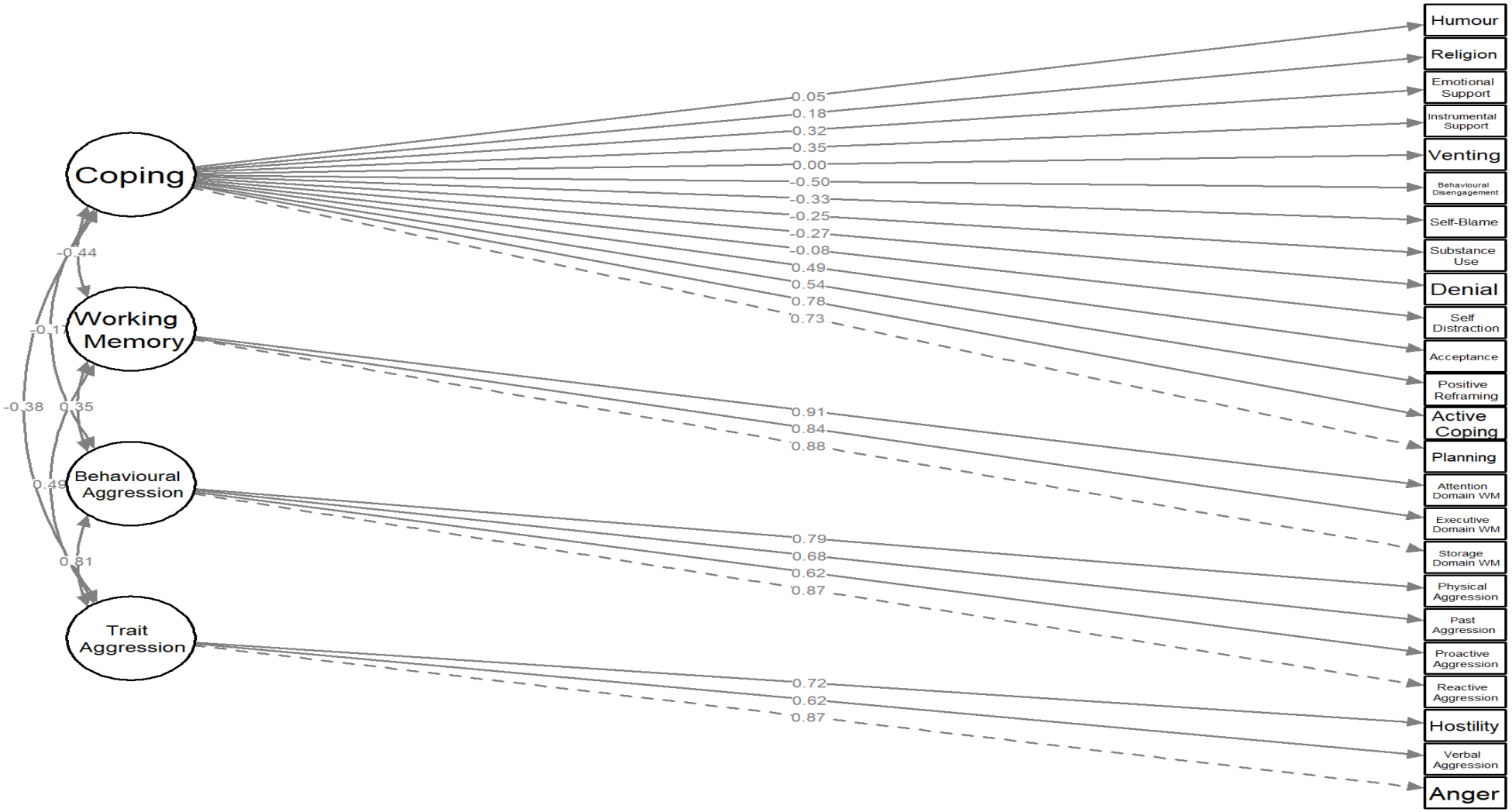

Figure 8.1 First measurement model

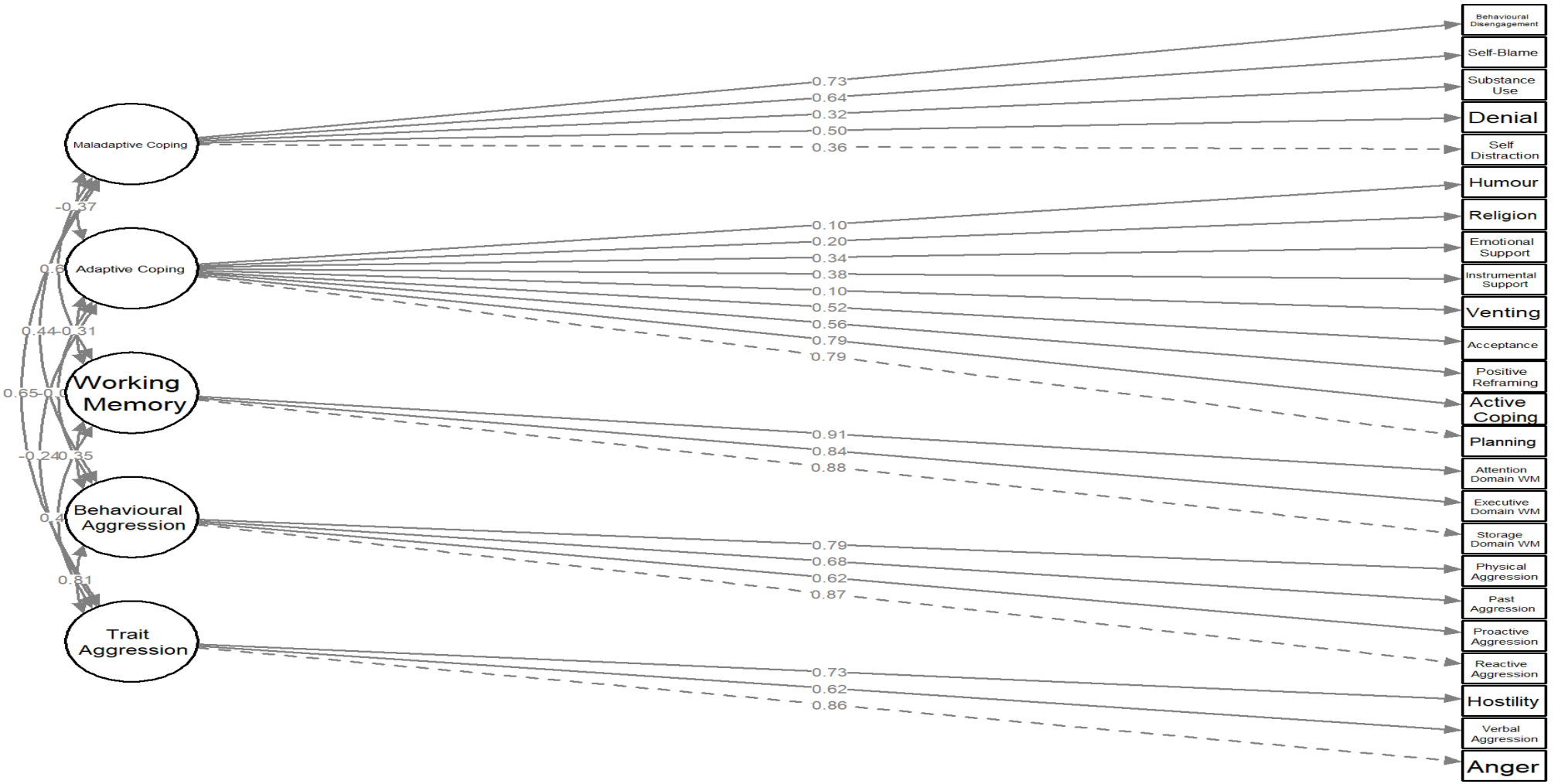

Figure 8.2 Second measurement model

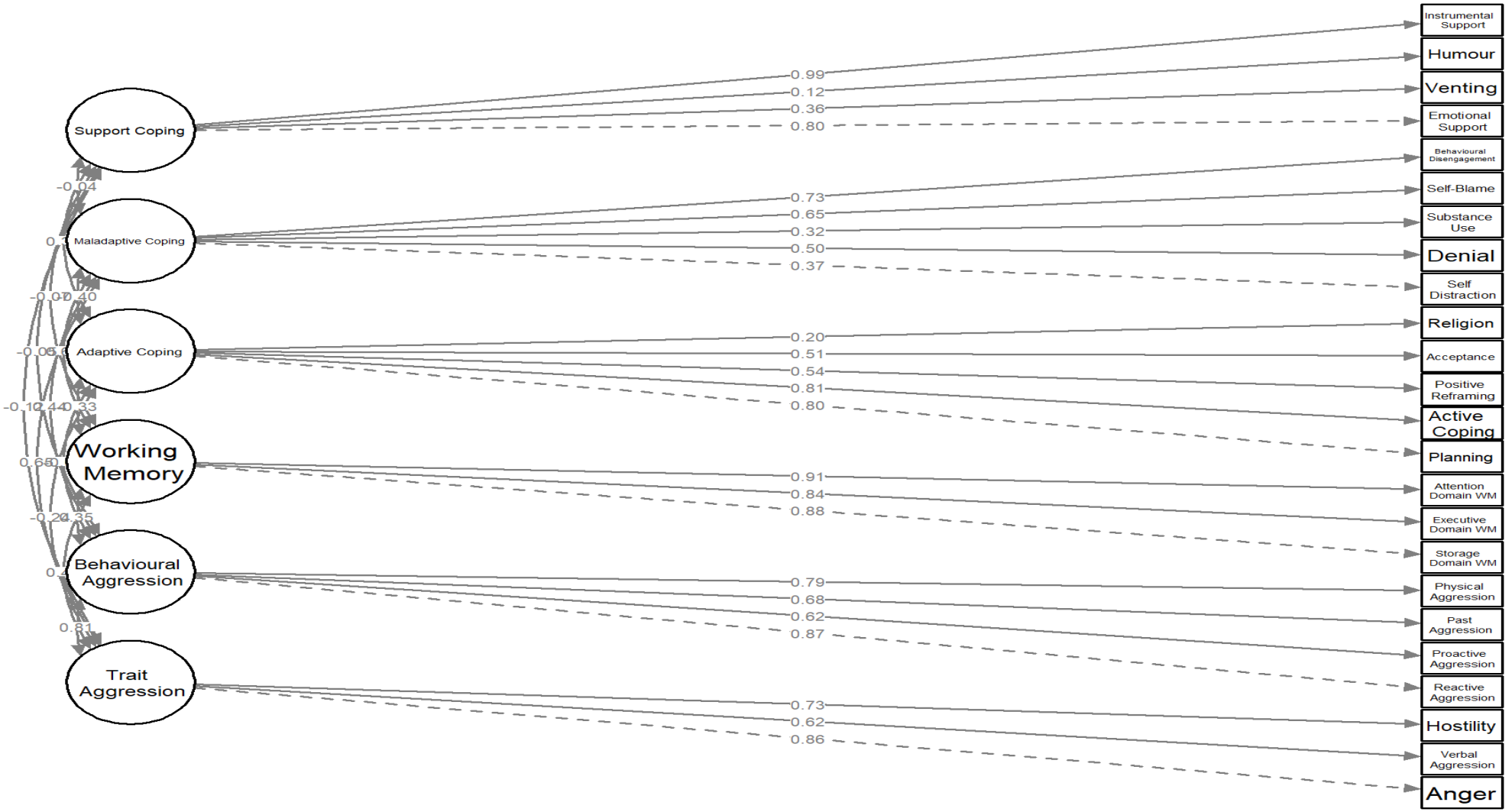

Figure 8.3 Third measurement model

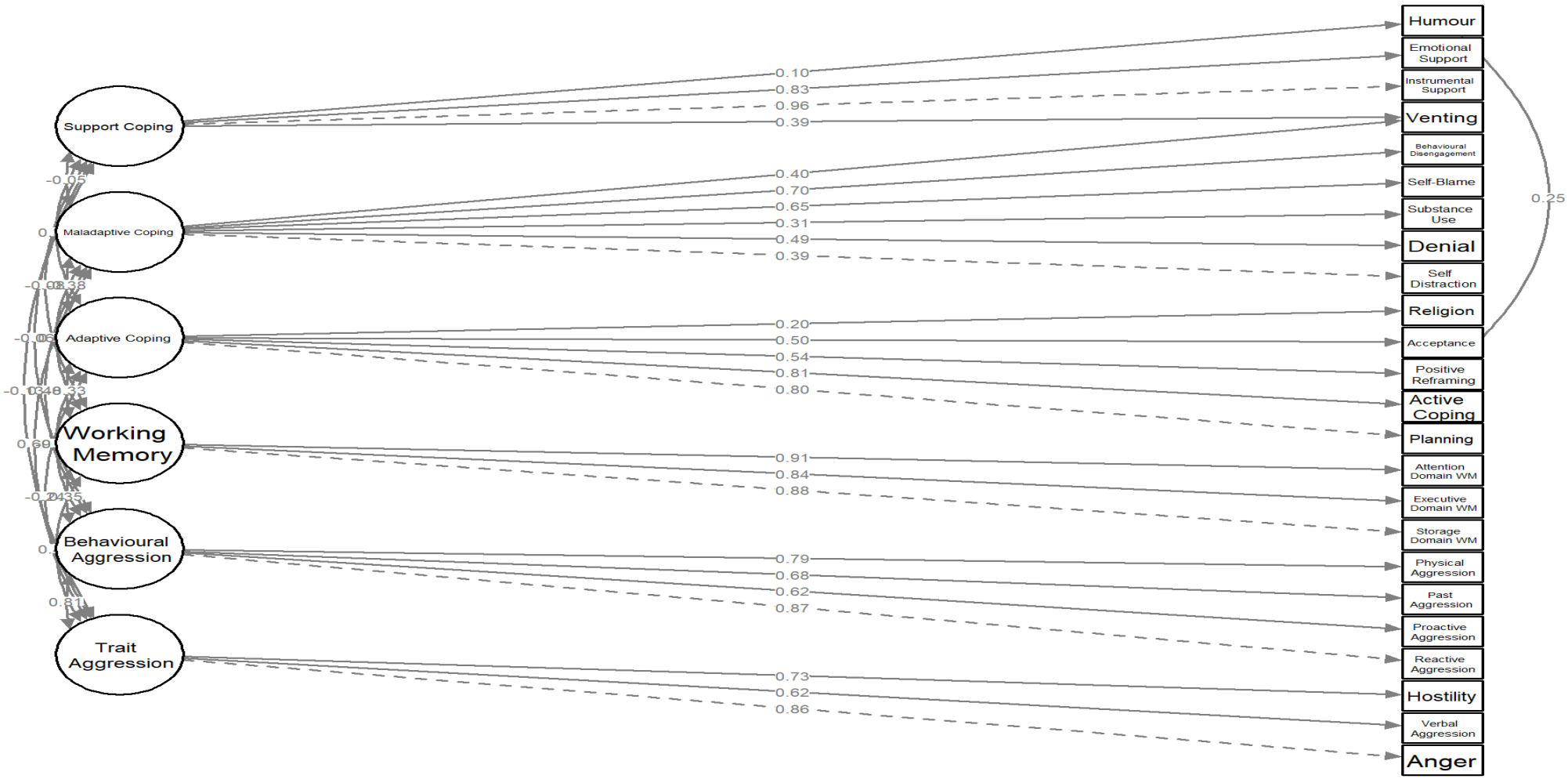

Figure 8.4 Fourth measurement model

Establishing a measurement model with acceptable fit allowed to use the identified latent variables in the full mediation model. It was guided by the predictions outlined in Chapter 5:

3.1. The effect of perceived stress on aggression will be mediated by adaptive and maladaptive coping styles.

3.2. The effect of the hostile attribution bias on aggressive behaviour and traits will be mediated by the criminal attitudes to violence.

3.3. The effect of the life stressors on aggressive traits will be mediated by the aggressive behaviour.

3.4. The effect of hostile attribution bias on aggressive behaviour will be mediated by working memory problems.

3.5. The coping styles that modulate the experienced stress will be associated with working memory problems, as stress has been shown to affect working memory.

8.4.2 Mediation Models

Using the latent variables identified in the measurement model, an SEM model with multiple parallel mediations based on the proposed hypotheses was built. The covariance between coping styles, as well as between positive reframing and humour were retained. The resulting Model 1 showed borderline fit, CFI = .91, RMSEA =.06 [.05, .06], SRMR = .07. Table A3.1 in Appendix 3 shows the weights in the saturated model (M1). To establish whether the indirect effect shown as negative are genuinely absent, the saturated model was compared with the model where the indirect non-significant paths were dropped. Specifically, paths from adaptive coping to aggressive traits (b = .02, [-.16, 22], $p > .05$) and to aggressive behaviour (b = - .013, [-.55, .27], $p > .05$) were removed but the path from perceived stress to adaptive coping was retained (b = - .06, [-.08, -.04], $p < .001$). Meanwhile, for support based coping all segments of the indirect effect were removed: the path from perceived stress to support coping (b = -.01, [- .04, .02], $p > .05$), and the paths from support coping to aggressive traits (b = -.09,

[-.21, .04], $p > .05$) and behaviour (b = .20, [-.06, .49], $p > .05$). Lastly, the path from criminal attitudes to violence to aggressive traits (b = -.02, [-.03, .003], $p > .05$) was removed.

