

**Identification of Night Eating Behaviour and Investigation
Into its Characteristics in an Obese Population**

by

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of the degree of Doctor of Philosophy at the University of Central Lancashire**

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Student Declaration

Concurrent registration for two or more academic awards

I declare that while registered as a candidate for the research degree, I have not been a registered candidate or enrolled student for another award of the University or other academic or professional institution.

Material submitted for another award

I declare that no material contained in the thesis has been used in any other submission for an academic award and is solely my own work.

Jacqueline Cleator

Methought I heard a voice cry, 'Sleep no more!
Macbeth does murder sleep, the innocent sleep;
Sleep that knits up the ravell'd sleeve of care,
The death of each day's life, sore labour's bath,
Balm of hurt minds, great nature's second course,
Chief nourisher in life's feast;'

Macbeth : Act 2, scene 2. W. Shakespeare

Abstract

Background

Night Eating Syndrome (NES) was characterised in 2003 as comprising; morning anorexia, evening hyperphagia, night-time awakenings, consumption of high calorie snacks during awakenings and an absence of other eating disorders (ED).

Method

An identification study was conducted in a hospital-based UK obesity clinic with 81 individuals undertaking a diagnostic interview and completing a proposed screening tool. Full NES (n=7) and partial NES (n=24) individuals were combined into one Night Eating Behaviour (NEB) group (n=31) and compared to all other participants (n=50). NEB characteristics were identified through qualitative thematic analysis of interview data, based on techniques used by Grounded Theorists. NEB individuals (n=28) were compared with matched controls. In a separate prevalence study, night-eating, sleep quality and suspected Obstructive Sleep Apnoea (OSA) were estimated in 103 participants using the Pittsburgh Sleep Quality Index, Epworth Sleepiness Scale and a validated Night Eating Questionnaire. Correlational analysis was also performed.

Results

In the identification study, full NES was rare (9%). Comparison of the NEB and non-NEB group showed significant differences in mood (p=0.001), work status (p= 0.03), perceived lack of control over eating (p= 0.03) and variability in sleep duration (p<0.01). The study tool successfully identified NEB, but not other ED. Interview analysis identified the compulsive nature of night-eating and chaotic eating patterns. A variety of physical factors affected night-time awakenings. The matched control comparison showed no difference in levels of significant life events, childhood-onset obesity and poor sleep quality. Prevalence study results showed; suspected NES 14.6% (n=15), based on a lower cut score of >25, 3.9% (n=4), based on a higher cut score of >30, poor sleep quality 74% (n= 76) and suspected OSA 32% (n= 33). A strong relationship ($r = 0.55$, $p = < 0.001$) between night-eating and poor sleep quality was found, with day-time sleepiness having no influence over this relationship.

Conclusion

Comparison of the study findings with new NES criteria (2010) shows poor differentiation between NES and morbid obesity. In obese populations, a shift of focus is proposed to an alternative 'Impaired Sleep Syndrome' of which night-eaters may be a sub-group, often with severe depression. Night-eaters also exhibit too many features of other ED for this relationship to be ignored.

Table of Contents

1.	OVERVIEW OF THESIS	17
1.1.	Introduction	17
1.2.	Literature Review	17
1.3.	Identification Study	17
1.3.1.	Rationale	17
1.3.2.	Methods	18
1.3.3.	Results	18
1.4.	Characterisation Study	19
1.4.1.	Rationale	19
1.4.2.	Methods	19
1.4.3.	Results	19
1.5.	Prevalence Study	20
1.5.1.	Rationale	20
1.5.2.	Methods	20
1.5.3.	Results	20
1.6.	Discussion Of Thesis	20
2.	LITERATURE REVIEW	22
2.1.	Introduction	22
2.2.	Prevalence and Evolution of Diagnostic Criteria	22
2.3.	Behavioural Characteristics	26
2.4.	Clinical Characteristics	27
2.5.	Is NES an Eating Disorder?	28
2.6.	Is NES a Sleep Disorder?	29
2.7.	Is NES a Mood Disorder?	33
2.8.	Is NES an Anxiety Disorder?	33
2.9.	Is NES an Obesity-related Disorder?	34
2.10.	Obesity and Depression	35
2.11.	Influence of Obesity-related Co-Morbidities on NES	37
2.12.	Treatments for NES	37
2.12.1.	Obesity Treatments	37
2.12.2.	Mood Treatments	39
2.13.	Therapeutic Level of Intervention	40
2.14.	Clinical Significance	41
2.15.	Identification	42
2.16.	Screening Tools for NES	43
2.17.	Discussion	43
2.18.	Conclusion	45
3.	IDENTIFICATION STUDY	46
3.1.	NES in UK Populations	46

3.2.	Relationship between NES and Obesity-related Co-morbidity	46
3.2.1.	NES and Mood	46
3.2.2.	NES and Sleep	47
3.3.	Aims	47
3.4.	Design	47
3.4.1.	Identification of Individuals with NES	47
3.4.2.	Interview Tools	48
3.4.3.	Development of NES Screening Tool	59
3.4.4.	Testing of the Tool and Sample Size	62
3.5.	Methods	63
3.5.1.	Ethical Approval	63
3.5.2.	Recruitment	63
3.5.3.	Completion of Screening Tool	65
3.5.4.	Additional Data	65
3.5.5.	Identification of Co-morbidity	66
3.5.6.	Statistical Analysis	66
3.5.7.	Confirmation of Diagnosis	66
3.6.	Results of Diagnostic Interviews	69
3.6.1.	Demography	69
3.6.2.	Identification of Cases	70
Table 13 - Diagnosis of Eating Disorder at Interview (JC)		70
3.6.3.	Discussion	71
3.6.4.	Conclusion	75
3.7.	Subgroup Analysis: Comparison between NEB and Non-NEB Subgroups	75
3.7.1.	Demography	75
3.7.2.	Co-morbidity	78
3.7.3.	Eating Disorder Examination Subscales	81
3.7.4.	Stunkard's Night Eating Questionnaire (NEQ 2004)	83
3.8.	Screening Tool Results	84
3.9.	Discussion	85
3.9.1.	Demographics: Age/Gender/BMI	85
3.9.2.	Obstructive Sleep Apnoea, Type 2 Diabetes and Depression	86
3.9.3.	Sleep	88
3.9.4.	Eating Behaviour	89
3.9.5.	Screening Tool	90
3.9.6.	Conclusion	91
4.	CHARACTERISATION STUDY	92
4.1.	Introduction	92
4.2.	Mixed Methods Approach	92
4.3.	Interview Data: Qualitative Theme Analysis	93
4.4.	Glaser and Strauss: Social World Perspective	93
4.5.	Grounded Theory Procedures	94
4.5.1.	Stage 1 - Open Coding	95
4.5.2.	Stage 2 - Axial Coding	95
4.5.3.	Stage 3 - Selective Coding	95
4.6.	Study Methodology	96

4.6.1.	Data Preparation	96
4.6.2.	Coding Procedures	96
4.7.	Results of Qualitative Interviews	97
4.7.1.	Presentation of the Data	97
4.7.2.	Results of Coding Process	98
4.7.3.	Characteristics of the NEB Group	99
4.7.4.	Discussion of Key Findings from the Characterisation Study	118
4.7.5.	Unanswered Questions	125
4.8.	Comparison with Non-NEB Matched Control Group	126
4.8.1.	Rationale for Matching	126
4.8.2.	Gender	126
4.8.3.	BMI	126
4.8.4.	Work Status	127
4.8.5.	Age	127
4.8.6.	Matching Process	128
4.8.7.	Statistical Analysis	129
4.8.8.	Results	130
4.8.9.	Discussion	139
4.8.10.	Unanswered Questions	143
5.	PREVALENCE STUDY	144
5.1.	Rationale	144
5.2.	Methodology	144
5.2.3.	Screening for Obstructive Sleep Apnoea: The Epworth Sleepiness Scale	144
5.2.4.	Measuring Sleep Quality: The Pittsburgh Sleep Quality Index	148
5.2.5.	Screening for NES: The Night Eating Questionnaire (2008)	150
5.3.	Ethical Approval	152
5.4.	Data Collection	152
5.5.	Statistical Analysis	153
5.6.	Results	153
5.6.1.	Comparison between Gender	153
5.6.2.	The Night Eating Questionnaire (NEQ 2008)	154
5.6.3.	The Epworth Sleepiness Scale	155
5.6.4.	The Pittsburgh Sleep Quality Index	156
5.6.5.	Comparison of Demographic Data according to Cut Points	157
5.6.6.	Correlations between Tools	159
5.7.	Discussion	163
5.7.1.	Demographics: Age/Gender/BMI	163
5.7.2.	The Epworth Sleepiness Scale	163
5.7.3.	The Pittsburgh Sleep Quality Index	164
5.7.4.	The Night Eating Questionnaire (2008)	164
5.7.5.	Limitations of the Prevalence Study	166
5.7.6.	Relationship between OSA, Night Eating and Sleep Quality	166
5.7.7.	Conclusion	166
6.	GENERAL DISCUSSION OF THESIS	168
6.1.	Chapter Overview	168
6.2.	Section 1 : Key Findings	168
6.2.1.	Identification Study	168
6.2.2.	Characterization Study	168

6.2.3.	Prevalence Study	169
6.2.4.	Current Thinking in NES	169
6.2.5.	Summary of Criteria Data	180
6.3.	Section 2: Study Design and Study Tools	180
6.3.1.	Emergent Design	180
6.3.2.	The Night Eating Syndrome History and Inventory	181
6.3.3.	The Night Eating Questionnaire (2008)	181
6.3.4.	The Eating Disorder Examination (version 12)	182
6.3.5.	The Beck Depression Inventory	184
6.3.6.	The Epworth Sleepiness Scale	186
6.3.7.	The Pittsburgh Sleep Quality Index	186
6.3.8.	Thematic Analysis	187
6.4.	Section 3: Implications for Future Research and Current Practice	188
6.4.1.	Generalisation of Findings	188
6.4.2.	Future Research.	188
6.4.3.	Implications for Practice	190
6.5.	Personal Reflection	191
6.6.	Conclusion	192
Appendix 1.	The Eating Disorder Examination	193
Appendix 2.	The Night Eating Syndrome History and Inventory (NESHI)	194
Appendix 3.	The Beck Depression Inventory	197
Appendix 4.	NES Study Screening Tool	200
Appendix 5.	Weight Management Clinic Pathway	204
Appendix 6.	Matching of Sub-NES Group Characteristics with 2003 NES Criteria	205
Appendix 7.	List of NVivo Codes	207
Appendix 8.	Pathway of Obesity, NEB and Life Events for each NEB Participant.	212
Appendix 9.	Sleep and Eating Patterns of NEB Group and Matched Controls	223
Appendix 10.	The Prevalence Study Questionnaire	224
	List of References	229

Table Of Tables

Table 1- Changes in diagnostic criteria for NES from 1955-2003.	23
Table 2 - Prevalence of Night Eating Symptoms in Adults	25
Table 3 - Differential characteristics between NES, Noct ES and SRED	31
Table 4 - Differentiation between Overeating and Bulimia according to the EDE	49
Table 5 - EDE version 12 item scores on which AN and BU diagnoses are based.	50
Table 6 - EDE version 12 subscale items	50
Table 7 - Items on which a diagnosis of Binge Eating Behaviour is based	54
Table 8 - Study criteria for NES and sub-threshold NES	55
Table 9 – Level of agreement between JC and dietitian in identifying individuals with NES with a diagnostic interview. Agreement expressed as a kappa coefficient.	68
Table 10 - Level of agreement between JC and dietitian in identifying individuals with BU with a diagnostic interview. Agreement expressed as a kappa coefficient.	68
Table 11 - Level of agreement between JC and dietitian in identifying individuals with BED with a diagnostic interview. Agreement expressed as a kappa coefficient.	69
Table 12 - Demographic details of study population compared to obesity clinic audit conducted in January 2005.	69
Table 13 - Diagnosis of Eating Disorder at Interview (JC)	70
Table 14 - Comparison of characteristics between the NEB group and non-NEB group. Differences reported as mean (SD) and using independent samples t tests unless otherwise stated.	76
Table 15 - Comparison of the proportion of males vs females in the study group with type 2 diabetes, OSA and depression. Significance tests performed using chi square analysis.	78
Table 16 - Comparison of characteristics of participants with and without type 2 diabetes, OSA and depression within the whole study group. Differences reported as mean (SD) using independent samples t tests.	79
Table 17 - Comparison of the proportion of individuals in the NEB group and Non-NEB group with type 2 diabetes, OSA and depression. Significance tests performed using chi square analysis.	79
Table 18 - Comparison of characteristics of participants with type 2 diabetes, OSA and depression between the NEB and non-NEB group. Differences reported as mean (SD) and using independent samples t tests unless otherwise stated.	80
Table 19 - Comparison of characteristics between participants with and without type 2 diabetes, OSA and depression within the NEB group only. Differences reported as means (SD) and using independent samples t tests.	81

Table 20 - Comparison of the NEQ item scores and total score by NEB classification using independent samples t tests.	83
Table 21 - Example of categories and sub-categories identified from a section of narrative from a study participant using open coding.	96
Table 22 - List of all categories, examples of 4 sub-categories per category and total number of sub-categories identified through the code of 30 NEB transcriptions.	98
Table 23 - Categorisation of traumatic life events occurring in childhood/teenage years experienced by the NEB group (n=30)	100
Table 24 - Categorisation of traumatic life events occurring in adulthood experienced by the NEB group (n=30)	102
Table 25 - Patient reported causes by category of current night-time awakenings in the NEB group (n=30).	105
Table 26 - Table of activities undertaken either before bed or during the night by the NEB group (n=30).	107
Table 27 - Strategies used by NEB group participants to change their eating and sleep patterns	108
Table 28 - Table showing the sleeping arrangements of the NEB group (n=30)	109
Table 29 - Types of foods chosen by participants when night-eating and the number of participants choosing each item.	110
Table 30 - Range of eating and food-related emotions reported by the NEB group.	113
Table 31 - Range of general emotions described by the NEB group	114
Table 32 - Table showing number of NEB individuals and non-NEB individuals who were matched according to gender, BMI, age and work status strata.	129
Table 33 - Pairwise comparison of the numbers of individuals with childhood onset obesity in the matched NEB group and non-NEB matched control group.	130
Table 34 - Pairwise comparison of the numbers of individuals experiencing traumatic life events in the matched NEB group and non-NEB matched control group.	130
Table 35 - Categorisation of traumatic life events experienced by the matched non-NEB and non-NEB matched control groups and number of individual experiencing such events. Some individuals experienced more than one event.	131
Table 36 - Comparison of the NEQ and BDI sleep-related item scores between the matched NEB and non-NEB matched control group using paired t tests (A higher score in both the NEQ and BDI items indicates a greater degree of dysfunction).	132
Table 37 - Categorisation of activities undertaken either before bed or during the night by the non-NEB matched control group (n=28) and the matched NEB group (n=28) and the number of individuals undertaking each activity. NB some individuals undertook several activities.	133
Table 38 - Categorisation of sleeping arrangements of the non-NEB matched control group and matched NEB group and number of individuals per sleeping arrangement (n=28).	133

Table 39 – Categorisation of reasons for current night-time awakenings in the non-NEB matched control group and matched NEB group and number of participants per reason for night-time awakening. N.B. some individuals in both groups give more than one reason for waking	134
Table 40 - Pairwise comparison of the NEQ (2004) eating-related items between the matched NEB and non-NEB matched control group using paired t-tests. A higher score on all items indicates a greater degree of dysfunction.	136
Table 41 - Comparison of the EDE v12 subscale and global scores between the matched NEB and non-NEB matched control group using the Paired Sample Sign test (n=28 unless otherwise stated) ¹ .	136
Table 42 - Number of references to control over eating per individual in the non-NEB matched control group	137
Table 43 - Pairwise comparison of the BDI item scores and NEQ (2004) mood-related item scores between the matched NEB and non-NEB matched control group using paired t tests. A higher total score and higher score on each item indicates a greater degree of dysfunction.	138
Table 44 - Comparison of the characteristics and screening tool total scores between males and females of the whole study group. Differences reported as mean (SD) and using independent samples t tests unless otherwise stated (¶ Binomial test).	154
Table 45 – ¹ Mean (SD) NEQ (2008) individual item scores, mean (SD) total score (excluding item 13) and percent scoring >25 and >30 of the whole prevalence study group. ² Mean (SD) scores from the verbally administered NEQ (2004) used in the Identification study.	155
Table 46 - Mean Epworth Sleepiness Scale individual item scores, mean (SD) total score and percent scoring above 10 for the whole study group.	156
Table 47 - Mean Pittsburgh Sleep Quality Index individual component scores, mean (SD) total score and percent scoring above 5 for the whole study group.	156
Table 48 - Comparison of characteristics between participants with suspected OSA and those without suspected OSA. Differences reported as means (SD) and using independent samples t tests. (¶ Chi square analysis)	157
Table 49 - Comparison of characteristics between participants with poor quality sleep and those with good quality sleep. Differences reported as means (SD) and using independent samples t tests. (¶ Chi square analysis)	157
Table 50 - Comparison of characteristics between participants with suspected NES and those without suspected NES at a cut point of > 25 on the NEQ. Differences reported as means (SD) and using independent samples t tests. (¶ Chi square analysis).	158
Table 51 - Comparison of characteristics between participants with suspected NES and those without suspected NES at a cut point of > 30 on the NEQ. Differences reported as means (SD) and using independent samples t tests (¶ Chi square analysis).	158

Table 52 - Comparison of the proportion of participants in the study group with a PSQI score > 5 and NEQ >25. Significance tests performed using Fisher's Exact Test (p= 0.34).	160
Table 53 - Comparison of the proportion of participants in the study group with a PSQI score > 5 and NEQ >30. Significance tests performed using Fisher's exact test (p= 0.57).	160
Table 54 - Comparison of the proportion of participants in the study group with an ESS score >10 and PSQI >5. Significance tests performed using Fisher's exact test (p = 0.09).	161
Table 55 - Comparison of the proportion of participants in the study group with an ESS score > 10 and < 10 and NEQ >25 and < 25. Significance tests performed using Fishers exact test (p= 0.35).	162
Table 56 - Comparison of the proportion of participants in the study group with an ESS score >10 and NEQ >30. Significance tests performed using Fisher's exact test (p= 1.0).	162
Table 57 - Comparison between the 2003 and 2010 criteria for NES	171
Table 58 - Matching of sub-threshold NES group characteristics with the 2003 NES criteria	205
Table 59 - Partial NES Developing in Childhood and Teenage Years	212
Table 60 - Full NES Developing in Childhood and Teenage Years	215
Table 61 - Partial NES Developing in Adult Years	216
Table 62 - Full NES Developing in Adult Years	220

Table of Figures

Figure 1- Flow chart of recruitment	65
Figure 2 - Percentage distribution of participants in the NEB and Non-NEB group according to age category.	77
Figure 3 - Percentage distribution of participants in the NEB and Non-NEB group according to BMI category	77
Figure 4 - Percentage distribution of BDI scores per BDI category indicating degree of depression for the NEB and non-NEB groups (the black lines are super-imposed linear trend lines for each group).	78
Figure 5 - Comparison of the median EDE v12 individual item scores between the NEB and non-NEB groups using the Mann Whitney U test.	82
Figure 6 - Comparison of the median EDE v12 subscale and global scores between the NEB and non-NEB groups using the Mann Whitney U test.	82
Figure 7 - Relationship between NEQ total score and BDI total score for all study participants (n=81).	84
Figure 8 - Embedded design using Morse's notation system (1991)	93
Figure 9 - Diagrammatic representation of the pathways between childhood onset NEB and obesity	118
Figure 10 - Diagrammatic representation of the pathways between adult onset NEB and obesity	119
Figure 11 - Relationship between NEQ total score and PSQI total score for all study participants (n=103).	159
Figure 12 - Relationship between ESS total score and PSQI total score for all study participants (n=103).	161
Figure 13 - Relationship between NEQ total score and ESS total score for all study participants (n=103).	162

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List of Abbreviations

ADL	Activities of Daily Living
AN	Anorexia Nervosa
ANCOVA	Analysis of Covariance
ANOVA	Analysis of Variance
APMRT	Abbreviated Progressive Muscle Relaxation Technique
AR	Adiposity Rebound
ASQ	Attributional Style Questionnaire
BDI-I	Beck Depression Inventory version I
BDI-IA	Beck Depression Inventory version IA
BDI-II	Beck Depression Inventory version II
BED	Binge Eating Disorder
BES	Binge Eating Scale
BITE	Bulimic Investigatory Test, Edinburgh
BMI	Body Mass Index
BN	Bulimia Nervosa
BSQ	Body Shape Questionnaire
BU	Bulimia subscale on EDE version 11
BULIT	Bulimia Test
CAGE	CAGE questionnaire (screening tool for alcohol dependency)
CI	Confidence Interval
COPD	Chronic Obstructive Pulmonary Disease
CPAP	Continuous Positive Airway Pressure
DEBQ	Dutch Eating Behaviour Questionnaire
DIMS	Disorders of initiating and maintaining sleep
DOES	Disorders of excessive somnolence
DSM III	Diagnostic & Statistical Manual of Mental Disorders 3 rd edition
DSM IV	Diagnostic & Statistical Manual of Mental Disorders 4 th edition
EAT	Eating Attitudes Test
EC	Eating Concern subscale on EDE version 12
ED	Eating disorders
EDE v12	Eating Disorder Examination version 12
EDI	Eating Disorder Inventory
EDNOS	Eating Disorder Not Otherwise Specified
EDS	Eating Disorder Service
ESS	Epworth Sleepiness Scale
EWL	Excess weight loss
GP	General Practitioner
GSQ	Good Sleep Quality
HRQL	Health-Related Quality of Life
HRSD	Hamilton Rating Scale for Depression
IQR	Interquartile range
IRT	Item Response Theory
LCA	Latent class analysis
MAACL	Multiple Affect Adjective Checklist Depression Scale
MET	Metabolic equivalent
MMPI-D	Minnesota Multiphasic Personality Inventory Depression Scale
MSLT	Multiple Sleep Latency Test
MWT	Maintenance of Wakefulness Test
NHS	National Health Service
NEB	Night Eating Behaviour
NEQ	Night Eating Questionnaire
NES	Night Eating Syndrome
NESHI	Night Eating Syndrome History and Inventory

NESQ	Night Eating Syndrome Questionnaire
Noct ES	Nocturnal Eating Syndrome
NREM	Non-rapid eye movement sleep
NSRED	Nocturnal Sleep-Related Eating Disorder (now known as SRED)
OPD	Outpatient Department
OSA	Obstructive Sleep Apnoea
PIS	Patient information sheet
PRIME-MD	Primary Care Evaluation of Mental Disorders
PSQ	Poor Sleep Quality
PSQI	Pittsburgh Sleep Quality Index
PTSD	Post Traumatic Stress Disorder
R	Restraint subscale on EDE version12
RAFFT	RAFFT questionnaire (screening tool for substance misuse)
REM	Rapid eye movement sleep
ROC	Receiver Operator Curve
RS	Revised Restraint Scale,
SADS	Schedule for Affective Disorders and Schizophrenia
SC	Shape Concern subscale on EDE v12
SCL-90-R	The Symptom Checklist -90- revised
SCOFF	SCOFF Eating Disorders Questionnaire
SD	Standard deviation
SEM	Standard error of the Mean
SF- 36	Short Forms 36
SPECT	Single photon emission computed tomography
SPSS	Statistical Package for the Social Sciences
SRED	Nocturnal Sleep-Related Eating Disorder (previously NSRED)
SSRI	Selective serotonin reuptake inhibitor
TEFQ	Three-Factor Eating Questionnaire
UK	United Kingdom
WALI	Weight and Lifestyle Inventory
WAS	World Assumptions Scale
WC	Weight Concern subscale on EDE version12
WMC	Weight Management Clinic
ZSDS	Zung Self-Rating Depression Scale

1. OVERVIEW OF THESIS

1.1. *Introduction*

Levels of obesity in the United Kingdom continue to rise at an alarming rate and the development of obesity services is gathering pace to meet the increased demand for successful and enduring treatments. Many patients in specialist weight management clinics (in which most have a body mass index (BMI) $> 40 \text{ kg/m}^2$) experience refractory obesity and the influences on the development and maintenance of morbid obesity continue to be investigated. In recent years, interest has grown in the relationship between obesity and Night Eating Syndrome (NES), which features disordered eating, mood and sleep. Early investigations into NES were hampered by evolving diagnostic criteria and its characteristics and clinical significance continue to be debated. The current prevalence and impact of NES in UK obesity clinics is unknown. The overall aim of this study was to identify NES and explore its characteristics in an obese UK population.

1.2. *Literature Review*

This chapter examines the evolution of the diagnostic criteria for NES, which despite Stunkard's attempt at standardisation in 2003, continue to be refined. Evidence of the behavioural and clinical characteristics of NES and its clinical significance in diverse populations is reviewed. The relationship between NES, sleep, mood, other eating disorders, obesity and obesity-related co-morbidity in particular is examined. The chapter concludes with a discussion of the problems in identifying NES and the lack of qualitative evidence exploring the individual's experience of NES.

1.3. *Identification Study*

1.3.1. *Rationale*

This section presents the rationale for conducting a study aimed at identifying NES in a UK hospital-based weight management clinic (WMC). When this study was first conceived, there was no tool available to identify NES and this section discusses the rationale for developing a screening tool for NES in order to identify what proportion of an obese clinic population would screen positive for NES using the tool. The rationale for using existing tools to explore whether low mood and depression would also feature in obese UK individuals with NES is discussed.

Identification of individuals with NES would then allow for the relationship between NES and other NES related co-morbidity to be explored.

1.3.2. *Methods*

The study design adopted for the identification study is discussed. The development of a screening tool based on the 2003 criteria, Stunkard's unvalidated questionnaire (NEQ 2004) and items to exclude other eating disorders is presented. Testing the tool would require a cohort of individuals identified with NES through a diagnostic interview. The rationale for the interview design is discussed along with justification for the choice of interview tools, which includes The Eating Disorder Examination (EDE) version 12, supplementary questions to exclude Binge Eating Disorder, The Night Eating Syndrome History and Inventory (NESHI) and Stunkard's unvalidated NEQ. The Beck Depression Inventory version IA (BDI-IA) was also included. Sample size calculations are also presented. The methods used to recruit eighty four individuals from a local obesity clinic for an interview to determine NES and to complete the proposed screening tool are discussed. Recruitment strategies included the use of purposive sampling, with individuals referred for interview if they reported symptoms suggestive of NES, or self-referring after being given a flyer asking 3 questions about night-eating. Methods for the collection of demographic data, sleep duration, work status and obesity-related co-morbidity are also presented.

1.3.3. *Results*

Complete data sets were obtained on 81 individuals and analysis of these data and key findings are presented. Full NES was rare (n=7). Individuals with full or partial NES were combined to form one Night Eating Behaviour group (NEB) (n=31) and compared with all other subjects (n=50). Differences were noted in mood, work status, eating behaviour and sleep patterns. Night-eating items of the study tool were successful in identifying NES, although items used to identify other eating disorders identified too many false positive cases. The section concludes by explaining the rationale for halting further development of the tool and taking a sideways step to undertake conceptual analysis of the interview data in order to identify the key constructs of 'night-eating' as experienced by individuals with NEB.

1.4. *Characterisation Study*

1.4.1. *Rationale*

The interviews had a very structured design with response categories determined in a close-ended scoring system allowing for the collection of quantitative data. During interviews participants spoke qualitatively about their personal experience of NEB and offered in-depth knowledge into the different factors affecting their behaviour. This section explains the rationale for combining qualitative analysis of this interview data within an overall quantitative structure, in an ‘embedded mixed method’ design.

1.4.2. *Methods*

The methodological approach used to analyse interview data is explained, including the use of the NVivo data collection system and analysis techniques developed by Grounded Theorists. Data was coded systematically and organised into categories until common themes emerged. Comparisons were also made using quantitative data where appropriate. A quantitative and qualitative comparison of the themed analysis results was also undertaken with a matched control group, in order to establish the likely impact of obesity on key findings. Twenty eight NEB individuals were matched with individuals from the non-NEB group according to age, gender, BMI and work status. Justification for the choice of matching variables is given.

1.4.3. *Results*

The results of the interview analysis are described in detail, including the influence of obesity and significant life events on the development of NEB. Other key themes described include the effect of poor sleep on the onset and maintenance of NEB, the compulsive nature of night-eating, chaotic daytime eating patterns, conflictful relationships and the presence of negative emotions relating to eating in general and general daily living. A variety of physical factors, both directly and indirectly obesity – related, appear to influence current night-time awakenings. Comparison with the matched control group showed significant differences in mood, eating patterns and control over eating. Disturbed sleep was a distinctive feature of both groups. A wide range of physical causes for waking and poor sleep quality were prevalent across both groups making it difficult to determine the role of NEB in this relationship.

1.5. *Prevalence Study*

1.5.1. *Rationale*

This chapter describes the rationale for conducting a more systematic evaluation of the relationship between sleep quality, NEB and co-morbidity in a separate cohort of individuals from the WMC. Justification is given for the use of validated tools, including the Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS) and an updated NEQ validated in 2008. The strengths and weaknesses of each tool are described and cut points for use in data analysis are identified. (ESS > 10, PSQI >5, NEQ \geq 25 (lower cut point), NEQ \geq 30 (higher cut point)).

1.5.2. *Methods*

The methods used to conduct the prevalence study in the WMC are described. Analysis of data was based on 103 individuals with complete data sets. Differences in characteristics and individual screening tool scores between gender were compared and the proportions of individuals above and below the screening tool cut points were also compared. Pearson Correlation Coefficients were calculated to examine relationships between the screening tool scores.

1.5.3. *Results*

Prevalence study results showed suspected NES in 14.6% (n=15) of the study cohort based on the lower cut score of >25 and 3.9% (n=4) based on the higher cut score of >30. Poor sleep quality was noted in 74% (n= 76) and suspected Obstructive Sleep Apnoea (OSA) in 32% (n= 33). A strong relationship ($r=0.55$, $p < 0.001$) between night-eating and poor sleep quality was found, with day-time sleepiness having no influence over this relationship. This suggests that OSA, of which day-time sleepiness is a symptom, may be a separate construct to NES.