The Chi-Square difference tests showed no significant distinction between the models suggesting that refined version can be retained, $\chi 2$ (6) = 8.89, p = .18. Indeed, Model 2 (Table A3.2 in Appendix 3) had similar fit, CFI = 0.92, RMSEA = 0.06 [0.05, 0.06], SRMR = 0.07. Afterwards to establish the extent of the mediation the refined model was tested against the model without the direct paths that were non-significant. The new model excluded: the path from perceived stress to aggressive traits (b = .01, [-.08, .07], $p > .05$); from life stressful events to aggressive traits (b = -.02, [-.10, .06], $p > .05$), and from working memory problems to aggressive behaviour (b = .01, [-.12, .12], $p > .05$).

The Chi-Square difference test showed that Models 2 and 3 did not differ significantly, $\chi 2$ (3) = .38, p = .95, suggesting that these direct effects paths do not need to be retained in the model. The resulting Model 3 had borderline acceptable fit, CFI = .92, RMSEA = .05 [0.5, .06], SRMR = .07. Next, Model 3 was compared to the model without the direct effect between perceived stress and aggressive behaviour (b = - .21, [-. 41, - .10], $p < .01$). The Chi-Square difference test showed that models differed significantly, $\chi 2$ (1) = 12.72, p < .001, suggesting that this effect cannot be removed. The only direct effect that could not be tested by model comparison corresponded to the path between direct hostility and behavioural aggression, as it is part of the indirect effect of working memory problems on behavioural aggression. Consequently, the Model 3 was adopted as the final model for the adult sample (Table A3.3 in Appendix 3 and Figure 8.5).

Several indirect effects were present in Model 3 (all significant mediation effects are presented in the Figure 8.6). The effect of perceived stress in last month on both aggressive traits (b = 0.11, [.08, .14], $p <$. 001) and behaviour (b = .31 [.18, 49], $p < .001$) was indirect.

However, full mediation was present only for the aggressive traits, as the removal of this direct effect did not alter the model fit. Meanwhile, the direct effect between perceived stress and aggressive behaviour could not be removed without significantly affecting the model fit, suggesting that for these variables the mediation was partial. Moreover, this direct effect was negative (b = -0.21, [-0.41, -0.10], $p < .01$), suggesting that low levels of perceived stress are associated with an increased frequency of aggressive behaviour. It is important to note that as seen in Table 8.6, the standardised estimate total effect for the path from perceived stress to behavioural aggression through maladaptive coping is non-significant as its 95% CI includes 0 (std. b = 0.22, [-0.12, 0.55], $p > .05$). Meanwhile, the standardized estimate for this indirect effect is significant, as its 95% CI does not include 0 (std. b = 0.64, [0.23, 1.05], $p < .01$), suggesting that the mediation is likely to be full. However, taking into account the significant non standardised total effect (b = 0.11, [0.06, 0.16], $p < .001$) and significant alteration of the model fit when the direct path was excluded, the mediation of the relationship between perceived stress and aggressive behaviour by increased maladaptive coping within this study is considered partial.

The other coping styles did not mediate the effect of perceived stress on aggressive traits and behaviour. However, perceived stress was positively associated with maladaptive coping (b = .08 [.06, .10], $p < .001$) and negative associated with adaptive coping (b = -.06 [-.08,.-.04], $p < .001$). Only maladaptive coping was significantly associated with working memory problems (b = 6.14 [4.54, 8.59], $p < .001$).

Meanwhile, hostile attribution tendency was shown to have an indirect effect, through criminal attitudes to violence[34], only on aggressive behaviour (b = 0.09, [.03, .16], $p < .01$), but

[34] Although the scale name appears to be focus on attitudes to crime, as elaborated in the methods section, it in fact, assess aggression supportive cognitions.

not on aggressive traits, as removal of the indirect pathway for the latter did not alter the fit of the model. As the direct effect of hostile attribution tendency could not be removed from the model and was significant, b = .20 [.10, .30], $p < .001$, the criminal attitudes to violence were shown to be a partial mediator. Moreover, hostile attribution tendency was the route through which the indirect effect of working memory problems on aggressive behaviour operated, b = .04 [.02, .06]. $p < .01$. This showed that problems with working memory facilitate aggressive conduct, when an individual has expectations that others have hostile motives. Interestingly, criminal attitudes to violence were not associated with aggressive traits as removal of the path between them did not alter the model fit. Similarly, the hostile attribution tendency had a non-significant relationship with aggressive traits, b = .05 [-.01, .10], $p > .05$. However, aggressive behaviour was a full mediator of the effect that stressful life events have on aggressive traits, b = .08 [.04, .12], $p < .001$ since the direct effect was removed without affecting the overall model. This demonstrated that traits are formed through behaviour. Repetition of specific acts solidifies corresponding personality traits.

To compare the TAY and Adult sample, the saturated model was also applied to the TAY sample. The resulting fit was poor, CFI = .85, RMSEA = .07 [.06, .08], SRMR = .09. When following the same procedure as outlined above, the non-significant indirect paths were removed, Chi-square difference test indicated a significant change to the model fit, $\chi 2$ (9) = 55.10, $p < 0.001$. This suggested that the saturated model needs to be retained. However, its poor fit indicated that it is not applicable. This means that the pathways facilitating aggressive

acts and forming aggressive traits in adults are different from those in the transitional aged youth.

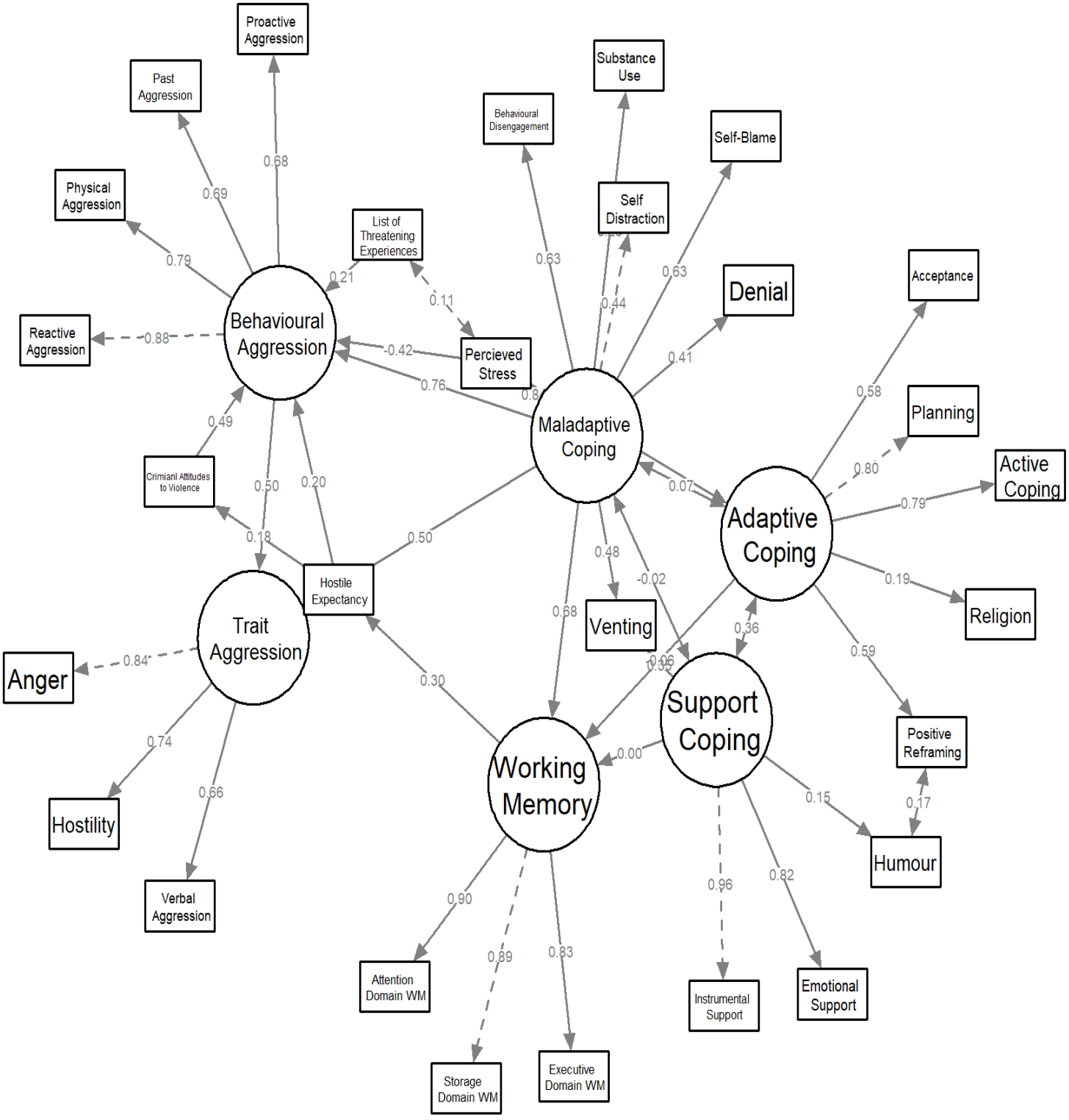

Figure 8.5 Model 3: Total direct and indirect effects of the model for adults (n = 300)

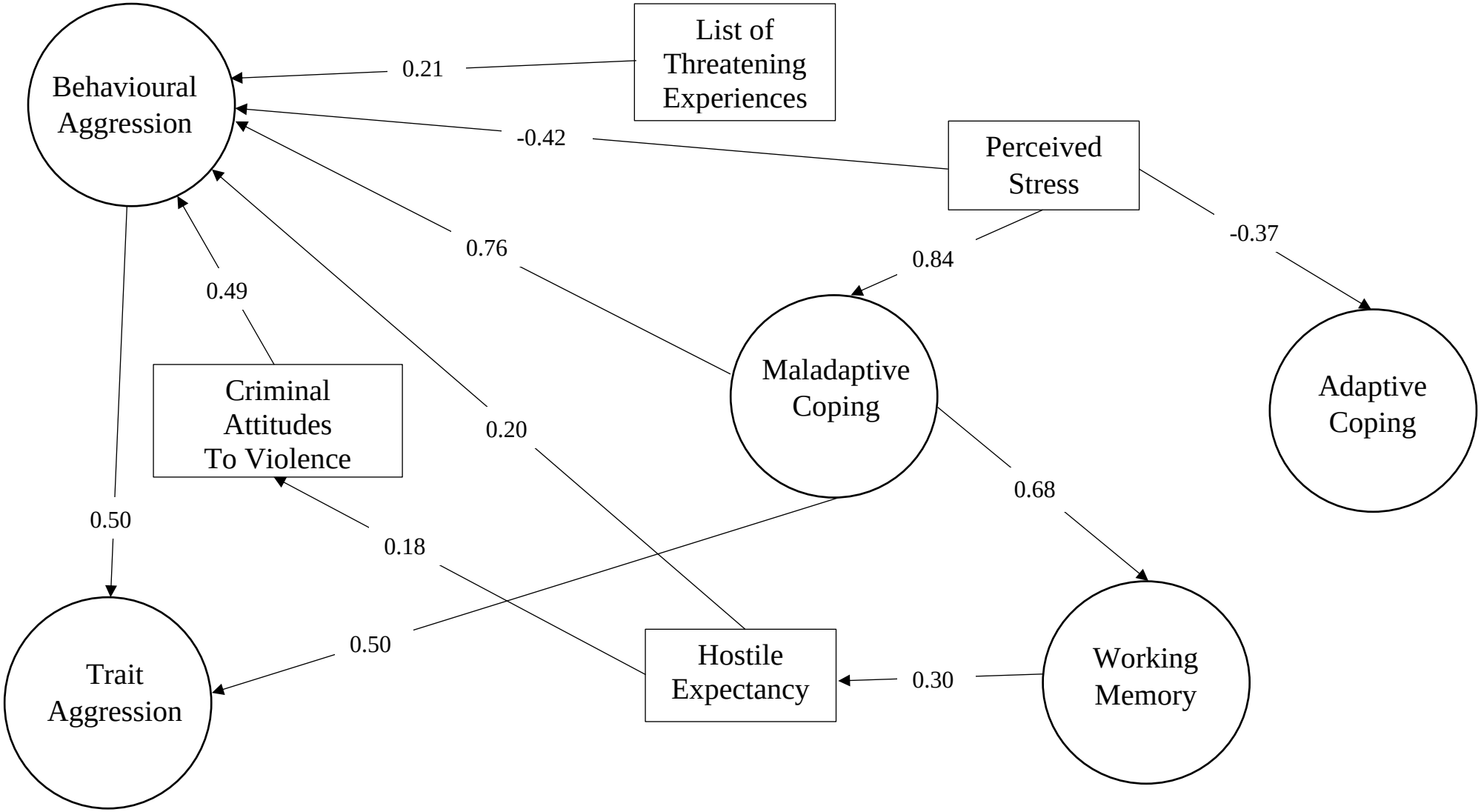

Figure 8.6 Only significant paths of Model 3

8.5 Discussion

The current study showed that aggressive behaviour is directly influenced by aggression supportive cognitive structures and indirectly by experienced stress. The results of the study supported the existence of two main pathways to aggression. The first pathway, originating in perceived stress, facilitated aggressive behaviour and aggressive traits through maladaptive coping. This highlighted not only an indirect relationship between stress and aggression but also the importance of the habitual reaction to the situation in guiding the behaviour. The latter was also reinforced by the indirect effect of life stressors on aggressive traits through aggressive behaviour, as it exemplified the entrenchment of behaviour in traits. However, the inverse direct path between perceived stress and aggressive behaviour showed that there are likely further modulators of this relationship. The second cognitive pathway demonstrated the relevance of cognitive structures in guiding conduct. Poor working memory functioning was related to the tendency to attribute hostile intentions to actions of others, which in turn, through the existence of violence supportive cognitions, facilitated aggressive behaviour. Importantly this pathway was only present for behaviour but not the traits, thereby suggesting that cognitions outline conduct.

However, the borderline acceptable fit of the refined model for the adult sample suggested that current results can be interpreted but with caution. In other words, they represent a preliminary model identifying primary and secondary factors facilitating aggression. Nevertheless, the expected cognitive and stress pathways to aggression were present in the results.

The results partially confirmed prediction 3.1 stating that the effect of perceived stress on aggression will be mediated by adaptive and maladaptive coping styles. The analysis has

demonstrated that perceived stress in the past month exerts an indirect effect on aggressive behaviour *and* aggressive traits. This effect was partially mediated only by increased maladaptive coping that included such responses to encountered problems as self-distraction, denial, substance use, self-blame, behaviour distraction, and venting, particularly venting not to a support network. Increased perceived stress was associated with increased maladaptive coping, which in turn was associated with increased aggressive behaviour and aggressive traits. Although the mediation effect was through positive associations between all three variables, the direct path from perceived stress to aggressive behaviour was negative. This suggests that those who experienced low stress in the past months demonstrate increased behavioural aggression. Contrary to prediction 3.1, adaptive coping did not mediate the effect of perceived stress on aggression. Although stress facilitated lower engagement with adaptive approaches to coping, they were not associated with aggressive behaviour or traits, highlighting the importance of maladaptive coping only. These results are similar to those reported by Gardner, Archer, & Jackson, (2012), who reported that maladaptive coping mediates the relationship between borderline personality traits and aggression. The current results extend the mediating role of maladaptive coping to perceived stress. Another point of distinction was that while Gardner and colleagues (2012) utilised reactive and proactive aggression in their study separately, the present study used unified constructs of aggressive behaviour (i.e. both reactive and proactive aggression as one construct) and aggressive traits. This suggests a possibility that both reactive and proactive of aggression can be facilitated by similar mechanisms. Hence the results provide an additional argument in favour of Bushman and Anderson's (2001) proposition that proactive and reactive aggression may not be utterly separate and that they can co-exist in an individual, with similar mechanisms contributing to both motivations. In addition, Blair (2016) also noted that impairment in vmPFC, resulting in valuing outcomes of aggressive behaviour, can advance both types of aggression. Since the same variable can affect

both forms of aggression, they in some cases they can be analysed as a unitary construct. Furthermore, the results for the mediating role of maladaptive coping were in agreement with the results of Carlo, Mestre, McGinley, Samper, Tur, & Sandman, (2012) who had shown that increased emotional coping, which included avoidance and counter-productive approaches, mediates the relationship between emotional instability and aggression. Similarly, the intermediary role of maladaptive coping agreed with the review by Roberton, Daffern, and Bucks, (2012) highlighting the association between poor emotion regulation and aggression. These findings also correspond to the conclusions made in the Chapters 3 and 5 stating that the effect of stress on aggression is contingent on an extraneous third variable. Using the I^3 terminology this result places maladaptive coping style as an impellent of aggressive behaviour in stressful situations.

The refined model also confirmed prediction 3.3 stating that the effect of the life stressors on aggressive traits will be mediated by aggressive behaviour. Stress evoking experiences were shown to facilitate aggressive behaviour, which in turn could reinforce aggressive traits. The mechanism of repetitive acts informing individual traits partly corresponded to the socio-cognitive models such as General Aggression Model (GAM) (Anderson & Bushman 2002) and Integrated Information Processing Model (IIPM) (Huesmann, 2018) that place emphasis on learning behavioural scripts. However, contrary to the importance placed by these models on observational learning, the present study highlighted the role of learning from personal experience (i.e. from behaviour) with the association of traits in adulthood. Furthermore, the findings were in partial agreement with previous research showing a positive association between reactive aggression and experience of stressful events (Brown, Fite, DiPierro, & Bortolato, 2017). However, opposite to Brown and colleagues (2017) the current study used a unidimensional construct of aggression suggesting that the relationship is present for proactive *and* reactive aggression. Taking into account the indirect effect of perceived stress on

aggressive behaviour and aggressive traits, it could be argued that the association between experiencing stressful events would be more pronounced among those who use maladaptive rather than adaptive or support based coping.

Overall, the refined models showed that the pathway from stress to aggressive traits is unlikely to be a direct one. This reflects the findings of Sprague, Verona, Kalkhoff, and Kilmer, (2011), who demonstrated that the effect of perceived stress on aggressive traits is moderated by executive functions. The current study adds maladaptive coping and aggressive behaviour as the indirect routes. Although, for aggressive behaviour indirect effect was also present, the mediation was partial as removal of the direct effect altered the model fit. Moreover, contrary to the positive indirect path, the direct one was negative. Given that the two other coping styles did not mediate this relationship, it is likely that further variables can affect the association between stress and aggression.

The other major pathway in the study was cognitive. The refined model showed hostile attribution tendency to be a full mediator for the relationship between working memory problems and aggressive behaviour. This confirmed prediction 3.4 positing that the effect of working memory problems on aggressive behaviour will be mediated by hostile attribution bias. Impairments in daily working memory functioning were shown to increase the tendency to attribute hostile intention to the behaviour of others, which in turn facilitated aggressive conduct. This suggests that poor information processing is related to aggression in cases when there already is a potential for it, in this case hostile interpretations. In line with this suggestion, Model 3 showed that part of the effect of hostile attribution tendency can be explained by criminal attitudes to violence. Prediction 3.2, stating the effect of the hostile attribution bias on aggressive behaviour and traits will be mediated by the criminal attitudes to violence, was confirmed. The results have demonstrated an indirect positive effect of hostile attribution tendency via increased criminal attitudes to violence on aggressive behaviour but not on

aggressive traits. The consistent relationship between hostile attribution tendency and aggressive behaviour reflects existing research (Martinelli, Ackermann, Bernhard, Freitag, & Schwenck, 2018; Tuente, Bogaerts, & Veling, 2019, Quan et al., 2019), and extends it by highlighting the role violence related cognitions. Specifically, interpreting the behaviours of others as hostile is more likely to facilitate aggression among those with higher number of aggression supportive cognitive structures than among those who have fewer of them. Taken together these findings support the sociocognitive models of aggression (Anderson & Bushman 2002, Huesmann, 1988; 1998; 2016) as they demonstrate that information processing capacities, operationalised using the working memory problems, are related to aggressive behaviour via aggression supportive cognitions or biases. Possibly this can be explained as low working memory functioning limiting the pool of available behavioural scripts for a given situation, and when aggression supportive cognitions are present, they represent the majority of available choices. Within the I^3 framework these findings place aggression supportive cognitions and hostile expectancy bias as impellance processes that can amplify proclivity to aggress independently of each other (Finkel & Hall, 2018). However, when considering that aggressions supportive cognitive structures partially mediate the contribution of hostile attribution bias to aggression, these structures can also be considered as reverse inhibitors.

Although the criminal attitudes to violence scale was designed to assess primarily attitudes (Polaschek et al, 2004), Nunes and colleagues (2015), suggested that it measures normative beliefs. The current study supports the latter proposition. There was no indirect effect of hostile attribution tendency on aggressive traits through criminal attitudes to violence, as there was no association between the latter two variables. Instead, only a direct effect was present. Aggressive traits were composed from scales assessing individual differences in aggression-associated domains (Webster et al., 2014) rather than simply instances of such behaviour. This suggests that items in the CAV describe conduct viewed as acceptable or

unacceptable, which is closer to Ajzen's (1991) conceptualisation of normative beliefs, than to personal evaluations (i.e. attitudes).

The results also partially confirmed prediction 3.5 stating that the coping styles that modulate the experienced stress will be associated with working memory problems. Model 3 showed that only maladaptive coping was positively associated with problems in daily working memory functioning. While the current study used self-report measures for both perceived stress and for working memory functioning, the findings are supported by previous research. An acute experience of stress has been shown to negatively affect executive functioning, including working memory (Shields et al., 2016). The results showing an association only for maladaptive coping style but not for adaptive or support based approaches to problems suggest that the former does not decrease the experienced stress, which in turn leads to poor working memory functioning in daily life. Indeed, previous research has shown that stressful experiences in the past two weeks are associated with poor working memory performance (Shields, Doty, Shields, Gower, Slavich, & Yonelinas, 2017).

While the poor fit of the refined model to the TAY sample precludes assessing the specific pathways identified in the adult sample, it also suggests a potential difference. In other words, the variables exerting effects on aggressive behaviour and traits in the adult sample do not necessarily function similarly in the TAY sample. This suggests the presence of distinct pathways of mechanisms facilitating aggression in this population. The result falls in line with previous research indicating distinct features of the TAY in terms of cognitive and social skills (Wilens & Rosenbaum, 2013), and the differences in aggressive behaviour between age groups (Petersen, Bates, Dodge, Lansford, & Pettit, 2015). However, a more detailed group comparison is needed to confirm this finding. A larger sample of the TAY is required in order to adjust the model to it.

8.6 Limitations

The current study had a number of limitations, which need to be acknowledged. The paths identified in the adult sample need to be interpreted with caution. Firstly, borderline acceptable fit suggests an imprecise match between data and the hypothesised variance-covariance matrix. This might be accounted for by the moderate number of participants with respect to the number of items included in the analysis (Cheung, & Rensvold, 2002), or by the poor association between adaptive and support based coping with other variables in the analysis. However, while the indices of the refined model were borderline acceptable[35] the combination of the RMSEA and SRMR were within the reasonable parameters for error rates (Bu & Bentler, 1990). Furthermore, to avoid overfitting the model, the distinction between adult and TAY sample was ended after it was established but without investigation into the specifics. In addition, the cross-sectional design of the study restricts the inferences about causality of the identified pathways, with a use of questionnaires constraining results to reports about behaviour or cognitive processes. However, as this was an exploratory analysis of the proposed model, further replications with an experimental or longitudinal design would be able to disprove or confirm, as well as specify the identified associations between the variables.

[35] i.e. CFI > .95, RMSEA < .06, SRMR < .08

CHAPTER NINE: GENERAL DISCUSSION

9.1 Recap of Findings

The current set of studies have shown the complex nature of the relationship between cognitive structures and processes, stress and aggression. Based on existing sociocognitive and neurocognitive models of aggression, two primary factors and one secondary factor explaining aggressive behaviour were derived. First, aggressive behaviour is enactment of cognitions supporting aggression (Huesmann, 1988; 1998; 2016). Second, aggression results from decision-making, where aggression supportive cognitions were chosen as an appropriate guide for action (Anderson & Bushman, 2002; Blair, 2004; 2016; Crick & Dodge, 1994). Furthermore, stress affects memory, where such cognitions are stored, by enhancing encoding yet impeding the retrieval of information (Wolf, 2009; Schwabe, Joëls, Roozendaal, Wolf, & Oitzl, 2012). Stress also alters decision-making process by prioritising short-terms rewards over costs (Starcke & Brand, 2012; 2016). Thus, in the Chapter 3 it was proposed that stress is another guiding, but *secondary*, factor influencing aggressive behaviour.

The systematic literature review demonstrated that there most likely is a relationship between stress and aggression. However, the results of the studies that used similar measurements for stress response markers and aggression varied between studies. Consequently, it was suggested that the stress-aggression relationship was indirect, with a thematic analysis of the studies utilised to identify the influential modifiers. Nevertheless, from the resulting six themes, only one identified a consistent pattern, highlighting sex as a possible moderator. Specifically, acute activation of the Sympathomedullary Pathway (SAM) was shown to facilitate aggressive behaviour among males, but to decrease it among females (Verona & Curtin, 2006; Verona & Kilmer, 2007; Verona et al., 2006). However, the same distinction was not consistently present in studies assessing acute activation of the Hypothalamic-Pituitary-Adrenal (HPA) axis (Böhnke et al., 2010b; Mehta et al., 2017; Prasad

et al., 2017). Moreover, a number of studies showed a lack of significant effect of sex on the relationship between stress markers and reported aggression (Armstrong & Boutwell, 2012; Feinberg et al., 2011; Hagan et al., 2014; Murray-Close et al., 2017; Rausch et al., 2015; Ruiz-Robledillo & Moya-Albiol, 2015; Schwartz & Portnoy; 2017; Steptoe et al., 1996). Thus, despite strongly suggesting that the stress-aggression relationship is contingent on a third variable(s), the systematic literature review could not identify them with consistency. Given that the relationship was still reported as present by the majority of the studies included in the systematic literature review, Studies 2 and 3 attempted to identify the relevant third variable(s).

The results of the Study 1 suggested that even within a male sample, acute activation of the SAM system, operationalised as an increase in skin conductance level (SCL), does not always facilitate aggressive behaviour. This effect was only present for the patients of a high secure hospital, but not for university students. Importantly, however, this is a preliminary result from a small scale study. Yet, such variation in the stress-aggression relationship within sex extends the results of Verona et al. (2006), who demonstrated that stress facilitated aggressive behaviour only among men with a homozygous short genotype. In other words, participants' sex was shown to be an inconsistent intermediary for the stress aggression relationship on its own. Consequently, in the ensuing studies, male and female participants were analysed together.

Study 1 established the possible basic pathways to aggressive behaviour in a specific situation. The duration and intensity of an unpleasant noise delivered to an "opponent" in the first trial of a competitive reaction time task was selected as the operationalisation of such conduct. Since it was the first trial, the participants' actions were not influenced by the behaviour of the "opponent". Instead, following stress exposure, they showed aggressive behaviour towards a previously unencountered person who was not responsible for the stressor (i.e., "stranger"). Given this nature of aggression, its significant explanation by a core cognition

representing the right to decide to whom and when harm can be inflicted (i.e., Implicit Theory "I am the law") is understandable. Furthermore, it exemplifies the proposition of socio-cognitive models that aggressive conduct is an enactment of aggression supportive cognitive structures (Anderson & Bushman, 2002; Huesmann, 2016). Figure 9.1[36] presents the pathways to aggression suggested by the results of Study 1.

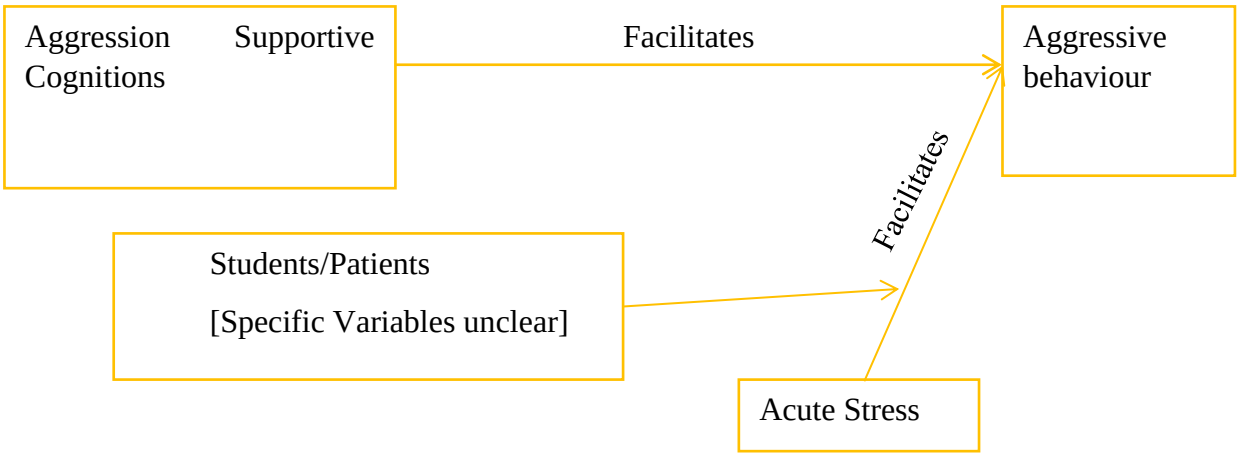

Figure 9.1 Results of Study One: Basic Paths

While the Study 1 demonstrated the difference between male students and male patients from a high secure hospital, the particular mechanism responsible for it was not identified. Comparison of the descriptive statistics of the sample suggest that forensic patients have more extensive histories of aggressive and antisocial behaviour and more entrenched aggression supportive implicit theories. As was shown in Study 3, use of maladaptive coping strategies appears to be another likely candidate. Meanwhile, since stress affects information processing (Starcke & Brand, 2012; 2016), which in turn is argued to be an antecedent of aggressive

[36] All figures in this chapter utilise Knowledge Integration Map (KIM) approach, where labelled arrows represent relationships between concepts (Schwendimann, 2014)

behaviour (Anderson & Bushman, 2002; Blair, 2004; 2016), its role was the focus of Study 2, which found that information processing did indeed influence aggression.

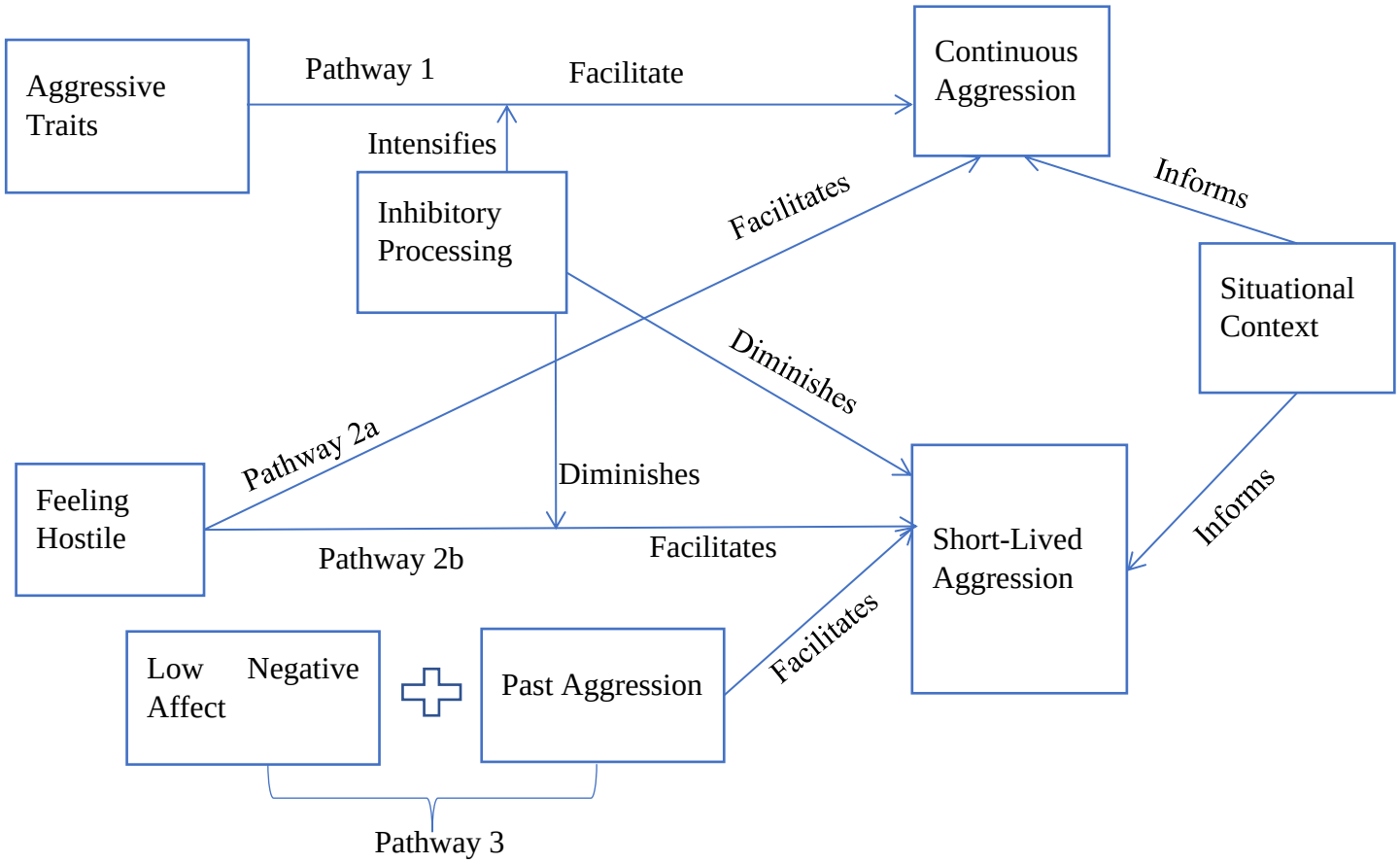

Figure 9.2 Results of Study Two: continuous and short lived aggression

In contrast to the Study 1, Study 2 focused on aggression during a series of interactions with an "opponent". Furthermore, it first captured aggression as linear or continuous, reflecting aggressive conduct that steadily grows in intensity over time. Second, it captured quadratic, or short lived, aggression, corresponding more to explosive aggression, which rapidly increases in intensity, yet quickly ceases afterwards. Instead of examining dynamic physiological stress, participants' subjective feelings or state before aggression was investigated. The results showed utility in considering aggressive behaviour as linear/continuous and quadratic. Continuous aggression could be viewed more as proactive aggression, as it increases as long as there are opportunities to engage in it. Quadratic-explosive aggression could be viewed as

more as reactive, since it represents the initial response to a situation that then stops. As shown in Figure 9.2, the factors affecting these two types had a distinct pattern.