1.6. *Discussion Of Thesis*

Strengths and weaknesses of study methodology are examined. In particular, the appropriateness of using these studies' tools for future NES research is discussed. Results from the identification study, characterisation study and prevalence study are summarised and the key findings discussed in the light of revised NES criteria published in 2010. The new criteria appear to differentiate NES poorly in morbidly obese populations. The predominance of sleep difficulties in this population suggests a shift of focus is required and an alternative 'Impaired Sleep

Syndrome' is proposed, of which night-eaters may be a sub-group likely to experience severe depression. Results also suggest that night-eaters exhibit too many features of other ED for this relationship to be ignored and that previous guidance to separate NES from other ED was probably counter-productive. Future treatment options are proposed based on Social Rhythm Theory.

2. LITERATURE REVIEW

2.1. Introduction

It is estimated that of the 1 billion people worldwide who are overweight, 300 million are clinically obese (BMI >30kg/m²) (World Health Organisation 2003). As obesity-related co-morbidities, such as cardiovascular disease and type 2 diabetes, continue to impact heavily on the health economy, the need for successful and enduring obesity treatments is essential. During the last decade there have been sustained attempts to understand how disordered eating patterns contribute to the development of obesity. Attention has focused particularly on Bulimia Nervosa (BN) and Binge Eating Disorder (BED), two patterns of eating which share common features. BN is defined as eating larger than normal amounts of food in a discrete time period (usually 2 hours), with associated loss of control and the presence of compensatory mechanisms such as purging. Compensatory mechanisms are absent with BED (Spitzer et al. 2006; Spitzer et al. 1993).

A less well established cluster of behaviours called Night Eating Syndrome (NES) has been described more recently as a unique disorder of eating, mood and sleep, characterised by a phase onset delay of morning appetite and continuation of evening eating (O'Reardon et al. 2004). NES was defined by Stunkard (Stunkard & Allison 2003), a psychiatrist specialising in eating disorders as:

1. Morning anorexia, even if subject eats breakfast.
2. Evening hyperphagia. At least 50% of the daily caloric intake is consumed in snacks after the last evening meal.
3. Awakenings at least once a night, at least 3 nights a week.
4. Consumption of high calorie snacks during the awakenings on frequent occasions.
5. The pattern occurs for a period of at least 3 months.
6. Absence of other eating disorders.

2.2. Prevalence and Evolution of Diagnostic Criteria

Establishing the true prevalence of NES has traditionally been problematic due to evolving diagnostic criteria (Table 1). Stunkard, Grace and Wolff (1955) proposed the following diagnostic criteria; 'morning anorexia', 'hyperphagia until midnight on 50% of nights' and 'sleep

onset insomnia' and using these, suggested that 80% of 25 American subjects with obesity from a research clinic had features supporting a diagnosis of NES.

Table 1- Changes in diagnostic criteria for NES from 1955-2003.

<i>Author</i>	<i>Year</i>	<i>Criteria</i>
<i>Stunkard</i>	1955	<ul style="list-style-type: none"> • morning anorexia, • nocturnal hyperphagia until midnight on 50% of nights, • sleep onset insomnia
<i>Kuldau</i>	1986	<ul style="list-style-type: none"> • morning anorexia, • eating later in day, • on and off evening eating without enjoyment, • sleep onset insomnia, • evening tension
<i>Rand</i>	1993	<ul style="list-style-type: none"> • morning anorexia, • excessive evening eating, • evening tension and/or feeling upset, • insomnia
<i>Stunkard</i>	1996	<ul style="list-style-type: none"> • no appetite for breakfast, • 50% or more of food intake after 7pm, • trouble getting to sleep and/or staying asleep.
<i>Powers</i>	1999	<ul style="list-style-type: none"> • More than 25% of total energy intake after evening meal, • trouble sleeping, • appetite in morning
<i>Ceru-Bjork</i>	2001	<ul style="list-style-type: none"> • As per Stunkard 1996 and • waking up at night and getting out of bed to eat and/or after having gone to bed, getting out of bed to eat or eating in bed
<i>Napoletano</i>	2001	<ul style="list-style-type: none"> • morning anorexia, • pm hyperphagia, • emotional distress, • sleep difficulties
<i>Adami</i>	2002	<ul style="list-style-type: none"> • morning anorexia, • more than 25% of total energy intake after evening meal, • trouble falling and/or staying asleep most nights
<i>Stunkard</i>	2003	<ul style="list-style-type: none"> • morning anorexia, even if subject eats breakfast. • Evening hyperphagia. At least 50% of the daily caloric intake is consumed in snacks after the last evening meal. • Awakenings at least once a night, at least 3 nights a week.

- Consumption of high calorie snacks during the awakenings on frequent occasions.
 - The pattern occurs for a period of at least 3 months.
 - Absence of other eating disorders
-

Thirty years later an attempt was made to re-establish the extent of NES in the obese through a series of comparisons between healthy individuals of normal weight and subjects awaiting obesity surgery in the US. Subjects were assessed using an unpublished self-report questionnaire with 2 extra items: 'eating with tension' and 'without enjoyment' added to the 1955 criteria. Comparison between healthy weight (89m/143f) controls and morbidly obese individuals (16m/84f) indicated a prevalence of 0.5% and 15% respectively (Kuldau & Rand 1986; Rand & Kuldau 1986) (Table 2). A larger study followed (888m/1209f controls, 3m/252f obese), indicating prevalence levels of 1.5% and 25% (Rand, Macgregor, & Stunkard 1997; Rand & Kuldau 1993). These studies suggest prevalence much lower than Stunkard's original estimates, but inconsistencies in reporting the degree of overweight across studies make it difficult to assess which factors contribute to the disparity.

In 1996, Stunkard revisited the syndrome. He conducted individual standardised interviews with subjects recruited through the American mass media for a study investigating Binge Eating Disorder (BED) (Stunkard et al. 1996). Subtle changes to the original NES criteria were made in this study to include no appetite for breakfast, 50% or more of food intake after 7pm and trouble getting to sleep and/or staying asleep, as well as nocturnal awakenings to eat. Prevalence of NES was 13.7% in this group of 102 subjects (mean BMI 37.8kg/m²) and a similar prevalence of 15% was found in a second sample of 79 BED subjects (mean BMI 35.3kg/m²) in a pharmacology trial. A lower prevalence of 8.9% was described in a sample of female participants (n=40) in a weight loss programme (mean BMI 35.5kg/m²) (Stunkard et al. 1996) and Adami et al. (2002), interviewing an Italian population with a slightly higher BMI, reported a similar prevalence of 7.8%, to Stunkard's weight loss programme group, although criteria were modified to include insomnia on most nights. Powers et al. (1999), interviewing subjects before obesity surgery, noted a NES prevalence of 10%, whilst Colles, Dixon & O'Brien (2008b) noted higher levels (17.1%) in a lighter pre-surgery group.

Table 2 - Prevalence of Night Eating Symptoms in Adults

<i>Author</i>	<i>Year</i>	<i>Population</i>	<i>Degree of overweight Mean BMI (kg/m²)% BMI >30</i>	<i>N</i>	<i>Prevalence (%)</i>
Stunkard	1955	Obesity OPD	68% overweight normal weight	25 38	80 0
Kuldau	1986	General population Pre obesity surgery	normal weight morbidly obese	232 100	0.5 15
Rand	1993	General population Pre obesity surgery	24.9 52	2097 255	1.5 25
Stunkard	1996	Self report BED Weight loss study BED subjects	37.8 35.3 35.5	102 79 40	13.7 8.9 15
Powers	1999	Obesity surgery	53.4	116	10
Aronoff	2001	Obesity OPD	55	110	51
Ceru-Bjork	2001	Obesity OPD	40	194	14
Gluck	2001	Obesity OPD	36.5	76	14
Napoletano	2001	Obesity OPD Some BED subjects	41.1	83	43
Adami	2002	Obesity OPD	43.5	166	7.8
Anderson	2004	General population (MONICA project)	10%	2111	8
Morse	2006	Diabetes 1&2 OPD	32 %	714	9.7
Colles	2008	Obesity OPD	44.3	129	17.1%
Lundgren	2010	Psychiatric OPD	37.2	68	25

Other studies that appear to be based on the 1996 criteria have shown prevalence similar to that found in Stunkard's mass media sample despite using questionnaires instead of interviews as the diagnostic tool. A study using an unpublished 'Night Eating Questionnaire' with obesity-research-centre subjects revealed a prevalence of 14% (Gluck, Geliebter, & Satov 2001). Ceru-Bork, Anderson & Rossner (2001), using an eating-and-sleep habit questionnaire, in a larger population of Swedish subjects with a slightly higher mean BMI of 40 kg/m² showed a similar prevalence of 14%. A study of self-reported levels of NES in subjects with diabetes based on a

single question about eating late at night revealed a prevalence of 9.7% (Morse et al. 2006). Lundgren et al. (2010) reported a prevalence of 25% in a psychiatric population based on 'conservative criteria'. Exceptionally, Aronoff, Geliebter & Zammit (2001), reported a prevalence of 51% in American obesity clinic attendees (mean BMI of 55 kg/m²) although as the authors acknowledge, the methodology used may have resulted in biased reporting. The only other similar reported prevalence of 43% in a comparable population of obese clinic subjects used a more structured NES interview but was acknowledged by the author to include some subjects with features of BED (Napolitano et al. 2001).

Interpretation of prevalence data, must take the differing criteria into account, although generally, levels are lower when more stringent criteria are adopted. Striegel-Moore et al. (2006b) analysed food diary data from subjects in a national survey and found 33% of individuals ate more than 25% of their daily calories between 7pm and 5am, 11.5% ate more than 50% in this period and only 11% ate between 11pm and 5am. Similarly, of 106 responders to a newspaper advert about night-eating, 31% met Stunkard's 1955 criteria and 13.2% his later 1996 criteria (de Zwaan et al. 2006).

2.3. *Behavioural Characteristics*

Although early work has suggested a possible link between obesity, disrupted eating, sleep, mood and NES, it is clear that without an understanding of the key characteristics of the syndrome on which to base clearly defined criteria, early researchers refined and adapted the criteria to incorporate either their own observations or study population characteristics, or concentrated on criteria for which they had readily available data. In 1999 a landmark study explored the behavioural and clinical characteristics of NES in Norwegian overweight females (BMI 28.5kg/m²) and concluded that the main feature of NES was eating and sleep desynchronisation (Birketvedt et al. 1999). This involved disassociation between eating and sleeping rhythms characterised by a phase onset delay of morning appetite and continuation of evening eating.

On average, subjects woke 3.6 times a night and snacked in order to return to sleep. Significantly more of the total energy intake was consumed at night as compared to non-NES subjects (56% versus 15%) although total 24hr energy intake varied only moderately between the

two groups. This was confirmed by retrospective psychometric modelling on the temporal eating and amount of intake of the study subjects (Boston et al. 2008). A later replication study on American subjects (BMI 34.9 kg/m²) showed similar patterns. Subjects in both groups had similar sleep onset, offset and duration times, but the NES group woke on average 1.5 times per night and ate during 74% of the awakenings. The control group woke on average 0.5 times and did not eat. Food intake after the evening meal was three fold greater in the NES group (34.6% v 10%) (O'Reardon et al. 2004). Lundgren et al. (2008a) confirmed these differences.

2.4. *Clinical Characteristics*

An understanding of the neuro-endocrine patterns of NES may help to understand its pathogenesis. Healthy adults have a nocturnal rise in plasma leptin and melatonin levels which suppress appetite and induce sleep respectively. Cortisol levels can be raised as a response to stress. Participants in a study by Birkedvedt et al. (1999) showed a distinctive neuro-endocrine pattern with high cortisol levels and an attenuation of the nocturnal rise in plasma leptin and melatonin levels. Similar leptin and melatonin responses have also been found in normal weight, healthy, sleep-deprived volunteers (Mullington et al. 2003; Qin et al. 2003). The replication study of O'Reardon et al. (2004), however, showed only a trend towards higher Thyroid Stimulating Hormone levels for the NES subjects throughout the 25 hour testing period and significantly lower ghrelin and higher insulin and glucose levels overnight, as would be expected with an increased nocturnal food intake (Qin et al. 2003). Although ghrelin levels were not measured in the early study, the lowest ghrelin level in the replication study was at 2am. It is hypothesised that this would be a typical wake time for an NES subject and is consistent with evidence that ingested food suppresses ghrelin in a dose-dependent manner, as high ghrelin levels are associated with hunger. However, the low level is at variance with findings by Rosenhagen et al. (2005) that ghrelin levels were consistently raised in one NES female (BMI 23.5 kg/m²) in comparison to normal weight controls and consistent with reports of increased night-time hunger in NES (Boseck et al. 2007).

Both studies have confirmed the circadian shift in meal patterns, despite no difference in energy intake, although the degree of shift and number of nocturnal awakenings was different (Birkedvedt et al. 1999; O'Reardon et al. 2004). The reasons for these differences are not entirely

clear. Both studies had a relatively small sample size (12 and 15 subjects), although mean BMI differed somewhat (28.5kg/m² v BMI 36 kg/m²) and there were some methodological differences, with a rigid meal pattern imposed in the early study which is not typical for NES. Crucially, criteria used to define NES differed, with the first using Stunkard's 1996 criteria and the latter the 2003 criteria, which required at least 50% of calorie intake after the evening meal and awakenings to eat 3 or more times a week (Stunkard & Allison 2003).

2.5. *Is NES an Eating Disorder?*

It is clear that NES sufferers have 'disordered' eating patterns and much early debate centred on the theory that NES is a subgroup of BED as suggested by early prevalence studies (Napolitano et al. 2001;Stunkard et al. 1996). BED was included in the Diagnostic and Statistical Manual of Mental Disorders (DSM IV: 1994) as a research category in need of further study and is not officially classified as an eating disorder. Greeno,Wing & Marcus (1995) examined 40 American obese female subjects with BED and 39 controls for crossover with NES, but concentrated on some NES features only, exploring self -reports of night-time eating episodes, sleep and mood. The BED group had 6 night eating episodes in 10 days and the control group had none, leading the author to conclude that NES is associated with BED, although it is unclear whether the eating episodes relate to 1 or to 6 separate individuals. A similar study of 207 subjects with BED found that 28% reported 'waking up at night and eating'(Grilo & Masheb 2004). Colles (2007), studying a combined sample of general community, weight loss support group and bariatric surgery subjects found a NES prevalence of 11%, with co-existing NES and binge eating in 4.4% of the total cohort. Other studies report a more modest overlap (de Zwaan et al. 2006).

At the time of developing the 2003 criteria, Stunkard favoured the view that NES and BED are separate constructs with little cross-over and a growing body of evidence supported this (Geliebter 2002;Stunkard & Allison 2003). NES appears to have a stronger family link, with children seven times more likely to have NES if their parents suffer from it (Lamerz et al. 2005;Lundgren, Allison, & Stunkard 2006). A longitudinal study of 63 (15m/48f) Italian patients before and after bilio-pancreatic surgery found 27 (42%) to have BED at pre-operative interview and 7.9% (n=5) to have NES. Three years later, however, only 1 individual had BED but 4 out of

5 still had NES, suggesting that these are two separate behavioural constructs responding differently to treatment (Adami, Meneghelli, & Scopinaro 1999).

Subjects with BED also had higher disinhibition and hunger scores on the Three Factor Eating Questionnaire used by Adami et al. (2002). Other studies support these observations, with Allison finding that NES subjects ate fewer meals in the day and more in the night (1300 calories are consumed during an average binge as opposed to 271 per average night-time snack) than BED and control subjects. BED subjects reported less sleep disturbance and morning anorexia and more objective bulimic and overeating episodes, shape and weight concerns, disinhibition and hunger (Allison et al. 2005b). Interesting emerging work in bariatric surgery subjects puts less emphasis on the similarities and differences between the disorders and more on the influence of control as a predictor of outcome. Subjects with higher levels of uncontrolled eating post operatively, regardless of eating disorder diagnosis, had poorer percentage weight loss and elevated psychological distress (Colles, Dixon, & O'Brien 2008b).

2.6. *Is NES a Sleep Disorder?*

One prominent feature of the studies attempting to characterise NES is that night snacks are typically carbohydrate rich (73%), with a high carbohydrate to protein ratio (7:1). This has led to the theory that NES is driven by disrupted sleep, with night snacks chosen to increase the availability of tryptophan for transport into the brain and conversion to serotonin, thus facilitating the restoration of sleep (Birketvedt, Sundsfjord, & Florholmen 2002). The clustering of disordered eating and sleeping patterns together in one syndrome is of interest to different research disciplines, with 'sleep researchers' highlighting the disordered sleep characteristics. Confusion regarding interpretation and differential diagnosis is also possible because there are several similar sounding syndromes reported in the literature (Table 3).

Nocturnal Eating and Drinking Disorder, for example, is a semantic variant of NES (Ceru-Bjork, Andersson, & Rossner 2001). Nocturnal Eating Syndrome (Noct ES) however, is viewed as a distinct sleep disorder, which gained international classification in 1990 and is described as 'recurrent awakenings associated with an inability to return to sleep without eating or drinking'. This diagnosis focused mainly on young children who were nursing but has somewhat awkwardly been applied to adults (Allen 2000). Using unpublished questionnaires

featuring details of nocturnal eating, prevalence of Noct ES in a sample of 120 (51m/69f) Italian adult subjects with insomnia was estimated to be 5.8% (Manni, Ratti, & Tartara 1997).

Interestingly, in a mixed sample of 700 (190m/510f) American students, eating disorder subjects and obese subjects, Nocturnal Eating Syndrome was shown to be prevalent in 9.4% (Winkelman, Herzog, & Fava 1999).

Nocturnal Sleep-Related Eating Disorder (NSRED), more recently known as SRED, is a much rarer condition and considered to be the opposite pole of the disordered eating spectrum to NES (Howell, Schenck, & Crow 2008; O'Reardon, Peshek, & Allison 2005). Sufferers describe 'a half asleep/half awake' state and impaired consciousness whilst eating at night, often accompanied by sleep walking and restless leg syndrome and may be brought on by sedative hypnotics (Schenck & Mahowald 1994). Food choices are often bizarre and sufferers exhibit a compulsion to eat before returning to sleep. Due to the partial amnesia for night-eating episodes, Noct ES and SRED are classed as parasomnias and viewed as arousal disorders (Allen 2000). The amnesia is assumed to be the key distinction from NES and yet although the level of awareness in NES is assumed to be complete, it is not always reported consistently across studies (Winkelman 2003). One videopolysomnographic study showed all NES subjects to be fully aware (Vetrugno et al. 2006) and yet de Zwann et al. (2006) found 17% of 'night-eaters' to have low awareness whilst night-eating, together with frequent parasomnias such as nightmares and somnambulism and suggests that NES and SRED are the same construct. Parasomnias have been commonly associated with other eating disorders.

Table 3 - Differential characteristics between NES, Noct ES and SRED

	<i>Night eating Syndrome(NES)</i>	<i>Nocturnal Eating Syndrome (Noct ES)</i>	<i>Sleep-related eating Disorder (SRED).</i>
<i>Timing of food intake</i>	After last meal and prior to final awakening	After initiation of sleep and prior to final awakening	After initiation of sleep and prior to final awakening
<i>Level of consciousness</i>	Fully awake	Fully awake	Unconscious
<i>Unusual food intake (inedible food)</i>	Rare	No	Common
<i>Associated problems</i>	Obesity Depression	Restless leg syndrome Depression	Sleepwalking Restless leg syndrome Obesity Depression
<i>Associated medication</i>	None	None	Zolpidem Triazolam Olanzapine Risperidone

Normal sleep comprises 4 distinct phases: Non-rapid eye movement (NREM) sleep stages 1-3 and Rapid Eye Movement (REM) sleep. Stage 1 is known as drowsy sleep and 45-55% of total sleep occurs in stage 2. Stage 3 is also known as slow wave, or delta sleep. The final REM stage is associated with dreaming and accounts for 25% of the total sleep cycle (Silber et al. 2007). Some degree of sleep disturbance is obviously present in NES. Rogers et al. (2006) suggest the main problem may actually be that of sleep maintenance insomnia (staying asleep once having dropped off). During sleep studies, Australian NES subjects (BMI 36 kg/m²) showed normal sleep-wake behaviour, but experienced less phase 2 and phase 3 sleep than non-NES controls, resulting in a lower total sleep time and reduced sleep efficiency (amount of time in bed spent sleeping). In recent years, a gradual rise in obesity in the general population has corresponded with a general reduction in adult sleep from an average of 9 hours to 7.5 hours a night, although slow wave sleep appears less affected than Rapid Eye Movement sleep, suggesting individuals still get sufficient REM sleep, despite the reduction in total hours (Broman, Lundh, & Hetta 1996; Spiegel, Leproult, & Van 1999). Several large population studies have consistently reported a dose-response relationship between short sleep duration and excess

body weight in infants, adolescents and adults of varying ethnicities (Agras et al. 2004;Hasler et al. 2004;Kohatsu et al. 2006;Padez et al. 2005;Patel et al. 2008;Reilly et al. 2005;Vioque, Torres, & Quiles 2000;von Kries R. et al. 2002;Vorona et al. 2005). Long (≥ 9 hours) and short sleep (≤ 6 hours) duration is also linked to increased mortality (Cizza, Skarulis, & Mignot 2005;Youngstedt & Kripke 2004). It is possible that sleep duration may play a part in the pathogenesis of NES, through its association with obesity (Gangwisch et al. 2005;Vorona et al. 2005). Taheri (2006) examined the sleep habits of 1024 adult participants in the Wisconsin Sleep Cohort study and found a correlation between decreased sleep and increases in BMI. Short sleep duration was associated with lower leptin and higher ghrelin levels in fasting morning blood, hormone changes which are typically associated with increased appetite and also seen in NES subjects (Gangwisch et al. 2005;Taheri et al. 2004). It is theorized that sleep debt may contribute to chronic illness through its effects on metabolism (Bass & Turek 2005;Spiegel, Leproult, & Van 1999). Serotonin promotes satiety and is replaced during sleep. Disturbed sleep may reduce natural levels and increase the need for exogenous sources such as carbohydrates, resulting in obesity. This link between obesity, short sleep duration and poor sleep quality is apparent in both adolescent and older adult populations (Gupta et al. 2002;Hasler et al. 2004;Patel et al. 2008).

The Heartfelt study of 383 11-16 yrs olds showed that fewer hours of sleep were associated with a higher prevalence of obesity ($r = - 0.36$ in boys and $- 0.29$ in girls) (Gupta et al. 2002). Percent body fat and BMI were used together to define obesity. Study strengths were objective measures of activity using actigraphs and the control of confounding behavioural variables such as alcohol use. One major weakness was its cross-sectional design over one 24 hr period. Obesity was the single most consistent correlate of sleep time, and lack of daytime physical activity the single most consistent correlate of sleep disturbance. Whilst the study design did not allow for inferences to be made about causation, the link between obesity and sleeping a few hours was nonetheless significant. A similar link has been identified between short sleep and co-morbidities associated with obesity such as type 2 diabetes and heart disease (Ayas et al. 2003a;Ayas et al. 2003b;Bass & Turek 2005;Spiegel et al. 2005;Wolk et al. 2005).Whilst proof of concept studies are currently underway to establish whether increased sleep can ‘treat’ obesity

and by inference, obesity-related conditions such as NES, further work is required to understand the significance of factors such as sleep duration and sleep quality in the development of NES.

2.7. *Is NES a Mood Disorder?*

NES appears common in patients with mental illness with prevalence levels ranging from 12.3% to 25% in American Out-Patient populations and there is some evidence that anti-psychotic medication may exacerbate the symptoms (Cohrs 2008;Lundgren et al. 2006;Lundgren et al. 2010). Indeed, throughout many NES studies, there has been a consistent tendency for sufferers to report significantly lower self-esteem and lower mood than non-NES individuals, although the mood pattern is atypical with distinctive circadian features, appearing minimal in the morning and increasing during the evening and night as the mood falls, unlike in depression, which is usually characterised by low mood that is worse in the morning (Birketvedt et al. 1999;Birketvedt, Sundsfjord, & Florholmen 2002;Gluck, Geliebter, & Satov 2001;Lundgren et al. 2008a). Allison et al (Allison et al. 2007b) demonstrated higher self-reported rates of neglect and emotional abuse, correlating with elevated depression levels in both BED and NES subjects, in comparison with matched obese controls although physical and sexual abuse were not more common. Similar levels of childhood maltreatment and depression were found in a sample of NES subjects with diabetes (Morse et al. 2006). When O'Reardon et al. (2004) sought to replicate the behavioural and clinical findings of Birkedvedt et al. (1999), as many as 70% of subjects were found to be suffering from a depressed mood and yet despite this, low mood has never been included as a core criteria for NES.

2.8. *Is NES an Anxiety Disorder?*

It has been suggested that NES may start out as a low level coping response to some psychological stressor that turns into a habit (Stunkard, Grace, & Wolff 1955). There is evidence that it is a chronic condition with 5 years being the mean duration of night -eating in an American survey sample (de Zwaan et al. 2006). In addition, non-obese NES subjects were found to have greater levels of stress and anxiety and were significantly more likely to have developed other coping mechanisms such as substance abuse (30.6% v 8.3%) (Lundgren et al. 2008a), although others consider the substance abuse to be linked to depression rather than NES per se (Striegel-Moore et al. 2008). Colles, Dixon & O'Brien (2007) found similar elevated levels of

psychological stress as well as depression in morbidly obese nocturnal snackers. Biochemical findings of increased circadian secretion of cortisol, support the theory that NES subjects have an over-expressed hypothalamic pituitary adrenal axis with an attenuated response to stress (Birketvedt, Sundsfjord, & Florholmen 2002;Lundgren et al. 2008a;Takeda et al. 2004).

2.9. *Is NES an Obesity-related Disorder?*

The role of NES in the pathogenesis and maintenance of obesity remains unclear. Behavioural studies reported to date demonstrated that total daily energy intake did not differ between night-eaters and controls (Allison et al. 2005a;Birketvedt et al. 1999), although the findings from one survey suggested more calories are consumed by night-eaters than by evening-eaters (Striegel-Moore et al. 2008). However, the majority of evidence suggests a general trend for prevalence to be low in general population samples and to increase with the degree of obesity (Adami et al. 2002;Aronoff, Geliebter, & Zammit 2001;Ceru-Bjork, Andersson, & Rossner 2001;Colles, Dixon, & O'Brien 2007;Gluck, Geliebter, & Satov 2001;Kuldau & Rand 1986;Napolitano et al. 2001;Rand, Macgregor, & Stunkard 1997;Rand & Kuldau 1993;Stunkard, Grace, & Wolff 1955). Psychiatric out-patients with NES are also more likely to have a higher BMI (mean BMI 33.1 kg/m² v 27.7 kg/m²) than psychiatric out-patients without NES (Lundgren et al. 2006).

Whether obesity influences the development of NES, or vice versa is unclear as studies comparing NES in obese and non-obese populations have variable results, with evidence limited mainly to cross-sectional studies, with self-reports of weight change, diagnosis and varying diagnostic criteria, making interpretation of causality more difficult. The questionnaire findings of 21 obese out-patients (mean BMI 37.3 kg/m²), 40 normal weight individuals, (mean BMI 22.5 kg/m²) and 40 obese individuals (mean BMI 37.9 kg/m²), who completed a website and subsequent phone interview were compared. Subjects either self-reported NES or had been previously diagnosed. NES was present in lean individuals, but was more common in the obese. Although the study was cross-sectional in design, 52% of obese NES sufferers reported normal weight before the onset of NES. Normal weight night-eaters were significantly younger than obese NES subjects (33.1 versus 43.1yrs $p < 0.01$) suggesting NES may be a risk factor for obesity (Marshall et al. 2004). Conversely, 60% of a general population sample who responded to

a newspaper advert about night-eating reported being overweight before night-eating with no significant difference in the age of onset of night-eating between normal and overweight participants noted (de Zwaan et al. 2006). In a prospective study of members of the general population, which included a longitudinal design with a 5 year and 10 year follow up, 9% of women and 7% of men responded 'yes' to the question, 'do you get up at night to eat?' at baseline. Obese females responding 'yes' experienced an average 6 year weight gain of 5.2kg ($p=0.004$) compared to 0.9 kg in obese females who responded 'no'. Night-eating and weight change were not associated among men (Andersen et al. 2004). This suggests night-eating may only influence weight gain in women who are already obese. A cross-sectional survey in young adults with NES used latent class analysis (LCA) to identify 4 subtypes of night-eating symptomatology (depressed and non-depressed late and night-eaters). Obesity was not associated with any of the four classes, or night-eating in general, with the mean (SD) BMI of night-eaters slightly lower than non-night-eaters (24.8 (5.9) versus 25.4 (6.0)) (Striegel-Moore et al. 2008).

The relationship between obesity and NES in childhood is difficult to establish as data on children are somewhat limited to surveys and produce estimates in line with the general adult population prevalence. In one survey 1.1% of German school children aged 5 -7 were identified by their parents as getting up at night to eat (Lamerz et al. 2005). Similar results were identified in 10 year olds, who completed diaries as part of a 10 year annual survey. One point five percent reported eating more than 25% of total kcal intake between 11pm and 5am at the age of 11. By the age of 19, this had increased to 3.5%, with larger numbers reporting positive on the more relaxed criteria of eating >25% of total calorie intake after the last evening meal. Further follow-up of 1341 of these individuals when aged 21 used questionnaires and phone interviews to reveal an NES prevalence of 1.6%, in keeping with prevalence in a general adult population (Striegel-Moore et al. 2005; Striegel-Moore et al. 2004). No association with obesity was found in any of these studies.

2.10. *Obesity and Depression*

More is becoming known about the links between obesity and depression with various hypotheses being proposed. Rosmond (2004) considers both obesity and depression to be manifestations of brain serotonin deficiency with obesity as the clinical manifestation of a

subtype of depression similar to that of atypical depression. Others consider obesity to develop subsequent to depression as a result of reduced activity and comfort eating. Longitudinal tests of the effect of depression on follow-up obesity status were meta-analysed, combining data from 16 studies. Those depressed at baseline were 1.8 times more likely than non-depressed to develop obesity (mean follow-up 7.6 years), although this effect was reduced when controlling for baseline BMI. The risk was particularly high for adolescent females (odds ratio 2.57, 95% CI: 2.27, 2.91) (Blaine 2008). An alternative hypothesis is that health impairments caused by obesity, such as fatigue and sleep disturbance, result in depression. In a large cross-sectional study of 2003 Americans, extreme obesity was associated with an increased risk for depression across gender and racial groups, even after controlling for chronic physical disease, familial depression and demographic risk factors (Dong, Sanchez, & Price 2004). Other cross-sectional studies support these findings (Atlantis & Baker 2008). The Look Ahead study of 5145 overweight and obese individuals showed higher depression levels to be linked to lower quality of life and a reduced metabolic equivalent (MET) level (indicating reduced physical fitness) in morbidly obese individuals (Rejeski et al. 2006). Others relate depressed mood in the obese to weight-related stigma (Chen et al. 2007).

Studies in morbidly-obese groups show improvements in depressed mood when physical appearance improves after weight loss interventions. This supports further the theory that obesity precedes depression. In a longitudinal study, the paired pre-operative, and 1,2,3 and 4 year post-operative Beck Depression Inventory (BDI) scores of 262 bariatric surgery subjects (mean pre-operative BMI 44 kg/m²) were used to compare changes in BDI score and identify predictors of change. Higher depression scores correlated with poorer physical and mental quality of life measures and significant reductions in depression post-operatively correlated with improvements in appearance evaluation ($r = -0.31$) (Dixon, Dixon, & O'Brien 2003). Mean (SD) BDI scores improved from 17.7(9.5) to 9.6 (7.7) at 4 years follow up ($p < 0.001$). If obesity results from coping strategies for depression, one would expect to see an increase in depressive symptoms post-operatively and not the reverse, although it is interesting to see a general trend upwards for the mean BDI score from one year to 4 year follow-up (7.8 ± 6.5 to 9.6 ± 7.7). Whether this increase would stabilize over time is unclear, although greater falls in BDI were seen in women,

younger subjects and those with greater excess weight loss. Others consider improvements in disordered eating patterns after surgery, as opposed to changes in physical appearance to be the main reason for reduced depressive symptoms (Mazzeo, Saunders, & Mitchell 2006).

2.11. *Influence of Obesity-related Co-Morbidities on NES*

Little is known about the influence on NES of co-morbidities such as Obstructive Sleep Apnoea (OSA) and type 2 diabetes, obesity-related conditions also associated with sleep disturbances, which can result in poor sleep quality and excessive day-time sleepiness (Bixler et al. 2005). Insomnia-related symptoms concomitant with daytime tiredness are in themselves independent risk factors for obesity (Fogelholm et al. 2007; Resta et al. 2003). OSA is defined by repeated episodes of apnoea and hypopnoea (interruptions in breathing and low oxygen saturation levels) during sleep, resulting in arousal from sleep to increase depth of respiration and restore oxygen levels. Though present in lean individuals, it is more common in obese males (Coughlin et al. 2004). It is also independently associated with an increase in the cardiovascular risk factors that comprise the metabolic syndrome and are common in obese subjects (Coughlin et al. 2004). Likewise, type 2 diabetes has a known relationship with sleep. Short and long sleep durations increase the risk of developing diabetes independent of confounding factors (Yaggi, Araujo, & McKinlay 2006). In addition, poor sleep quality and excessive daytime sleepiness have been associated with worsening HbA1c (marker of diabetes control) (Knutson et al. 2006b). One study in diabetic subjects has shown night-eating to be associated with higher obesity levels and increased diabetic complications, although subjects were asked one question only, to estimate their food intake after dinner and other criteria were not assessed (Morse et al. 2006).

2.12. *Treatments for NES*

2.12.1. *Obesity Treatments*

Examination of the various treatment strategies proposed for NES gives some insight into the relative importance attached to some characteristics in relation to others. Given the suspected link between NES and obesity and limited evidence that obese NES subjects do less well in standard weight loss programmes (Gluck, Geliebter, & Satov 2001), it would be expected that appropriate treatment options for NES would need to take obesity into account, yet there are no published data on the effect of standard anti-obesity agents on NES. Orlistat, a pancreatic lipase

inhibitor, prevents the absorption of approximately 30 % of dietary fat but requires a regular eating pattern of three meals a day for optimal effect (Hollander et al. 1998;Morse et al. 2006). It is likely that night-eaters would find this requirement problematic. Sibutramine, a selective serotonin reuptake inhibitor (SSRI), causes feelings of fullness and increases energy expenditure (Finer et al. 2000). Although now withdrawn from use, anecdotal evidence showed it to have a particularly favourable effect on evening overeating, suggesting it may have been of some benefit to night-eaters.

There is a surprising lack of evidence regarding the effect of cognitive and behavioural therapy (CBT), a cornerstone of obesity treatment, on NES. Reports are limited to a few behavioural change strategies which are recommended in a self-help manual (Allison, Stunkard, & Thier 2004;Vinai et al. 2008). More recently, a pilot study of a 10 session CBT programme used with 25 NES subjects has shown significant reductions in weight, number of nocturnal ingestions and percent of daily calorie intake after dinner (all p values <0.0001) (Allison et al. 2010a). The authors call for a controlled treatment trial to fully assess the efficacy of the programme. Although the mean weight of participants reduced in the pilot study from 82.5kg to 79.4kg the mean baseline weight suggests these were not morbidly obese individuals and larger studies which include such groups are required.

Several studies have examined the effect of restrictive weight loss surgical procedures on NES. It is difficult to draw definitive conclusions as the management of eating disorders pre-surgery varies widely and NES is poorly recognised at present (Devlin et al. 2004). Only 52.5% of American bariatric surgeons taking an internet survey included NES in the pre-surgery screen. Busetto et al. (2005) found the percent excess weight loss (% EWL) in the first 5 years after laparoscopic gastric banding was similar in patients with and without disordered eating, although the eating disordered group had slightly more complications directly related to the frequency of post-operative vomiting. Latner et al. (2004) found similar results after gastric bypass with greater weight loss related to a reduction in night-eating episodes. Colles, Dixon & O'Brien (2008b) found differing levels of success for BED as opposed to NES subjects. The prevalence of BED and NES in Australian subjects reduced from 14% and 17.1% pre-operatively to 3.1% and

7.8% post-surgery respectively and although not significantly different, the NES subjects lost less weight than the BED subjects (BMI at one year post treatment 33.1 kg/m² v 27.7 kg/m²).

2.12.2. *Mood Treatments*

Other proposed treatments promote the theory that NES is primarily a mood disorder. A small-scale open label study suggested that sertraline, a selective serotonin reuptake inhibitor (SSRI) anti-depressant, which is also known to improve anxiety, improves NES through the restoration of inadequate serotonin levels. Responders experienced weight loss, reductions in nocturnal ingestions and in calorie intake after the evening meal (O'Reardon, Stunkard, & Allison 2004). A larger, placebo-controlled trial of 8 weeks duration in 34 American subjects with a mean BMI of 32.6 kg/m² confirmed these findings. In addition, a weak, non-significant improvement in depressive symptoms suggested the improvements in NES symptoms were independent of the usual anti-depressant effect of sertraline and led the authors to suggest that the drug may effect a more normal circadian rhythm of food intake through the suprachiasmic nucleus (an area of the brain responsible for regulation of circadian rhythm) (O'Reardon et al. 2006; O'Reardon, Peshek, & Allison 2005). A pilot SPECT (single photon emission computed tomography) study comparing the serotonin transport uptake ratios of night-eaters with healthy controls has proposed the involvement of the serotonin system as opposed to other neuroendocrine systems in the pathophysiology of NES (Lundgren et al. 2008b). Stunkard supports this view, particularly given the success of sertraline as a treatment (Stunkard & Lu 2010). Other anti-depressants have helped alleviate night-eating symptoms, with one study reporting remission of symptoms after 2 weeks using paroxetine and fluoxetine in 4 subjects with Nocturnal Eating/drinking Syndrome and another using d-fenfluramine (now withdrawn) to treat 10 Italian subjects suffering from Noct ES (Miyaoka et al. 2003; Spaggiari et al. 1994).

Non-pharmacological anti-depressant treatments such as Gotu Kola and other herbs have also been promoted in the treatment of NES (O'Brien 2005). A study using Abbreviated Progressive Muscle Relaxation Technique (APMRT) significantly reduced stress, anxiety, fatigue, anger and depression after 1 week of therapy in a group of 10 subjects, and was associated with trends towards higher morning and lower evening hunger ratings and less night-time eating (Pawlow, O'Neil, & Malcolm 2003). One individual with co-morbid seasonal

affective disorder was successfully treated with light therapy (10,000 lux for 50 minutes) (Friedman et al. 2004). Interestingly, topiramate, a neurotherapeutic agent normally used to treat epilepsy, but which has been shown to help weight loss in obesity both with or without BED was shown to reduce night-eating episodes in two patients with NES and one with co-morbid post traumatic stress disorder (McElroy et al. 2007; Tucker, Masters, & Nawar 2004; Wilding et al. 2004; Winkelman 2003). Others argue that topiramate is a more effective treatment for SRED as opposed to NES (Howell, Schenck, & Crow 2008). Unfortunately the small participant numbers in all these studies makes the variable results difficult to interpret meaningfully.

2.13. *Therapeutic Level of Intervention*

Recent work appears to be focusing on establishing a therapeutic level of intervention whilst the debate about core characteristics and criteria persists. Colles, Dixon & O'Brien (2007) identify 3 separate groups with NES in a mixed sample of subjects from an obesity clinic, a weight loss group and the general community. Controversially, they class subjects **without** nocturnal snacking as 'NES' and those with nocturnal snacking as 'NES plus nocturnal snacking'. The third group of 'NES plus binge eating' includes subjects with BED and sub-threshold BED symptoms. These classifications suggest an attempt to define NES along a continuum of severity, with those in the latter two groups reporting greater psychological distress and depressive symptoms than the simple 'NES' group. Striegel-Moore et al. (2008) make a similar distinction between evening-eaters and night-eaters, with night-eaters consuming on average 433 more calories a day than non-night-eaters and suggesting NES criteria should be based on eating very late at night. On the other hand, others conclude that evening hyperphagia and nocturnal eating are probably the same construct, whilst acknowledging that stress variables and functional impairment should be taken into account (de Zwaan et al. 2006). Allison et al. (2008a) used item response theory (IRT) to analyse the responses from 1481 night eating questionnaires. IRT is a model-based theory of measurement which assumes that a person's trait level can be estimated from responses to individual items. Results showed the key features to be nocturnal eating and/or evening hyperphagia, initial insomnia and night awakening with morning anorexia and delayed morning meal less important. Indeed others note that morning hunger is less important as it is difficult to define objectively (Striegel-Moore et al. 2008). In 2008, Stunkard appeared to

acknowledge this debate and proposed relaxing the core criteria to: eating more than 25% kcal after the evening meal and/or waking to eat three times a week (Stunkard, Allison, & Lundgren 2008).

2.14. *Clinical Significance*

NES is currently classed as an Eating Disorder Not Otherwise Specified (EDNOS) in the DSM-IV scheme for classifying eating disorders. Stunkard, Allison & Lundgren (2008) argue that NES should be included in its own right in the new DSM-5 scheme due for publication in 2013 on the basis that it may lead to obesity and is a significant source of distress. The DSM criteria generally are intended to be reliable, theory-neutral diagnostic criteria that provide necessary and sufficient conditions for differential diagnosis of genuine mental disorders as opposed to non-disordered problems in living. However, even if NES were to be given a separate identity, it must be acknowledged that there is some disagreement as to the validity of the DSM scheme generally. Whilst some have sought to demonstrate the validity of the scheme arguing that in the case of bulimic disorders for example, the scheme has good construct and predictive validity (Hay & Fairburn 1998), others consider the criteria to be too broad, with an emphasis on sensitivity rather than specificity, resulting in false positives and a failure to make true distinctions (Wakefield 1997). Indeed until recently, Stunkard & Alison (2003) appeared to concur with this view. Whilst promoting the identification of NES, they initially seemed less convinced of its clinical significance, suggesting it should be considered less as an object of therapeutic endeavour and more as a marker of psychiatric co-morbidities such as depression and anxiety. In the case of BED they argued that standard behavioural weight loss treatments which concentrate on improving the psychopathological co-morbidities of BED, such as depression and low self esteem, can be more effective than treatments which focus primarily on the behaviour surrounding a binge. Although they now appear to have changed this view, inclusion into the DSM-5 scheme looks unlikely based on published provisional listings for DSM-5. For a syndrome to be included in DSM-5 stringent criteria must be satisfied; namely the existence of an ample body of literature, clearly defined criteria, sufficient differentiation from other similar syndromes, diagnostic reliability and syndrome validity (Blashfield, Sprock, & Fuller 1990). Other NES researchers argue that these 5 criteria are not yet fulfilled and that more insight into

NES is required before these criteria can be met (Striegel-Moore et al. 2006a; Striegel-Moore, Franko, & Garcia 2009).

2.15. *Identification*

The lack of standardisation across studies in both the choice of methodology of assessing and interpretation of NES criteria has adversely affected NES research. Whilst a battery of tools validated for diagnosing eating and mood disorders has often been used for supporting evidence, the absence of a consistent method for identifying NES itself has encouraged researchers to use various methods to establish diagnosis, such as structured interviews, questionnaires based solely on Stunkard's criteria and questionnaires combining other syndromes (Adami et al. 2002; Ceru-Bjork, Andersson, & Rossner 2001; Gluck, Geliebter, & Satov 2001).

Stunkard himself recognised this problem and attempted to standardise the methodology used by proposing that an interview conducted by a therapist specialising in eating disorders should be the gold standard diagnostic tool for NES. This was based on the finding that many respondents thought to have BED and NES using self-report questionnaires were found **NOT** to have these conditions during a subsequent follow-up interview (Allison et al. 2007a; Stunkard & Allison 2003). Two of Spitzer's early BED studies used self-report questionnaires and estimated prevalence to be 29% in people seeking treatment for obesity and 30% respectively. Later interview-based studies found lower prevalence rates ranging from 8.9% and 18%. One study of 1450 persons identifying themselves as binge eaters following a TV appeal yielded only 50 subjects (3.4%) who met interview-based criteria for BED (Spitzer et al. 2006).

In 2004 Stunkard's team proposed that the interview should include the Eating Disorder Examination (EDE) a validated diagnostic tool specifically designed to be delivered in an interview setting to identify AN and BN in order to exclude other eating disorders in the first instance, along with a 14 item NES questionnaire (NEQ 2004) which can be used to confirm a diagnosis of NES. Once diagnosis has been established, findings are supplemented using a Night Eating Symptom and History Inventory (NESHI), a 17 item interview schedule that assesses night-eating characteristics such as age of onset, level of control, foods eaten, treatment strategies tried and family history (Allison 2004; O'Reardon, Stunkard, & Allison 2004).

2.16. *Screening Tools for NES*

The unvalidated 14 item Night Eating Questionnaire (NEQ 2004) (Marshall et al. 2004) was adapted from the Night Eating Symptom Questionnaire (NESQ), which in turn, reflects items that were added to the Weight And Lifestyle Inventory (WALI) (Wadden & Phelan 2002) , a behavioural assessment questionnaire designed for use with the obese, but neither tool includes items which could be used to identify other eating disorders, the absence of which were suggested by Stunkard in 2003 as a core criterion for the diagnosis of NES. Whilst some aspects of the WALI have undergone reproducibility studies, the NES additions have been added subsequent to this (Wadden T & Foster 2001). As a result, very little data are available on the performance of the NES items. One small study has compared the responses of 59 (14m/45f) ‘night snackers’ to the first 9 items of the NESQ with 6 nested definitions of NES ranging from the broad criterion of ‘eating more in the evening than any other time of day’, to the more restrictive ‘eating at least 50% of intake after 7pm, with no morning appetite and sleep disturbance’. The authors concluded that the NESQ lacked cut points that would increase its sensitivity and specificity (Vander Wal et al. 2005). A second study used the 14 item NEQ (2004) to identify the prevalence of NES in 399 (154m/245f) outpatients from 2 separate psychiatric clinics. 12.4% met the rather loose criterion of either eating more than 1/3 of total daily intake after the last evening meal, or nocturnal awakenings to eat. Two hundred and five (more than 50%) scored >20 on the NEQ (2004), although only 49 of these were diagnosed at interview as having NES, a further 28 were found to have sub-threshold levels and 44 were negative for NES (84 did not attend for interview). Unfortunately the diagnostic interview was not based on the 2003 criteria. The authors report the point prevalence of the NES to be 12.3% increasing to 15.6%, when the 84 drop-outs are excluded, with a positive predictive value of 40% when the cut-off score is 20, 52% at 25, and 68% at 30, although the statistical analysis on which these conclusions are based is not reported (Lundgren et al. 2006).

2.17. *Discussion*

It is apparent that NES is not a simple, clearly defined phenomenon, but a multifaceted construct with several characteristics of varying significance. Eating, sleeping and mood are clearly disrupted but it is likely that emphasis on one aspect as opposed to another is probably the

result of researcher bias towards their specialties. The syndrome can be either relatively common or rare, depending on which criteria are adopted. Evidence from European and American studies would suggest that it may feature strongly in some high risk groups, such as obesity clinic populations. The presence of other obesity and sleep related co-morbidities, such as OSA and type 2 diabetes in these populations makes understanding NES even more complex. Lack of consensus on the diagnostic criteria is clearly hampering further investigation and yet paradoxically, until the characteristics are better defined, debate over the criteria will continue. NES does appear to be a significant clinical issue for many sufferers (D'Arrigo 2007), although there is a notable lack of literature describing the subject's experience of NES (Allison et al. 2008b; Colles & Dixon 2006).

Routine identification of NES is problematic. Others agree with Stunkard that clinical interviews are the most accurate method for the diagnosis of eating disorders, whilst at the same time acknowledging that they are time consuming and require specifically-trained investigators (Ricca et al. 2000). Although this resource may be available in some specialist centres, due to cost considerations it is not generally available for the assessment of eating behaviours in large populations, such as obese patients seeking treatment for weight loss in NHS routine outpatient clinics. A more realistic approach may be to use self-report questionnaires as a 'first-line' screening method, with diagnosis of positive screens confirmed at a follow-up diagnostic interview. Care must be taken in choosing the correct tool. Distinction should be made between diagnostic tools which have been developed to identify cases and prognostic tools which seek to identify at risk individuals, as these have different objectives and require different methods of validation. In general, a good screening instrument, developed to aid diagnosis, should have good sensitivity to identify a large majority of the patients affected, and whilst a high specificity is less essential, a test with low specificity that identifies many false positive cases can lead to the unnecessary expense of too many inappropriate follow-up diagnostic interviews (Offord et al. 1998).

Screening tools are often used to identify eating disorders, although, what is considered an acceptable level of sensitivity and specificity appear to vary between tools. Commonly used tools such as The Binge Eating Scale (BES), Bulimic Investigation Test Edinburgh (BITE) and

SCOFF (anacronym created from key words in tool questions: Sick,Control, One, Fat, Food) screening tools have a sensitivity of 84.8%, 91% and 100% and specificity of 74.6%, 51.4% and 87.5% respectively (Garner & Garfinkel 1979;Henderson & Freeman 1987;Morgan, Reid, & Lacey 1999). Such tools appear to perform broadly in line with tools developed to test other problematic behaviours such as the RAFFT, a five-question instrument developed for screening adolescent substance-abuse disorders (sensitivity 89%, specificity 69%) and the CAGE, a screening tool for alcoholism (sensitivity 90 %, specificity 83.7%) (Bastiaens, Riccardi, & Sakhrani 2002;Buchsbaum et al. 1991).

Jacobi, Abascal & Taylor (2004) advise caution in interpreting the results of sensitivity and specificity testing. Tools such as the Eating Attitudes Test (EAT) for example, a screening tool for anorexia (AN), may have a high sensitivity and specificity in highly selected clinical case-control samples, but a low positive predictive value for identifying cases of full syndrome AN in community-based populations (Keski-Rahkonen et al. 2006) . In addition, during the process of development, many tools have failed to establish basic psychometric properties such as reliability or construct and discriminant validity prior to establishing concurrent validity with sensitivity and specificity testing. Consideration must also be given to the validity of the ‘gold standard’ and the definition of a case versus non-case against which tools are tested, as the diagnostic criteria for eating disorders in particular are often in a state of flux.

2.18. *Conclusion*

To conclude, identifying NES in clinical populations remains problematic due to inconsistent criteria and a lack of appropriate tools. The definition and characterization of NES is still ongoing with little qualitative evidence currently available to help explain NES from patients’ perspectives (Colles & Dixon 2006). More evidence is needed to assess the clinical significance of each of the associated features and the syndrome as an entity in order to establish a threshold at which the cluster of behaviours causes marked distress, mental or physical impairment or generates a risk to health. A better understanding of the syndrome may then result in the availability of effective treatment choices.

3. IDENTIFICATION STUDY

3.1. *NES in UK Populations*

Specialist Weight Management Services in the UK typically treat individuals with a BMI $> 40 \text{ kg/m}^2$ (or a BMI $> 35 \text{ kg/m}^2$ with existing co-morbidities). At the time of instigating this study, the prevalence of NES in such populations and its potential impact on the mental and physical health of sufferers was unknown. As many individuals in such clinics suffer from refractory obesity and response to treatment is often low, it was considered clinically useful to identify and characterize NES in this population.

Identification of prevalence requires a validated and consistent approach, based on defined criteria. Stunkard's 2003 criteria were current when this investigation was conceived. Whilst items for identifying NES are included in these criteria, the exclusion of other eating disorders (ED) was also a key feature. Although Stunkard recommended a 'gold standard' diagnostic interview with an eating disorder specialist to identify NES and exclude other ED in the first instance, this approach is impractical in the UK as such specialists are not routinely available in weight management clinics and current resources allow for only a small proportion of patients to be referred to the local Eating Disorder Service (EDS). A more pragmatic and cost-effective approach was considered to be the use of a screening tool for NES to identify what proportion of an obesity clinic would screen positive. The tool would need to be both simple enough to aid detection of NES features and accessible to health professionals generally. No tool was available that had undergone reliability and reproducibility studies or included items based on all six 2003 criteria.

3.2. *Relationship between NES and Obesity-related Co-morbidity*

3.2.1. *NES and Mood*

Prior to the 2003 criteria, researchers had consistently found an association between depressive symptoms and NES (Birketvedt et al. 1999; Birketvedt, Sundsfjord, & Florholmen 2002; Gluck, Geliebter, & Satov 2001), but the nature of this relationship remained unclear. Although Stunkard & Allison (2003) included two questions about mood in the NESHI they did not feature in the 2003 diagnostic criteria, despite their suggestion at times that NES was a

feature of psychiatric co-morbidity. Other NES researchers considered it significant enough to warrant further exploration and included screening tools for depressive symptoms when characterising NES (Allison et al. 2005b; Allison et al. 2005a; Grilo & Masheb 2004; O'Reardon, Stunkard, & Allison 2004; Pawlow, O'Neil, & Malcolm 2003). It seemed appropriate therefore, to investigate whether the depression characteristics found in other NES populations were replicated in an obese UK population.

3.2.2. *NES and Sleep*

Investigation into the relationship between obesity and sleep duration was in its infancy when this study was conceived in 2004, with no data available on sleep in UK obese populations. In addition, the prevalence of sleep-related co-morbidity such as OSA in obesity clinic populations was unknown and the relationship between OSA and NES had not been explored. As there were no known prevalence studies of NES in UK OSA populations, the effect of OSA on NES could only be theorised.

3.3. *Aims*

Given the above, the aims of the first phase of the study were to:

- Identify NES in a UK obese population
- Develop a screening tool for NES and identify what proportion of an obese population would screen positive for NES using the tool and how this compared with classification via a diagnostic interview
- Explore the relationship between NES and depression
- Explore the relationship between NES, sleep and sleep-related co-morbidity such as OSA.

3.4. *Design*

3.4.1. *Identification of Individuals with NES*

The first stage of the study was to identify individuals from a UK obesity clinic population with and without NES on whom a screening tool could be tested. In accordance with the procedures advocated by Stunkard's group in the USA, a diagnostic interview was chosen as the method for identifying individuals with NES. As previously discussed, Stunkard proposed the

interview as the ‘gold standard’ for diagnosis and in the absence of any other validated method based on the 2003 criteria or the availability of an eating disorder specialist, it seemed reasonable to adopt the same approach and to use his interview schedule. Although unvalidated, no other interview schedules currently exist for identifying NES and its use by other NES researchers has become common place (Allison et al. 2007b;Lundgren et al. 2008a;Lundgren et al. 2011;O’Reardon, Stunkard, & Allison 2004).

3.4.2. *Interview Tools*

The interviews comprised the following items in the order listed below:

- The Eating Disorder Examination (EDE) version 12
- Supplementary questions for Binge Eating Disorder (BED)
- The Night Eating Syndrome History and Inventory (NESHI)
- The Beck Depression Inventory version IA (BDI-IA)

3.4.2.1. *Eating Disorder Examination Version 12*

3.4.2.1.1. **Background**

As well as diagnosing NES, the interviews required a formal method of identifying other eating disorders (ED), namely Bulimia Nervosa (BN), Anorexia Nervosa (AN) and BED in order to fulfil the diagnostic requirement for NES that other ED be excluded. The EDE version 12 is a validated diagnostic tool specifically designed to be delivered in an interview setting to identify AN and BN. The EDE was initially developed by Cooper and Fairburn (Cooper & Fairburn 1987) as a tool to aid detailed studies of key ED attitudes and behaviour such as disturbed eating habits and extreme methods of weight control. At the time of developing the EDE, various general and specific self-report tools for identifying ED were available, however, Cooper and Fairburn considered it preferable to examine the nuances of shape and weight attitudes in an interview format, which, whilst more difficult to score, would allow for in-depth analysis and thus greater validity. They theorised that ED sufferers place undue importance on shape and weight as a means of evaluating self-worth. Whilst society generally views fatness negatively and thinness positively, ED sufferers have a morbid fear of fatness which is considered dysfunctional because

it is rigid, extreme, imbued with personal significance and of primary importance in maintaining disordered eating.

In addition, a face-to-face interview allows for attention to be paid to defining ‘overeating’, a behavioural construct which often presents difficulties of interpretation. Although sufferers of BN experience florid episodes of overeating and self-induced vomiting, not every episode is identical. For an overeating episode to be classed as ‘bulimic’, the subject must report a loss of control, whilst at the same time eating an objectively large amount of food (Table 4). Probe questions included in the EDE are specifically designed to establish these subtleties, an option not available with self-report tools.

Table 4 - Differentiation between Overeating and Bulimia according to the EDE

	<i>Amount Eaten</i>	
	<i>‘Large’ EDE definition</i>	<i>Not ‘large’ but viewed by subject as excessive</i>
<i>Loss of control</i>	Objective bulimic episodes	Subjective Bulimic episodes
<i>No loss of control</i>	Objective overeating	Subjective overeating

3.4.2.1.2. Scoring System

Version 12 of the EDE was used in this study and contains 33 items, covering behaviour over the preceding 4 weeks, with some diagnostic items covering a 3 month time period. An example of one EDE item is included in Appendix 1. The scoring system is based on the 4 subscales of Restraint, Eating Concern, Weight Concern and Shape Concern. The majority of items are rated on a seven point scale ranging from 0-6 on which either frequency or severity of attitudes and behaviour is rated. Individual items of each subscale are totalled to provide subscores, then added together and divided to provide a global score, an overall measure of the degree of ED pathology. Training is recommended in both the technique of interviewing and in the concepts and rules governing the ratings (Fairburn & Cooper 1993). The criteria used to generate ED diagnoses are listed in Table 5 and subscale items in Table 6.

Table 5 - EDE version 12 item scores on which AN and BU diagnoses are based.

ANOREXIA NERVOSA (Fairburn 1993)	
1. Body weight should be 15% or more below expected 2. Maintained low weight rated 1 3. Fear of weight gain rated 4,5 or 6 for each of past 3 months 4. Importance of shape rated 4,5 or 6 for each of past 3 months 5. Importance of weight rated 4,5 or 6 for each of past 3 months 6. Menstruation should be rated 0 or 7*	
SUB THRESHOLD – body weight 15% or more below expected. *7 =not applicable	
BULIMIA NERVOSA (Fairburn 1993)	
1. At least 12 objective bulimic episodes over the past 3 months 2. Longest period of abstinence of such episodes no greater than 2 weeks 3. Abstinence from extreme weight-controlled behaviour rated 0,1or 2 4. Importance of shape or Importance of weight rated 4,5 or 6 for each of last 3 months 5. Negative for diagnosis of anorexia 6. Presence of any one of vomiting, laxative misuse, diuretic misuse, fasting outside bulimic episodes, or excessive exercise	
SUB THRESHOLD - < 12 objective bulimic episodes, presence of compensatory measures	

Table 6 - EDE version 12 subscale items

<i>Restraint</i>	<i>Shape concern</i>
Restraint over eating	Flat stomach
Avoidance of eating	Importance of shape
Food avoidance	Preoccupation with shape or weight
Dietary rules	Dissatisfaction with shape
Empty stomach	Fear of weight gain
	Discomfort seeing body
	Avoidance of exposure
	Feelings of fatness
<i>Eating concern</i>	<i>Weight concern</i>
Preoccupation with food, eating or calories	Importance of weight
Fear of losing control over eating	Reaction to prescribed weighing
Social eating	Preoccupation with shape or weight
Eating in secret	Dissatisfaction with weight
Guilt about eating	Desire to lose weight

3.4.2.1.3. Normative Scores in an Obese Population

The EDE v12 is primarily designed to diagnose and characterise AN and BN, ED which are both associated with normal or underweight individuals. Normative values for individual

subscales range from 0.22 to 0.91 in normal controls and are generally higher, ranging from 2.43 to 3.55 in ED subjects (Beumont et al. 1993; Cooper, Cooper, & Fairburn 1989; Wilson & Smith 1989). However, caution should be taken in interpreting these results which are from studies conducted prior to the EDE v12 being published and which include almost exclusively thin, young females. Establishing norms for obese populations is problematic, as despite its widespread use in the obese ED research field, subscale scores are often not published, with researchers concentrating more on the diagnostic utilities of the EDE. One early study (unpublished) using the EDE v 12 examined overweight subjects, with sub-scale scores ranging from 0.64 to 1.97 in subjects with BMI >30 kg/m². Recent studies in morbidly obese samples have generally included more males and yielded higher scores, with a range from 1.60 to 3.28 in one study of pre-bariatric surgery patients (mean BMI 52.2 kg/m²) (Grilo, Masheb, & Wilson 2001; Kalarchian et al. 2000; Wilfley et al. 1997).