Three pathways to aggressive behaviour were observed. First, there was a path from aggressive traits to continuous aggression. This association was intensified by inhibitory processing. Although these results were not in line with previous research showing an inverse association between P3 amplitude and aggression (Fanning et al., 2014; Jabr et al., 2018), a closer look at the aggression paradigm provided a possible explanation. Specifically, in the Study 2, aggressive behaviour was predicated on the "incorrect" answers of the "employee," in response to which participants could choose to deliver a shock. P3 amplitude can also serve as an indicator of the allocation of cognitive resources (Hajcak, MacNamara, & Olvet, 2010). Taking this into account, together with the specific paradigm and Theory of Planned Behaviour (Ajzen, 1991), it appears that increased linear aggression among those with larger P3 amplitudes reflected processing of a high number of cues for "incorrect" answers that would justify an aggressive response.

The second pathways (2a+2b) showed how hostile feelings give rise to both continuous and short-lived aggression. While continuous aggression was directly associated with feeling hostile, the association of short-lived aggression was predicated on poor inhibitory processing. For those with higher cognitive resource allocation, feeling hostile was not associated with aggressive behaviour. These results were consistent with previous research linking hostility and aggression (Ramirez & Andreu, 2006). Moreover, as was discussed in the Chapter 2, hostile perception of a given situation increases the likelihood of an aggressive response to it (Bushman & Anderson, 2002; Tuente, Bogaerts, & Veling, 2019, Quan et al., 2019).

The third pathway showed that low negative affect can facilitate aggression, yet only when a person has a history of aggression. The first and third pathways demonstrated the dominant influence of stable personality traits on aggressive behaviour over transient states.

Specifically, they showed that the stable personality trait of aggressiveness is directly related to aggressive behaviour, while the transient state of negative affect is related to aggressive behaviour indirectly. This highlights that the inner state a person experiences can affect aggressive behaviour, but only when such conduct is already habitual.

Although there was discord between some of the obtained results and the extant literature, in a dialectic manner this was used to inform part of the Study 2. As expected from previous research, inhibition processing (as measured by P3 amplitude) was inversely related to short lived aggression and, when poor, afforded a positive relationship between hostility and short lived aggression (Ramirez & Andreu, 2006; Rubio-Garay Carrasco & Amor, 2016; Verona & Bresin, 2015). However, for the relationship between trait aggressiveness and aggressive behaviour, inhibition processing was shown to be an amplifier rather than an inhibitor, which was inconsistent with previous studies (Fanning et al., 2014; Jabr et al., 2018). In the context of a specific paradigm, this unexpected finding highlighted another important variable that most likely influences aggressive behaviour: the situational context. In order to address the possible role of this variable, the Study 3 included hostile attribution bias into the model to account for an individual's perception of social interactions. Furthermore, contrary to the Studies 1 and 2, the third study used a cross-sectional design as it aimed to extend the patterns identified for specific situations to aggressive behaviour in daily life.

Fitting the model to the data collected for the Study 3 highlighted that pathways to aggression among adults and Transitional Aged Youth (TAY) differ. This is consistent with previous research showing that aggressive behaviour changes with age (Petersen, Bates, Dodge, Lansford, & Pettit, 2015) and suggests that mechanisms facilitating it do so as well. Nevertheless, as Figure 9.3 shows, the last study demonstrated two pathways to aggression. Importantly, in a similar manner to the Study 2, the third study also utilised two aggression-related variables for outcomes: first, past aggressive behaviour or act based aggression that

captures the extent to which a person has engaged in such conduct while disregarding specific motivations; and second, trait aggression reflecting proclivity for aggressive behaviour. The good fit of the measurement model with divided aggression variables was in line with previous research showing that measures of aggressive conduct and traits reflect different phenomena (Archer & Webb, 2006).

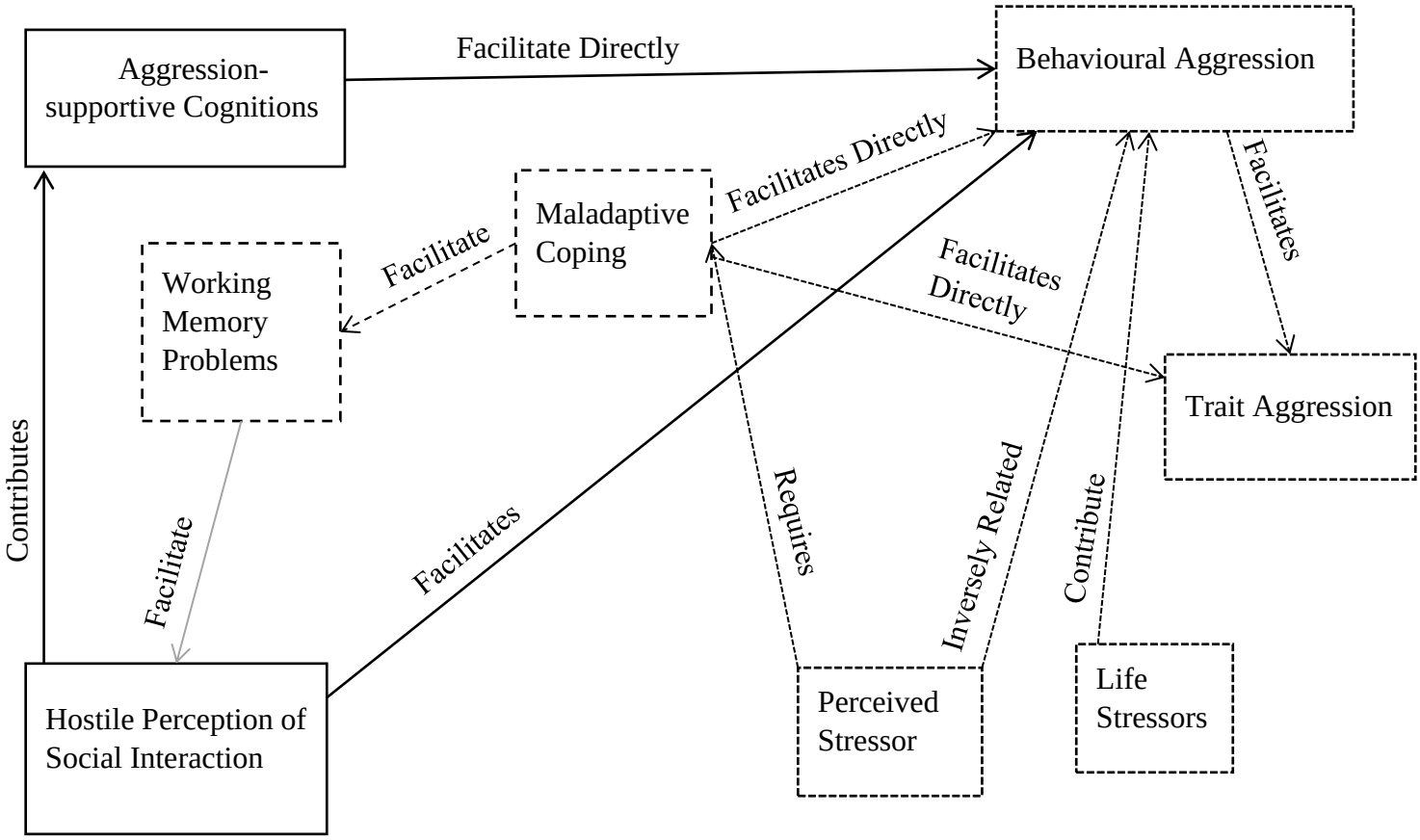

Figure 9.3 Results of Study Three: cognitive and stress pathways to stress.

The cognitive route (presented in Figure 9.3 with solid lines) demonstrates that the tendency to perceive situations as hostile facilitates aggressive behaviour through the presence of aggression supportive cognitions. This pathway supports and extends the findings from Study 1. Although in a specific situation where aggression is inflicted upon a stranger, only a particular cognitive structure was found to contribute to this (i.e., Implicit Theory "I am the law"), in daily life the general presence of cognitions commending or justifying use of

aggression (i.e., all of the Criminal Attitudes to Violence together) was associated with aggressive behaviour. Moreover, the importance of perception, noted in the Study 2, and the role of hostility were clarified. Indeed, the tendency to attribute hostile intent to others was associated with more instances of aggressive behaviour in the past. Taken together, the three studies exhibited support for the main proposition of the socio-cognitive models that aggression is an enactment of cognitive structures promoting aggression as the right or suitable course of action (Anderson & Bushman, 2002; Huesmann, 2016). Furthermore, as aggression is a form of behaviour, the effect of beliefs and perceptions on it further supports the Theory of Planned Behaviour (Ajzen, 1991) since it posits that behaviour results from favourable existing attitudes towards it and by the subjective norms that approve it.

Meanwhile, the second route to aggression (presented in Figure 9.3 with dotted lines) showed that the stress-aggression relationship is affected by the individual's preferred responses to stress. Traits and maladaptive coping styles mediated the relationship between perceived stress in the past month and aggressive behaviour. This finding is in line with previous research (Carlo, Mestre, McGinley, Samper, Tur, & Sandman, 2012; Gardner, Archer, & Jackson, 2012; Whitman & Gottdiener, 2015) reporting association of maladaptive coping styles and aggression. It also extends it, as coping style was shown to regulate the effect of stress. This path provides a possible explanation for the conflicting results in the systematic literature review, as the studies included in the review did not take into account coping styles. However, even when coping styles were part of the model, the direct path from perceived stress to aggressive behaviour was inversed. Furthermore, the experience of life stressors was associated with the development of aggressive traits through acts of aggression. Given that stress has been shown to facilitate habitual behaviour (Smeets et al., 2019), the second pathway highlights that it is the typical behavioural responses to stressful experiences that might lead to aggressive behaviour, rather than the experience on its own.

In addition to the two pathways, the Study 3 identified a link between them, which is presented in Figure 9.3 with dashed lines. Maladaptive coping styles were associated with daily problems in working memory, which in turn were related to aggressive behaviour via hostile attributions. This provided evidence in support of the second proposition derived from socio- and neurocognitive models stating that aggression results from decision-making processes (Anderson & Bushman, 2002; Blair, 2004; 2016; Crick & Dodge, 1994; Huesmann, 2016). The full mediation role of the tendency to perceive social interactions as hostile demonstrated that poor information processing, exemplified by problems with working memory, is not related to aggressive behaviour by itself. Instead, a cognitive structure facilitating aggressive conduct, in this case an assumption that others have harmful intent, is required for aggression to occur across situations.

Furthermore, the relationship between maladaptive coping and working memory problems demonstrates two aspects. First, it helps to define maladaptive coping styles as engagement in the behaviours harmful to mental health. Second, it shows the interrelation between cognitive and stress pathways to aggression. Through working memory problems among those who have justifications for aggression, a poor response to perceived stress can facilitate aggressive behaviour. The unifying role of this index of information processing between cognitions and stress for aggressive behaviour in daily life resembles the Study 2. Another index of information processing played a central role as an amplifier and buffer for aggressive behaviour in a specific situation.

9.2 Overall Model

Taking together the findings from this thesis as a whole, the *Stratified Integrated Model of Aggressive Behaviour* (SIMBA) is proposed and presented in Figure 9.4. Although it is guided mostly by the obtained results, it also takes into account previously established explanations of aggression. As such it is presented as a model that describes mechanisms that

facilitate and impair aggressive behaviour. Consequently, while SIMBA cannot replace more seminal explanatory models, such as GAM or IIPM, it serves as an extension to try and further explain how various variables could be presented and interact. It further captures aggressive behaviour across situations, as well as within specific situations and specifies the interaction between stress and coping styles in their effect on aggression. For the factors located outside of the Aggressive Acts frame in Figure 9.4, the SIMBA represents generally occurring aggressive behaviour. As such, it is proposed to be driven by pre-existing expectations for social encounters through aggression supportive cognitive structures. The expectation that others have hostile intent, which can evoke hostile feelings in an individual, is likely to activate *existing* aggression supportive cognitive structures, which can manifest as behaviour. However, for the components within the Aggressive Acts frame, the aggressive acts variable reflects the *history* of aggressive behaviour that impacts continuous aggression in a situation through the activation of aggressive traits or by directly facilitating short lived aggressive behaviour. Positioning aggression supportive cognitions and the behaviour that solidifies them as root antecedents of aggressive behaviour reflects the findings from the studies of this thesis, the General Aggression Model (GAM) (Anderson & Bushman, 2002) and the Integrated Information Processing Model (IIPM) (Huesmann, 2016). Meanwhile, within the I^3 terminology, aggression supportive structures are deemed as main impellants of proclivity for aggressive behaviour.

The information processing component, which includes cognitive resource allocation, inhibitory processing and working memory functioning assessed in the Studies 2 and 3, is an exception to the component placement in Figure 9.4. Their unique positioning on the border of aggressive acts shows its indirect effect on aggressive conduct across as well as within particular situations. Importantly, despite the position in the centre, information processing is linked with aggression only *indirectly.* This extends the inference from neurocognitive models

that evaluation of a behaviour precedes its enactment (Blair, 2004; 2016), yet it is *predicated* on existing cognitive structures and traits. Consequently, the information processing component of the SIMBA corresponds to what are named 'inhibitors' in the I^3 model (Finkel

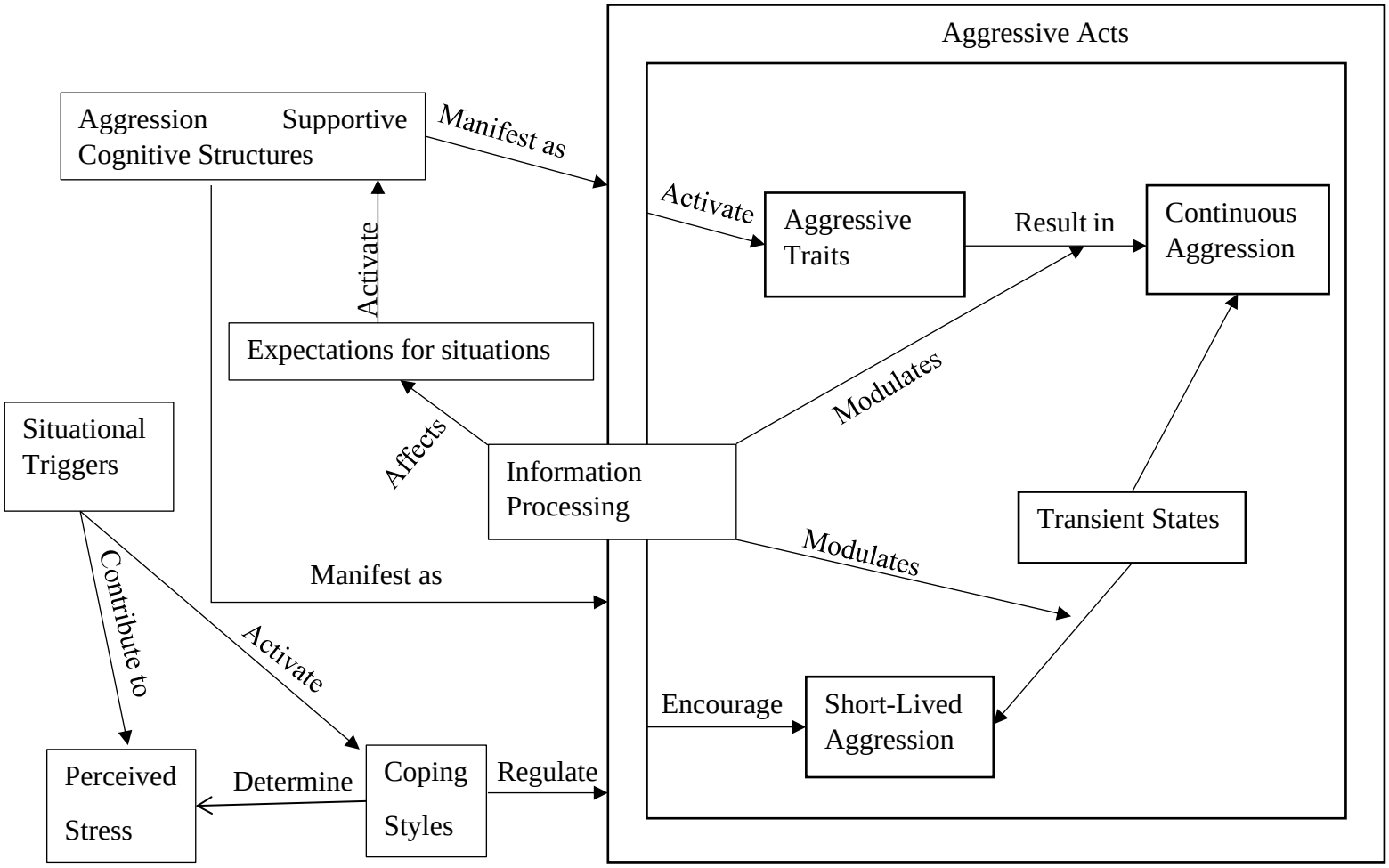

Figure 9.4 Proposed Model: Stratified Integrated Model of Aggressive Behaviour (SIMBA) & Hall, 2018).

In Figure 9.4, perceived stress is shown to be created by the situational triggers, but determined by coping styles, which are directly liked to repeated aggressive acts. This serves as a reminder of the conflicting results regarding the stress-aggression relationship when it is considered as direct. Consequently, the effect of stress is shown as being mediated by coping styles, as the former is not connected with aggressive acts, whereas the latter is. Although the

Study 3 showed only maladaptive coping strategies to act as a mediator, perceived stress was inversely related to adaptive coping strategies. In other words, while maladaptive approaches to dealing with stress are likely to facilitate aggressive behaviour by allowing stress to fuel it, adaptive coping appears to deny this fuel access to aggression. With such an interpretation, for generally occurring aggression, preferred coping styles represent the impellants from the I^3 model (Finkel & Hall, 2018).

Lastly, the inclusion of stress in a model of aggressive behaviour does not signify that the presence of stress is a necessary or sufficient component for it to occur. Rather, stress reflects the demands exerted on an individual. Using the terms of the I^3, stress appears to be an instigator, as it originates externally from the individual (Finkel & Hall, 2018). However, its intensity and subsequent effect on aggression is regulated by subjective individual factors, for instance preferred coping styles (i.e., impellants). Consequently, the SIMBA posits that aggressive behaviour is facilitated not simply by a situation *taxing* an individual, but by the individual's *poor response* to such circumstances, resulting from constrained decision-making. Moreover, given the importance of psychosocial elements for a stress response, it is likely that the perception of the intensity of situational demands is of higher influence than the actuality. However, as this proposition is an inference from the obtained results, it needs to be tested in further studies.

9.3 Limitations

The current research is not without limitations. The scales used to assess aggression supportive cognitive structures in the Studies 1 and 3 lacked precision. Although the Implicit Theories (ITs) (Polaschek et al., 2009) were identified as present among violent offenders, this study was the first to establish their presence through semi-structured interview, and the internal reliability of them together was low. However, the subsequent use of the ITs separately helped to uncover the specific core cognition related to aggressive behaviour at the cost of

increasing the number of predictors in the regression model. Meanwhile, in regard to the Criminal Attitudes to Violence scale (CAV) (Polaschek, Collie, & Walkey, 2004), Nunes et al. (2015) have questioned what specific type of aggression supportive cognitive structures it measures: attitudes or beliefs. While both represent cognitive structures (Anderson & Bushman, 2002), the lack of specificity hinders accurate identification of the mechanisms involved in facilitation of aggression. Nevertheless, due to the association of the CAV with aggressive behaviour rather than traits, the current thesis agrees with the suggestion of Nunes and colleagues (2015) that this scale is related to normative beliefs about aggression.

Another arguable limitation of this thesis is that it does not address possible sex differences in aggressive behaviour. Since the thematic analysis of the studies included in the systematic literature review did not find a consistent sex difference in the effect of stress response markers on aggression, the Studies 2 and 3 analysed male and female participants together. The results on the direct influence of aggression supportive cognitive structures and aggressive behaviour were comparable between the Study 1, which had an exclusively male sample, and Study 3, which used both men and women. This suggests that there is likely to be a certain degree of heterogeneity in the aggression-facilitating mechanism across sexes. This pattern is also consistent with the gender similarity hypothesis, which states that in most psychological variables the effect size of differences between men and women is small or very small (Zell, Krizan, & Teeter, 2015).

It is also important to note that while Studies 1 and 2 employed experimental designs allowing for a certain degree of speculation about causality, the use of a retrospective cross-sectional design in the Study 3 limits it. In other words, the reported findings show relationships between variables with a highly likely direction that need to be confirmed with longitudinal studies. Consequently, the proposed SIMBA model is a first stepping-stone, representing a synthesis of obtained results and requiring further testing.

A further limitation relating to the different methodologies in this thesis was assessment of the same latent construct using different measures. For instance, in the Study 2, information processing was indexed by the P3 difference amplitude during the Go/No-Go task, and in the Study 3 it was assessed via self-report questionnaire. Similarly, while in Studies 1 and 2 the outcome variable was aggressive behaviour in a given situation, in the Study 3 it was generally occurring aggression. However, it was the use of different methodologies that allowed a more comprehensive understanding of the roles of latent constructs in the SIMBA, and for its stratification.

9.4 Directions for Future Research

The SIMBA model posits aggression supportive cognitive structures as the primary facilitators of aggressive behaviour. However, it only partially addresses their formation and entrenchment. The results showed that experiencing stressful life events is associated with aggressive traits that facilitate aggressive responses via aggressive acts in the past. This highlights learning from behaviour rather than from observational learning, which is emphasised in socio-cognitive models (Huesmann, 2016). Nevertheless, this is consistent with a meta-analysis that showed only a small effect size for the influence of violent video games (a medium for observational learning of aggression supportive cognitions) on aggressive behaviour (Mathur & VanderWeele, 2019). Further research is needed that would be able to compare the rate of formation of aggression supportive cognitions from observation and from experiences, as well as the intensity of their effects on aggressive behaviour.

Given the noted limitations, the main focus of future research addressing or incorporating the proposed model should be identification of the finite aspects of the proposed components. As noted, the main drawback of the SIMBA is lack of specificity. Consequently, further research needs to address this. In this thesis, information processing was represented by P3 amplitude and daily problems with working memory. However, there are other mechanisms

that are likely to be relevant. Given the proposition that aggression results from "choosing" aggression supportive cognitive structures, the role of variables related to decision-making would be beneficial. In this respect, the next line of research would be to establish the effects of intuitive and analytic cognitive styles, measured using the Cognitive Reflection Test (CRT) (Frederick, 2005), on the association between aggression supportive cognitions and according conduct. The reasoning behind this suggestion is that the CRT allows assessment of the motivation for engaging in a certain type of thinking (Pennycook, Cheyne, Koehler, & Fugelsang, 2016). Consequently, if the standard CRT does affect the selection of behavioural scripts, the next step would be to create a version of it specific to aggressive and non-aggressive behaviour in given situations.

Furthermore, a proposal to investigate information-processing requires future studies to address the role of attention. Contrary to decision-making processes, which can only be observed indirectly, attention can be followed more precisely. For instance, eye tracking can identify the order in which situational cues are encoded and the time spent attending to each of them. Moreover, since eye tracking can be combined with a fixation-related potentials (FRP) analysis while participants observe naturalistic scenes (Simola, Le Fevre, Torniainen, & Baccino, 2015), a precise understanding of the mechanisms through which perception of the situation affects behaviour in it is possible. Additionally, the use of a similar approach, but with varying levels of emotional induction, will highlight a range of transient states that can affect aggression.

A closer investigation of the intermediary role played by coping styles between stress and aggression represents another line of future research. This could be considered using experimental designs, where participants engage in specific techniques representing distinct coping styles before taking part in the aggression behaviour paradigm. Such approaches will highlight the function of coping styles for aggressive conduct in specific situations. However,

this would require a systematic overview of coping styles with theoretically and statistically justified clustering, and establishment of their effect on the markers of perceived and physiological stress. In addition to furthering the understanding of aggressive behaviour, such research would also help to evaluate potential therapeutic interventions targeting aggression.

Due to the bottom-up foundation for the SIMBA, it does not incorporate all potential mediators for the relationship between stress and aggression or factors that could be directly linked to aggressive behaviour (e.g. such as impulsivity, self-control and risk-taking). As discussed in Chapter 3, acute stress evoked by situational triggers might influence aggressive conduct through the encouragement of habitual behaviour (Fournier et al., 2017; Smeets et a., 2019) or through facilitating higher risk-taking (Starcke & Brand, 2016). Meanwhile, as noted in Chapter 2, poor impulse control can be a direct antecedent of aggressive behaviour (da Cunha-Bang et al., 2017). Explicit incorporation of these factors into any future adaption of a model is likely to increase the accuracy with which it reflects aggression.

9.5 Implications for Clinical Practice

The SIMBA does not only aid in the understanding of aggressive behaviour, but it also has potential to guide therapeutic interventions aimed at reducing such conduct. Although such programmes can be formed on the basis of a series of treatment evaluation studies informing one another, there is a need for a theoretical background to guide it (Polaschek, 2011). The primary roles of aggression supportive cognitive structures in facilitating aggression highlight the utility of cognitive components in therapies targeting violent conduct. Furthermore, despite the indirect effect, the presence of stress in the model suggests that development of adaptive coping strategies would help to sustain the non-aggressive behaviour obtained during the sessions. Life Minus Violence-Enhanced (LMV-E) (Ireland, 2009) is an example of one intervention that addresses these two pathways and has been shown to reduce aggressive behaviour among adult male forensic inpatients (Daffern, Simpson, Ainslie, & Chu, 2018) and

young male offenders (Derbyshire, Tarrant, Fitter, & Gibson, 2019). Consequently, the current research suggests that a decrease in aggression is achieved by working with distal factors as well as with proximal ones. It is impossible to completely eliminate stressors, largely because most of them are external to individuals and are beyond their immediate control. However, an individual has control over their coping strategies. The presented studies suggest that while development of adaptive coping strategies can "break the link" between stress and aggression, the intervention needs to also address existing maladaptive coping strategies. Since they represent one of the paths through which stress was found to result in aggression, changing them could be incorporated in aggression interventions.

Furthermore, the current model can be relevant to violence risk assessment. The findings of the current thesis converge with structured risk assessments such as the Historical Clinical Risk Management-20 (HCR-20) (Douglas, Hart, Webster, Belfrage, Guy, & Wilson, (2014) in terms of the role of past aggressive behaviour which, according to the SIMBA, increases the likelihood of engaging in aggression. They are also clearly present in structured risk assessment tools (e.g., HCR-20, Douglas Hart, Webster, & Belfrage, 2013). However, aggression supportive cognitive structures and coping styles are present only partially, and the current thesis argues for more promotion of these factors. The results of the presented studies highlight the relevance of cognitive structures closer to normative beliefs than to attitudes, especially for aggression in a given situation. Moreover, structured risk assessments deal with general problems of coping skills without distinguishing their categories (e.g., adaptive vs. maladaptive or emotional vs. problem-focused vs. avoidant). Given the call for a more aetiological approach to risk assessment (Ward & Fortune, 2016), the addition of specifications for these categories would be beneficial.

9.6 Concluding Comments

The aim of the current thesis was to understand and outline the contribution of aggression supportive cognitive structures, stress and information processing to aggressive behaviour. Based on the preliminary findings, the SIMBA proposed that the primary route towards aggression originates in preconceived expectations for social situations and aggression supportive cognitions. Although the information processing components were also associated with aggressive behaviour through multiple paths, their effect was predicated on interaction with other variables. Consequently, rather than representing a route to aggression, they reflect omnipresent inhibitors and disinhibitors. Instead, the second contributor to aggression originated in stress, which broadly reflects situational demands on a person, and was suggested to affect aggressive behaviour only through other variables. One such variable was identified to be coping styles, specifically maladaptive coping style. It is, however, not the only possible mediator. Currently, the SIMBA is in its basic form, and does not include all variables that are drive or facilitate aggression. Adding them is the aim of future studies. The present thesis was devoted to establishing the foundation, meaning that the model is not all encompassing.

The discussed order of components raises a vital issue for the understanding of aggression and for therapeutic interventions. The first pathway with aggression supportive cognitive structures appears to be a necessary component for aggressive behaviour, as it is arguably challenging to select or behave according to a non-existent behavioural script. For a behaviour (including aggression) to be informed by a cognitive structure approving and outlining it with expected benefits, such a structure *needs* to be present. Otherwise, it cannot be enacted. However, it is not a sufficient component, as other factors related to information processing or coping styles can preclude the behaviour despite the presence of a script justifying or promoting it. Therefore, it is suggested that using either of these components on

their own would result in an incomplete understanding of, or a short-lived change in, aggression.

Lastly, aggression is a form of behaviour, and investigation into its origins, facilitators and inhibitors should not separate it from other behaviours. In particular, investigations should not neglect opposing behaviours, such as those that lead to offering help to others rather than inflicting harm. Stating that aggressive behaviour results from decision-making, whereby aggression supportive cognitive structures are selected as guides to conduct, necessarily implies that prosocial behaviour in underpinned by the processes that are similar in form but differ in substance.

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APPENDIX ONE: STRESS AND AGGRESSION MEASURES

Stress Response Markers

There was a variation in the biological markers measured to assess stress response. The most frequently used were salivary cortisol (k = 40), heart rate (k = 20) serum cortisol (k = 16), blood pressure (k = 14). The other markers were assessed in less than ten studies and include: norepinephrine (k = 8), epinephrine (k = 7), and adrenocorticotropic hormone (ACTH, k = 5) estimated from blood samples, norepinephrine (k = 5), epinephrine (k = 5) estimated from urine sample, skin conductance level (k = 5), eye blinking (k = 4), cortisol levels estimated from urine sample (k = 3), Dehydroepiandrosterone Sulphate (DHEA, k = 3) and Dehydroepiandrosterone (k = 2) estimated from blood samples, and cortisol levels estimated from hair sample (k = 2). There were measures used only by one study such as Peripheral-type benzodiazepine receptors (PBR), Beta- and Alpha- adrenergic receptors responsiveness, norepinephrine estimated from cerebrospinal fluid, and cortisone and DHEA estimated from hair samples. While majority studies used only one measure of stress (k = 47), others used two (k = 12), three (k = 6), four (k = 4), five (k = 3) and six (k = 5).

Aggression Measures

Methods used to assess aggressive behaviour varied considerably between the studies. Four paradigms, specifically, the TAP (Taylor, 1967) with aversive noise blasts (k = 5), the PSAP ((Cherek et al., 1996, (k = 5)), Teacher-learner task (Verona & Curtin, 2006; (k = 3)), social decision game (Von Dawans et al., 2012, (k = 2)), were used in more than one study. Meanwhile the other tasks were only used in a single study: the TAP with electric shocks, the STAP (Buades-Rotger et al., 2016), the Social Distance Dictator game (Margittai et al. 2015), the Hawk and Dove game (Mehta et al. ,2017), the Ultimatum game (Prasad et al., 2017). Despite variation between the studies, these paradigms represent one of the most valid

assessments of aggressive behaviour as participants consciously decide to harm, though in minor degree, an opponent whom they believe to be a living person. Another measures of aggression that were used in more than one study included: STAXI (Spielberger, 1988; 1999; both versions: k = 20), BPAQ (Buss & Perry, 1992; k = 8), BDHI (Buss & Durkee, 1957; k = 6), file trawl for aggressive incidents (k = 5), aggressive behaviour during an interaction (k = 5), hostility subscale of SCL-90-R (Derogatis, 1994, k = 3), KSP (Schalling, 1978; k = 3), judgment in military or police training (k = 2). Meanwhile, the rest of the instruments were used only once and included: anger subscale of STAI , intent to commit a crime descried in a vignette, aggression scale from DIS (Matthews et al., 1996), MOAS (Kay, Wolkenfeld, & Murrill, 1988), CMHS (Cook & Medley, 1954), externalising scale from ASR (Achenbach & Rescorla, 2003), self-reported crime history, criminal record, clinical observations, YASR (Achenbach, 1991), LHAS (Coccaro, Berman, Kavoussi, 1997), SRASBM (Murray-Close et al., 2010), behaviour during martial arts match, combination of DMI (Gleser, & Ihilevich, 1969) and LSI (Plutchik, Kellerman, & Conte, 1979) scales, and distance at which participant gave way to a confederate in a narrow corridor (Cohen et al., 1996).

ASR - Adult Self-Report (Achenbach & Rescorla, 2003) is 126-item questionnaire that assesses externalising and internalising problems. The externalising domain includes subscale of aggression behaviour.

BDHI – Buss Durkee Hostility Inventory (Buss & Durkee, 1957) is a 75 dichotomous item self-report measure that assess hostility. It includes eight subscales assessing: direct aggression, indirect aggression, verbal aggression, irritability, negativism, resentment, suspicion, and guilt.

BPAQ – Buss and Perry Aggression Questionnaire (Buss & Perry, 1992) is a 29 true-false item questionnaire that assesses trait aggression. It includes four subscales: physical aggression, verbal aggression, hostility, and anger.

CMHS - Cook-Medley Hostility Scale (Cook & Medley, 1954) is a 50 true-false item questionnaire that assess predisposition towards hostility which includes aggression proneness.

Derogatis, L. R. (1994). The SCL-90-R: Administration, scoring and procedures manual. Baltimore, MD: Clinical Psychometric Research.

DIS – Driver Stress Inventory (Matthews et al., 1996) is a 48-item self-report questionnaire that assesses stress related behaviours among drivers. It includes an aggression scale.

DMI - Defense Mechanism Inventory (Gleser & Ihilevich, 1969) is a self report questionnaire that consists of 10 vignettes, which are followed by questions about: proposed actual behaviour, impulsive behaviour (in fantasy), thoughts, and feelings. It is suggested to identify the defence mechanisms used by participants. They include: hostility, projection, principalization, turning against self, and reversal.