3.4.2.1.4. **Validity and Reliability**

The EDE v12 is widely reported as having acceptable validity and reliability (Fairburn & Cooper 1993; Rosen et al. 1990) and yet detailed data are severely lacking on factor analysis and construct validity of early versions of the tool. There were 9 apparent revisions before testing was first reported on what appears to be version 10 in 1989 (Cooper, Cooper, & Fairburn 1989). Original development was based on key constructs which were highlighted from AN and BN literature and from a series of lengthy unstructured interviews with patients with AN and BN and these constructs were developed by Cooper & Fairburn into items for the tool, although there are no published papers describing this development. Items to generate operationally defined diagnoses were later editions to version 12 when an earlier Bulimia subscale was dropped as these items were now replicated in the diagnostic section (Fairburn & Cooper 1993).

3.4.2.1.5. **Internal Consistency**

Development of the weight and shape subscales in particular, raises some concerns about the internal consistency of the tool. The EDE (version unknown) scores of 100 subjects with AN and BN identified from 5 different clinic settings (mean ages 20.5yrs and 22.1yrs respectively) with 42 controls identified from general practice (mean age 21.3yrs) were compared (Cooper,

Cooper, & Fairburn 1989). Weight was not reported, although the controls and participants with BN were noted to be comparable, with the AN group substantially underweight.

Cronbach alpha coefficients for the 5 subscales were: Restraint (R) 0.75, Bulimia (BU) 0.90, Eating Concern (EC) 0.78, Weight Concern (WC) 0.67 and Shape Concern (SC) 0.79. The WC alpha coefficient was somewhat low and inconsistencies were centred around a lack of independent factor structure between the shape and weight subscales generally. The item 'Sensitivity to weight gain' correlated poorly with its own WC subscale ($r = 0.26$), but better with the R ($r = 0.59$) and SC ($r = 0.61$) subscales. When dropped from the WC subscale, the WC alpha coefficient, whilst still low, rose slightly to 0.68 and the mean item total correlation to 0.44. There was a similar effect with the 'Pursuit of thinness' item which correlated poorly with the SC subscale and once removed, raised the alpha coefficient to 0.82 and the item total correlation to 0.54. Both items were subsequently dropped from their respective subscales, although the rationale for doing so seems weak. The authors argued that both items helped to differentiate from controls and should therefore be left in the tool, but argued against including the items in other subscales on empirical grounds as this weakens their initial allocation on rational grounds. Without a detailed explanation of the original item allocation, it is difficult to comment on this argument, although there is a suspicion that items are being manipulated to raise the alpha coefficients to acceptable levels. The lack of an independent factor structure between weight and shape items also calls into question the separating out of the weight and shape constructs. For many individuals the most objective way they have of measuring their shape is to record their weight. If these constructs are blurred in this young, thin population, the effect may be more striking in an obese population.

Studies reporting reliability of the tool are limited. Beumont et al. (1993) compared the EDE (v 11) scores of 112 female and 4 male Australian participants (mean age 22.3 yrs, including some children) with Cooper's Oxford group ($n=143$) and found variable results, with some differences noted in the internal consistency measures. Cronbach alpha coefficients were higher in the Sydney patients for R (0.78) and WC (0.90), though lower for EC (0.68), SC (0.70) and BU (0.76). Although both were relatively large sample sizes, the method of diagnosis of ED between studies was variable, so more evidence would be required to comment fully. Inter-rater reliability

was noted to be high in 3 separate studies (Cooper & Fairburn 1987; Rosen et al. 1990; Wilson & Smith 1989).

3.4.2.1.6. **Conclusion**

Although Stunkard's team recommended the use of the EDE for investigations into NES and it was used by other researchers at the time this study was initiated, its weaknesses must be acknowledged, particularly with regard to the subscales. In addition, most validity and reliability studies appear to have been conducted on versions 10 and 11, rather than on version 12 and there is little evidence available on its performance in obese populations. Its primary purpose in this study however, was to identify other ED and subscale items have a relatively minor role in this. Rosen et al. (1990) found very high correlations between the ED diagnostic items such as vomiting questions and actual vomiting reported in self-report food diaries and good correlations between the EDE overeating scale items and food diary reported episodes of binge eating ($r = 0.40, p < 0.0001$) and frequency of binge eating ($r = 0.46, p < 0.0001$). It would seem reasonable therefore to use the EDE v12 as a diagnostic aid to exclude other ED according to Stunkard's 2003 criteria.

3.4.2.2. *Additional items for Binge Eating Disorder*

At the time the EDE v12 was developed, BED was not fully recognised as a separate condition and was classed as an Eating Disorder not otherwise specified (EDNOS).

Classifications of Eating Disorders recognised only one disorder, Bulimia Nervosa, involving binge eating although Spitzer et al. (1993) argued that this excluded many individuals with marked distress about binge eating who couldn't be diagnosed with bulimia as they did not engage in compensatory measures such as vomiting or laxatives.

For the purposes of the EDE v12, the authors concluded that not enough specific information was available to formally include BED in the tool, but suggested a working definition in keeping with the EDE diagnostic criteria for bulimia, based on 12 bulimic episodes in the past 3 months and the absence of compensatory measures (see Table 7). This differed slightly from criteria proposed by Spitzer et al. (2006) and other members of the Eating Disorders Work Group of the DSM-IV Task force, which suggested diagnosis should be based on a greater frequency of binges, i.e. at

least twice a week for 6 months. In addition, Spitzer proposed supplementary diagnostic criteria such as the presence of marked distress in the patient and specific behavioural indicators of loss of control, such as rapid eating. For the sake of consistency, the frequency proposed by the EDE authors have been adopted for this study, but the additional items proposed by Spitzer were included in the EDE schedule to further assist in the identification of BED. Similar approaches have been taken by other NES researchers (Allison et al. 2005b).

Table 7 - Items on which a diagnosis of Binge Eating Behaviour is based

BINGE EATING DISORDER (Items 1&2 Cooper & Fairburn (1993) Items 3&4 Spitzer 1993)

1. Presence of at least 12 objective bulimic episodes over the past 3 months in the absence of compensatory mechanisms such as vomiting.

2. Negative for diagnosis of anorexia

3. Presence of positive response to any of the supplemental questions below

- Eat much more quickly than usual
- Eat until feel uncomfortably full
- Eat large amounts of food when not physically hungry
- Eat alone because of embarrassment
- Feel disgusted with self or very guilty

4. Marked distress regarding binge eating.

SUB THRESHOLD - < 12 objective bulimic episodes, absence of compensatory measures

3.4.2.3. *Identification of NES*

NES was identified using the Night Eating Syndrome History and Inventory (NESHI) (Appendix 2). This semi-structured unpublished interview contains a schedule of questions suggested by Stunkard's team for identifying and characterising NES, based on the 2003 diagnostic criteria for NES (Allison 2004). Questions cover meal patterns, the amount of food intake throughout the 24hr day, history of NES symptoms, sleeping routine, weight and diet history and previous treatment strategies for NES. The schedule also contains the 14 items which form the basis of the NEQ (2004) as a 5 point Likert scale, although its performance as a diagnostic aid or screening tool had not been evaluated by Stunkard's team at the time this study was developed. For the purposes of this study, the presence of NES was defined by subjects testing positive for all disorder-specific criteria as listed in Table 8.

Table 8 - Study criteria for NES and sub-threshold NES

NIGHT EATING SYNDROME (Stunkard 2003)

1. Morning anorexia, even if subject eats breakfast
2. **Evening hyperphagia. At least 50% of the daily caloric intake is consumed in snacks after the last evening meal**
3. **Awakenings at least once a night, at least 3 nights a week**
4. **Consumption of high calorie snacks during the awakenings on frequent occasions**
5. The pattern occurs for a period of at least 3 months
6. Exclusion of other eating disorders

SUB THRESHOLD - < 50% consumed in snacks after last evening meal, waking up to eat and /or eating prior to going to bed very late regardless of frequency and duration of behaviour, amount eaten, presence of morning anorexia or other ED. Reversal of day and night (excluding night work)

NEQ Likert scale scores were also used to supplement diagnosis, in particular for the criteria of morning anorexia and evening hyperphagia. In response to the question, ‘How hungry are you usually in the morning?’ subjects were asked to choose between:

0	1	2	3	4
Not at all	A little	Somewhat	Moderately	Very

Morning anorexia was defined as a score of 0 or 1. As per Stunkard’s specific instructions in the NESHI, the degree of evening hyperphagia after the last evening meal was an educated estimate based on discussion with the patient about their description of a typical day and responses to other NES questions. Subjects were asked ‘How much of your daily food intake do you consume *after* supertime?’

0	1	2	3	4
0% None	1-25% up to a quarter	26-50% about half	51-75% More than half	76-100% almost all

Evening hyperphagia was defined as a score of 3 or 4. Stunkard’s Likert scale questions do contain some American terminology. In particular, the word ‘supertime’ can be confusing, with ‘supper’ meaning either evening meal or a separate later meal or snack depending on the culture. As Stunkard’s criteria and the NESHI instructions refer specifically to ‘after the last evening meal’ clarification during the interview was given and subjects were asked to choose ‘after the

last evening meal'. The presence of AN, BU and BED was defined by subjects testing positive for all disorder-specific criteria as listed in Table 5 and Table 7. Individuals were classified as having sub-threshold BU and BED, if the key behaviours of bingeing and/or purging were present, but not at the frequencies specified by the core criteria. (Key constructs are highlighted in tables in bold).

3.4.2.4. *Beck Depression Inventory (BDI)*

3.4.2.4.1. **Background**

The BDI (Beck et al. 1961) has been used in 2000 studies world-wide as a measure for assessing the intensity of depression in psychiatric populations and for identifying depressive symptoms in non-psychiatric populations. Developed in 1961, the BDI-I was designed to reflect clinical observations about the attitudes and symptoms displayed frequently by depressed psychiatric patients and infrequently by non-depressed psychiatric patients, rather than to reflect a particular theory of depression. It contains 21 items with a potential score of 63, (<10 indicates no, or minimal depression, 10-18 mild to moderate, 19-29, moderate to severe and above 30 severe depression). It was revised in 1979 (BDI-IA) when alternative wordings of the same responses were eliminated and double negatives for 15 items were removed (Beck et al. 1979) (Appendix 3). A further revision in 1996 (BDI-II) reflected changes in the management of depression over time, with some milder symptom items substituted for symptoms typical of severe depression or depression warranting hospitalisation (Beck, Steer, & Brown 1996).

3.4.2.4.2. **Validity and Reliability of the BDI-I and 1A**

Studies into factorial validity show a lot of variability in earlier studies, with the number of factors varying between 1 to 7 depending on methods used and sampling effects, such as age of subjects and degree of depression, although all studies were reasonably large with more than 100 subjects (Beck, Steer, & Garbin 1988). Campbell (1984) for example identified the 3 factors of negative self attitudes, physiological symptoms and sadness using the principal components method. A later study in 414 college students suggested a 2 factor split of cognitive-affective symptoms and somatic symptoms (Storch, Roberti, & Roth 2004). Whilst Beck himself had earlier proposed a 2 factor split in a study of 500 outpatients of mixed psychiatric out patients, he could only account for 19 of the 21 items with Pessimism and Loss of Interest in Sex having loadings less than 0.35 (Beck, Steer, & Brown 1996). Whistman, Perez & Ramel (2000) were

able to load all 21 items onto the 2 factors in a sample of 576 college students, although this was using the BDI-II version and the other studies used the BDI-IA. In the face of such variable factor analysis findings, Beck concludes that the BDI reflects a general syndrome of depression composed of highly inter-correlated first-order symptom dimensions. This generalised view implies it may have limited use in differentiating different types of depression and should be viewed more as a symptom tool.

Nonetheless it appears to have acceptable concurrent validity, comparing well with other tools. Beck, Steer & Garbin (1988) conducted a meta-analysis of studies using either the BDI-1 or BDI-1A and other tools concurrently. Similar correlations of 0.41 to 0.86, 0.41 to 0.80 and 0.41 to 0.96 respectively were found in 3 separate groups of 15 psychiatric populations (sample sizes ranging from 30-325), 16 non-psychiatric populations (range 34 -1354) and 4 mixed groups (range 39-169). Correlations of BDI samples with clinical ratings in psychiatric patients were 0.72, though less strong in non-psychiatric patients (0.60). Generally, lower coefficients were noted with observer scales than with self-report tools and in more depressed psychiatric patients. This suggests self-report tools generally may be less effective than face-to-face interview and is in keeping with the construct validity findings, that the BDI-1 and 1A may be less helpful in identifying severe cases of depression.

3.4.2.4.3. **Internal Consistency**

Beck's meta-analysis of studies using the BDI-I and BDI-IA in 9 psychiatric populations and 15 non psychiatric populations showed high internal consistency, with mean alpha coefficients of 0.86 (0.76 - 0.95) and 0.81 (0.73 - 0.92) respectively (Beck & Steer 1984). Differences in sample sizes were accounted for, as although generally large, samples ranged from 78 to 409 in psychiatric populations and from 65 (female prisoners) to 1091 (general health survey) in other populations. Lowest alphas tended to be in very young or very old populations (young psychiatric adolescents and non-depressed older adults). Although these findings should be viewed cautiously as the meta-analysis mixes the two versions, Beck & Steer (1984) have argued that the internal consistencies between the two are comparable. Others support these findings. (Lightfoot & Oliver 1985; Richter et al. 1998).

3.4.2.4.4. **Test-retest Reliability**

Interpretation of test-retest reliability data is problematic as studies vary considerably in the timings between tests and results are often reported as a Pearson Product Moment Correlation Coefficient (measure of correlation between 2 variables), rather than a measure of agreement. In Beck's meta-analysis, the Pearson Product Moment Correlation Coefficients range from 0.48 to 0.86 in psychiatric populations (samples sizes ranging from 32 - 91) and 0.60 – 0.83 in much larger non-psychiatric populations (sample range 204 - 498). The higher lower boundary in the non-psychiatric populations suggests they display more stable BDI scores and that the tool is more sensitive to change in psychiatric populations, as would be expected if treatment effects were reflected in changes over time (Richter et al. 1998). Generally, reliability appears better in non-psychiatric samples and in shorter time distances, with some notable exceptions. A high Pearson Product Moment Coefficient of 0.82 was found in substance abusers after a 1 month retest, but only 0.65 after 1 week in a more generalised depressed population (Beck, Steer, & Garbin 1988).

3.4.2.4.5. **Use in an Obese Population**

Significantly higher mean total scores in psychiatric, as opposed to non-psychiatric populations have led others to use the BDI to identify potentially depressed individuals in a wide range of populations predisposed to depression such as chronic pain sufferers or hospitalised patients (Cavanaugh, Clark, & Gibbons 1983;Turner & Romano 1984). It is used commonly by obesity researchers and is used in a third of all pre-operative assessments for bariatric surgery in the US with negative results often used as a basis on which to deny patients surgery (Munoz et al. 2007). Yet the BDI is often challenged by others on its lack of representative norms, as studies on 'normal' populations often comprise highly specific groups such as University students and prisoners (Gotlib 1984;Richter et al. 1998;Tanaka-Matsumi & Kameoka 1986).

3.4.2.4.6. **Conclusion**

The BDI-1A appears to be a well-used tool with high internal consistency, reasonable test-retest reliability, sensitivity to change and high convergent validity with other depression rating scales (Beck, Steer, & Garbin 1988;Richter et al. 1998). Despite these strengths, it has some weaknesses which must be taken into consideration. Its controversial construct validity

limits its ability to identify depression per se, although it is still useful for identifying features suggestive of depression. The purpose of this study was not to ‘diagnose’ depression, track changes over time, or make comparison with a normal population, but to explore whether individuals with NES in an obese population are more likely to have depressive symptoms than non-NES obese individuals. Given that, at the time this study was initiated, other NES researchers were using the BDI-1A (Allison et al. 2005b; Allison et al. 2005a; Grilo & Masheb 2004), it would seem reasonable to use the same tool, as the benefits of allowing for easy comparison of results outweigh the limitations of the tool.

3.4.3. *Development of NES Screening Tool*

3.4.3.1. *Selection of Items*

The 2003 NES criteria require other ED to be absent before a diagnosis of NES can be made. WMC clinic resources did not allow for an in-depth assessment of every individual by an eating disorder specialist. Validated tools already existed which could be used to eliminate other eating disorders. In order to develop one, easily administered, composite NES screening tool which would be suitable for an obese UK population, core elements of tools used to identify other ED were combined with items from Stunkard’s NEQ (2004). As there were no known subjects with NES with whom key constructs could be debated, it was necessary to focus on the four main behavioural constructs contained in Stunkard’s 2003 diagnostic criteria. The four constructs are morning anorexia, evening hyperphagia, nocturnal awakenings and overnight snacking. Questionnaire items based on these constructs, along with other items, had originally been included in the 2003 NESQ (Wadden & Phelan 2002) which was adopted into the NEQ. For the purposes of this study, items from the NEQ were either chosen by JC for inclusion in the study screening tool, or rejected, depending on whether they reflected the four main constructs. Included items are listed below.

Morning anorexia

- How hungry are you usually in the morning?
- When do you usually eat for the first time?

Evening hyperphagia

- How much of your daily food intake do you consume after suppertime?

- Do you have urges to eat snacks after suppertime but before bedtime?

Nocturnal awakenings

- How often do you have trouble getting to sleep?
- Apart from getting up to go to the toilet, how often do you get up at least once in the middle of the night?

Overnight snacking

- When you get up in the middle of the night, how often do you snack?
- If you snack in the middle of the night, how aware are you of your eating?

Low mood

Two questions relating to mood were also included. Although low mood is not listed as a core criterion for NES, these items were included as studies have consistently noted the distinctive circadian features of the mood patterns of NES sufferers and Stunkard includes mood items in the NESQ and NEQ (Birketvedt et al. 1999;Birketvedt, Sundsfjord, & Florholmen 2002;Gluck, Geliebter, & Satov 2001;Marshall et al. 2004). Stunkard uses American terminology to ask whether subjects ever feel ‘blue’ and the items were adapted to reflect more familiar English terminology.

- Do you ever feel low in mood?
- When you are feeling low in mood, is your mood better in the early morning / late morning / afternoon / early evening/ late evening?

Breakfast

The question ‘Do you find it difficult to eat breakfast?’ had originally been included in the WALI but removed by Stunkard, on the grounds that it was similar to the question ‘how hungry are you usually in the morning?’ After discussion with 3 local clinic dietitians, it was decided to reintroduce this item, in order to characterise better the construct of morning anorexia as most subjects in obesity clinics report not feeling hungry in the morning, even if they are compliant with eating breakfast.

3.4.3.2. *Excluded Items*

Two items, ‘Do you need to eat in order to get back to sleep when you awake at night?’ and ‘Do you have cravings or urges to eat snacks when you wake up at night?’ were excluded on the grounds that they were potentially open to misinterpretation. Positive responses would not necessarily signify that an individual had actually eaten anything. Two further items relating to control over night snacking, ‘How much control do you have over your eating while you are up at night?’ and ‘how much control do you have over your eating between supper and bed time?’ were also excluded as the issue of control is not included in the diagnostic criteria.

3.4.3.3. *Items Included Based on Other Diagnostic Criteria*

Extra items were also included in the tool which reflected the two other components of the diagnostic criteria, namely duration of the behaviours and absence of other eating disorders. ‘How long have you experienced your current pattern of eating and sleeping?’ had been asked by Stunkard as a stand-alone question at the end of the NEQ. In order to standardise the format of the study screening tool, it was included here as a Likert scale question, with scores ranging from 0-4 depending on whether the duration was zero, less than 3 months, less than 6 months less than 12 months or above 12 months. Behaviour of longer duration was given a higher score as a chronic problem is more likely to have a greater impact on the individual.

With regard to the exclusion of other eating disorders, identifying suitable items for inclusion in the screening tool was problematic as other validated tools such as the EAT (Eating Attitudes Test) or BITE (Bulimic Investigatory Test) tend to be lengthy, including 40 and 30 items respectively and would be impractical for adding to another tool (Garner & Garfinkel 1979; Henderson & Freeman 1987). By contrast, the SCOFF questionnaire is a short, 5-item, validated and reliable diagnostic tool with good sensitivity and specificity for diagnosing BU and AN, which has been previously administered both verbally and in writing (Morgan, Reid, & Lacey 1999). Four of the SCOFF items listed below were included in the screening tool:

- Have you recently lost more than 1 stone in a 3 month period? (please circle yes or no)
- Would you say that food dominates your life?
- Do you worry you have lost control over how much you eat?
- Do you ever make yourself sick because you feel uncomfortably full?

The fifth SCOFF item, ‘Do you believe yourself to be fat when others say you are thin?’ was not included as it was considered to be inappropriate for morbidly obese subjects. A positive response to two or more of the SCOFF items indicates a likely case of either AN or BU. With regard to BED, one question was included in the screening tool which is based on Spitzer’s validated core criteria for BED (Spitzer et al. 1993).

- Do you ever eat large amounts of food in a short period of time (larger than most people would eat during a similar time and under similar circumstances)?

Also, one of the SCOFF items, ‘Do you worry you have lost control over how much you eat?’ concerns loss of control over eating which reflects the second key construct of BED (Spitzer et al. 1993). In total, the final version consisted of a 17 item Likert scale screening tool (Appendix 4), the contents of which were discussed with the 3 obesity clinic dietitians in order to establish face validity. Each item had a potential score ranging from 0-4 and item scores, when combined, produced a total score ranging from 0-68. It was envisaged that testing of the tool in the WMC would produce cut points that supported its use as a screening tool for NES in an obese clinic population.

3.4.4. *Testing of the Tool and Sample Size*

In order to test the sensitivity and specificity of the tool it was necessary to identify both individuals with and without NES in the obesity clinic population. As the prevalence of NES in this clinic was unknown at the time of developing the study, the sample size was based on prevalence data from other obesity clinic populations. On the assumption that 15-20% of a clinic population of 400 would have some features suggestive of NES and be willing to undertake an in-depth diagnostic interview, this would result in 60-100 research volunteers. Based on previous studies, it was estimated that this would produce approximately 50 cases of NES, the minimum required to estimate the sensitivity of the screening tool to within $\pm 10\%$ (with 95% confidence, assuming a sensitivity of 85% or better). In order to estimate the specificity of the tool to within $\pm 10\%$ (assuming a specificity of 70% or better), a second group of 81 subjects identified at interview as not having NES was also required.

It was also planned to perform test-retest reliability on the tool by posting it to all clinic attendees (n=400) and repeating the process 1 month later to all respondents. This process would be blinded to the researcher in order to ensure the anonymity of the participants. Although the questionnaire would be anonymous, respondents would be asked to indicate gender, age, weight, height and socio-economic group in order to stratify the results.

3.5. *Methods*

3.5.1. *Ethical Approval*

Ethical approval to conduct the study was obtained from St Helen's and Knowsley Ethics Committee (ref no. 04/Q1508/9) on 21st July 2004 and the UCLan Faculty of Health Ethics Committee (ref no. CA 142) on 11th August 2004. Management approval from the hospital R&D Committee (ref no. 04DE006) was obtained on 17th August 2004. An amendment was granted to include Dr. Sutton as a supervisor on 3rd April 2005.

3.5.2. *Recruitment*

Participants were recruited from a Specialist Weight Management Clinic (WMC) in the North West of England, which is part of an acute NHS Foundation Trust. It is a secondary care service, established with the aim of providing comprehensive assessment and treatment for severely obese patients as part of a local Primary Care Trust strategy for obesity management. Referrals are currently accepted from general practitioners and physicians for patients with a BMI > 40 kg/m² or a BMI > 35 kg/m² with existing co-morbidities. The patient treatment pathway is shown in Appendix 5, although since the commencement of this study, some pharmacological treatment options are no longer available. Patients have an initial assessment by a physician and 3-4 monthly follow-up appointments with either a doctor or nurse for a period of 2 years. Where appropriate, patients are treated with pharmacotherapy and expected to attend for monthly dietetic review and a physiotherapy-led exercise programme. After 2 years they may be discharged to appropriate community-based services, or considered for bariatric surgery.

From May 2005 to December 2007, 7 practitioners in the weight management clinic (5 doctors, the specialist nurse (JC) and the dietician) assisted with patient recruitment. The assistance of doctors was required as the clinic protocol did not allow JC to see patients at their first clinic appointment. Individuals were shown a flyer containing 3 simple questions about

night-eating during their clinic appointment, if it became apparent during discussion that they had some features of NES. The questions were:

- Do you find it difficult to eat breakfast?
- Do you often eat during the night time?
- Do you have trouble sleeping?

Patients were then invited to take a copy of the Patient Information Sheet attached to the flyer and asked to provide a phone number for JC to contact them in a week's time if they expressed an initial interest in taking part. Patients were also given another option to contact JC directly. It was originally envisaged that recruitment of the additional participants to the control group would occur after the NES group was identified, but in practice, recruitment for both ran concurrently, as subjects who did not feel the poster applied to them were still willing to take part. Some individuals with 'standard' eating patterns who had not seen the poster were also approached by JC. Recruitment of individuals with features suggestive of NES was slower than predicted and extended from 6 months to 18 months, after which numbers of individuals both with and without NES were assessed. Although the original sample size was not attained, it was considered large enough to evaluate the screening tool, and the decision to stop recruitment was taken. Time constraints on the researcher made further recruitment impractical.

A flow chart outlining the recruitment process is shown in Figure 1. Participants were recruited from a pool of 400 clinic attendees. One hundred and twenty one participants expressed an interest in taking part. After a follow-up phone call, seven individuals with suspected features of NES declined to take part and approximately 30 others who did not consider the study applicable to themselves also declined to take part. It is not known how many individuals declined a Patient Information Sheet whilst in clinic. Of the 84 individuals who agreed to take part, 35% percent were recruited at their first clinic appointment and 65% at a subsequent follow up appointment. Three individuals attended for the diagnostic interview but did not return to complete the screening tool, thus complete data are available on 81 subjects. Interviews were performed in the Patient Assessment Area of a dedicated research unit, separate to the Outpatient clinic facility.

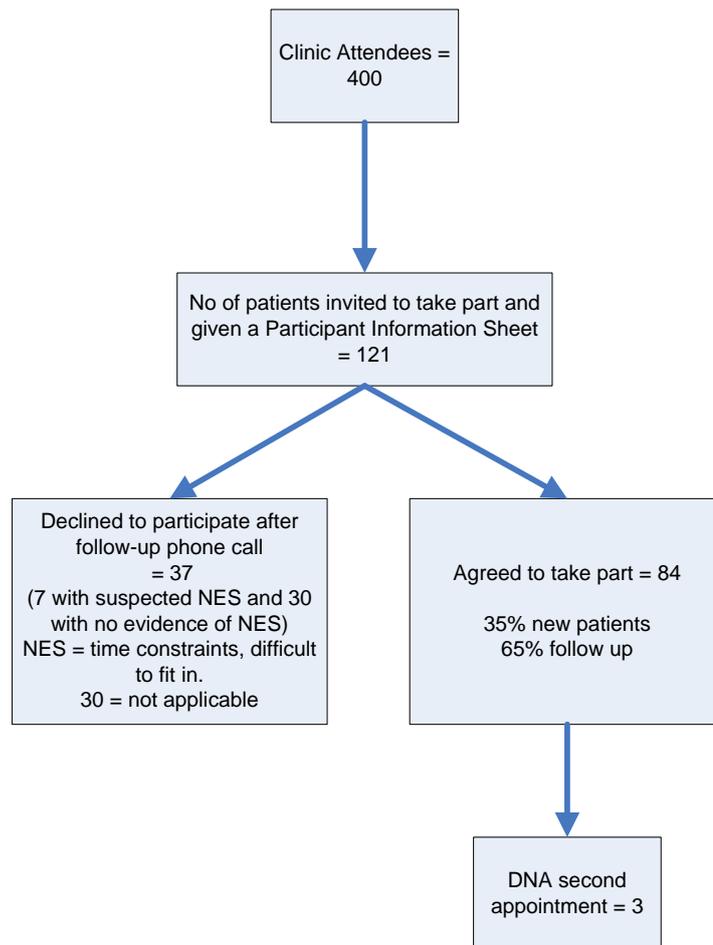


Figure 1- Flow chart of recruitment

3.5.3. *Completion of Screening Tool*

All subjects who attended for the initial interview were invited to return to the research facility for a second appointment to complete the proposed screening tool so that the results of the interview process and the questionnaire could be compared and tested for concurrent validity. Subjects were encouraged to complete the screening tool without supervision by the researcher.

3.5.4. *Additional Data*

Demographic data were also collected on all subjects in order to characterise better any difference between the NES group and non-NES group. Data collected included weight (kg), BMI (kg/m^2), gender (male/female), age (years) and work status. Participants were asked to self-report usual sleep and wake times, including differences on weekdays and weekend, work and non-work days. An average total sleep duration (hours) was calculated.

3.5.5. *Identification of Co-morbidity*

Evidence of type 2 diabetes was identified from medical records, together with documented evidence of anxiety and depression. No distinction was made between individuals diagnosed with anxiety and depression by their GP or by a psychiatrist. Evidence of a diagnosis of OSA was also obtained from medical records. Individuals currently under investigation for OSA were also included in the analysis.

3.5.6. *Statistical Analysis*

Interview subjects were categorised as suffering from BU, BED, NES or sub-threshold BU, BED and NES, according to the criteria listed in Table 5, Table 7 and Table 8. SPSS (version 14) and Inter Cooled Stata (version 9) were used to perform data analysis. Data was generally presented as means (SD). Independent samples t tests were used to perform between-groups analyses of means on approximately normally distributed data and Mann Whitney U tests on other ordinal data. Frequencies (%), with chi-square analysis, were calculated for gender, work status and co-morbidity with Fisher's exact test and bootstrapping used on small sample sizes either approaching significance or just reaching significance (p-values between 0.01 and 0.1). Between-group comparisons of variances (SD) were made for approximately normally distributed data using F-tests. Post-hoc analysis was performed on sleep duration using an F test for equality of variances. Corrections have not been made for multiple testing due to the exploratory nature of the analysis. The sensitivity and specificity of the study tool were estimated; with exact 95% confidence intervals estimated using Inter Cooled Stata (version 9). NES tool items were totalled to produce an overall score in keeping with Stunkard's approach and a receiver operator curve (ROC) was produced to identify an appropriate cut point for defining cases of NES or sub-threshold NES based on the findings of the diagnostic interview. Due to the impracticalities of analysing items for other ED and sub-threshold ED groups, items were converted into a 'positive' or a 'negative' response and compared with the interview diagnosis.

3.5.7. *Confirmation of Diagnosis*

In order to confirm the reliability of the interview-based diagnosis of NES, a sample of 18 participants who were recruited early into the study were asked to have a second interview by

a dietician from the WMC. This dietician has a background in eating disorders and was blinded to the results of the original diagnostic interview. It was originally envisaged that all study participants would be subjected to a second interview, but due to practical considerations (maternity leave of the dietician) it was decided to analyse the data for agreement after the first 18 participants.

The level of agreement at interview between JC's and the dietician's diagnostic interviews (inter-rater reliability) was measured using the kappa coefficient. Three classifications were compared between raters; subjects identified with NES or sub-threshold NES, subjects identified with BED or sub-threshold BED and subjects identified with BU or sub-threshold BU. Initial interviews by both interviewers were tape recorded and compared to ensure consistency of approach during the interview process. There was a high level of agreement between JC and the dietician in identifying subjects with NES or sub-threshold NES. Both interviewers agreed on 9 out of 10 positive cases and 8 out of 8 negative cases (Table 9) yielding a kappa coefficient of 0.89 (95% CI 0.6,1.0). There was disagreement on one individual only. Whilst both interviewers agreed that he had sub-threshold BU, he was not counted as NES or sub-threshold NES by the dietician as he consumed only drinks overnight. Following discussion, it was agreed to include this individual in the sub-threshold NES group, as the high calorific content of the drinks could be contributing to his obesity.

Similar high levels of agreement were seen in identifying positive and negative cases of BU and sub-threshold BU with a kappa coefficient of 0.77 (95% CI 0.3, 1.0). Only one case was identified as being BU by the dietician and not by the nurse (Table 10). This individual had been classed as suffering from BED by JC as no vomiting was reported during JC's interview. Greater disagreement was seen however, in the identification of BED and sub-threshold BED, with a much lower kappa coefficient of 0.45 (95% CI -0.2, 0.9) (Table 11).

Eleven negative cases of BED or sub-threshold BED were identified by both interviewers, but agreement was reached on 3 positive cases only. The interviewers disagreed on 4 subjects, with both identifying 2 separate subjects as having BED or sub-threshold BED. One of these cases was the individual previously discussed who was classed by JC as having BED on the grounds that no vomiting was reported. In the other 3 cases, two factors appear to have

contributed to the discrepancies. Firstly, subjects appeared to ‘change their story’ and report different amounts of food during episodes of overeating between interviews. This was compounded by the difficulty of distinguishing episodes of bingeing from overeating, especially in obese individuals whose energy intakes are naturally higher. Whilst the average binge is considered to be 1300 calories, this would not constitute an abnormally large amount of food for some morbidly obese individuals to consume in one episode and all 3 individuals had a high BMI (47, 65.3 and 63.5 kg/m² respectively).

As the discrepancies in the classification of the BED, sub-threshold BED, BU and sub-threshold BU groups appeared to be as a result of inconsistent reporting; it was decided to retain subjects in the original diagnostic groups allocated by JC.

Table 9 – Level of agreement between JC and dietitian in identifying individuals with NES with a diagnostic interview. Agreement expressed as a kappa coefficient.

	<i>No night eating (dietitian)</i>	<i>Presence of night eating (dietitian)</i>	<i>Total</i>	<i>Kappa coefficient and 95% Confidence Interval (lower limit, upper limit)</i>
<i>No night eating (nurse)</i>	8 (100 %)	0 (0 %)	8	
<i>Presence of Night eating (nurse)</i>	1 (10%)	9 (90 %)	10	0.89 (0.65, 1.0)

Table 10 - Level of agreement between JC and dietitian in identifying individuals with BU with a diagnostic interview. Agreement expressed as a kappa coefficient.

	<i>No AN/BU/sub BU (dietitian)</i>	<i>Presence of AN/BU/sub BU (dietitian)</i>	<i>Total</i>	<i>Kappa coefficient and 95% Confidence Interval (lower limit, upper limit)</i>
<i>No AN/BU/sub BU (nurse)</i>	15 (94 %)	1 (6. %)	16	
<i>Presence of AN/BU/sub BU (nurse)</i>	0 (0 %)	2 (100 %)	2	0.77 (0.34, 1.0)

Table 11 - Level of agreement between JC and dietitian in identifying individuals with BED with a diagnostic interview. Agreement expressed as a kappa coefficient.

	<i>No BED/sub BED (dietitian)</i>	<i>Presence of BED/sub BED (dietitian)</i>	<i>Total</i>	<i>Kappa coefficient and 95% Confidence Interval (lower limit, upper limit)</i>
<i>No BED/sub BED (nurse)</i>	11 (84.6 %)	2 (15.4 %)	13	
<i>Presence of BED/sub BED (nurse)</i>	2 (40.0 %)	3 (60 %)	5	0.45(-0.15,0.87)

3.6. Results of Diagnostic Interviews

3.6.1. Demography

The demographic characteristics of the study group (n=81) appeared broadly similar to those of the clinic population identified in a clinic audit conducted in January 2005 (Table 12), although the study population included proportionally more men (43% compared to a clinic population of 33%). Participants were on average 45 years of age, with a mean body weight of 145.0 kg, reflected in the higher BMI of 50.0kg/m² (compared to the clinic average of 48.5 kg/m²).

Table 12 - Demographic details of study population compared to obesity clinic audit conducted in January 2005.

<i>Variable</i>	<i>Clinic audit 2005 (n=100) Mean (SD)</i>	<i>Study pop (n=81) Mean (SD)</i>
<i>Age (yrs)</i>	44.5 (12.2)	44.6 (11.6)
<i>Male (%)</i>	33	43
<i>BMI (kg/m²)</i>	48.5 (9.9)	50.0 (10.7)
<i>Weight (kg)</i>	133.9 (28.0)	145.0 (36.5)

3.6.2. Identification of Cases

Analysis of JC's diagnostic interview revealed 7 cases of NES, 6 of BED and 1 of BU (Table 13). Two cases of sub-threshold BED and 2 of sub-threshold BU were also identified. One individual with NES also had sub-threshold BED, 1 with BED had sub-threshold BU and 1 with BU had sub-threshold NES. Unsurprisingly, no subject was identified with AN or sub-threshold AN as a core criterion is a body weight of at least 15% below normal. Thirty nine individuals had no disordered eating.

Table 13 - Diagnosis of Eating Disorder at Interview (JC)

<i>Diagnosis category</i>	<i>(n)</i>	<i>Number of subjects with sub-threshold symptoms of other ED</i>
<i>NES (all 6 criteria)</i>	7	1 (BED)
<i>BED</i>	6	1 (BU)
<i>Bulimia</i>	1	1 (NES)
<i>Anorexia</i>	0	
<i>Sub-threshold category</i>		
<i>NES</i>	24	1 (BED), 3 (BU)
<i>BED</i>	2	
<i>Bulimia</i>	2	
<i>No disordered eating</i>	39	
<i>Total</i>	81	

A much larger group (n=24) displayed some, but not all, of the NES criteria and their individual characteristics are matched against the 2003 criteria in Appendix 6. The number of participants failing each criterion is listed below.

- 5 participants failed criterion 1 (am anorexia),
- 14 participants failed criterion 2 (evening hyperphagia)
- 10 participants failed criterion 3 (night awakenings)
- 9 participants failed criterion 4 (eating during awakenings), and
- 1 participant failed criteria 5 (pattern occurring for at least 3 months) and 6 respectively (absence of other ED).

Nineteen of the 24 sub-threshold NES group (79%) reported morning anorexia, although 14 participants failed the requirement to eat more than 50% of calories after the last evening meal, with 1-25% being a more typical estimate. Seven reported intakes of 50% and 3 participants reported intakes of more than 50%. One individual woke frequently to drink high calorie drinks,

but did not eat food at night. Two other individuals with no other NES symptoms, but with BED, reported evening hyperphagia, as did 2 others with no disordered eating. Individuals ‘failed’ the sleep criterion for diverse reasons. Four had bed-times ranging from 1.30am to 6am and frequently ate after midnight, but did not wake up at all to eat once they had gone to bed. Two individuals went to bed very late and occasionally woke in their sleep, but not at the frequency specified by the criterion. Two others reported night-time awakenings, but not at the required frequency. A further 2 individuals woke to eat on frequent occasions, but slept in the day and stayed awake at night. Another individual went to bed at 3 am and also woke frequently and was classed as positive according to the ‘sleep’ criterion.

Thirteen others had no other NES features, but reported waking at night. Two of these had BED, 1 had sub-threshold BU and 10 individuals had no disordered eating. Generally, those who failed the ‘night-time awakenings’ criterion also failed the ‘snacking during awakenings’ criterion. The 4 individuals who went to bed very late and slept all night did not eat once asleep and the 2 other late sleepers who woke occasionally did not eat when awake. The 2 individuals who slept in the day and woke to eat at night were classed as positive for criterion 4 as they did eat when they woke up, although this was not during the night. Only one individual reported his current pattern of behaviour to have developed less than 3 months prior to the interview. With regard to other eating disorders, only one individual with some NES features was diagnosed with BU, although three others had sub-threshold BU and 1 had sub-threshold BED. One individual fitting all the NES criteria also had sub-threshold BED and one with BED also had sub-threshold BU.

3.6.3. *Discussion*

Care must be taken not to interpret the levels of NES and sub-threshold NES identified in the identification study as prevalence estimates. Individuals were identified through purposive sampling, with the intention of identifying a cohort of likely NES individuals on which to test a tool. Despite the limitations of the sample, it is still noteworthy that there were few participants identified with NES (n=7, 9%) using the 2003 criteria, although this was in keeping with prevalence data from some studies (Adami et al. 2002;Stunkard et al. 1996). The majority of individuals with disordered eating at night identified in this study would not be classified as

having NES according to the 2003 criteria. This calls into question the appropriateness of the criteria generally, with some criteria appearing too inclusive and others too restrictive. Although most participants with either full NES or sub-threshold NES reported morning anorexia (84%), this behaviour was not exclusive to the NES group, with 73% of the group as a whole reporting morning anorexia. As this is a feature associated with morbid obesity and not exclusive to NES, its value as an indicator of NES in morbidly obese populations must be questioned.

The requirement to consume more than 50% of calories after the last evening meal appears unnecessarily restrictive and most other researchers have relaxed this criterion downwards when recruiting for studies (Allison et al. 2005b; Lundgren, Allison, & Stunkard 2006; Morse et al. 2006). This may be because subjects thought to be suffering from NES in one study reported eating 51% of their daily energy intake after the evening meal during interview, but the actual intake recorded in their food diaries was only 34.9% (O'Reardon, Stunkard, & Allison 2004). Others typically report intakes of 36.8% (Allison et al. 2005b). Allison et al. (2006) used 25% as a cut off when examining 210 (38m/172f) pre-bariatric surgery subjects, (mean BMI 50.42 kg/m²) for NES and only 23.7% of subjects self-reported an energy intake of >25% after the last evening meal. In this study four subjects classed as having NES reported eating 50% of their calories after the last evening meal. Whilst the NESHI criteria technically require subjects to eat more than 50%, it is a blunt cut-off point which is difficult to estimate accurately during a verbal recall of daily dietary intake. As these individuals fulfilled all other criteria and their behaviour appeared to be having a significant impact on their lives, they were still classed as having full NES, as the level of impairment may be a more important marker of dysfunction than the amount itself. The wording of the criterion with regard to energy intake is also confusing. Although the term 'high calorie snacks' suggests items of food, one subject woke frequently to drink high calorie drinks but did not eat at night. His behaviour was clearly impacting on his obesity, although it was unclear whether this would be considered NES and he was included in the sub-threshold NES group. Likewise, the four individuals who stay up until very late eating clearly have a problem which may be impacting on their obesity, but they are technically excluded from the 2003 criteria.

In addition to the limitations of the eating criteria, the sleep component of the criteria is underplayed and appears to exclude a large number of 'night-eaters'. NES sufferers are thought to experience a circadian rhythm dysfunction with a disassociation between eating and sleeping rhythms, characterized by a phase onset delay of morning appetite and continuation of evening eating (Rogers et al. 2006). Laboratory studies have suggested that sleep and wake times are not disturbed (Allison et al. 2005a; Birketvedt et al. 1999; Birketvedt, Sundsfjord, & Florholmen 2002). As previously discussed, there is limited evidence that subjects with NES experience less phase 2 and phase 3 sleep than non-NES controls, resulting in a lower total sleep time and reduced sleep efficiency (percent of time in bed spent asleep) (Rogers et al. 2006). The authors conclude that sleep duration may have a role to play in the pathogenesis of NES. Clearly, disturbed and irregular sleep patterns may be contributing to NES, but the 2003 criteria do not account for individuals who stay up until very late eating, but do not get out of bed to eat once they have settled to sleep, or individuals who wake to eat on frequent occasions, but sleep in the day and stay awake at night. Fourteen individuals from the non-disordered eating group also reported frequent night-time awakenings suggesting that obesity may be a risk factor for disordered sleep. Only one individual reported eating and sleeping patterns of less than 3 month's duration, suggesting these are chronic patterns of behaviour which have been established for some time.

Although the 2003 criteria for NES explicitly exclude other eating disorders, categorising individuals into standard eating disorder groups was not so simple in practice due to the significant numbers of patients with low level eating disorder pathology. Cross-over was noted between the diagnostic groups, with some subjects appearing positive for more than one category. For example one subject in the NES group and four in the sub-threshold NES group had either BU, sub-threshold BU, or sub-threshold BED. This may be a particular feature of morbid obesity, but it does call into question the appropriateness of the DSM-IV scheme for classifying eating disorders generally, suggesting it may be less important to identify and categorize disordered eating behaviour in a particular individual than to view it as a marker of psychiatric co-morbidity (Stunkard & Allison 2003).

All individuals identified with NES or sub-threshold NES in this study were recruited through the same process, with some features identified by the referrer during discussion in clinic and after seeing the poster. During the study interviews, all had a standardised approach in keeping with Stunkard's team. Although JC has extensive experience in interviewing obese individuals in a clinic setting the EDE v12 is not commonly used in the WMC clinical practice. To mitigate this, the clinic dietician (MP) who had previously used the tool extensively in an eating disorder setting provided appropriate training to JC during a mock interview in which MP acted as a subject.

One possible weakness of this study was the reliance on verbal estimates of energy intake as opposed to food diary data when identifying evening hyperphagia and disordered eating behaviour generally. Other NES researchers identified a tendency towards overestimation when reporting energy intake (Allison et al. 2005b; O'Reardon, Stunkard, & Allison 2004), yet under-reporting is a common phenomenon and is more likely in overweight and obese individuals (women more so than men) compared to their lean counterparts. Evidence suggests that the degree of under-reporting of energy intake in epidemiological studies ranges from as low as 10% (Garriguet 2008) to as high as 47% (Lichtman et al. 1992). In a UK based study of the 2000 National Diet and Nutrition Survey underreporting of energy intake was calculated to be 25% (Rennie, Coward, & Jebb 2007). Under-reporting in obese women in particular has been shown to be associated with depression. Kretsch, Fong & Green (1999) found a positive correlation ($r = -0.67$, $p = 0.2$) with the BDI and energy intake difference in 22 obese women (mean BMI 34.2kg.m^2). This suggests under-reporting may be influenced by psychological state and that reporting of energy intake may be less about the ability to comply with instructions, than the emotional and moral feelings that influence the perceived value of making a true record of events (Blundell 2000; Goris, Westtererp-Plantenga, & Westtererp 2000). It is possible then, that an obese individual with NES and depression is more likely to minimise overeating and this must be taken into account when considering estimates of energy intake in this study along with the natural inclination of humans to overestimate symptoms when being investigated for a particular problem.

3.6.4. *Conclusion*

Whilst it is clear that adherence to the very restrictive 2003 criteria would exclude many individuals with disordered night-time eating and sleeping patterns, the clinical significance of this broader spectrum of behaviour is not known. In particular, the impact of disordered sleep and obesity on the severity of NES remains unclear. As previously noted, others currently investigating NES in other countries have had similar dilemmas in categorising NES cases and have suggested classifications based on a continuum of severity (Colles, Dixon, & O'Brien 2007). In 2008, Stunkard acknowledged the restrictions of the 2003 criteria and proposed instead 2 core criteria of hyperphagia of at least 25% of daily calories after the last evening meal and/or awakenings to eat 3 times a week (Stunkard, Allison, & Lundgren 2008). When these were retrospectively applied to this study sample, 87% of those classified as having NES and sub-threshold NES were classed as full NES: exceptions were 2 late sleepers and 2 others who woke infrequently to eat small amounts only. A further 7 individuals with evening hyperphagia but a normal bedtime and sleeping pattern would also be classed as NES. It was considered appropriate therefore to class all NES and sub-threshold NES subjects identified in this study as one heterogeneous 'Night Eating Behaviour' (NEB) group in order to characterize further its clinical and behavioural characteristics.

3.7. *Subgroup Analysis: Comparison between NEB and Non-NEB Subgroups*

3.7.1. *Demography*

Preliminary comparisons between the NEB group (n=31) and all other subjects (n=50) were performed using statistical analyses as described in section 3.5.6. The groups had similar representation of males (14 (45%) NEB group; 21(42%) non-NEB group) but the NEB group was, on average, slightly younger (42 yrs versus 46yrs, $p = 0.21$) (Table 14).

Table 14 - Comparison of characteristics between the NEB group and non-NEB group. Differences reported as mean (SD) and using independent samples t tests unless otherwise stated.

<i>Characteristic</i>	<i>Mean (SD)</i>		<i>P value</i>	<i>95% Confidence Interval of the difference (NEB-Non-NEB)</i>
	<i>NEB (n=31)</i>	<i>Non-NEB (n=50)</i>		
<i>Age (yrs)</i>	42.3 (14.1)	46.0 (9.7)	0.21	-9.5, 2.1
<i>Number (%) of males ¶</i>	14 (45)	21 (42)	0.78	-20%, 30%
<i>BMI (kg/m²)</i>	52.5 (11.7)	48.4 (9.8)	0.09	-0.7, 8.9
<i>Wt (kg)</i>	155.3 (37.5)	138.3 (34.7)	0.04	0.7, 33.3
<i>Number (%) employed ¶¶</i>	9 (29)	27 (54)	0.03	-50%, -4%
<i>BDI total score</i>	24.8 (10.9)	17.7 (10.8)	0.01	2.1, 12.0
<i>Sleep duration (hrs) ¶¶¶</i>	7.59 (2.03)	7.82 (1.21)	0.57	-1.0, 0.6

¶ Chi square analysis ¶¶ (F test. F = 4.47, df = 30, 49, p<0.01).

The frequency distribution of both groups per age category is shown in Figure 2. It is noteworthy that a large proportion of the NEB group (41.9%) were aged under 35. Mean body weight and BMI were both higher in the NEB group, with weight only being significant (p=0.04). The frequency distribution of both groups per BMI category is shown in Figure 3.

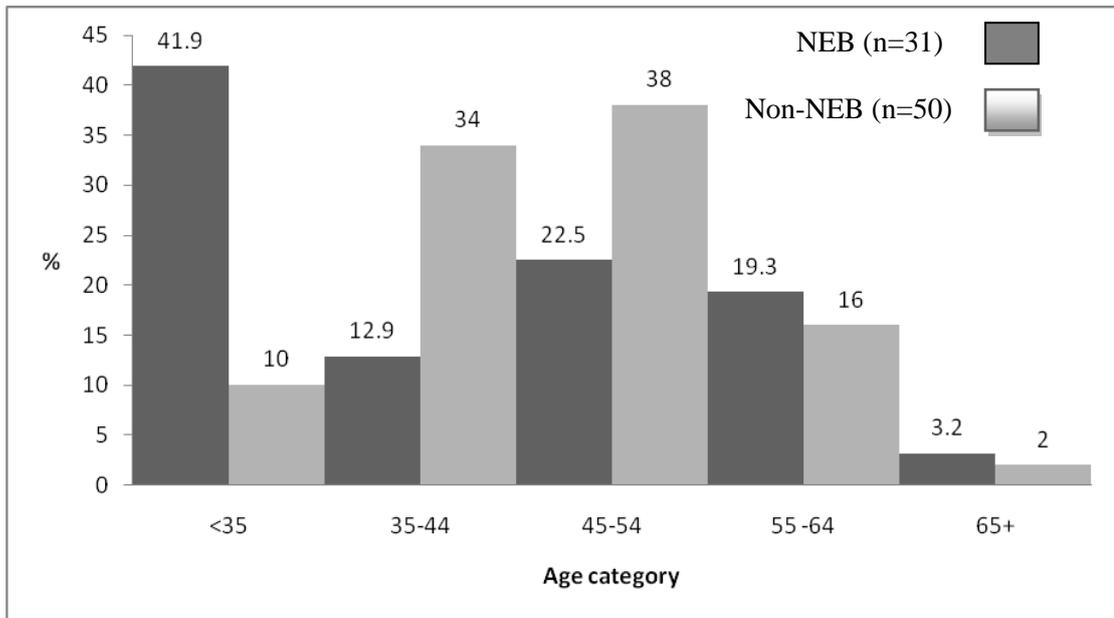


Figure 2 - Percentage distribution of participants in the NEB and Non-NEB group according to age category.

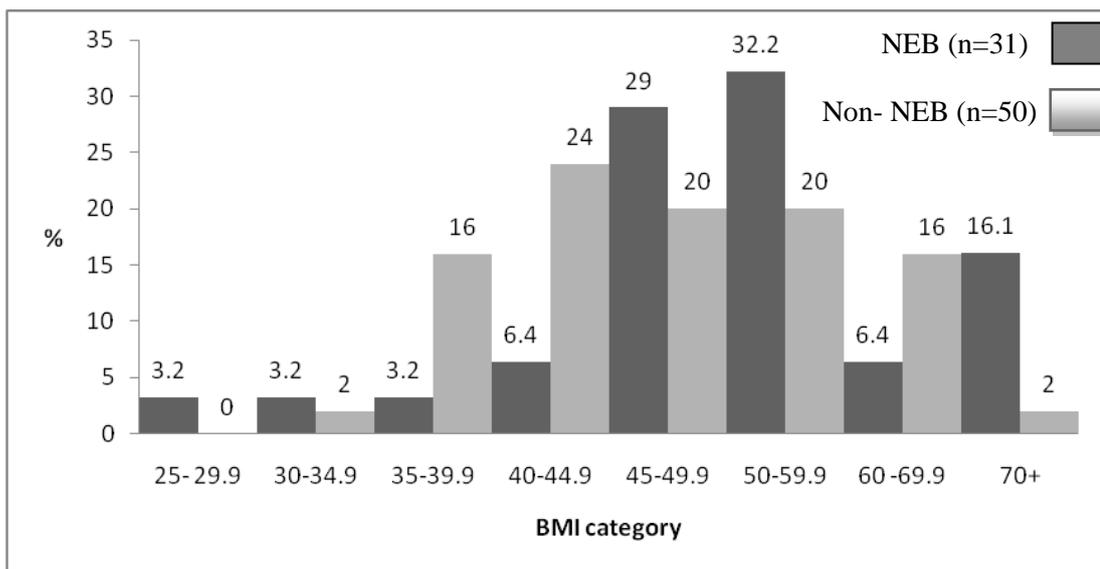


Figure 3 - Percentage distribution of participants in the NEB and Non-NEB group according to BMI category

Participants in the NEB group were less likely to be working ($p=0.03$) and scored significantly higher on the BDI scale ($p=0.01$), indicating greater depressed mood (Figure 4). Both groups reported less than 8 hours total sleep time on average with significantly greater variability noted in the NEB group ($p<0.01$).

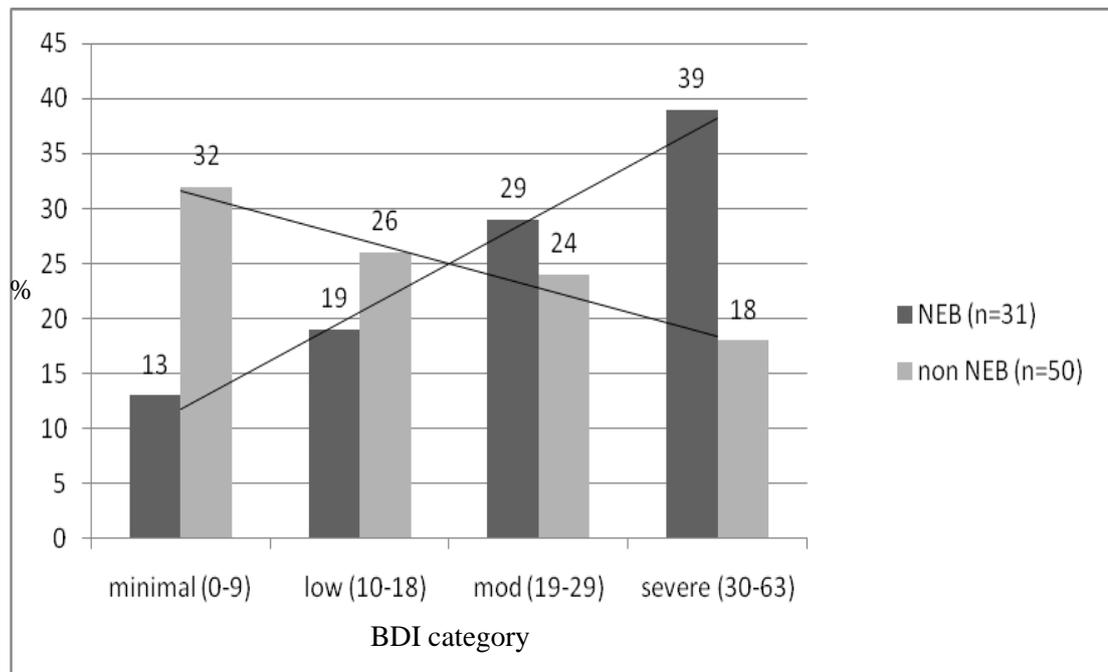


Figure 4 - Percentage distribution of BDI scores per BDI category indicating degree of depression for the NEB and non-NEB groups (the black lines are super-imposed linear trend lines for each group).

3.7.2. Co-morbidity

Twenty (24.7%) of the study group as a whole were diagnosed with, or were under investigation for OSA and the OSA group contained proportionally more males than the non-OSA group 70% vs 34.4% (p=0.005) (Table 15) .

Table 15 - Comparison of the proportion of males vs females in the study group with type 2 diabetes, OSA and depression. Significance tests performed using chi square analysis.

<i>Characteristic</i>	<i>Co-morbidity</i>		<i>P value</i>	<i>95% Confidence Interval of the difference (Co-morbidity-no co-morbidity)</i>
	<i>Type 2 diabetes N= 18</i>	<i>No Type 2 diabetes N=63</i>		
<i>Males (%)</i>	11 (61)	24 (38)	0.08	-3%,50%
	<i>OSA N= 20</i>	<i>No OSA N= 61</i>		
<i>Males (%)</i>	14 (70)	21 (34.4)	0.005	10%,60%
	<i>Depression N= 31</i>	<i>No Depression N= 50</i>		
<i>Males (%)</i>	10 (32)	25 (50)	0.12	-40%,4%

Study participants with type 2 diabetes were significantly older than those without type 2 diabetes (50 (11.4) yrs versus 43 (11.2) yrs (p=0.02)) (Table 16). Differences in mean BMI for study participants with OSA were approaching significance (53.0 (11.3) kg/m² OSA group; 49.0 (10.4) kg/m² non-OSA group. Almost half of the NEB group (14 (45.2%)) had a diagnosis of depression and/or anxiety compared to 17 (34%) of the non-NEB group (Table 17).

Table 16 - Comparison of characteristics of participants with and without type 2 diabetes, OSA and depression within the whole study group. Differences reported as mean (SD) using independent samples t tests.

<i>Characteristic</i>	<i>Co-morbidity (Mean (SD))</i>		<i>P value</i>	<i>95% Confidence Interval of the difference (Co-morbidity-no co-morbidity)</i>
	<i>Type 2 diabetes n= 18</i>	<i>No Type 2 diabetes n=63</i>		
<i>Age (yrs)</i>	50 (11.4)	43 (11.2)	0.02	1.2,13.2
<i>BMI (kg/m²)</i>	46.5 (9.6)	51 (10.9)	0.12	-10.1,1.2
	<i>OSA n= 20</i>	<i>No OSA n= 61</i>		
<i>Age (yrs)</i>	45.6 (11.8)	44.0 (11.6)	0.66	-4.7,7.3
<i>BMI (kg/m²)</i>	53.0 (11.3)	49.0 (10.4)	0.15	-1.5,9.4
	<i>Depression n= 31</i>	<i>No Depression n= 50</i>		
<i>Age (yrs)</i>	42.3 (10.9)	46.0 (11.9)	0.15	-9.0,1.5
<i>BMI (kg/m²)</i>	52.2 (11.1)	48.6 (10.3)	0.15	-1.3,8.4

Table 17 - Comparison of the proportion of individuals in the NEB group and Non-NEB group with type 2 diabetes, OSA and depression. Significance tests performed using chi square analysis.

<i>Co-morbidity</i>	<i>NEB group(n=31)</i>	<i>Non-NEB group (n=50)</i>	<i>P value</i>	<i>95% Confidence Interval of the difference in percentages (NEB-non-NEB)</i>
<i>Number (%) with Type 2 diabetes</i>	8 (26)	10 (20)	0.54	-13% ,28%
<i>Number (%) with Obstructive Sleep Apnoea</i>	6 (19)	14 (28)	0.50	-12% ,28%
<i>Number (%) with Depression</i>	14 (45)	17 (34)	0.32	-11% , 33%

OSA was present in 6 (19%) participants in the NEB group and type 2 diabetes in 8 (25.8%) participants. Differences were not significant (Table 17). Participants with type 2 diabetes in the NEB group had a higher BMI than those with type 2 diabetes in the non-NEB group, with the difference approaching significance (50.8(10.3) kg/m² versus 43.1 (8.0) p= 0.09) (Table 18). As with the study group as a whole, participants in the NEB group with type 2 diabetes were significantly older than those without type 2 diabetes (52.4 (11.3) yrs NEB group; 38.8 (13.4) yrs non-NEB group p=0.02) (Table 19).

Table 18 - Comparison of characteristics of participants with type 2 diabetes, OSA and depression between the NEB and non-NEB group. Differences reported as mean (SD) and using independent samples t tests unless otherwise stated.