Hawk and Dove game (Mehta et al., 2017) – is a decision game where two participants are play against each other and it is the combined decision that results in the amount of money that both players earn. During a trial, a participant can select one of two choices. If both participants select option A then both earn equal amount of money. If both participants select option B then neither participant gains any money. Lastly, when different options are selected, then a player who selected option B receives more money than the player who selected option A. Consequently, for each player, option A represents a Dove strategy and option B – Hawk.

KSP - Karolinska Scales of Personality (Schalling, 1978) is a collection of tests which include 6 scales related to aggression: indirect aggression, verbal aggression, irritability, suspicion, guilt, inhibited aggression.

LHAS – Life history of Aggression (Coccaro, Berman, Kavoussi, 1997) is a checklist that assesses frequency of three types of aggression in the past: aggression, antisocial conduct, and self-direct aggression.

LSI - Life Style Index (Plutchik, Kellerman, & Conte, 1979) is a 97 true-false item self-report questionnaire. It is suggested to measure eight defence mechanisms compensation, denial, displacement, intellectualization, projection, reaction formation, regression, and repression.

MOAS - Modified Overt Aggression Scale (Kay, Wolkenfeld, & Murrill, 1988) is a checklist that assess presence of four types of aggression among patients in the last week. It includes verbal aggression, physical aggression, aggression against property, and autoaggression (direct at oneself).

PSAP – In Point Subtraction Aggression Paradigm (Cherek et al., 1996) participants are led to believe that they are playing an interactive game with a real-life opponent. A participant is instructed to earn as much game points as possible in a fixed amount of time and after the game the points will be converted into money. To do so a participant needs to press button A repeatedly (e.g. 100 presses add 20 cents Bjork et al., 2000). However, there also is button B, pressing which will subtract game points from an opponent (e.g. 10 presses lead to 20 cents subtracted from opponent, Bjork et al., 2000). Some studies include button C which provides immunity from subtraction for certain amount of time (Gerra et al., 2001). In fact there is no opponent and participant is playing against scripted responses.

Social decision game – (Von Dawans et al., 2012) is a sequential game that is played by two real life participants. In the first round one player makes a decision on how to distribute a sum of money between two players. If the sum is split into two equal sums the trial ends. However, if the decision is unfair towards one of the participants, the other player can either accept the unfair offer, or reject it leaving both players without monetary units.

Social Distance Dictator Game – (Margittai et al. 2015) in this game a participant is asked to donate some part of a given monetary sum to people who are positioned at different social distances. Participants are also led to believe that after the game ends, one of their decisions will be selected to be paid out.

SRASBM - Self-Report of Aggression & Social Behavior Measure (Murray-Close, Ostrov, Nelson, Crick, & Coccaro, 2010) is 11-item self-report questionnaire which assesses reactive and proactive relational aggression.

STAP – Social Threat Aggression Paradigm (Buades-Rotger et al., 2016) is a variation of the TAP, where participants are shown a video of opponent's facial expression (angry or neutral) while he sets the noise intensity and duration for them. The rest of the paradigm follows TAP.

STAXI – State Trait Anger Expression Inventory (Spielberger, 1988) and State Trait Anger Expression Inventory II (Spielberger, 1999) are 57 item self-report questionnaires that assess state anger, trait anger and anger expression, which is composed of anger control and expression (in or out).

TAP – Taylor Aggression Paradigm (Taylor, 1967) is a reaction time task where a participant is led to believe that he or she is playing against a real-life opponent. Before each trial participant set noise or shock level intensity and duration that his or her opponent will receive is the participant wins. A participant does not see what levels of feedback the opponent

has set for him until the end of a trial. However, in reality the game is scripted. In some studies (e.g., Buades-Rotger, Beyer, & Krämer, 2017) TAP is modified to include avoidance option, which allows them to avoid a trial.

Teacher-Lerner Task (Verona & Curtin, 2006) - participants are led to believe that another participant, who in fact is a confederate, will be playing a role of student who needs perform a memory task, and they need to supervise his or her performance. After correct responses participants press "correct" button, but after incorrect response participants can press either one of ten buttons with varying shock intensity. Unknowingly to participants no shocks were delivered.

Ultimatum Game (Prasad et al., 2017) – in this paradigm participants are led to believe that they are playing against 20 real life opponents with one interaction per each opponent. In every trial participant was shows how an opponent, in fact a computer, distributed a sum of money, which varies from trial to trial, between the two players. After the participant saw how the money is divided, he or she could either accept an offer, which lead to both players received the accorded sum or reject it, thereby denying any earning to both players. Before the game participants were informed that one of the trials will be randomly selected a money will be awarded to both players.

YASR – Young Adult Self Report (Achenbach, 1991) is a 138 item self-report measures that assess internalising and externalising problems. The externalising domain includes aggression subscale.

APPENDIX TWO: SUPPLEMENTARY FIGURES FOR STUDY TWO

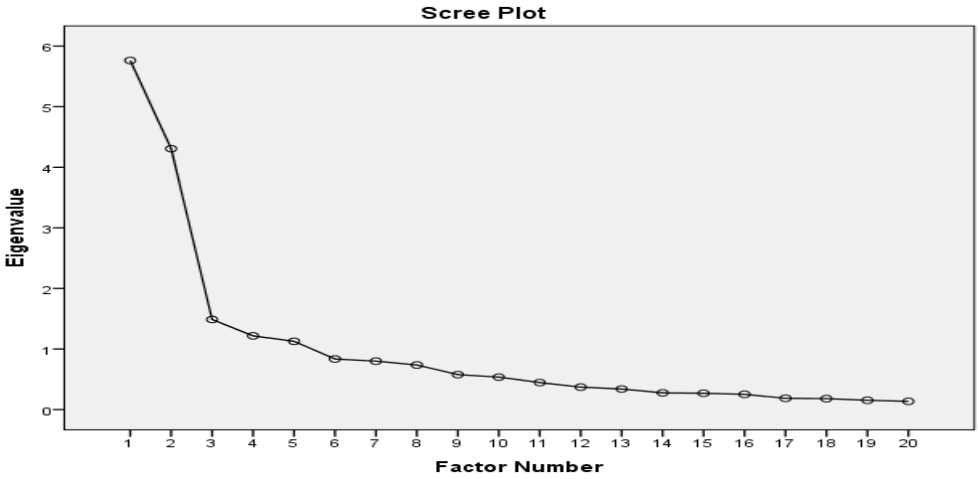

Figure A2. 1 Scree Plot for PANAS

Table A2. 1 Factor loadings for PANAS

	Factor 1	Factor 2
Proud	.824	
Enthusiastic	.792	
Excited	.788	
Strong	.770	
Determined	.762	
Inspired	.730	
Active	.663	
Interested	.651	
Alert	.532	
Attentive		
Afraid		.794
Nervous		.789
Distressed		.778
Upset		.658
Scared		.652
Ashamed		.603
Jittery		.551
Guilty		
Irritable		
Hostile		

Table A2. 2 Moderation analysis predicting aggressive responding from interaction between disposition to worry and P3 difference amplitude

	β [95% CI]	S.E.	*t*	*p*
Average shock across blocks, R^2= .01, *p* = .89				
Constant	1.34 [1.93, 2.75]	.21	11.03	<.0001
PSWQ	.005 [-.02, .03]	.01	.41	.68
Frontocentral P3 GNG	-.13 [-.48, .16]	.19	-.71	.48
PSWQ X Frontocentral P3 GNG	-.002 [-.03, .02]	.01	-.13	.90
Quadratic shock across blocks, R^2= .04, *p* = .49				
Constant	.43 [.007, .86]	.23	.06	.06
PSWQ	-.002 [-.03, .02]	.01	.87	.87
Frontocentral P3 GNG	-.29 [-.75, -.01]	.19	.12	.12
PSWQ X Frontocentral P3 GNG	-.006 [-.03, .009]	.01	-.53	.59
Average shock across blocks, R^2= .02, *p* = .66				
Constant	2.32 [1.93, 2.74]	.21	11.16	<.0001
PSWQ	.006 [-.02, .03]	.01	.42	.67
Parietal P3 GNG	-.04 [-.26, .19]	.12	-.36	.72
PSWQ X Parietal P3 GNG	.009 [-.006, .02]	.009	.99	.32
Quadratic shock across blocks, R^2= .002, *p* = .94				
Constant	.44 [.03, .90]	.22	1.97	.05
PSWQ	-.003 [-.03, .02]	.01	-.19	.85
Parietal P3 GNG	-.04 [-.23, .14]	.08	-.52	.61
PSWQ X Parietal P3 GNG	-.002 [-.01, .008]	.004	-.50	.62

APPENDIX THREE: SUPPLEMENTARY TABLES FOR STUDY THREE

Table A3. 1 First model (M1) for adult sample (n = 300)

Measurement Model	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Trait Aggression- > Anger	1 [1, 1] ***	0	0.844 [0.758, 0.931] ***	0.044
Trait Aggression- > Verbal Aggression	1.004 [0.849, 1.167] ***	0.081	0.657 [0.524, 0.79] ***	0.068
Trait Aggression- > Hostility	1.291 [1.101, 1.48] ***	0.097	0.742 [0.632, 0.853] ***	0.057
Aggressive Behaviour - > Reactive Aggression	1 [1, 1] ***	0	0.884 [0.838, 0.931] ***	0.024
Aggressive Behaviour - > Proactive Aggression	0.565 [0.45, 0.698] ***	0.061	0.684 [0.598, 0.769] ***	0.044
Aggressive Behaviour - > Past Aggression	1.028 [0.897, 1.163] ***	0.065	0.692 [0.613, 0.77] ***	0.04
Aggressive Behaviour - > Physical Aggression	0.774 [0.678, 0.883] ***	0.051	0.787 [0.716, 0.858] ***	0.036
Adaptive Coping - > Planning	1 [1, 1] ***	0	0.799 [0.727, 0.872] ***	0.037
Adaptive Coping - > Active Coping	0.972 [0.848, 1.136] ***	0.072	0.794 [0.72, 0.868] ***	0.038
Adaptive Coping - > Positive Reframing	0.746 [0.573, 0.932] ***	0.091	0.589 [0.478, 0.7] ***	0.057
Adaptive Coping - > Acceptance	0.676 [0.533, 0.84] ***	0.079	0.584 [0.474, 0.694] ***	0.056
Adaptive Coping - > Religion	0.242 [0.075, 0.427] **	0.087	0.19 [0.062, 0.318] **	0.065
Maladaptive Coping - > Self-Distraction	1 [1, 1] ***	0	0.446 [0.273, 0.618] ***	0.088
Maladaptive Coping - > Denial	0.804 [0.492, 1.181] ***	0.181	0.423 [0.234, 0.612] ***	0.096

Maladaptive Coping - > Substance Use	0.714 [0.357, 1.247] **	0.215	0.265 [0.1, 0.43] **	0.084
Maladaptive Coping - > Self Blame	1.734 [1.291, 2.349] ***	0.277	0.641 [0.472, 0.81] ***	0.086
Maladaptive Coping - > Behavioural disengagement	1.314 [0.967, 1.829] ***	0.218	0.642 [0.478, 0.806] ***	0.084
Maladaptive Coping - > Venting	1.015 [0.736, 1.461] ***	0.184	0.477 [0.293, 0.661] ***	0.094
Support Coping - > Instrumental Support	1 [1, 1] ***	0	0.959 [0.881, 1.037] ***	0.04
Support Coping - > Emotional Support	0.865 [0.714, 0.999] ***	0.076	0.824 [0.746, 0.901] ***	0.04
Support Coping - > Venting	0.304 [0.162, 0.421] ***	0.065	0.359 [0.222, 0.497] ***	0.07
Support Coping - > Humour	0.163 [0.034, 0.309] **	0.07	0.152 [0.024, 0.279] **	0.065
Working Memory Problems - > Storage Domain WM	1 [1, 1] ***	0	0.891 [0.843, 0.939] ***	0.025
Working Memory Problems - > Executive Domain WM	0.86 [0.743, 0.945] ***	0.052	0.826 [0.751, 0.901] ***	0.038
Working Memory Problems - > Attention Domain WM	1.065 [0.967, 1.17] ***	0.052	0.901 [0.852, 0.95] ***	0.025
Structural Model	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Direct Effects				
Hostile Attribution Tendency - > Working Memory Problems	0.176 [0.09, 0.256] ***	0.042	0.302 [0.154, 0.45] ***	0.075
Behavioural Aggression - - > Working Memory Problems	0.016 [-0.109, 0.122]	0.057	0.027 [-0.163, 0.218]	0.097
Working Memory Problems - > Adaptive Coping	-0.405 [-1.111, 0.321]	0.362	-0.08 [-0.222, 0.062]	0.072
Working Memory Problems - > Maladaptive Coping	6.068 [4.539, 8.207] ***	0.972	0.681 [0.518, 0.845] ***	0.083

Working Memory Problems - > Support Coping	0.056 [-0.353, 0.579]	0.234	0.016 [-0.113, 0.144]	0.066
Trait Aggression - - > Perceived Stress	0.033 [-0.055, 0.102]	0.039	0.129 [-0.202, 0.459]	0.169
Behavioural Aggression- > Perceived Stress	-0.183 [-0.382, -0.054] *	0.08	-0.374 [-0.7, -0.049] *	0.166
Trait Aggression- > Hostile Attribution Tendency	0.032 [-0.023, 0.092]	0.03	0.061 [-0.055, 0.177]	0.059
Behavioural Aggression- > Hostile Attribution Tendency	0.204 [0.101, 0.307] ***	0.054	0.201 [0.092, 0.31] ***	0.056
Trait Aggression - - > List of Threatening Experiences	-0.035 [-0.115, 0.039]	0.04	-0.048 [-0.155, 0.06]	0.055
Maladaptive Coping - - > Perceived Stress	0.077 [0.057, 0.097] ***	0.01	0.833 [0.68, 0.985] ***	0.078
Trait Aggression - - > Maladaptive Coping	0.906 [-0.106, 2.053]	0.55	0.33 [-0.051, 0.711]	0.194
Behavioural Aggression - > Maladaptive Coping	3.512 [1.568, 6.664] **	1.277	0.667 [0.199, 1.134] **	0.239
Adaptive Coping - > Perceived Stress	-0.062 [-0.079, -0.043] ***	0.01	-0.379 [-0.618, -0.14] ***	0.122
Trait Aggression - - > Adaptive Coping	0.024 [-0.159, 0.223]	0.096	0.015 [-0.105, 0.136]	0.062
Behavioural Aggression - > Adaptive Coping	-0.134 [-0.552, 0.272]	0.207	-0.045 [-0.18, 0.091]	0.069
Support Coping - - > Perceived Stress	-0.006 [-0.038, 0.023]	0.016	-0.027 [-0.228, 0.174]	0.102
Trait Aggression - - > Support Coping	-0.094 [-0.215, 0.038]	0.064	-0.086 [-0.205, 0.033]	0.061
Behavioural Aggression - > Support Coping	0.197 [-0.06, 0.493]	0.138	0.094 [-0.033, 0.221]	0.065

Criminal Attitudes to Violence - > Hostile Attribution Tendency	0.72 [0.3, 1.175] **	0.224	0.177 [0.075, 0.278] **	0.052
Trait Aggression- > Criminal Attitudes to Violence	-0.015 [-0.034, 0.003]	0.009	-0.113 [-0.473, 0.248]	0.184
Behavioural Aggression- > Criminal Attitudes to Violence	0.125 [0.089, 0.159] ***	0.018	0.501 [0.337, 0.664] ***	0.083
Behavioural Aggression- > .Life Traumatic Experiences	0.301 [0.149, 0.423] ***	0.069	0.216 [0.116, 0.315] ***	0.051
Trait Aggression- > Behavioural Aggression	0.336 [0.204, 0.482] ***	0.069	0.643 [0.377, 0.909] ***	0.136
indirect effects	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Perceived Stress - - > Maladaptive Coping --> Trait Aggression	0.07 [-0.003, 0.16]	0.041	0.274 [-0.056, 0.605]	0.168
Perceived Stress - - > Maladaptive Coping --> Behavioural Aggression	0.272 [0.119, 0.483] **	0.091	0.555 [0.109, 1.001] **	0.228
Perceived Stress - - > Adaptive Coping --> Trait Aggression	-0.001 [-0.014, 0.01]	0.006	-0.006 [-0.052, 0.04]	0.023
Perceived Stress - - > Adaptive Coping --> Behavioural Aggression	0.008 [-0.015, 0.035]	0.013	0.017 [-0.035, 0.069]	0.027
Perceived Stress - - > Support Coping - -> Trait Aggression	0.001 [-0.002, 0.007]	0.002	0.002 [-0.015, 0.02]	0.009
Perceived Stress - - > Support Coping - -> Behavioural Aggression	-0.001 [-0.016, 0.004]	0.004	-0.003 [-0.022, 0.017]	0.01
Hostile Attribution Tendency --> Criminal Attitudes to violence - > Trait Aggression	-0.011 [-0.034, 0]	0.008	-0.02 [-0.085, 0.045]	0.033
Hostile Attribution Tendency --> Criminal Attitudes to violence - > Behavioural Aggression	0.09 [0.031, 0.158] **	0.032	0.088 [0.027, 0.15] **	0.031
Life Stressful Experiences --> Aggressive Behaviour - > Aggressive Traits	0.101 [0.055, 0.176] ***	0.029	0.139 [0.055, 0.222] ***	0.043