<i>Characteristic</i>	<i>Co-morbidity (Mean (SD))</i>	<i>P value</i>	<i>95% Confidence Interval of the difference (NEB-Non- NEB)</i>
Type 2 diabetes			
	<i>NEB n=8</i>	<i>Non NEB n=10</i>	
<i>Age (yrs)</i>	52.4 (11.3)	48.4 (11.8)	0.48
<i>BMI (kg/m²)</i>	50.8 (10.3)	43.1 (8.0)	0.09
<i>Number (%) of males ¶</i>	5 (62.5)	6 (60)	0.91
			-7.7,15.6
			-1.5, 16.8
			-40%, 50%
OSA			
	<i>NEB n=6</i>	<i>Non NEB n=14</i>	
<i>Age (yrs)</i>	45.7 (13.1)	45.5 (11.7)	0.65
<i>BMI (kg/m²)</i>	52.5 (12.1)	53.2 (11.4)	0.85
<i>Number (%) of males ¶</i>	6 (100)	8 (57)	0.22
			-8.5,13.2
			-10.1,12.1
			-13%, 80%
Depression			
	<i>NEB n=14</i>	<i>Non NEB n=17</i>	
<i>Age (yrs)</i>	42.7 (13.8)	41.8 (8.1)	0.83
<i>BMI (kg/m²)</i>	53.9 (10.7)	50.8 (11.6)	0.45
<i>Number (%) of males ¶</i>	5 (35.7)	5 (29.0)	0.70
			-7.8,9.6
			-5.2,11.3
			-30%,40%

¶ comparison performed using Fisher's exact test

Table 19 - Comparison of characteristics between participants with and without type 2 diabetes, OSA and depression within the NEB group only. Differences reported as means (SD) and using independent samples t tests.

<i>Characteristic</i>	<i>Co-morbidity (mean (SD))</i>		<i>P value</i>	<i>95% Confidence Interval of the difference (Characteristic positive vs negative)</i>
	<i>Type 2 diabetes n= 8</i>	<i>No type 2 diabetes n=23</i>		
<i>Age (yrs)</i>	52.4 (11.3)	38.8 (13.4)	0.02	2.7, 24.5
<i>BMI (kg/m²)</i>	50.8 (10.3)	53.1 (12.3)	0.63	-12.3,7.6
	<i>OSA n= 6</i>	<i>No OSA n= 25</i>		
<i>Age (yrs)</i>	45.7 (13.1)	41.5 (14.4)	0.20	-4.2, 19.2
<i>BMI (kg/m²)</i>	52.5 (12.1)	52.5 (11.9)	0.65	-7.8, 12.2
	<i>Depression n= 14</i>	<i>Depression n= 17</i>		
<i>Age (yrs)</i>	42.7 (13.8)	42.0 (14.7)	0.88	-9.8,11.3
<i>BMI (kg/m²)</i>	53.9 (10.7)	51.4 (12.7)	0.57	-6.3, 11.2

3.7.3. *Eating Disorder Examination Subscales*

No significant differences emerged between groups for the median EDE global scores (Figure 6). The Restraint sub-scale score was approaching significance (p= 0.06). The individual items of ‘restraint over eating’ and ‘dietary rules’ accounted for this (p=0.03 and p= 0.02 respectively), along with the ‘food avoidance item’, which although not statistically significant showed a median difference between groups (Figure 5). The Eating Concern subscale was also approaching significance (p=0.06) with the ‘social eating’ and ‘eating in secret’ items accounting for this (p= 0.04 and p=0.03). The non-NEB group scored higher on the Restraint subscale, but lower on the Eating Concern subscale, contributing to a balancing out of the median global scores. Only one item in the Shape and Weight Concern subscales, the ‘Avoidance of Exposure’ item showed a significant difference between the 2 groups (p= 0.02). Participants in both groups generally scored higher (i.e. worse) on the Shape and Weight Concern items than on other subscale items (Figure 6).

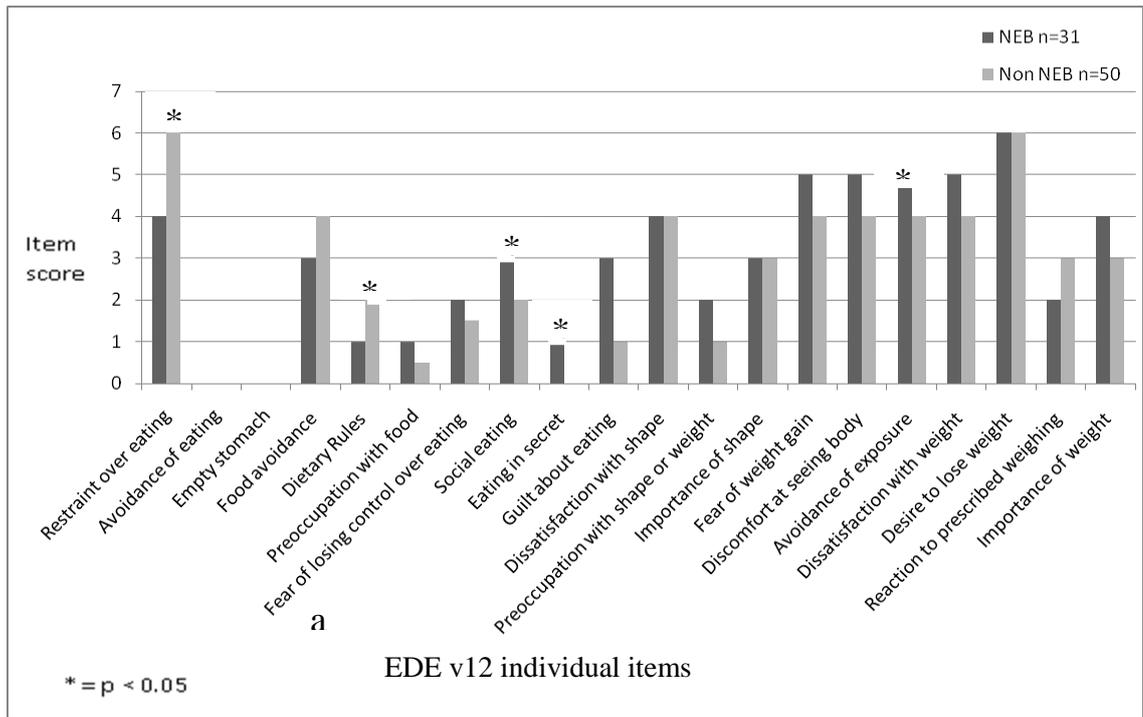


Figure 5 - Comparison of the median EDE v12 individual item scores between the NEB and non-NEB groups using the Mann Whitney U test.

^a n = 23 (NEB) vs 44 (non-NEB)

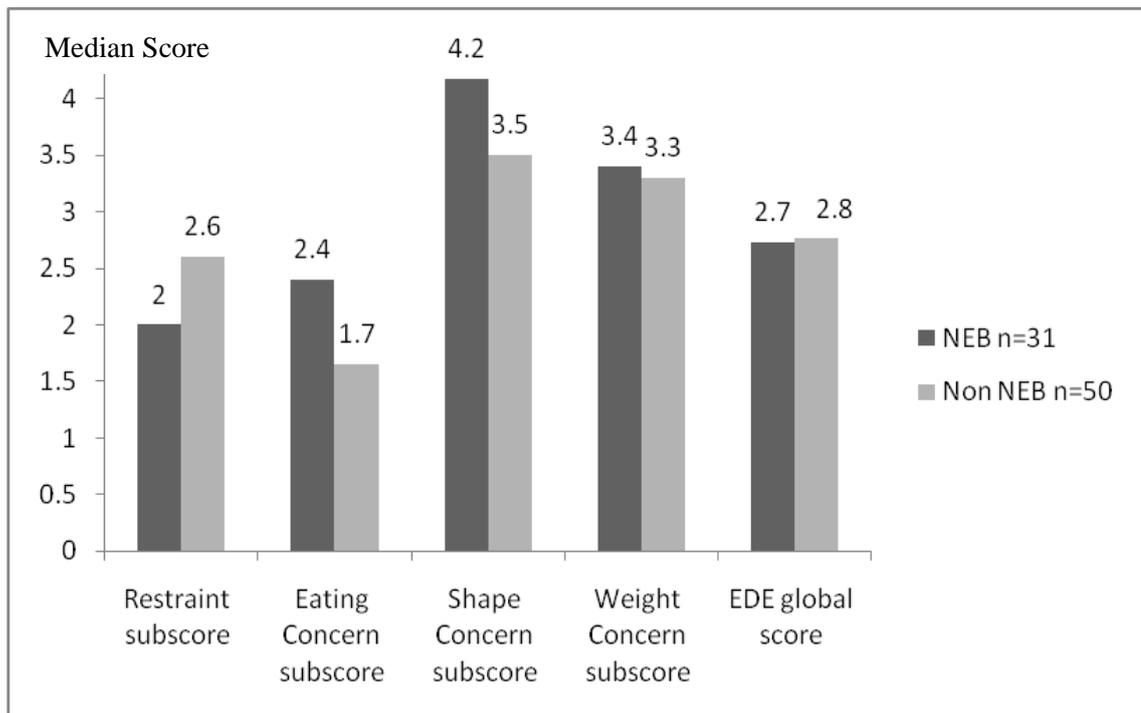


Figure 6 - Comparison of the median EDE v12 subscale and global scores between the NEB and non-NEB groups using the Mann Whitney U test.

3.7.4. *Stunkard's Night Eating Questionnaire (NEQ 2004)*

Total scores of the NEQ (2004) items included in Stunkard's NESHI were significantly greater in the NEB group (28.3 versus 13.0 $p < 0.001$) (Table 20). Significant differences were noted between all individual item scores except for morning anorexia and both mood-related items. Highest levels of significance ($p < 0.001$) were noted in all items directly referring to late night-eating and night-time disturbances, although subjects who do not wake to eat are instructed not to complete these items and automatically score 0.

Table 20 - Comparison of the NEQ item scores and total score by NEB classification using independent samples t tests.

<i>NEQ item</i>	<i>Mean (SD)</i>		<i>P value</i>	<i>95% Confidence Interval of the difference (NEB-non NEB)</i>
	<i>NEB (n=31)</i>	<i>non-NEB (n=50)</i>		
<i>Total score</i>	28.3 (7.6)	13.0 (5.3)	<0.001	12.0, 18.5
<i>Morning hunger</i>	2.9 (1.2)	3.0 (1.1)	0.75	-0.6, 0.4
<i>Eat for the first time</i>	1.3 (0.9)	0.9 (1.0)	0.05	0.0, 0.8
<i>Cravings before bedtime</i>	2.9 (1.4)	1.5 (1.6)	<0.001	0.6, 2.0
<i>Control before bedtime</i>	2.2 (1.4)	1.5 (1.3)	0.03	0.1, 1.3
<i>Intake after suppertime</i>	1.9 (0.8)	0.8 (0.8)	<0.001	0.7, 1.5
<i>Feeling blue</i>	2.1 (1.5)	1.7 (1.4)	0.32	-0.3, 1.0
<i>When feel blue?</i>	2.2 (1.6)	1.6 (1.6)	0.09	-0.1, 1.4
<i>Trouble getting to sleep</i>	2.5 (1.5)	1.6 (1.5)	0.01	0.2, 1.6
<i>Up at night</i>	2.6 (1.6)	0.7 (1.3)	<0.001	1.3, 2.6
<i>Cravings at night</i>	2.4 (1.6)	0.1 (0.3)	<0.001	1.7, 2.9
<i>Need to eat to sleep?</i>	1.9 (1.7)	0.0 (0.0)	<0.001	1.3, 2.6
<i>How often snack?</i>	2.1 (1.7)	0.0 (0.2)	<0.001	1.5, 2.7
<i>How aware when eating at night?</i>	2.6 (1.7)	0.2 (0.8)	<0.001	1.8, 3.1
<i>Control at night</i>	1.9 (1.7)	0.0 (0.1)	<0.001	1.3, 2.5
<i>Duration of night eating (yrs)</i>	9.3 (12.7)	N/A	N/A	N/A

Pearson's correlation between the NEQ (2004) total score and BDI total score was moderate (0.47 $p= 0.01$) (Figure 7). This further confirms the positive association between NEB and depressive symptoms as identified by the significant difference in BDI total score between groups.

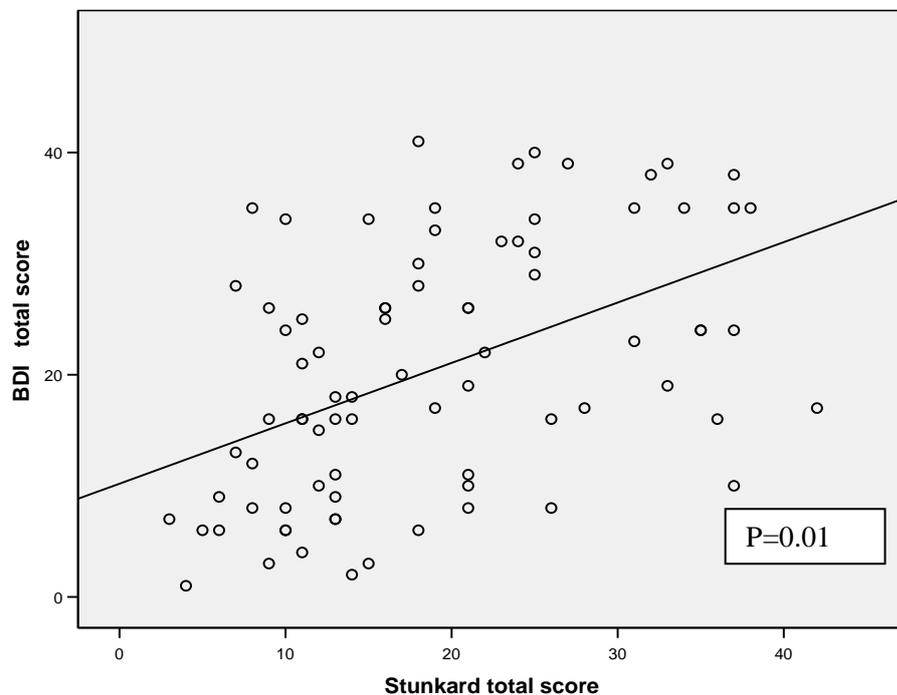


Figure 7 - Relationship between NEQ total score and BDI total score for all study participants (n=81).

3.8. *Screening Tool Results*

Screening tool items fell into three groups: those designed to identify NES, those designed to identify BED and those designed to identify AN or BU. Although BED and BU in particular share similar features and some crossover exists between eating disorders generally, for the purposes of the tool analysis all three groups were considered separately, although the item relating to loss of control over eating was included in both the BED and the AN/BU analysis. It was proposed that the screening tool should be expected to show a sensitivity of 0.85 or better, with a lower confidence limit of 0.75 or higher and a specificity of 0.70 or better with a lower confidence limit of 0.60 or higher. Based on data from 80 individuals as one interview had not been undertaken at the time of data analysis, the optimum sensitivity and specificity was seen with a score of 23 and above, (sensitivity of 0.93 with a CI width of 21 and lower limit of 0.78 and a specificity of 0.78 with a CI of 0.64-0.89). This suggests that a score of 23 is the optimum cut point for the tool and is a good predictor of subjects who will be positive for night-eating in a

diagnostic interview. Unfortunately, despite a high sensitivity for BED and sub-threshold BED (1.0 (95% CI 0.66, 1) specificity was only 0.14 (95% CI 0.07, 0.24). Likewise, sensitivity for the BU, and sub-threshold BU was 1.0 (95% CI 0.59, 1), with an extremely low specificity of 0.10 (95% CI 0.04, 0.19).

3.9. *Discussion*

3.9.1. Demographics: Age/Gender/BMI

A wide range of BMI was noted in both groups (27-76 kg/m² NEB group; 33-72 kg/m² non-NEB group). More than 90% of the NEB group and 82% of the non-NEB group had a BMI above 40 kg/m². Given that a BMI \geq 35 kg/m² is an essential requirement for referral to the clinic, it is unsurprising that the higher BMI ranges were over-represented in both groups, however the larger numbers of night-eaters with morbid obesity supports the association of NES with obesity found in other studies (Aronoff, Geliebter, & Zammit 2001;Kuldau & Rand 1986;Napolitano et al. 2001;Rand, Macgregor, & Stunkard 1997;Rand & Kuldau 1993;Stunkard, Grace, & Wolff 1955; 1955).

The mean age of all study participants is in keeping with an Australian pre-bariatric surgery group, (mean age 45.2yrs) although the pre-bariatric night-eaters were slightly older (mean age 47yrs) compared to the NEB group in this study (Colles, Dixon, & O'Brien 2007). Crucial differences in defining night-eating are noted between these studies with Colles, Dixon & O'Brien (2007) including consumption of at least 50% of calories after 7pm and sleep 'difficulties' more than 3 nights a week. It is striking that 41% of the NEB group were aged less than 35 years, compared to only 10% of the non-NEB group. NEB was relatively less common in participants aged over 55 than in other age groups. This may be partially a cohort effect as the exponential rise in obesity over the past 20 years has seen more individuals developing obesity at a younger age and it is possible that NES contributes to a more rapid rise in weight in those who are already overweight as proposed by Striegel-Moore et al. (2008). Others have found significant differences in ages in participants with NES at much lower BMI levels than those seen in this study. Marshall et al.(2004) found a mean age of 33.1 yrs in NES subjects with a BMI < 25 kg/m² and 41.5 yrs in those with a BMI >30 kg/m². The mean duration of NES behaviour in

Marshall's study was 10.4 years and 17.4 years respectively. De Zwann et al. (2006) report a mean duration of 5 years. Participants in this study reported a mean duration of 9 years and 3 months, although the range was very broad, ranging from just under 3 months to 47 years. It is possible that some individuals developed obesity as a result of their night-eating, but others may have already been obese when they started to night-eat. Marshall et al. (2004) found 52% of obese subjects to be normal weight before they started night-eating, suggesting NES was a risk factor for obesity. The relationship between obesity and NES seems particularly complex, with the evidence at times contradictory. It is possible that more than one causal pathway can be identified, in which obesity may be either the cause or the effect of NES depending on the initial trigger for night-eating behaviour and the age at which it first developed.

Almost half (45%) of the NEB group were male, with a similar level of 42% in the non-NEB group, as compared to 33% in the clinic audit population as a whole. It is unclear whether more male volunteers came forward for the study because they exhibited more NES features, or whether clinic staff was more successful in inviting males to take part. In many of the earlier landmark NEB characteristics studies, females appear to be over-represented, although, once again, it is unclear whether recruitment strategies contributed to this (Birketvedt et al. 1999; Birketvedt, Sundsfjord, & Florholmen 2002; Marshall et al. 2004). In a large population study, gender differences were less marked, with 9% of women and 7.4% men reporting eating at night (Andersen et al. 2004). A prevalence study in the WMC cohort would be required to determine the true level of NEB in males although other obesity researchers have found similar high levels of males. An investigation into NES in an obesity clinic population found that those who wake to eat are more likely to be male (Colles, Dixon, & O'Brien 2007) and others have suggested a level as high as 60% (Aronoff, Geliebter, & Zammit 2001).

3.9.2. *Obstructive Sleep Apnoea, Type 2 Diabetes and Depression*

It is possible that the presence of co-morbidity may have influenced the proportion of males in the NEB group as OSA in particular is known to be more common in obese men (Coughlin et al. 2004). Significantly more of the participants with OSA taking part in this study were male than female. The true prevalence of OSA in the WMC is unknown although unpublished audit data from patients attending the clinic in 2001 suggest a level of 9%, much

lower than in our study group. Participants under investigation for OSA were also included in the OSA group as, in order to be referred for assessment, they had reported disturbed sleep and excessive daytime sleepiness. It is possible that these individuals are artificially raising the estimated prevalence of OSA, although it is more likely that increased awareness has raised detection levels since 2001. Nonetheless, 19% of the NEB group were experiencing some OSA features which could be driving or worsening the disordered eating and sleep behaviour of a significant number of individuals.

Similar levels of type 2 diabetes to OSA were also found in the NEB group. Most morbidly obese patients with type 2 diabetes referred to our practice are managed within the diabetes service rather than in the WMC, so these figures are probably an under-representation of the level of type 2 diabetes one would expect in such a morbidly obese population. Night-eating behaviour was reported by 9.7% of 714 diabetes clinic subjects and 8.4% of the diabetes 'Look Ahead study' participants (Allison et al. 2008a; Morse et al. 2006). Participants with type 2 diabetes, both within the study group as a whole and within the NEB group were older than participants without type 2 diabetes, although the significance of this must be viewed cautiously, given the likely under-representation of type 2 diabetes in this study sample. It is, nonetheless, reasonable to assume that features of type 2 diabetes may be influencing the night-eating behaviour of some of the NEB group in our study and influencing the level of impairment. Comparison with other studies is problematic as the level and impact of OSA and type 2 diabetes are not reported often in NES studies (Aronoff, Geliebter, & Zammit 2001; Colles, Dixon, & O'Brien 2007; Lundgren et al. 2006).

The mean BDI-1A score of the non-NEB group (17.7 (10.8)) was similar to the mean score of a pre-bariatric surgery population (16.5 (9.2)) using the same version of the tool (Munoz et al 2007). Dixon, Dixon & O'Brien (2003) found similar scores in a similar population (17.7 (9.5)). A diagnosis of depression and/or anxiety was more common in the NEB group and may have influenced the higher scores of the NEB group, although levels of depression were also notably high in the non-NEB group (45.2% versus 34%). Higher BDI scores are consistently found in NES subjects in comparison to control groups, but at lower levels than the 24.8 (10.9) of this study. Allison et al. (2005a) found a mean score of 19.7 (10) using version IA in NES

subjects with a mean BMI of 36 kg/m² and a lower mean score of 15.7 (10.6) in subjects with mean BMI of 33.7 kg/m² using version II (Allison et al. 2007b). It is possible that the degree of obesity may have an impact on the score. It is interesting that, despite a significant correlation between Stunkard's NEQ (2004) scores and BDI scores across the whole group ($r = 0.47$), the two mood items of the NEQ (2004) differentiated poorly between the two groups, with both scoring relatively highly on both items. It is possible that obesity is confounding the relationship between depression and NES.

One weakness of this study was the lack of standardisation in the approach to the diagnosis of depression and anxiety, with some participants reporting assessment from psychiatrists in psychiatric units and others being diagnosed by their GP. It is possible that participants assessed by a non-specialist such as a GP have been inappropriately diagnosed and that the true level of depression in the WMC subjects is lower than stated. This must be accounted for when considering its effect on NES.

3.9.3. *Sleep*

Both groups slept on average for less than 8 hours, reflecting findings in other obese populations (Taheri et al. 2004). Preliminary examination of the sleep duration times showed an apparent wide variety of sleep behaviour exhibited by the NEB group, with some individuals appearing to sleep for very short periods and others for very long periods. Post-hoc analysis confirmed this variability in the NEB group to be significantly greater than the non-NEB group. Stunkard's 2003 criteria do not include those who eat and go to bed very late, but did not wake once asleep and interrupted sleep is only captured if it results in eating. The NEB group reported significantly more difficulty in getting to sleep, but whether this is due to a psychological factor or the physical effect of co-morbidity is unclear. Research on NES in morbidly obese populations has tended to concentrate on eating behaviour, with little focus on sleep aspects, possibly as sleep patterns are currently not measured routinely in obesity clinic settings. It is unclear for example, if going to bed at 1am and sleeping all night, having consumed a large amount of calories before bed, or going to bed earlier, but having a very disrupted sleep pattern and waking three times to consume a snack will have a bigger impact on health.

Sleep quality is not included in the 2003 criteria, although an item about awareness when eating at night is included in the NEQ (2004). The standard deviation of 1.7 on the ‘awareness whilst eating at night’ item in the NEB group suggests there is some variability in reported awareness. Although sufferers of NES are assumed to be fully aware of their night-eating behaviour, this finding supports others who have found variation in levels of awareness in this group (de Zwaan et al. 2006; Vetrugno et al. 2006). It is reasonable to assume that disturbed sleep would have an impact on day-time functioning and significantly less of the NEB group were in employment, although whether night eating and/or co-morbidity is the cause or result of unemployment remains unclear.

3.9.4. *Eating Behaviour*

The EDE scores were in keeping with a morbidly obese group awaiting bariatric surgery (1.60 for Restraint, 1.34 for Eating Concern, 3.28 for Shape Concern and 3.30 for Weight Concern) (Kalarchian et al. 2000). High median scores on both the Weight and Shape Concern subscales in both the NEB and non-NEB groups and in other morbidly obese groups suggest these may be of limited use in such populations, as concern over weight and shape at this level of BMI could be considered entirely rational. The non-NEB group scored lower on the ‘Eating Concern’ subscale suggesting that they have fewer psychological issues related to their eating behaviour than the NEB group. The higher ‘Restraint’ subscale score in the non-NEB group suggests that these individuals found it easier than the NEB group to control their eating. The fact that 8 of the NEB group (26%) as opposed to only 6 (12%) of the non-NEB group were unable to score the ‘Fear of Losing Control over Eating’ item as they reported having lost control already also supports this. Scoring guidelines specifically exclude these individuals from the subscale analysis. The substantial number of individuals unable to score this item calls into question its appropriateness as a measure of control in this population. Significant differences were also seen in the control items on the NEQ (2004). Colles, Dixon & O’Brien (2008b) have identified control as the main predictor of outcome in bariatric surgery patients, regardless of ED behaviour and preliminary findings suggest this may be a key influence on night-eating behaviour.

3.9.5. *Screening Tool*

With regard to the screening tool, the original aim was to develop a tool which would identify NES based on the 2003 NES criteria and this was only partially achieved. Although a cut-off score of 23 on the night-eating items demonstrated an acceptable level of sensitivity (0.93 CI 0.78, 0.99) and specificity (0.78 CI 0.64, 0.89) for identifying night-eating, items used to identify other eating disorders were less successful. Despite being chosen originally for their strong performance in previous validation studies, too many false positive cases were identified. On the surface this would suggest that the elements of the tool included with the purpose of identifying other eating disorders have little diagnostic utility in this particular population. However, weaknesses in the methodology used to construct and score the tool may account for the tool's poor performance. Items included to exclude other ED were taken from tools which require a yes /no response to individual items and converted into a Likert scale in keeping with the NES items. However, analysing these item scores on a Likert scale proved impractical, thus for the BED and sub-threshold BED group and for the BU and sub-threshold BU group, item scores were converted back into a 'positive' or a 'negative' response. Subjects were considered positive for BED or sub-threshold BED if they were positive on one or both of the BED items. A numerical score of ≥ 1 on the Likert scale of an individual item was counted as positive. With regard to the BU items, only 3 of the SCOFF items could be included in the final analysis. Unfortunately, 12 subjects were unable to answer the question 'have you recently lost more than 1 stone in a 3 month period?' as they had been unable to access scales with sufficient capacity to provide an accurate weight. The SCOFF scoring system normally requires positive responses to two or more items and is based on 5 items in total. As only 3 items were included in the analysis, the presence of BU or sub threshold BU was based on a positive response to one or more items. As with BED, a score of ≥ 1 on an individual item was counted as a positive score. It is possible that the inclusivity of this scoring system resulted in too many false positive results. It is also possible that this was due in part to a similarity of behavioural constructs and cognitive processes in severe obesity, binge eating and bulimia.

3.9.6. *Conclusion*

It is clear that a complex interplay of factors, such as demography, obesity, sleep, disordered eating and psychiatric and clinical co-morbidity may be underpinning the behaviour of the NEB group with some characteristics more dominant than others. A large proportion of individuals with NEB were under the age of 35, in comparison to the non-NEB group and significantly more were not working and scored higher than the non-NEB group on the BDI scale. The presence of sleep-related co-morbidity in some NEB participants and not others, the variable duration of night-eating and diversity in sleep patterns suggest that the causes of NEB may be multi-factorial and different for each individual in this group. The degree of perceived dietary control may also be contributing to differing levels of impairment. However, it is not clear whether the degree of severity of one factor will have a greater impact on an individual and his/her weight than the presence of several factors in a milder form, as little is known about NES from the patient's perspective. It is clear that there are many unanswered questions regarding the characteristics of NES, with some features such as sleep variability and work status appearing more prominent than when the identification study was originally conceived. As it became apparent that the 2003 criteria for NES excluded many obese individuals with night-eating behaviour it did not seem appropriate to continue further development of a tool that ran the risk of excluding key behaviours. In addition, the process of combining elements of different tools was not successful. There would be no value therefore in pursuing a reliability and validity analysis of the proposed tool without first undertaking further conceptualisation of the syndrome, particularly from the individual's perspective. The next stage of this study therefore was to take a sideways step and undertake conceptual analysis of the interview data to identify the key constructs of 'night-eating' as experienced by individuals with NEB.

4. CHARACTERISATION STUDY

4.1. Introduction

The diagnostic interviews have a very structured design with response categories determined in a close-ended scoring system. However, during the interviews, participants spoke at length about their personal experience of NEB and offered in-depth knowledge and insight into the different factors affecting their behaviour. It was felt that further in-depth analysis of this interview data using a qualitative approach may help to identify causal relationships, multiple perspectives and degrees of impairment of NEB. Findings could be cross-validated with generalisations identified in the first phase of study and subtleties clarified. In addition, the large number of individuals interviewed would allow for further comparison with a matched group of non-NEB individuals.

This combination of quantitative and qualitative methodologies, referred to as a ‘mixed methods’ approach was used as it allows for both the identification of NEB and investigation into its characteristics; research questions which require both inductive and deductive approaches (Creswell 2003). It places less emphasis on the divergent research paradigms of post-positivism and constructivism with pragmatism as the overarching paradigm (Creswell 2003; Rossman & Wilson 1985). The pragmatic world view emphasises pluralism and ‘what works’ and values subjective and objective knowledge equally.

4.2. Mixed Methods Approach

The type of mixed methods approach used usually depends on where the main focus of the study is placed (Morgan 1998). An embedded mixed method design was adapted for use in this study as this design is used when one data set provides a supportive, secondary role to a primary data set (Creswell, Plano, & Clark 2007). The design is described in Figure 8 using Morse’s notation system to indicate the emphasis of each component (Morse 1991). This approach is often used when quantitative results need further interpretation, or when more detailed views of select participants can help explain the quantitative results. Although based on the same data set, the primary purpose of this study was to identify NEB and the secondary purpose was to build on these initial results by exploring the participants’ experiences of NEB

through qualitative analysis of the interviews and a matched control comparison. Data collection for both approaches was performed concurrently during a single phase of the study, but analysed sequentially (Morgan 1998).

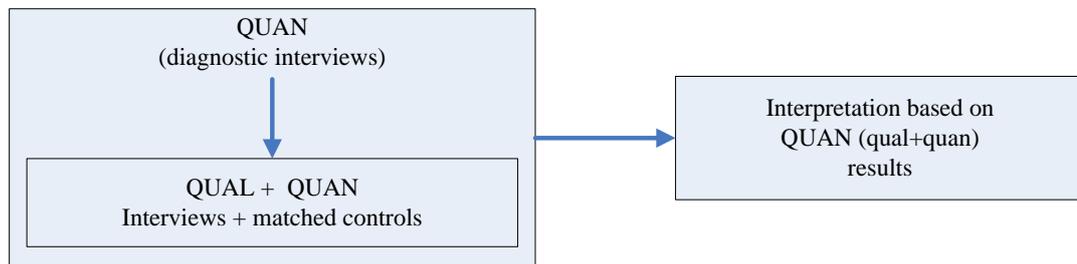


Figure 8 - Embedded design using Morse's notation system (1991)

4.3. *Interview Data: Qualitative Theme Analysis*

Collection of the interview data was based on a standardised interview approach using quantitative tools delivered in a set order. However, qualitative theme analysis of the interview data used a process for coding and analysing data which was developed for use in Grounded Theory approaches. Grounded Theory is a specific methodology first identified by Glaser & Strauss (1967) for developing theoretical frameworks for social phenomena. Its purpose is to generate new theory by providing predictions, explanations, interpretations and applications for human conduct which are grounded in the data under study.

4.4. *Glaser and Strauss: Social World Perspective*

Glaser and Strauss' social world perspective reflects both the complexity and processual nature of social phenomena. It has its origins in the philosophical doctrines of the Neo-classical Pragmatists (Peirce/James/Dewey/Mead) and the traditions of Chicago Interactionism (Hughes 1971; Park 1967; Thomas 1966). They describe social worlds which segment into specifiable sub-worlds and countless discernible worlds, which in turn intersect under a variety of conditions. Humans are members of these diverse 'worlds' and membership is complex, overlapping, contrasting, conflicting and not always apparent to other interactants (Strauss 1993). Thus human experience is located within and cannot be divorced from larger events in the social, political and cultural framework. Organisations themselves may be relatively embedded within a social world, while others stand at intersections, or may have been intentionally constructed that way. Strauss summarises:

'Intersection and segmentation imply that we are confronting a universe marked by tremendous fluidity: it won't and can't stand still. A universe where nothing is strictly determined. Its phenomena should be partly determinable via naturalistic analysis including the phenomenon of men participating in the construction of the structures which shape their lives.' (Strauss 1993).

As Glaser and Strauss describe, this is indeed a complex world and capturing this complexity is an essential part of research into social worlds. It is more important to describe, explain, predict and interpret through discovery and serendipity than restrict inquiry to the testing of variables. The focus should be on identifying and explaining the inner experience of participants and their often conflictful relationships in the context of the larger conditional frame in which it is embedded, determining how meanings are formed and investigating the processes, sub-processes and relationships of the action/interaction/emotion/consequence dynamic (Strauss 1978).

4.5. ***Grounded Theory Procedures***

Glaser and Strauss' original theory reflects a positivist paradigm, relying on direct and often narrow empiricism to analyse a basic social process. Initially, they discouraged the researcher from influencing the data collection process, although in later years, Strauss, with co-author Corbin, developed data collection techniques which actively encourage the researcher to influence data collection (Corbin & Strauss 1990). They propose that initial interviews should be very unstructured so that categories which then emerge, form the core of the emerging theory and the basis of theoretical sampling. As this core usually emerges quickly, further sampling and data collection can be directed to achieve theoretical 'saturation' of these categories. The researcher then concentrates on the salient categories in subsequent interviews and 'drops' less relevant data. Data collection and analysis then become integrated processes, both systematic and sequential and follow a three-step cumulative process of open coding, axial coding and selective coding (Strauss & Corbin 2008).

As the interview data for this study had already been collected prior to the decision to submit it to in-depth qualitative analysis, such a constructivist Grounded Theory approach cannot be claimed. The EDE v 12 has a structured format and participants were steered towards discussing aspects of NES raised by the NESHI and NEQ (2004). A conscious effort was made to ensure all topics were raised with each participant in order to ensure consistency of approach.

Opportunities were therefore not available to perform theoretical sampling by dropping less relevant topics in later interviews in order to saturate emerging themes. Once the data was collected however, it was coded and analysed using the 3 step coding process used in Grounded Theory approaches and described below.

4.5.1. *Stage 1 - Open Coding*

All data are treated as potential indicators of phenomena. After the first pieces of data are collected, open coding is used to look for cues and 'fracture' the data into categories. Categories are the cornerstones of a developing theory and need detail and specification through exploration of their properties e.g. conditions which give rise to it, interaction by which it is expressed. Two types of preliminary categories and properties then emerge, those constructed by the researcher (explanations based on discovery and insight) and those direct from the language (process and behaviour) (Bazeley 2008). The researcher is encouraged to use memos systematically to assist in the development of these categories which point in turn, to the next steps in data collection.

4.5.2. *Stage 2 - Axial Coding*

Axial coding provides a structural matrix by linking together categories identified through open coding. Finding patterns and regularities gives order to the data and assists with integration. Integration, through a constant comparison of categories and their properties results in over-arching concepts, which suggest hypotheses and form the basis of the theory (Coffey & Atkinson 1996). Inadequately integrated categories often result in thin, tenuous theory. The researcher requires a degree of theoretical sensitivity to ensure emerging theories are meaningful. Concepts should be analytic, that is, sufficiently generalised to designate characteristics of concrete entities, not the entities themselves and be based around ranges, continua, degrees, types, uniformities, variations, causes, conditions, consequences, probabilities of relationships, strategies, process and structural mechanisms (Coffey & Atkinson 1996; Schatzman 1991).

4.5.3. *Stage 3 – Selective Coding*

With further comparison and integration of concepts, the theory solidifies as theoretical saturation is reached and 'pinpointing' hones in on finer detail. Selective coding at a higher level can then identify the main theoretical construct and ensure primacy of the core concepts and the emergence of a substantive model (Glaser & Strauss 1967). Once identified, a theory can be

presented as either a well – codified set of propositions or in a running theoretical discussion using the ‘novelist approach’ incorporating direct quotes, memos, telling phrases, case studies and background descriptions.

4.6. *Study Methodology*

4.6.1. *Data Preparation*

All NEB interviews were transcribed by a professional and independent transcription company. All transcriptions were quality checked by JC and items labelled as ‘unclear’ by the transcribers identified. These items mainly related to difficulties with interpreting a regional accent and the use of local expressions. Clarification was obtained by replaying the audio tapes or referring to interview notes.

4.6.2. *Coding Procedures*

Two transcriptions, one of a participant with full NES and one of a participant with sub-threshold NES were examined line by line using open coding. An example of open coding is seen in Table 21. Once all possible codes had been identified throughout the two texts, both transcriptions were then entered into NVivo, a computer software programme which assists in the management of data and the organisation of ideas (Bazeley 2008). NVivo allows for a section of text to be marked with a code (called ‘node’ in NVivo) .The codes from these first two transcriptions were then systematically organised into preliminary categories and sub-categories.

Table 21 - Example of categories and sub-categories identified from a section of narrative from a study participant using open coding.

<i>Text of narrative</i>	<i>Category / sub-category 1 / sub-category 2</i>
Interviewer: Are you snacking in between your meals on those days?	Activity/day to day Control/compulsion
Participant: No, I’m not. I’m not as bad in the day. You know in the daytime, in the morning. No I’m not, because I’ve got my husband there with me, so no and I don’t get that urge to keep going to the fridge. I’m satisfied in the day, yeah. I don’t have the sugar craving, or sweetie craving, it’s more later, you know in the night that I have the sugar craving. But in the day I’m not, not too bad, maybe because he is with me.	Eating at night/get up and eat /craving to eat Eating at night/get up and eat/current trigger Eating patterns/secret eating Eating patterns/snacking/variability of snacking Family and friends/ living arrangements Family and friends/relationship with family and friends/support Food emotions/satisfied Personality/self judgement/misbehaving

All other interviews were subsequently entered into NVivo and subjected to line-by-line coding. Adjustments were made to categories and subcategories as further data were analysed until categories reached saturation and no new categories were identified. Memos were used during the coding process to record spontaneous thoughts and link developing ideas. In order to ensure inference validity and demonstrate that the analysis was accurate, credible and trustworthy as proposed by Guba and Lincoln (1981), an independent check of 4 transcriptions was performed by a study supervisor. The purpose of this was twofold; firstly, to ensure that the narrative interpretation was performed to an adequate depth, with all salient categories identified and secondly, to check for adequate theoretical sensitivity. Experience in working with the client group under examination and exposure to their behaviours helps the researcher be more sensitive to the meanings embedded in data and develop emerging theory to an appropriate degree of abstraction (Strauss & Corbin 2008).

4.7. *Results of Qualitative Interviews*

4.7.1. *Presentation of the Data*

Presentation of the results of the interview analysis has taken into account the context in which the data were collected. Given the systematic approach of the data collection, a mixing strategy has been adopted, using data transformation approaches (Caracelli & Greene 1993). Where appropriate, qualitative findings have been quantified and presented in tabular form with direct quotes (in italics) from participants' experiences used to support and enrich the narrative discussion (Tashakkori & Teddlle 1998). The initials and study number of the relevant participant are included after each quote. As each subject was asked a standard set of questions, quantification of a characteristic has generally been based on the number of participants for which the characteristic occurred, rather than the number of times it occurred in the coding process. This was to minimise the risk of one vocal individual saying one thing ten times and adding undue weight to one factor. One individual with NEB declined to be tape recorded, thus NVivo analysis is based on the accounts of 30 NEB individuals.

4.7.2. Results of Coding Process

Thirteen broad categories with 185 sub-categories were identified through the coding process. The 13 broad categories and 4 examples of sub-categories for each category are listed in Table 22. A full list of all sub-categories is included in Appendix 8.

Table 22 - List of all categories, examples of 4 sub-categories per category and total number of sub-categories identified through the code of 30 NEB transcriptions.

<i>Category</i>	<i>Examples of sub-categories</i>	<i>Total no. of sub-categories per category</i>
<i>Activity</i>	Activity at night Exercise Need to be occupied Physical functioning	10
<i>Barriers</i>	Blame self Blame someone or something else Procrastination Sabotage by self	6
<i>Change</i>	Accept help Cant be bothered Negative thinking Take responsibility	10
<i>Control</i>	Compulsion Dietary rules Control after evening meal and before bed Restraint	10
<i>Eating at night</i>	Aware of night eating Crave to eat Current trigger Need to eat to return to sleep	17
<i>Eating patterns</i>	Meals one, two three, four Eat in public Secret eating Eat with family	15
<i>Episodes of overeating</i>	Awareness Amount Emotion during over eating Trigger emotion	14
<i>Family and friends</i>	Key players Living arrangements Relationship with helpers Relationship with family and friends (e.g concern, conflict)	16

<i>Food emotions</i>	Addiction Bored with food Comfort Emptiness	22
<i>General emotions</i>	Anxiety Bitter Content Crutch	24
<i>Personality</i>	Abrasive All or nothing Determined Self-judgment	13
<i>Sleep</i>	Patterns Quality of sleep Wake during sleep Location, partner	13
<i>Weight</i>	Attitude to weight Concern over weight gain Judged by others Key events	15

4.7.3. *Characteristics of the NEB Group*

4.7.3.1. *Introduction*

Analysis of coding categories identified the following major themes in relation to NEB:

- Influence of childhood obesity and significant life events on the development of NEB
- Effect of poor sleep on the onset and maintenance of NEB
- Compulsive nature of eating at night
- Chaotic daytime eating patterns
- Negative emotions relating to eating and general day-to-day living
- Conflictful relationships

The above themes will be explored individually from the participants' perspectives, including direct quotes where applicable and key findings will be summarized at the end of the analysis.

4.7.3.2. *Childhood Obesity and Significant Life Events*

Fifty percent (n=15) participants reported obesity which started in childhood. Childhood obesity was generally described in a matter of fact manner:

'I've always been a big kid'. (JP017)

'I've always been overweight; I've never been a normal weight'. (LR047)

Some individuals used deprecating language to describe themselves, though the word fat was usually avoided.

'I've always been big and tubby. I've never been thin'. (DLO58)

'I've always been on the chubbier side'. (KR009)

'I've always been ashamed of my size, from when I went to Primary School, I was the chubby one'. (CP065)

Childhood obesity often had traumatic consequences, with many recalling being taunted at school.

'we used to get a school bus and they'd pick on me all the way to school and all the way back from school'. (KR009)

'and people stare at you, because, you know, what they are saying and before I would just not look, but I don't do that now. And you can tell.. and you know what they are. And I stop and I turn around and I look at them and then they go.. and they run away'. (JG 062)

In addition two individuals were even hospitalised as children because of their weight. Other traumatic childhood events not directly related to obesity also featured prominently in participants' stories and included sexual abuse, death or absence of parents and parental maltreatment. (Table 23)

Table 23 - Categorisation of traumatic life events occurring in childhood/teenage years experienced by the NEB group (n=30)

<i>Traumatic event occurring in childhood/teenage years</i>	<i>Number of participants experiencing the event</i>
Childhood accident/hospitalization (without parents)	4
Sexual abuse (self, siblings)	3
Death parent/grandparents	2
Divorce/absence of parents	3
Childhood bullying	2
Teenage pregnancy	1
Maltreatment by family	4

Events were varied in nature, although loss (death/absence) of a significant individual featured prominently (n=9). All were similar in terms of their perceived severity and degree of impact on the individual.

Sexual abuse:

'Well up until ten I was absolutely normal. I've got pictures of me, when I was eight, a happy go lucky kid. I was then sexually abused between nine and eleven and my weight just ballooned from then. That was like the start of my problems'. (DE029)

'I can't remember now...I think it was before Christmas I started on them (taking tablets) and they said like, when I've got them out I was doing them but when me mum argued with me, I get down and everything and she started bringing stuff in the past back up (sexual abuse of siblings) and that's when I like, that's when I start, I get dead depressed and everything and I'll start having something (to eat)'. (PA020)

Hospitalisation:

'I was very sick as a baby though. I sometimes blame that in a way, don't know why, when I was born I had croup and everything and partly for my first 2 years I was in hospital more than I was out and I was fed through my leg. I've always said... I have actually got a lump in my leg where I was fed, so I've always said maybe that's what it was, the way I was fed when I was born. I don't know if there's anything in that or not, I don't know, but it's just something that seems strange'. (GD015)

'I believe I started to put weight on at the age of 7, I had ... my finger was crushed in an iron gate and I had to have an operation on it, which they saved the top and from then I started to put weight on apparently. We always put it down to how the shock to the system had changed things around.' (JG062)

Absent parents:

'Oh, I was always heavy. I was fat. I was brought up by my grandmother. I didn't live at home. It was only me and my granddad and if she did a meal...she used to have a big family, she used to cook for a big family. She was used to cooking for a big family and if you had something to eat and she would say 'there is a bit left over – do you want it?' and I would say 'no' and she would say 'go on, have it' and that's how I was brought up to have that little bit extra and I think that's why I've been fat all my life. It's a lifestyle I was brought up in.' (GR073)

Loss experienced as a child:

'I was always stick thin and that. I've got photos at home when I used to play football for the school side and the cubs. I've always been big, always tall, from 13 onwards it just went...spiralled. At about that time I lost 4 grandparents in a few years, 4 years and I was close to all my grandparents, I stayed with them for weekends. I lived with 2 of my grandparents, so it was a bit ...' (NC026)

Traumatic life events experienced as an adult included divorce, death, or illness of close family members and premature retirement (Table 24).

Table 24 - Categorisation of traumatic life events occurring in adulthood experienced by the NEB group (n=30)

<i>Traumatic events occurring in adulthood</i>	<i>Number of participants experiencing the event</i>
Cannabis addiction/ partner's alcoholism	2
Death/suicide/absence of family members	5
Divorce	3
Premature retirement/off work	3
Motherhood	1
Child with chronic illness	1

Loss experienced as an adult:

'Well, I looked after them all. I looked after my grandmother and my two uncles, now they died. I buried them three, or four, in 16 months...I buried them. And then my brother was 33, 6'3. Beautiful. And then he just developed cancer and a matter of a few weeks dead...I don't know, things just get on top of me. (MC 024)

Cannabis addiction:

'Cos I had me slum when I was younger, I was lazy. I used to get stoned all the time, munchie-ing and getting the munchies and going round to help me self and all that and that really put,,, I think that contributed a lot to me weight when I was younger, when I was getting stoned and it was from drugs and all that and no good when you kind of eat what you want.' (JMC 068)

4.7.3.3. *Patient Perceptions of Onset of NEB*

In total 23/30 (77%) reported the occurrence of traumatic life events (10 individuals reported childhood incidents, 9 adult incidents and 4 a mixture of both). Twelve of these individuals reporting significant life events also reported onset of childhood obesity. These two factors of childhood obesity and trauma appear to have had an overarching effect on the onset of NEB, with two distinct groups emerging; those who developed NEB in early or teenage years and those who developed it as adults. In this sample, 6 individuals reported childhood NEB, 6 teenage onset NEB and 18 adult onset NEB. Individual pathways are described in detail in Appendix 8. Of the 6 individuals who reported developing NEB as a child, 4 said it was in response to being forced to diet by parents as a result of childhood obesity:

'My mum and dad were always on at me, you know, 'you're getting chubby, you know, you're a chubby child'..... so I would try and be good, my mum would feed me a healthy diet, but I knew when my mum and dad was in bed, I can remember always sneaking down, a biscuit, a sandwich, or something out of the fridge'. (CP 065)

'I've got a feeling it was as a child but I mean it was sneaking food as a child because I mean we didn't have a fridge when we were kids, we used to have a larder but we used to sneak into the larder as well'. (AB007)

Individuals described a sense of awareness of 'being bad', sneaking about, 'pinching goodies', whilst desperately trying to be 'good' children:

'I used to go down, just thinking about it then, when I was at school, I used to sneak down when I went to bed when I lived at my mum's to pinch bread of a night'. (JP017)

'I don't know why but I always do it in the dark.....because the bedrooms were in the hallway and the pantry used to be in the hallway and I used to spend.... I always remember I'd become so experienced at opening the door without making the loud click that you do... sometimes when I opened the door, then 'who's that... what are you doing out there' you know, my dad, shouting from the bedroom and that. So I got to be an expert at opening the door without making a noise'. (AB007)

'I was always, I don't know whether it is cos of this, but I was always put on a diet anyways, so I think it was like, I think then it was more because I didn't want anyone to be disappointed at me for eating, so I'd get up and eat in the night and then no one can see, can't see what I'm having and that.....I think I've always been like, greedy and waking up at the night and going so like making something to eat. I've always been a bit like that, greedy and waking up'. (SLD 011)

For one individual developing NEB as a teenager, bullying as the result of childhood onset obesity was reported to be the initial trigger:

'I used to bunk off school so I didn't have to face them and started smoking cos I wanted to be in with them, so it's just all little stuff like that from the age of 13 really, that's when I started comfort eating and stuff and I just never stopped really.' (KR009)

For others developing teenage NES, trauma and conflict within the family itself appears to have been a more common cause. Two individuals reported moving out of the family home aged 16 and coping without parental boundaries on behaviour. One discovered as a teenager that her mother had tried to abort her in utero and another discovered that his father was sexually abusing his younger siblings.

Start of NEB as a result of teenage conflict with mother:

'We can actually trace things back to my mother's womb and they wanted me aborted and then I was born not breathing and blue and they had to put narcan in my heart to start it beating and I had a hole in my head and infection in there for 18 months'. (RB008)

Start of NEB as a result of sexual abuse within the family:

'Since we moved... when we moved up here really, because of all the problems what happened with me dad and stuff (sexual abuse) and really, 7 or 8 years now'. (PA020)

For those developing NEB as adults, traumatic events were more likely to be milestone life events such as retirement, divorce, pregnancy and bereavements of close family members such as parents or siblings. Their influence on the development of NES and obesity NES seems less clear, with some obese before the event and others developing obesity after it.

Start of NEB as a result of adult milestone life events:

It's probably when I moved house. I wasn't with my partner so I'd just had my baby, so it was probably then'. (weight gain prior to pregnancy) (KD055)

It happened really bad when I split from my husband. It got quite bad then, but before then, I wasn't really doing it. I was unhappy though, cos I was in an unhappy marriage, o my eating patterns were up and down then anyway.' (weight gain after separation) (MH004)

4.7.3.4. *Influence of Sleep on the Development of NES*

The relationship between the development of NES and disrupted sleep patterns appears particularly complex. Several individuals were able to identify reasons for developing poor sleep patterns initially, which may then have been a factor influencing the development of NEB. Three individuals described poor sleep patterns caused by work in earlier years. Reasons given were, 4 hours on - 4 hours off work/sleep shifts in the Navy, working nights as a nurse, and finishing work late when working as an entertainer in a holiday park. Three others reported disrupted sleep patterns and subsequent NEB after stopping work. The teenager with a cannabis addiction linked his subsequent NEB to staying up all night smoking and the night-time 'munchies'. The teenager who was bullied can clearly relate her disrupted sleep to the distress of the bullying and the start of NES:

'I think honestly the problem started when I was getting bullied in school, I think that's when (NEB started), my sleeping problem was there first, I think, because I used to be worrying about going to school... ..there was like about 2 years where I'd wake every hour on the hour and I was so tired in school that the school actually said that I kept falling asleep and I didn't know I was doing that'. (KR009)

The vast majority, however, did not particularly identify a causal relationship between NEB and sleep, but reported a general backdrop of sleep problems or abnormal sleep patterns of many years duration. Typical comments were:

'I've never had a normal bed time for years and years'. (MC024)

'I've never slept for 8 hours.' (MJ 084)

'I've had poor sleep for years'. (JG 062)

'I was never a morning person at school'. (KMC 068)

Only one individual who developed NEB as an adult described disturbed nights as a child:

'For as long as I can remember I've not slept well, even as a kid I can remember waking up during the night and looking out of my bedroom window at what was going on in the street and there was nothing most of the time... I use to wet my bed and he (hospital specialist)said to mum 'she's going straight into a deep sleep and she's not waking up from it' and he gave her some pills to make me sleep 'lighter' or something, they were alright, I thought I'd grow out of it'. (LR 047)

Poor sleep patterns per se were only identified by a minority of individuals (n=4) as the reason for current night-time awakenings. A variety of diverse reasons was reported and these are summarised in Table 25. Some individuals were unsure why they currently woke (n=4) and whilst others could clearly identify an emotional, or hunger-related cause, physical problems featured prominently, with 7 individuals citing Obstructive Sleep Apnoea and other breathing difficulties. The entire group reported going to the toilet once awake but only 3 individuals felt this was the primary reason for waking up.

Table 25– Patient reported causes by category of current night-time awakenings in the NEB group (n=30).

<i>Categories of reasons for night-time awakening</i>	<i>Cause (n= number of participants reporting cause)</i>
Eating-related	Day-time restraint (n=4) Up for drink (n=2)
Sleep-related	Day-time sleep, (n=2) Not tired (n=1) Early to bed(n=1)
Medical	Pain in legs - glowing hot (n=1) Hiatus hernia (n=1) Diabetes > problems sleeping (n=1) Psoriasis- awake scratching and washing hands (n=1) Woken by pressure of legs building up (n=1) ME (n=1) Dry mouth (n=2) Need for toilet (n=3)
Breathing	Sleep Apnoea and mask (n=5) Can't get my breath (n=1) Chronic obstructive Pulmonary Disease (n=1)
Emotional	Emotional stress (n=1) Bad dream (n=1) Feeling unhappy, crying (n=1) Stress at work (n=1)

External	Noises outside (n=2)
Unknown	Not sure why (n=4)

4.7.3.5. *Current Sleep Behaviour*

For all the participants in the NEB group, bed time and sleeping were difficult experiences. Most individuals struggled to get to sleep and viewed going to bed as an unpleasant experience. Typical general comments included:

'I just dread going to bed, I don't know why but I think it's because I don't fall to sleep straight away like I used to'. (DW039)

For some individuals concerns were related to emotional distress:

'So I'm constantly catching up on sleep all the time. And then my body gets into a routine..like when I want to go to sleep I can't. I'm that wound up you know'. (JP017)

'I'm lying in bed and I'm knackered, but I just can't get to sleep so I think that'd be when I'm thinking about all the stuff the most, so that's probably why I can't get to sleep'. (KR 009)

'I cry before I go to bed...my eyes are burning because I'm tired. I don't want to be tired. If I couldn't be tired I'd be happy. I just want to stop being tired'. (MC024)

For 2 individuals the concerns were more physical. One young male adult was frightened of physical violence from his older brother while he was asleep and for one single mother, night times were particularly fraught as she was on constant alert for her disabled son who suffered from episodes of apnoea:

'They had a meeting in the house with a family support worker and his boss and I said all I want is one night a week for someone to come in and take my role, so I can go to sleep like a normal person, which doesn't happen very often'.(AC 023)

Even those who stayed up very late eating before going to bed, still perceived getting to sleep to be a struggle and had become used to delaying their bed time until later due to the frustrations of not being able to sleep if they went to bed earlier.

'Occasionally as I say, sometimes I just have to knock myself out (with sleeping tablets) and sometimes that's the only way I can do it. I literally have to 'mong' myself you know – I take a stronger dose than normal and I feel sleepy and I can get to bed a lot easier'. (JMC 068)

Only one older participant felt he struggled to get to sleep due to his medical condition of COPD.

Unsurprisingly, only a very small number (n=4) felt their subsequent sleep was of good quality and all these were individuals who stayed up very late before going to bed. One individual went long periods without sleeping at all, preferring to work back to back double shifts for several days on end and two individuals had reversed night and day time completely. The majority of participants generally described having short periods of fitful sleep from which they were easily roused; something they found extremely frustrating and debilitating:

'I struggle to sleep. I'm very, very lucky if I can get four hours straight sleep. You know, I'll look on that as being a good night's sleep if I can get four hours. I mean the slightest thing, I mean believe it or not half past one this morning two kids playing football, kicking a ball against the wall.' (AB007)

'.. sometimes I just have periods where I can't sleep all night, so I just stay awake and toddle round the flat and things'. (SK040)

'That's probably the most annoying thing in me at the moment is not getting to sleep. It just does my head in, why can't I just go to sleep'. (GD015)

'One thing I do suffer with is sleep. It's a real struggle. You just get batty then and it's not good.' (NC026)

As well as eating, participants also resorted to a range of activities to either distract themselves or help them return to sleep (Table 26). Computer-based activities included games, chat rooms, on-line gambling, crib and searching the family tree.

Table 26 - Table of activities undertaken either before bed or during the night by the NEB group (n=30).

<i>Activity before sleep and during night</i>	<i>No of participants undertaking the activity</i>
Watch TV/DVD	16
Read/puzzles	7
Computer	9
Radio/Music	2
Late night shopping	1
Drink alcohol	1
Paperwork	1

As a result of having a poor night's sleep, getting out of bed in a morning was generally a huge struggle, with those who had the opportunity to do so, sleeping in wherever possible:

'I'm tired beyond exhaustion, even sometimes when I get up, I'm tired beyond exhaustion'. (KMC049)

'Well I do have two alarms and I drag myself out of bed'. (LR047)

Eleven of the participants resorted to sleeping in the day, either in the afternoon, or when they got home from work and struggled to stay awake when in work. This day-time sleeping often created problems with sleeping the night after, resulting in a vicious circle of sleep deprivation:

'It depends, sometimes I'll go to bed and I can't sleep because I've slept all day I think, although I'm tired when I wake up I'm exhausted all the time'. (RC052)

Frequent attempts were made by participants to change their eating and sleeping behaviour and are described in Table 27 although most found any changes they made unsustainable.

Table 27 - Strategies used by NEB group participants to change their eating and sleep patterns

<i>Attempts to change sleep/eating pattern</i>	<i>No of participants undertaking the activity</i>
No attempts to change	4
Weight loss/sleeping tabs	4
Sleeping tablets and change sleep routine	4
Healthier foods in fridge	2
Change sleep/wake times	2
Sleeping tablets and change eating regime	1
3 meals a day	1

'I was hoping that, or I'm thinking, OK, let's get meself into a routine. Breakfast, dinner, tea - don't skip them, breakfast, dinner, tea. And maybe after a few weeks or a month or two, the night time eating or the hunger, cos I've changed me body clock, might stop'. (MJ084)

4.7.3.6. *Eating at Night*

Once awake, almost a third of the group felt they got up and ate because they were 'hungry' (9/30), with one man feeling so ravenous he could 'eat a Sunday dinner'. Others acknowledged eating in response to negative affect, such as feeling alone, empty, unhappy and anxious:

'It's I'm not hungry in my tummy, but I'm, it's me mind ...saying, you know, go to the fridge and have a look'. (CP 065)

'No, it's just ...when I used to wake up I'd have this really empty pain kind of in the bottom of my stomach and I'd have to go and have something to eat to get rid of that emptiness feeling'. (JP017)

'...because, like I'll have some nights where I've been crying, like missing dad and that and then I fall asleep, so I could do it (night eat) that night'. (MH004)

Most individuals felt compelled to eat in order to return to sleep:

'It's as if it's waking me up to go and do it'. (JP017)

although individuals who stayed up very late eating and then went to bed did not experience this. Those who tried to resist the urge described the inevitability of fighting a losing battle and the eventual relief of finally being able to sleep:

'I've never tried to stop myself, because most times it's...before you realise it, it's too late, you're there. I can lie there saying don't go down, you know, it's wrong, don't go down, but it loses out, you know, I mean I'll tell myself not to go down but then I'll not go to sleep, so it's the worst of two evils. I've got to get some sleep. It's a horrible, horrible feeling..... you just eat and then you feel so bad about it, but at least I know then when I go back to bed I'll fall asleep whereas if I don't get up I'm just thinking and thinking and thinking. I just can't get it out of my mind'. AB007

Three individuals also described having to get out of bed to eat before being able to get to sleep in the first place:

'But if there's goodies there and I go to bed knowing that there's goodies down there, then it, it's just, you know ... I wouldn't sleep. I've got to have it'. (CP065)

4.7.3.7. **Secret Eating**

Twenty two individuals (73%) lived in a household with other family members (Table 28). Not all participants slept in a bed, with 3 sleeping in a chair, one on a sofa and one on a mattress on the living room floor with his head propped up against the couch.