Working Memory Problems --> Hostile Attribution Tendency --> Behavioural Aggression	0.036 [0.016, 0.067] **	0.013	0.061 [0.02, 0.101] **	0.021
Total Effects	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Perceived Stress - - > Maladaptive Coping --> Trait Aggression	0.103 [0.074, 0.137] ***	0.016	0.403 [0.223, 0.583] ***	0.092
Perceived Stress - - > Maladaptive Coping --> Behavioural Aggression	0.088 [0.027, 0.169] *	0.036	0.181 [-0.15, 0.511] *	0.169
Perceived Stress - - > Adaptive Coping --> Trait Aggression	0.031 [-0.052, 0.099]	0.037	0.123 [-0.195, 0.44]	0.162
Perceived Stress - - > Adaptive Coping --> Behavioural Aggression	-0.175 [-0.381, -0.058] *	0.076	-0.357 [-0.668, -0.047] *	0.158
Perceived Stress - - > Support Coping - -> Trait Aggression	0.033 [-0.056, 0.102]	0.04	0.131 [-0.201, 0.463]	0.169
Perceived Stress - - > Support Coping - -> Behavioural Aggression	-0.185 [-0.391, -0.056] *	0.081	-0.377 [-0.704, -0.05] *	0.167
Hostile Attribution Tendency --> Criminal Attitudes to violence - > Trait Aggression	0.022 [-0.036, 0.089]	0.033	0.041 [-0.095, 0.177]	0.069
Hostile Attribution Tendency --> Criminal Attitudes to violence - > Behavioural Aggression	0.294 [0.179, 0.406] ***	0.056	0.289 [0.18, 0.399] ***	0.056
Life Stressful Experiences --> Aggressive Behaviour - > Aggressive Traits	0.066 [-0.009, 0.137]	0.037	0.091 [-0.014, 0.196]	0.054
Working Memory Problems --> Hostile Attribution Tendency --> Behavioural Aggression	0.052 [-0.066, 0.157]	0.057	0.088 [-0.101, 0.277]	0.097
Covariance	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE

Adaptive Coping - - > Maladaptive Coping	0.038 [-0.057, 0.143]	0.051	0.091 [-0.143, 0.325]	0.119
Maladaptive Coping - - > Support Coping	-0.025 [-0.166, 0.129]	0.076	-0.039 [-0.273, 0.195]	0.119
Adaptive Coping - - > Support Coping	0.68 [0.403, 0.958] ***	0.139	0.361 [0.229, 0.494] ***	0.068
Positive Reframing - - > Humour	0.375 [0.079, 0.682] *	0.154	0.17 [0.037, 0.303] *	0.068
Anger - - > Anger	1.402 [1.009, 1.895] ***	0.211	0.287 [0.141, 0.434] ***	0.075
Verbal Aggression - - > Verbal Aggression	4.609 [3.785, 5.623] ***	0.456	0.568 [0.393, 0.743] ***	0.089
Hostility - - > Hostility	4.726 [3.857, 5.673] ***	0.476	0.449 [0.285, 0.613] ***	0.084
Reactive Aggression - - > Reactive Aggression	3.569 [2.543, 4.615] ***	0.506	0.218 [0.136, 0.301] ***	0.042
Proactive Aggression - - > Proactive Aggression	4.651 [3.231, 6.451] ***	0.804	0.532 [0.415, 0.649] ***	0.06
Past Aggression - - > Past Aggression	14.725 [12.48, 17.116] ***	1.212	0.522 [0.413, 0.63] ***	0.055
Physical Aggression - - > Physical Aggression	4.709 [3.845, 5.854] ***	0.508	0.381 [0.269, 0.493] ***	0.057
Planning - - > Planning	0.805 [0.573, 1.068] ***	0.122	0.361 [0.245, 0.477] ***	0.059
Active Coping - - > Active Coping	0.791 [0.566, 1.018] ***	0.115	0.37 [0.253, 0.488] ***	0.06
Positive Reframing- - > Positive Reframing	1.491 [1.223, 1.811] ***	0.15	0.653 [0.522, 0.784] ***	0.067
Acceptance - - > Acceptance	1.258 [1.014, 1.522] ***	0.133	0.659 [0.53, 0.788] ***	0.066
Religion - - > Religion	2.224 [1.701, 2.802] ***	0.279	0.964 [0.915, 1.012] ***	0.025

Covariance	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Self-Distraction - - > Self-Distraction	1.856 [1.591, 2.18] ***	0.151	0.801 [0.648, 0.955] ***	0.078
Denial - - > Denial	1.361 [1.103, 1.708] ***	0.154	0.821 [0.661, 0.981] ***	0.082
Substance Use - - > Substance Use	3.108 [2.523, 3.761] ***	0.308	0.93 [0.842, 1.017] ***	0.045
Substance Use - - > Substance Use	1.985 [1.635, 2.395] ***	0.194	0.589 [0.373, 0.806] ***	0.11
Behavioural disengagement - - > Behavioural disengagement	1.135 [0.896, 1.414] ***	0.132	0.588 [0.378, 0.799] ***	0.107
Venting - - > Venting	1.371 [1.137, 1.723] ***	0.146	0.658 [0.493, 0.823] ***	0.084
Instrumental Support - - > Instrumental Support	0.255 [-0.281, 0.635]	0.241	0.081 [-0.069, 0.23]	0.076
Emotional Support - - > Emotional Support	1.029 [0.665, 1.449] ***	0.202	0.321 [0.194, 0.449] ***	0.065
Humour - - > Humour	3.274 [2.861, 3.688] ***	0.206	0.977 [0.938, 1.016] ***	0.02
Storage Domain WM - - > Storage Domain WM	9.477 [7.066, 12.327] ***	1.358	0.206 [0.12, 0.292] ***	0.044
Executive Domain WM - - > Executive Domain WM	12.564 [9.952, 16.209] ***	1.553	0.318 [0.194, 0.442] ***	0.063
Attention Domain WM - - > Attention Domain WM	9.551 [6.731, 12.544] ***	1.497	0.188 [0.099, 0.276] ***	0.045
Hostile Attribution Tendency - - > Hostile Attribution Tendency	11.226 [9.153, 13.712] ***	1.139	0.909 [0.819, 0.998] ***	0.046
Criminal Attitudes to Violence - - > Criminal Attitudes to Violence	198.944 [159.505, 247.506] ***	22.017	0.969 [0.933, 1.005] ***	0.018

Trait Aggression - - > Trait Aggression	0.675 [0.416, 1.114] ***	0.166	0.194 [0.055, 0.333] **	0.071
Behavioural Aggression - - > Behavioural Aggression	4.782 [3.339, 6.984] ***	0.919	0.374 [0.205, 0.543] ***	0.086
Adaptive Coping - - > Adaptive Coping	1.219 [0.907, 1.578] ***	0.169	0.857 [0.675, 1.038] ***	0.092
Maladaptive Coping - - > Maladaptive Coping	0.141 [0.069, 0.25] **	0.044	0.307 [0.053, 0.56] *	0.129
Support Coping - - > Support Coping	2.905 [2.354, 3.662] ***	0.318	0.999 [0.988, 1.01] ***	0.006
Working Memory Problems - > Working Memory Problem	18.308 [12.557, 25.368] ***	3.343	0.502 [0.271, 0.732] ***	0.118
Perceived Stress - - > Perceived Stress	53.242 [53.242, 53.242] ***	0	1 [1, 1] ***	0
Perceived Stress - - > List of Threatening Experiences	2.095 [2.095, 2.095] ***	0	0.112 [0.112, 0.112] ***	0
List of Threatening Experiences - - > List of Threatening Experiences	6.548 [6.548, 6.548] ***	0	1 [1, 1] ***	0

*<0.05, **<0.01, ***<0.001

Table A3. 2 Second model (M2) for adult sample (n = 300)

Measurement Model	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Trait Aggression- > Anger	1 [1, 1] ***	0	0.841 [0.758, 0.925] ***	0.043
Trait Aggression- > Verbal Aggression	1.01 [0.871, 1.18] ***	0.076	0.658 [0.53, 0.786] ***	0.065
Trait Aggression- > Hostility	1.295 [1.106, 1.473] ***	0.095	0.742 [0.633, 0.85] ***	0.055

Aggressive Behaviour - > Reactive Aggression	1 [1, 1] ***	0	0.885 [0.841, 0.929] ***	0.023
Aggressive Behaviour - > Proactive Aggression	0.566 [0.429, 0.69] ***	0.062	0.685 [0.6, 0.769] ***	0.043
Aggressive Behaviour - > Past Aggression	1.029 [0.915, 1.155] ***	0.063	0.692 [0.613, 0.77] ***	0.04
Aggressive Behaviour - > Physical Aggression	0.773 [0.68, 0.871] ***	0.05	0.786 [0.717, 0.855] ***	0.035
Adaptive Coping - > Planning	1 [1, 1] ***	0	0.799 [0.726, 0.872] ***	0.037
Adaptive Coping - > Active Coping	0.973 [0.848, 1.116] ***	0.065	0.794 [0.719, 0.869] ***	0.038
Adaptive Coping - > Positive Reframing	0.743 [0.574, 0.954] ***	0.096	0.586 [0.478, 0.695] ***	0.055
Adaptive Coping - > Acceptance	0.674 [0.529, 0.829] ***	0.075	0.582 [0.471, 0.692] ***	0.057
Adaptive Coping - > Religion	0.241 [0.079, 0.406] **	0.085	0.189 [0.059, 0.318] **	0.066
Maladaptive Coping - > Self-Distraction	1 [1, 1] ***	0	0.444 [0.277, 0.611] ***	0.085
Maladaptive Coping - > Denial	0.796 [0.483, 1.172] ***	0.176	0.417 [0.23, 0.605] ***	0.096
Maladaptive Coping - > Substance Use	0.714 [0.305, 1.172] **	0.218	0.264 [0.104, 0.424] **	0.081
Maladaptive Coping - > Self Blame	1.732 [1.274, 2.333] ***	0.277	0.638 [0.47, 0.805] ***	0.085
Maladaptive Coping - > Behavioural disengagement	1.31 [0.964, 1.813] ***	0.227	0.637 [0.477, 0.797] ***	0.081
Maladaptive Coping - > Venting	1.017 [0.689, 1.404] ***	0.187	0.474 [0.3, 0.649] ***	0.089
Support Coping - > Instrumental Support	1 [1, 1] ***	0	0.962 [0.883, 1.042] ***	0.041

Support Coping - > Emotional Support	0.859 [0.715, 0.995] ***	0.069	0.821 [0.744, 0.898] ***	0.039
Support Coping - > Venting	0.297 [0.17, 0.4] ***	0.058	0.351 [0.229, 0.472] ***	0.062
Support Coping - > Humour	0.162 [0.034, 0.303] **	0.067	0.151 [0.036, 0.266] **	0.059
Working Memory Problems - > Storage Domain WM	1 [1, 1] ***	0	0.891 [0.842, 0.94] ***	0.025
Working Memory Problems - > Executive Domain WM	0.86 [0.752, 0.951] ***	0.051	0.826 [0.751, 0.901] ***	0.038
Working Memory Problems - > Attention Domain WM	1.064 [0.97, 1.165] ***	0.051	0.901 [0.852, 0.95] ***	0.025
Structural Model	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Direct Effects				
Hostile Attribution Tendency - > Working Memory Problems	0.176 [0.097, 0.257] ***	0.041	0.302 [0.161, 0.444] ***	0.072
Behavioural Aggression - - > Working Memory Problems	0.009 [-0.116, 0.116]	0.06	0.015 [-0.17, 0.201]	0.095
Working Memory Problems - > Adaptive Coping	-0.346 [-1.029, 0.333]	0.351	-0.068 [-0.206, 0.07]	0.07
Working Memory Problems - > Maladaptive Coping	6.109 [4.554, 8.559] ***	1.012	0.683 [0.52, 0.846] ***	0.083
Working Memory Problems - > Support Coping	0.01 [-0.36, 0.45]	0.218	0.003 [-0.121, 0.127]	0.063
Trait Aggression - - > Perceived Stress	0.011 [-0.08, 0.072]	0.036	0.045 [-0.278, 0.368]	0.165
Behavioural Aggression- > Perceived Stress	-0.193 [-0.41, -0.061] *	0.087	-0.394 [-0.753, -0.036] *	0.183
Trait Aggression- > Hostile Attribution Tendency	0.044 [-0.009, 0.098]	0.027	0.083 [-0.021, 0.188]	0.053

Behavioural Aggression- > Hostile Attribution Tendency	0.201 [0.095, 0.31] ***	0.053	0.198 [0.092, 0.304] ***	0.054
Trait Aggression - - > List of Threatening Experiences	-0.017 [-0.097, 0.057]	0.039	-0.024 [-0.128, 0.08]	0.053
Maladaptive Coping - - > Perceived Stress	0.077 [0.059, 0.098] ***	0.01	0.837 [0.691, 0.983] ***	0.074
Trait Aggression - - > Maladaptive Coping	1.233 [0.419, 2.452] *	0.519	0.449 [0.097, 0.8] *	0.179
Behavioural Aggression - > Maladaptive Coping	3.788 [1.557, 7.078] **	1.43	0.718 [0.216, 1.22] **	0.256
Adaptive Coping - > Perceived Stress	-0.061 [-0.078, -0.043] ***	0.009	-0.372 [-0.604, -0.139] **	0.119
Criminal Attitudes to Violence - > Hostile Attribution Tendency	0.72 [0.309, 1.186] **	0.226	0.177 [0.074, 0.28] **	0.053
Behavioural Aggression- > Criminal Attitudes to Violence	0.122 [0.09, 0.158] ***	0.017	0.489 [0.33, 0.648] ***	0.081
Behavioural Aggression- > .Life Traumatic Experiences	0.295 [0.156, 0.434] ***	0.068	0.211 [0.115, 0.308] ***	0.049
Trait Aggression- > Behavioural Aggression	0.271 [0.179, 0.36] ***	0.046	0.52 [0.334, 0.707] ***	0.095
Indirect effects	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Perceived Stress - - > Maladaptive Coping --> Trait Aggression	0.096 [0.034, 0.183] *	0.037	0.376 [0.057, 0.695] *	0.163
Perceived Stress - - > Maladaptive Coping --> Behavioural Aggression	0.293 [0.119, 0.54] **	0.104	0.601 [0.119, 1.082] **	0.246
Hostile Attribution Tendency --> Criminal Attitudes to violence - > Behavioural Aggression	0.088 [0.031, 0.158] **	0.032	0.086 [0.026, 0.147] **	0.031
Life Stressful Experiences --> Aggressive Behaviour - > Aggressive Traits	0.08 [0.041, 0.133] **	0.023	0.11 [0.044, 0.176] **	0.034

Working Memory Problems --> Hostile Attribution Tendency --> Behavioural Aggression	0.035 [0.015, 0.065] **	0.012	0.06 [0.021, 0.099] **	0.02
Total Effects				
Perceived Stress - - > Maladaptive Coping --> Trait Aggression	0.107 [0.079, 0.134] ***	0.014	0.421 [0.237, 0.605] ***	0.094
Perceived Stress - - > Maladaptive Coping --> Behavioural Aggression	0.101 [0.031, 0.171] **	0.036	0.206 [-0.128, 0.541] **	0.171
Hostile Attribution Tendency --> Criminal Attitudes to violence - > Behavioural Aggression	0.289 [0.183, 0.407] ***	0.056	0.284 [0.177, 0.392] ***	0.055
Life Stressful Experiences --> Aggressive Behaviour - > Aggressive Traits	0.062 [-0.022, 0.137]	0.039	0.086 [-0.023, 0.195]	0.056
Working Memory Problems --> Hostile Attribution Tendency --> Behavioural Aggression	0.044 [-0.079, 0.157]	0.059	0.075 [-0.109, 0.259]	0.094
Covariance	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Adaptive Coping - - > Maladaptive Coping	0.031 [-0.043, 0.127]	0.042	0.076 [-0.122, 0.273]	0.101
Maladaptive Coping - - > Support Coping	-0.01 [-0.136, 0.117]	0.063	-0.016 [-0.216, 0.185]	0.102
Adaptive Coping - - > Support Coping	0.682 [0.414, 0.95] ***	0.136	0.361 [0.224, 0.497] ***	0.07
Positive Reframing - - > Humour	0.377 [0.072, 0.655] **	0.147	0.17 [0.042, 0.299] **	0.066
Anger - - > Anger	1.419 [1.049, 1.854] ***	0.21	0.292 [0.151, 0.433] ***	0.072
Verbal Aggression - - > Verbal Aggression	4.584 [3.776, 5.449] ***	0.434	0.567 [0.398, 0.735] ***	0.086

Hostility - - > Hostility	4.722 [3.904, 5.843] ***	0.474	0.45 [0.289, 0.611] ***	0.082
Reactive Aggression - - > Reactive Aggression	3.524 [2.54, 4.71] ***	0.543	0.217 [0.139, 0.295] ***	0.04
Proactive Aggression - - > Proactive Aggression	4.621 [3.179, 6.577] ***	0.821	0.531 [0.416, 0.647] ***	0.059
Past Aggression - - > Past Aggression	14.657 [12.358, 17.384] ***	1.284	0.521 [0.413, 0.63] ***	0.055
Physical Aggression - - > Physical Aggression	4.704 [3.825, 5.783] ***	0.498	0.382 [0.274, 0.491] ***	0.056
Planning - - > Planning	0.803 [0.586, 1.063] ***	0.121	0.362 [0.245, 0.479] ***	0.06
Active Coping - - > Active Coping	0.786 [0.588, 1.04] ***	0.115	0.37 [0.251, 0.488] ***	0.06
Positive Reframing- - > Positive Reframing	1.496 [1.204, 1.814] ***	0.155	0.656 [0.529, 0.784] ***	0.065
Acceptance - - > Acceptance	1.261 [1.013, 1.536] ***	0.13	0.662 [0.533, 0.791] ***	0.066
Religion - - > Religion	2.225 [1.671, 2.779] ***	0.284	0.964 [0.916, 1.013] ***	0.025
Self-Distraction - - > Self-Distraction	1.86 [1.573, 2.18] ***	0.149	0.803 [0.655, 0.951] ***	0.076
Denial - - > Denial	1.37 [1.104, 1.769] ***	0.159	0.826 [0.669, 0.982] ***	0.08
Substance Use - - > Substance Use	3.11 [2.574, 3.709] ***	0.296	0.93 [0.846, 1.015] ***	0.043
Substance Use - - > Substance Use	1.998 [1.653, 2.418] ***	0.197	0.593 [0.38, 0.807] ***	0.109
Behavioural disengagement - - > Behavioural disengagement	1.146 [0.908, 1.429] ***	0.128	0.594 [0.391, 0.797] ***	0.104
Venting - - > Venting	1.372 [1.106, 1.684] ***	0.146	0.655 [0.496, 0.814] ***	0.081

Covariance	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Instrumental Support - - > Instrumental Support	0.235 [-0.299, 0.642]	0.23	0.074 [-0.079, 0.227]	0.078
Emotional Support - - > Emotional Support	1.043 [0.681, 1.47] ***	0.198	0.326 [0.2, 0.452] ***	0.064
Humour - - > Humour	3.274 [2.922, 3.755] ***	0.21	0.977 [0.942, 1.012] ***	0.018
Storage Domain WM - - > Storage Domain WM	9.46 [6.799, 12.427] ***	1.428	0.206 [0.119, 0.293] ***	0.044
Executive Domain WM - - > Executive Domain WM	12.563 [9.956, 15.969] ***	1.527	0.318 [0.195, 0.441] ***	0.063
Attention Domain WM - - > Attention Domain WM	9.57 [6.654, 12.509] ***	1.488	0.188 [0.101, 0.276] ***	0.045
Hostile Attribution Tendency - - > Hostile Attribution Tendency	11.225 [8.981, 13.535] ***	1.158	0.909 [0.823, 0.994] ***	0.044
Criminal Attitudes to Violence - - > Criminal Attitudes to Violence	198.944 [160.758, 247.171] ***	22.344	0.969 [0.932, 1.005] ***	0.019
Trait Aggression - - > Trait Aggression	0.709 [0.434, 1.205] ***	0.178	0.206 [0.062, 0.35] **	0.073
Behavioural Aggression - - > Behavioural Aggression	4.778 [2.864, 6.871] ***	1.017	0.376 [0.191, 0.561] ***	0.094
Adaptive Coping - - > Adaptive Coping	1.221 [0.896, 1.54] ***	0.167	0.862 [0.689, 1.035] ***	0.088
Maladaptive Coping - - > Maladaptive Coping	0.137 [0.073, 0.259] **	0.043	0.3 [0.055, 0.544] **	0.125
Support Coping - - > Support Coping	2.927 [2.343, 3.566] ***	0.316	1 [1, 1] ***	0
Working Memory Problems - > Working Memory Problem	18.374 [12.629, 26.331] ***	3.505	0.504 [0.273, 0.735] ***	0.118

Perceived Stress - - > Perceived Stress	53.242 [53.242, 53.242] ***	0	1 [1, 1] ***	0
Perceived Stress - - > List of Threatening Experiences	2.095 [2.095, 2.095] ***	0	0.112 [0.112, 0.112] ***	0
List of Threatening Experiences - - > List of Threatening Experiences	6.548 [6.548, 6.548] ***	0	1 [1, 1] ***	0

*<0.05, **<0.01, ***<0.001

Table A3. 3 Total Direct and indirect effects of the model 3 (M3) for adults (*n* = 300)

Measurement Model	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Trait Aggression- > Anger	1 [1, 1] ***	0	0.84 [0.76, 0.921] ***	0.041
Trait Aggression- > Verbal Aggression	1.011 [0.871, 1.177] ***	0.078	0.659 [0.532, 0.785] ***	0.065
Trait Aggression- > Hostility	1.298 [1.123, 1.47] ***	0.091	0.742 [0.636, 0.848] ***	0.054
Aggressive Behaviour - > Reactive Aggression	1 [1, 1] ***	0	0.885 [0.838, 0.931] ***	0.024
Aggressive Behaviour - > Proactive Aggression	0.567 [0.432, 0.687] ***	0.063	0.685 [0.599, 0.77] ***	0.044
Aggressive Behaviour - > Past Aggression	1.029 [0.907, 1.182] ***	0.067	0.692 [0.613, 0.771] ***	0.04
Aggressive Behaviour - > Physical Aggression	0.773 [0.681, 0.871] ***	0.049	0.786 [0.716, 0.856] ***	0.036
Adaptive Coping - > Planning	1 [1, 1] ***	0	0.799 [0.724, 0.874] ***	0.038
Adaptive Coping - > Active Coping	0.973 [0.847, 1.132] ***	0.073	0.794 [0.719, 0.87] ***	0.038

Adaptive Coping - > Positive Reframing	0.743 [0.569, 0.943] ***	0.096	0.586 [0.471, 0.701] ***	0.059
Adaptive Coping - > Acceptance	0.674 [0.54, 0.846] ***	0.076	0.581 [0.475, 0.688] ***	0.054
Adaptive Coping - > Religion	0.241 [0.065, 0.414] **	0.087	0.189 [0.061, 0.316] **	0.065
Maladaptive Coping - > Self-Distraction	1 [1, 1] ***	0	0.442 [0.274, 0.61] ***	0.086
Maladaptive Coping - > Denial	0.794 [0.466, 1.189] ***	0.184	0.414 [0.228, 0.601] ***	0.095
Maladaptive Coping - > Substance Use	0.713 [0.379, 1.2] ***	0.203	0.262 [0.105, 0.42] **	0.08
Maladaptive Coping - > Self Blame	1.732 [1.339, 2.406] ***	0.272	0.635 [0.468, 0.802] ***	0.085
Maladaptive Coping - > Behavioural disengagement	1.31 [0.968, 1.895] ***	0.225	0.635 [0.473, 0.797] ***	0.083
Maladaptive Coping - > Venting	1.023 [0.725, 1.43] ***	0.178	0.475 [0.301, 0.65] ***	0.089
Support Coping - > Instrumental Support	1 [1, 1] ***	0	0.962 [0.882, 1.043] ***	0.041
Support Coping - > Emotional Support	0.859 [0.718, 0.994] ***	0.071	0.821 [0.746, 0.896] ***	0.038
Support Coping - > Venting	0.297 [0.19, 0.417] ***	0.056	0.351 [0.232, 0.469] ***	0.06
Support Coping - > Humour	0.162 [0.038, 0.31] *	0.066	0.151 [0.031, 0.272] *	0.061
Working Memory Problems - > Storage Domain WM	1 [1, 1] ***	0	0.891 [0.842, 0.94] ***	0.025
Working Memory Problems - > Executive Domain WM	0.859 [0.758, 0.958] ***	0.051	0.826 [0.753, 0.898] ***	0.037
Working Memory Problems - > Attention Domain WM	1.064 [0.972, 1.17] ***	0.051	0.901 [0.852, 0.95] ***	0.025

Structural Model	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Direct Effects				
Hostile Attribution Tendency - > Working Memory Problems	0.176 [0.098, 0.253] ***	0.04	0.302 [0.165, 0.44] ***	0.07
Working Memory Problems - > Adaptive Coping	-0.329 [-0.986, 0.369]	0.342	-0.065 [-0.198, 0.069]	0.068
Working Memory Problems - > Maladaptive Coping	6.135 [4.541, 8.592] ***	1.005	0.683 [0.524, 0.842] ***	0.081
Working Memory Problems - > Support Coping	0.006 [-0.361, 0.43]	0.199	0.002 [-0.109, 0.112]	0.056
Behavioural Aggression- > Perceived Stress	-0.206 [-0.408, -0.095] **	0.073	-0.421 [-0.721, -0.122] **	0.153
Trait Aggression- > Hostile Attribution Tendency	0.046 [-0.007, 0.095]	0.026	0.088 [-0.018, 0.194]	0.054
Behavioural Aggression- > Hostile Attribution Tendency	0.202 [0.103, 0.301] ***	0.051	0.199 [0.095, 0.303] ***	0.053
Maladaptive Coping - - > Perceived Stress	0.078 [0.057, 0.098] ***	0.01	0.841 [0.698, 0.984] ***	0.073
Trait Aggression - - > Maladaptive Coping	1.386 [0.932, 2.016] ***	0.267	0.503 [0.361, 0.646] ***	0.073
Behavioural Aggression - > Maladaptive Coping	4.011 [2.319, 6.686] ***	1.087	0.757 [0.356, 1.158] ***	0.205
Adaptive Coping - > Perceived Stress	-0.061 [-0.079, -0.042] ***	0.009	-0.372 [-0.606, -0.137] **	0.12
Criminal Attitudes to Violence - > Hostile Attribution Tendency	0.72 [0.26, 1.211] **	0.232	0.177 [0.071, 0.282] **	0.054
Behavioural Aggression- > Criminal Attitudes to Violence	0.122 [0.087, 0.155] ***	0.017	0.49 [0.326, 0.653] ***	0.083
Behavioural Aggression- > .Life Traumatic Experiences	0.294 [0.168, 0.434] ***	0.067	0.211 [0.113, 0.309] ***	0.05

Trait Aggression- > Behavioural Aggression	0.257 [0.191, 0.335] ***	0.037	0.495 [0.348, 0.643] ***	0.075
Indirect effects	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Perceived Stress - - > Maladaptive Coping --> Trait Aggression	0.107 [0.079, 0.135] ***	0.014	0.423 [0.259, 0.587] ***	0.084
Perceived Stress - - > Maladaptive Coping --> Behavioural Aggression	0.311 [0.184, 0.491] ***	0.075	0.637 [0.226, 1.047] **	0.209
Hostile Attribution Tendency --> Criminal Attitudes to violence - > Behavioural Aggression	0.088 [0.032, 0.164] **	0.033	0.087 [0.024, 0.149] **	0.032
Life Stressful Experiences --> Aggressive Behaviour - > Aggressive Traits	0.076 [0.043, 0.122] ***	0.02	0.105 [0.046, 0.163] ***	0.03
Working Memory Problems --> Hostile Attribution Tendency --> Behavioural Aggression	0.036 [0.016, 0.063] **	0.012	0.06 [0.021, 0.099] **	0.02
Total Effects				
Perceived Stress - - > Maladaptive Coping --> Behavioural Aggression	0.105 [0.058, 0.157] ***	0.025	0.215 [-0.123, 0.553]	0.172
Hostile Attribution Tendency --> Criminal Attitudes to violence - > Behavioural Aggression	0.29 [0.184, 0.403] ***	0.055	0.286 [0.18, 0.391] ***	0.054
Covariance	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Adaptive Coping - - > Maladaptive Coping	0.029 [-0.043, 0.111]	0.039	0.072 [-0.119, 0.262]	0.097
Maladaptive Coping - - > Support Coping	-0.01 [-0.121, 0.11]	0.061	-0.016 [-0.208, 0.177]	0.098
Adaptive Coping - - > Support Coping	0.682 [0.411, 0.968] ***	0.144	0.361 [0.223, 0.498] ***	0.07
Positive Reframing - - > Humour	0.377 [0.096, 0.653] *	0.152	0.17 [0.039, 0.301] *	0.067

Anger - - > Anger	1.428 [1.06, 1.832] ***	0.196	0.294 [0.159, 0.429] ***	0.069
Verbal Aggression - - > Verbal Aggression	4.58 [3.788, 5.519] ***	0.446	0.566 [0.399, 0.733] ***	0.085
Hostility - - > Hostility	4.709 [3.911, 5.724] ***	0.464	0.449 [0.292, 0.606] ***	0.08
Reactive Aggression - - > Reactive Aggression	3.531 [2.538, 4.63] ***	0.522	0.218 [0.135, 0.3] ***	0.042
Proactive Aggression - - > Proactive Aggression	4.618 [3.21, 6.519] ***	0.813	0.531 [0.414, 0.648] ***	0.06
Past Aggression - - > Past Aggression	14.654 [12.316, 17.269] ***	1.281	0.521 [0.412, 0.631] ***	0.056
Physical Aggression - - > Physical Aggression	4.7 [3.813, 5.823] ***	0.501	0.382 [0.273, 0.492] ***	0.056
Planning - - > Planning	0.804 [0.572, 1.069] ***	0.126	0.362 [0.242, 0.482] ***	0.061
Active Coping - - > Active Coping	0.786 [0.573, 1.012] ***	0.113	0.369 [0.25, 0.489] ***	0.061
Positive Reframing- - > Positive Reframing	1.496 [1.22, 1.823] ***	0.156	0.656 [0.522, 0.791] ***	0.069
Acceptance - - > Acceptance	1.261 [1.025, 1.564] ***	0.134	0.662 [0.538, 0.786] ***	0.063
Religion - - > Religion	2.225 [1.696, 2.878] ***	0.285	0.964 [0.916, 1.013] ***	0.025
Self-Distraction - - > Self-Distraction	1.864 [1.603, 2.207] ***	0.143	0.805 [0.656, 0.953] ***	0.076
Denial - - > Denial	1.373 [1.105, 1.784] ***	0.158	0.828 [0.674, 0.982] ***	0.079
Substance Use - - > Substance Use	3.112 [2.586, 3.689] ***	0.285	0.931 [0.848, 1.014] ***	0.042
Substance Use - - > Substance Use	2.01 [1.691, 2.44] ***	0.188	0.597 [0.385, 0.809] ***	0.108

Covariance	Estimate and 95% CI	SE	Std Estimate and 95% CI	Std SE
Behavioural disengagement - - > Behavioural disengagement	1.152 [0.937, 1.414] ***	0.125	0.597 [0.392, 0.803] ***	0.105
Venting - - > Venting	1.371 [1.122, 1.707] ***	0.141	0.654 [0.496, 0.812] ***	0.081
Instrumental Support - - > Instrumental Support	0.235 [-0.246, 0.636]	0.25	0.074 [-0.081, 0.229]	0.079
Emotional Support - - > Emotional Support	1.043 [0.684, 1.428] ***	0.2	0.326 [0.202, 0.449] ***	0.063
Humour - - > Humour	3.274 [2.902, 3.742] ***	0.209	0.977 [0.941, 1.013] ***	0.019
Storage Domain WM - - > Storage Domain WM	9.445 [6.823, 12.478] ***	1.411	0.206 [0.118, 0.293] ***	0.045
Executive Domain WM - - > Executive Domain WM	12.573 [9.824, 15.596] ***	1.471	0.318 [0.199, 0.438] ***	0.061
Attention Domain WM - - > Attention Domain WM	9.575 [6.716, 12.873] ***	1.517	0.188 [0.101, 0.276] ***	0.045
Hostile Attribution Tendency - - > Hostile Attribution Tendency	11.224 [9.091, 13.551] ***	1.145	0.909 [0.826, 0.992] ***	0.042
Criminal Attitudes to Violence - - > Criminal Attitudes to Violence	198.944 [163.924, 250.063] ***	21.35	0.969 [0.931, 1.006] ***	0.019
Trait Aggression - - > Trait Aggression	0.689 [0.419, 1.136] ***	0.169	0.201 [0.064, 0.338] **	0.07
Behavioural Aggression - - > Behavioural Aggression	4.654 [3.084, 6.773] ***	0.937	0.366 [0.193, 0.54] ***	0.089
Adaptive Coping - - > Adaptive Coping	1.221 [0.927, 1.591] ***	0.175	0.862 [0.687, 1.037] ***	0.089
Maladaptive Coping - - > Maladaptive Coping	0.133 [0.071, 0.233] **	0.041	0.293 [0.052, 0.534] **	0.123
Support Coping - - > Support Coping	2.926 [2.388, 3.593] ***	0.321	1 [1, 1] ***	0

Working Memory Problems - > Working Memory Problem	18.432 [12.862, 26.364] ***	3.277	0.505 [0.28, 0.73] ***	0.115
Perceived Stress - - > Perceived Stress	53.242 [53.242, 53.242] ***	0	1 [1, 1] ***	0
Perceived Stress - - > List of Threatening Experiences	2.095 [2.095, 2.095] ***	0	0.112 [0.112, 0.112] ***	0
List of Threatening Experiences - - > List of Threatening Experiences	6.548 [6.548, 6.548] ***	0	1 [1, 1] ***	0

*<0.05, **<0.01, ***<0.001