Table 28 - Table showing the sleeping arrangements of the NEB group (n=30)

<i>Sleeping arrangements</i>	<i>No of participants</i>
Sleeps with partner	8
Lives alone	8
Intermittent partners	5
Lives with parents: own room	4
Spare room (disturbs partner)	2
Has partner, not discussed where sleeps	2
Shares room with sister	1

When getting up to eat at night, six individuals brought food back to bed with them although the majority (n=16) ate in either the kitchen, living room or an unspecified downstairs room. One individual ate in the bathroom. Night-time food choices are shown in Table 29. The most popular choices were foods which could be accessed and eaten quickly such as high fat/high sugar snacks and sandwiches. Although nine individuals denied attempting to conceal their night-eating, the popular food choices and location away from the bedroom appears for some participants at least to be an attempt to conceal their behaviour from other family members:

'He'd hear the wrapper or the paper and in the morning find sweet papers on the floor or he'd hear me eat crisps and he'd say 'going down? Gonna go past that fridge? What are you eating now? So I got a bit more cunning, going to the fridge before you went to the toilet and take that in with you. (Laughs). But I've come up with cakes and had them, pretend that I hadn't had anything and then in the night, you know, picked at them, you know and hide the papers'. (CP065)

'I have a partner at home and I might have a little something but all my bits and pieces (snacks) are hidden in a bag'. JC are they in the bedroom? 'I would take them up with me, yeah?'. (JG062)

Those who took the trouble to conceal their eating at night often reported similar efforts to conceal day-time eating.

'I'll come in, go to the fridge and my step-dad will go to me, you've done it again, you've walked straight to the fridge and I'll lie to myself saying, I'm getting a drink and then I'll pick on stuff and shut the kitchen door so they can't see me eating'. (KR 009)

'I tell loads of lies that I don't eat. 'Oh, I don't eat, I don't eat', but then again, I could go out shopping and that would be my stash. They don't know, they don't know because I just drop them (empty wrappers) in the bin'. (MC 024)

Table 29 - Types of foods chosen by participants when night-eating and the number of participants choosing each item.

<i>Foods eaten</i>	<i>Number of participants choosing the item</i>
Biscuits and cakes, sweets	16
Sandwich, toast	10
Crisps, chocolate	7
Dairy	6
Pies and meat	4
Cereal	3
Fruit	1
Oasis juice	1
Raw foods	1
Pasta	1
Anything in the fridge	1

4.7.3.8. Awareness of Night Eating

Fifteen individuals felt they were fully aware whilst eating at night, although 8 reported partial awareness, such as feeling in a daze, being 'half asleep' or being only 90% aware:

'I think I'm like half asleep, I'm awake and I know that.... Like if I wake up in the morning I know I know that I have done it, I hope so'. (KR 009)

Others seemed to relinquish responsibility for the night-eating, describing either being aware at the time, but not remembering in the morning, or a dual state of knowing what they are doing, but

also 'waking up' whilst eating. One individual describes being aware of the reasons for getting up and going downstairs, but being unaware that this has triggered an episode of eating:

'I go down to get a drink, but I find myself eating and sometimes I'm not even aware that I'm doing it. I don't know what triggers it off, but like, I'll say to myself 'oh, what am I doing?' You know, I've eaten half a packet of biscuits while I'm waiting for the kettle to boil and I'm like 'aah'.' (MH004)

These conflicting accounts of awareness may be more of a reflection of the general sense of compulsion and loss of control surrounding night-eating episodes, with many participants struggling to find an explanation for their apparently irrational behaviour:

'I'm not hungry and I don't want it, but I get up and eat it'.

'Hungry? I go...that's me. Cupboard. But I don't want them, I don't want it. I get up and get it and I don't want it, but I eat it'. (MC024)

'but it's strange because before I know it, I'm half asleep. I've gone in the cupboard and I'm eating it so I don't know what you'd class that as really'. (DW039)

'Of a morning, before I come down, I wake up in bed and I know I've been down'. (EC051).

'How much awareness? Oh I know I've got the food there, I can see it. But again I look at what I'm looking at and I go 'God, have I eat them already? They've gone quick.' I'm not always really aware that I've ate at all'. (MC024)

It is difficult to accurately determine perceived levels of control as different individuals can draw the opposite conclusions from the same behaviour:

'I don't think I've got much control over it because if I had control I'd be able to stop doing it'. (RB008)

'I do have complete control, but it doesn't stop me...'. (LR047)

'It's hard to say but I believe I was in control, I knew what I was doing, I knew what I was putting into my body and I knew what the results would be, but at the same time it was sort of like I knew I didn't want to do it but it was one of them things where you get something in your mind and you want to do it and you just go for it, it was basically sort of like that'. (SK040)

Many participants return frequently to the compulsive aspects of night-eating, with one participant describing the experience as if she was being pulled towards a magnet:

'It's literally just eating and eating and eating, as if like a magnet, like that where I was getting pulled towards it'. (SK040)

Others describe feeling powerless in the face of an irresistible force:

'In the back of your mind, you know it's going to put weight on you, but the urge is bigger. It's more powerful than me stopping eating it'. (CP065)

'Once I feel hungry, like really hungry in my stomach, I can't help it'. (DW039)

'Once curtains are closed and can't see out the window or see something, my nerve starts coming into it and then it makes you want to, just got to have, you know something. More hungrier than I do in the day'. (JP017)

Emotions during night-eating appeared neutral, with participants generally saying they either did not feel anything, or were not sure what they felt. Only two individuals reported actual distress.

After the episode, despite relief at being able to get to sleep, participants tended to perceive themselves and their actions negatively, feeling guilty, shameful, distressed and annoyed:

'Well, I don't feel anything. All that I feel is that when I get back I say to myself 'what did you eat that for?' You know, I feel sick, I feel ...how can I say it? I just feel that I want to cry. I don't like to face it'. (MC004)

It's like telling myself off before I go back up to bed because I really do feel bad. It's like as I said before, it's like the naughty boys' school, you know, 'you've been a naughty boy, why have you done that?'. (AB007).

Whilst 3 individuals felt better after eating:

'It's more of a satisfaction to my brain'.(JPO17)

The futility of their behaviour was also acknowledged.

'It's like trying to satisfy something that's never satisfied'.(MJ084)

4.7.3.9. **Day-time Eating Patterns**

Eating behaviour at other times of the day often appears chaotic and lacking in structure. Meal and sleep times of the 30 NEB sufferers included in the interview analysis and one NEB individual who declined to be taped have been presented visually on the first row of the spreadsheet included in Appendix 9. There appears to be considerable variability between NEB participants, with meal times generally occurring at irregular intervals and interspersed with frequent snacking. The first day-time meal often does not occur until the second half of the day resulting in a time shift of all subsequent meals to later in the day. The majority of participants also describe an overarching 'all or nothing' approach to eating, with eating patterns characterized by cycles of restraint through attempts at 'dieting' and subsequent overeating.

'Some days when you sort of think, 'Oh, I'm sick of this' and you give up. Then when I do stuff like that I feel worse, so I either just stuff my face even more, or I just stop it

altogether and eat healthily and then I'm putting myself back in the same place, it's just...I'm going mad'. (EC051)

'Well, I joined Slimming world 3 weeks ago and I've been really good and I lost 6 pounds in my first 2 weeks and that was it then and I didn't go back last week. I went yesterday and I've put 6 pounds back on and I nearly cried...it's just, I lose the weight and I'm on a high and I'm thinking, 'right, that's it' and then I just come off it and go back to my old eating habits'. (KD055)

4.7.3.10. **Eating-related Emotions**

NEB participants described a wide range of emotions experienced while eating, which are summarized in Table 30. Emotions are ranked in order from the highest number of references to a particular emotion in the interviews as a whole to the lowest.

Table 30 - Range of eating and food-related emotions reported by the NEB group.

<i>Food-related emotions</i>	<i>No. of different individuals describing each emotion</i>	<i>Total no. of references to each emotion</i>
Hunger	28	165
Enjoyment	22	90
Guilt	29	82
Cravings	25	53
Hunger in the morning	29	45
Struggle	18	38
Comfort	15	26
Obsessed with food	15	22
Disconnected	11	17
Temptation	7	17
Addiction	9	15
Satisfied	6	12
Habit	5	10
Bored with food	5	7
Respect for food	4	6
Entitlement	4	5
Feel need to eat	4	4
Feeling deprived	3	3
Too tired to eat	1	3
Torment	2	3
Emptiness	2	2
Greedy	1	1

The emotion of hunger was discussed most frequently.

Hunger:

'I've never had a feeling of being hungry. I can't tell you when I ever said 'I am hungry'. I eat and I don't have a feeling of hunger you know. I eat because it's there, it's them certain hours of the day that you eat on them certain hours or three, there's goodies there and I'm tormented because they're there but I've never ever, as far as I can remember, I've never had that feeling of me belly rumbling or feeling hungry. I never feel hungry'. C065

'I'm always hungry, every minute of every day'. (SK040)

'I think I need to time between meals because I don't, you know, I eat for appetite rather than hunger I think sometimes and I think it would be useful to have a routine of eating'. (RC052)

'Oh yeah, I never... you know when people say 'Oh God, I'm awful full' I could eat and eat and eat. I don't get that full feeling'. (MH004)

Whilst many individuals (n=22) acknowledged the enjoyment they derived during eating and 15 found it comforting to eat, other emotions associated with eating appeared to have a more negative impact on the individual, such as feeling guilty about eating or being obsessed with food.

Enjoyment:

'So, before, when I could have a roast dinner and I'd have 6 potatoes on it, God I loved it! I don't do that now'. (JC062)

'Unless it's during the adverts, you'll have a Homer Simpson moment, you know, you'll see something advertised on the TV and your like 'mmm profiterole', or I'll have to get myself some of them, something new'. (JMC068)

Guilt:

'I done it, you know and then I start calling myself things because you've spoilt it and cant do anything and just attack yourself then, well I do'. (KD055)

'I'm not getting guilt over food anymore. That's been my problem for a long time, I've felt guilty and then I'm thinking afterwards I shouldn't be feeling guilty for doing something like eating. If I'd murdered somebody I could feel guilty over it, but not because I want something to eat and it's been like that for years and years and years'. (LR047)

And then I get the guilt feeling. Why did I do that? I didn't want it or need it. Why did I do it? And I start kicking myself then but unfortunately that feeling doesn't last, like when I'm on the computer again, twenty four hours later, I'm thinking. 'What's in the fridge?'. (KM049)

A similar wide range of emotions related to day-to-day living was described by the NEB group and is summarized in Table 31.

Table 31 - Range of general emotions described by the NEB group

<i>General emotions</i>	<i>No. of sources</i>	<i>No. of references</i>
Low mood	29	111
Shame	22	109
Anxiety	15	61
Annoyance	22	58
Tired	16	58
Frustration	20	39
Unhappy	16	29
Upset	16	28
Emotional stress	11	27
Agoraphobia	3	14
Get bored	10	12

Grief	3	10
Distraction	3	9
Freedom	4	8
Relaxed	5	8
Emotional distance	2	7
Crutch	4	5
Delusion	4	5
Devastated	3	5
Lonely	3	5
Bitter	3	4
Content	3	3
Lazy	3	3
Shock	1	2

The vast majority are negative emotions with feelings of low mood in particular, shame related to obesity and anxiety most prevalent. Individuals often acknowledged such emotions to be influencing eating behaviour.

Low mood affecting eating:

'Sometimes, you know sometimes, it bugs me sometimes when I've got chocolate in, when I'm depressed. I have a chocolate 'group', I get myself a massive chocolate milkshake and then after that I'll have some, either bourbon biscuits and some chocolate squares, or a penguin and some chocolate squares, so I eat chocolate and I'm washing it down with chocolate and you know, sometimes I try not to do it when people are there and I feel self conscious about it. You know seeing people eat three kinds of chocolate and all that, but that was more down to depression and all that'. (JMC068)

'Mood terrible, moody, you know, bite people's heads off like basically, because I'm like stressing because I can't have this and then I start thinking well you know, what it's all for? Why should I be like this, if I like it, eat it, it's a vicious circle- I'm depressed because I'm on a diet and then when I eat it I'm depressed because I've eaten it, so I can't win either which way'. (LN 080)

Shame about obesity:

'I mean, it's been a couple of times where a couple of friends have been to gym and I only went twice and them two jumped in the shower and I'm like, 'well I don't want you to see me, I'll just get a wash'. (AB010)

'I'm always wearing black. I'm disguising myself in black all the time. They call me the black widow do you know what I mean, because I'm always in black. I'm trying to disguise my weight. And they say to me, 'D.. I'm not being funny, but even the way you dress now, in your back pants, your back jacket, your not disguising nothing because you know you're so big so.. and that's why I'm not bothered now. I just..., well I am bothered, but ..'. (DD027)

'If there's activities on that I can't do with the kids and they know that, like swimming, they've asked me to come swimming and I will not go swimming. I did abroad, like I say I'm not bothered, people don't know me. My daughter has asked me plenty of times to take her swimming and I said 'no' and she said 'why?', 'cos I don't want to' – she says, 'cos you're a big mum' and I go 'well yeah, you know I feel embarrassed getting in the water'.. It's not that I can't do it, I'd love to do it. I want to do it, but I won't do it because I'm ashamed'. (DD027)

Shame about eating:

'I have thought, sometimes when I have ate too much, 'go make myself sick'.. you know what I mean? I wish I had anorexia. Yeah and ashamed, very ashamed about the amount I used to eat, you know'. (CP065)

Anxiety:

'See, this is why "K" comes with me. I didn't want to come this morning but she shouted at me. So I've got no options then. I can cope with this. I might get a bit panicky but I can cope with it'. (JP017)

Anxiety about eating:

'Because I've tried over the years to lose weight and to try to be strict with myself in a sense that I know the weight is making my health worse and it frightens me that way and I'm still eating. I try and stop it and I'm still doing it knowing when.. I even sit and eat sometimes worrying about my weight while I'm eating and that doesn't stop me'. (EC051)

4.7.3.11. **Conflictful Relationships**

Participants with NEB generally describe strained work and social relationships, citing many instances of work-place discrimination, bullying and 'teasing' typically associated with morbid obesity. One individual describes protecting herself from such approaches with multiple face piercings.

'Like a lot of people in work, I don't think they like even dare say anything cos I'd bite their head off (multiple face piercings). (SLD011)

Family life in particular is not harmonious. For younger participants the main focus of conflict in the home is with parents and for older participants it is with partners. Arguments range from trivial disagreements about work life balance to more fundamental relationship issues.

'Then we had family problems....My husband now is not my son's father, there was a battle going on and stuff like that, I was seeing it all, getting emotional'. (DD027)

'One minute she's telling me she doesn't want me to move out and another time she's telling me to pack my bags'. (RB008)

Family members often express concern about the eating patterns and obesity of the participants.

'My mum's a counsellor, but she says to the doctors, 'I can't even get through to my own daughter''. (KR009)

'And they went 'did you get the pies?' I went 'no'. I couldn't say I ate 4 pies. He'd have went 'you dirty big fat baba'. (MC024)

Some NEB participants view this positively:

'I feel sorry for the missus because, you know, she's at her wits end at times you know'. (AB007)

JC - 'Did your mum know you were doing that, do you think? Going down and eating?'.
JP- 'Oh yeah. She used to shout at me, I've always had to have someone to tell me not to eat something, left to my own devices. I need telling off.'
JC to partner - 'Is that your job now, telling him off?'.
JP- 'She doesn't tell me off hard enough'. (JP017)

Others consider comments from family members as demeaning and disrespectful.

'My brother said to me years ago, we've never been best of mates, never ever, maybe because we're so different in ways and years ago, he said to me, I'll never forget it and my mum knows about it and she says, I'll never forget what he said to you, he said 'do you realize, you're only funny cos you're fat. And I said, why do you reckon that? Well, it isn't, no, I'm funny because I'm funny. What I do on stage is not funny cos I'm fat; it's funny cos of what I do, it's funny because I'm talented to do it'. (GD015)

I was like, stick thin as a child. A stick. Straight as a pin. And my mum said 'straight as a pin – look at you now, what shape are you, there's no shape to you'. (MC024)

'My husband just thinks like 'you're just waiting for the operation, that's what you're doing and you're not trying'. (DW039)

In the main, other family members are criticized whether they interfere or not.

'But then I say that, I want you to make me do stuff and then, when I've had enough of it, I'll be dead nasty to my mum then and I'll go off on one and I'll be like, no I don't want you.... Get out of my life, so I want it, but I don't want it at the same time, I just don't know what I want really'. (KR009)

'And it's sort of like, because I felt a bit tired and run down, I'd been eating less this time. I thought, well it serve them right if I eat too much...it's just thinking, well, if they're not bothered, they're not really helping me'. (LN080)

Some blame family members for sabotaging attempts at restraint

'My mother, like if she has words with me and that, she'll always give me food. Not that we do it that often, but if she, if we have a sort of biggish falling out, you'd say, right, I won't bother speaking to you then till you've calmed down sort of thing. Then she'll order a pizza and send it round. And I say to her, you always make up with food and to me that says I know I was in the wrong. But I was saying...why food?'. (LN080)

'He brings stuff in, but not the things I really ask for. There are things over and he'll say, well they're only going to get thrown out and go to waste, you may as well'. (MK016)

'More of the time when she's down it's 'Ey, what do you want from the chippie?'. (AB010)

'She can come across supportive in front of people, but you know, she can't be bothered cooking, I have to go the chippy, she knows that I'm desperate to lose weight and if I could cook I'd cook, but it doesn't help her comment saying, cook yourself if you don't want to eat what we're eating, things like that aren't helpful, cos I'm left to eat nothing'. (RB008)

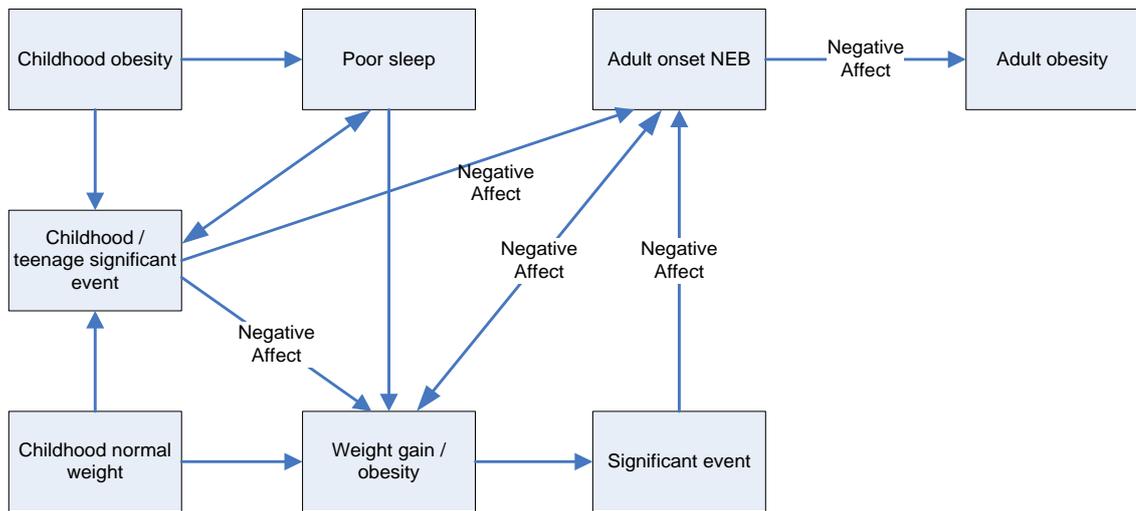


Figure 10 - Diagrammatic representation of the pathways between adult onset NEB and obesity

4.7.4.2. *Childhood Obesity and Traumatic Life Events*

All participants in this study were either obese or overweight with 15 (50%) of the NEB group reporting being obese as children. Childhood obesity is often linked to low birth weight, but it is unclear if the catch-up growth is genetic or biological. Stettler et al. (2002) showed that rapid weight gain during the first 4 months of life is associated with an increased risk of overweight status at the age of 7 years, independent of birth weight and weight attained at one year. Rolland-Cachera et al. (2006) evaluated several studies which compared the age of Adiposity Rebound (AR) in children to adult obesity and found a mean age of 3.2 years for AR in obese adults as opposed to 6 years for non obese. The early AR was associated with low fatness before the rebound and high fatness after the rebound, suggesting a period of energy deficit had occurred early in life which could have ‘programmed’ adaptive metabolism. The level of childhood obesity in this group is unsurprising as it is a known risk factor for adult obesity. Whitaker et al (1997) found that for those who were obese during childhood, the chance of obesity in adulthood ranged from 8 % for 1-2 year olds without obese parents to 79% for 10-14 year olds with at least one obese parent. It is possible therefore, that NEB developing as a result of childhood obesity has contributed to the maintenance and worsening of adult obesity.

A high number of significant life events were also noted in the NEB group. Although events varied in nature, 9 individuals reported the absence of a significant individual as having a major effect on their life. Published evidence of the links between trauma and the onset of

obesity is limited. In this sample, 14 /30 (46%) of individuals appeared to develop obesity as a result of trauma although the relationship between traumatic events and the onset of NEB was less clear.

4.7.4.3. *Eating Patterns and NEB*

Night-eating appeared secretive and compulsive with sufferers experiencing varying degrees of awareness of their night-eating behaviour. Eating episodes were perceived by the individuals to be uncontrolled and associated with post-prandial guilt. Day-time eating patterns generally appeared chaotic and were also associated with post – prandial guilt. Whether this develops prior to the onset of NEB or as a consequence is unclear. Nor is it clear whether it is a feature of all individuals with morbid obesity, or just those who have/go on to develop NEB.

Various models of perceived behavioural control have sought to explain its influence in relation to other eating behaviours. Conceptual models vary slightly in their emphasis. Ajzen (2002) describes the theory of planned behaviour as a hierarchical model, whereby self-efficacy and controllability are two separate components, each assessed by means of different indicators. Together they comprise the higher-order concept of perceived behavioural control. Perceived self-efficacy relates to ‘people’s beliefs about their capabilities to exercise control over their own level of functioning and over events that affect their lives’ (ease or difficulty of performing a behaviour) and perceived controllability relates to ‘beliefs about the extent to which performing the behaviour is up to the individual’. Whilst this model is conceptually independent of the more traditional ‘internal versus external locus of control theory’ described by Rotter (1966), it is likely that the two key features of self-efficacy and controllability are influenced by both internal and external factors.

Waller (1998), found women with established eating psychopathology to have a relatively external locus of control in that they saw themselves as having low levels of control over events and their own lives, resulting in feelings of inadequacy and ineffectiveness. The locus of control scores of 28 females with an eating disorder (ED) but without a history of sexual abuse were compared to 27 females with both ED and a history of abuse. Whilst all scores were higher than normative values (Nowicki & Hopper 1974), the sexual abuse group had significantly worse mean (SD) scores (15.8 (6.48) vs 12.2 (5.26) $p<0.05$). Dalglish et al.(2001) used the World

Assumptions Scale and The Attributional Style Questionnaire with 18 individuals with Anorexia, 15 with Bulimia and 22 healthy controls. Subjects with Anorexia and Bulimia reported having less personal control over outcomes in the world and a tendency to attribute the cause of 'bad' life events to aspects of themselves. It is possible that individuals with NEB are characterized by an external locus of control and poor self efficacy, making them less able to control their eating patterns although these may be characteristics shared by obese individuals generally.

4.7.4.4. *Low Mood and Depression*

Eating at night was generally due to perceived hunger or in response to negative affect. NEB individuals were characterised by negative emotions related to both eating and day-to-day living generally. Although the mean (SD) BDI score was significantly higher in the NEB group than in the control group (24.8 (10.9) versus 17.7 (10.8)), the lower mean score in the control group would still be considered indicative of moderate depression. As the relationship between obesity and depression is known, the influence of NEB on this association becomes less clear-cut. It is possible that other factors related to obesity such as low self-esteem and shame are contributing to the degree of depression.

Childhood overweight is known to have psychological and emotional consequences, with overweight children at increased risk of teasing and bullying, Ninety-six percent of a sample of 50 overweight female adolescents reported stigmatising experiences as a result of obesity (Neumark-Sztainer, Story, & Faibisch 1998). Society views fatness as a reflection of poor character with children as young as 3 associating overweight peers with being mean, stupid and ugly (Brylinsky & Moore 1994). Parents' attempts to encourage dietary restriction may reinforce these views and be internalized by the child as saying there is something wrong with the body, leading to feelings of shame and low self-esteem. Pierce & Wardle (1997) found high levels of low self esteem in 9 – 11 year old obese girls, which was further linked to the child's belief that their parents viewed their weight negatively and parental reports of dissatisfaction with the child's weight. Those with lowest self esteem believed they had caused their obesity themselves. Strauss (2000) found similar results in white and Hispanic girls aged 13-14. Those whose parents had greater concern about their body size were more likely to distort their bodies as too heavy and have an increased risk of depression (Wallace, Sheslow, & Hassink 1993).

4.7.4.5. *Sleep Quality and NEB*

The relationship between poor sleep quality and NEB appeared particularly complex, with a causal relationship between the two not clearly identified. Prevalence rates of self-reported sleep difficulties in general populations range from 10-40%, with higher rates for women and older subjects (Linton 2004; Mellinger, Balter, & Uhlenhuth 1985). As would be anticipated, all individuals with NEB, regardless of age or gender, self-reported poor quality sleep, apart from those who stayed up very late prior to going to bed. Whilst some recalled life events which contributed to poor sleep quality prior to the onset of NEB, a variety of physical factors appeared to influence current night-time awakenings, some of which appeared to be either directly or indirectly obesity-related through co-morbidity. Only 4 individuals perceived their awakenings to be due to emotional or psychological problems.

Obesity-related co-morbidities such as OSA appeared to account for some sleep disturbance with chronic pain also featuring heavily. Chronic pain is significantly associated with depression and anxiety (Karp et al. 2005). When pain and depression exist together, a 'synchrony of change' is often noted, with diminished self-efficacy associated with both depressive illness and chronic pain in later life. As sleep of older adults is known to contain more intra-sleep arousals, less slow wave 'deeper' sleep and more light sleep, the pain, aging and depression axis can have a profound effect on sleep quality (Karp & Reynolds 2009). However, this would not explain why some individuals get up to eat once awake and others return to sleep.

Many reported a backdrop of poor sleep for many years and all the co-morbidities experienced in the NEB group are obesity-related, making it difficult to determine the extent to which obesity and disrupted sleep were influencing each other, independently of NEB. The relationship between short sleep duration, its negative effect on mortality and relationship with obesity is known (Kohatsu et al. 2006; Vorona et al. 2005) and more recently, studies are starting to show a relationship between poor sleep quality and obesity. Resta et al. (2003) compared 78 obese subjects (mean BMI 39.7 (5.95) kg/m²) without Obstructive Sleep Apnoea to normal weight controls and found the obese group to have significantly higher sleep latency, lower percent of REM sleep and lower sleep efficiency. Chaput et al. (2005) investigated improvements in sleep quality in 11 men losing weight with a lower baseline BMI (mean age 38 years, mean

BMI 33.4 kg/m²) and saw a significant improvement in sleep quality with a 5% weight loss (p<0.01).

4.7.4.6. *Sleep Quality and Stress*

The most frequently reported cause of sleeping difficulties due to increased arousal is work-related stress. Data are limited to cross sectional studies and longitudinal data would be needed to make a causal inference and determine the direction of the link. In the demand-control-support model discussed by Nordin et al.(2005), workload can be viewed as a psychological demand and when demand exceeds the perception of control, the individual may perceive stress. In a random sample of 2066 20-60 year old Swedish inhabitants (52% male, mean age 41 years) subjects were classified as good sleepers, poor sleepers and insomniacs (i.e. a problem in initiating and maintaining sleep for more than 30 minutes at least 3 times a week). Stress during the day and stress at work were reported as related to sleep by 64% of the poor sleepers and 70% of the insomniacs with sleep problems reported as having a negative effect on aspects of work capacity such as poor concentration, headaches and poor memory. The 2002-3 National Employee Survey measured sleep difficulties and psychological work stressors over a longer time period of one month and found a similar positive link between high work demands, disturbed and non-restorative sleep (Ota et al. 2005). Although few of the NEB group were working and only one individual reported work-related stress as a reason for waking up, this does not exclude NEB and stress as mitigating factors in the reason for stopping work. Personal vulnerability to stress is complex and mediated by many factors such as biological make-up, genetic predisposition, conditioning and learning, past history and current life situation, personality characteristics and attitudes. The resultant state of hyperarousal is in itself a core mediating feature of insomnia. Morin, Rodrigue & Ivers (2003), proposed that insomniacs have different coping mechanisms to good sleepers, tend to internalize conflicts and ruminate over things they should have done or said. An individual's perception of control over a stressful situation can determine the emotional response and predict the most likely coping strategies that will be adopted. It is possible that individuals with NEB adopt a similar external locus of control model when dealing with stress. In a study examining coping strategies of insomniacs, forty subjects with insomnia and 27 good sleepers with a mean age of 39.6 years were submitted to a battery of scales to measure perceived

stress, coping, pre-sleep arousal and insomnia (Morin, Rodrigue, & Ivers 2003). A significant group difference on the perception of control over stress ($F(1,64) = 8.83, p < 0.005$) was noted, with insomniacs reporting lower levels. Insomniacs also relied more on emotion-orientated coping strategies. ($F(1,64) = 3.90, p < 0.05$) and reported higher pre-sleep cognitive and somatic arousal states. For a given day, a high level of stress was associated with a higher level of cognitive and somatic arousal at bedtime. A significant association between a negative appraisal of stressors and sleep disturbances was noted. Both groups reported equivalent numbers of minor stressful life events, but insomniacs rated both the impact and the intensity of the events as higher. In the NEB group, conflictful relationships with others also appeared common, suggesting the use of conflict as a coping mechanism to perceived stressors. Of course this may also be a relationship feature of obese individuals without NEB.

4.7.4.7. *Sleep and Depression*

The significant levels of depression seen in the NEB group must be considered as an important contributor to both the development and maintenance of NEB through its relationship with poor sleep. The neurobiology of disturbed sleep in depression is well documented with disturbances in the electrophysiological architecture noted as well as functional deviations in the different brain regions of individuals with depression, such as a decrease in slow wave sleep and increase in awakenings (Drevets 2001). Several studies have demonstrated a link between poor subjective sleep quality and depression. Agargun, Kara & Solmaz (1997) examined 41 patients with major depressive disorder using the Pittsburgh Sleep Quality Index (PSQI) and Schedule for Affective Disorders and Schizophrenia (SADS suicide subscale). The SADS subscale scores positively correlated with the scores of subjective sleep quality ($r = 0.46, p < 0.01$), sleep latency ($r = 0.47, p < 0.01$), sleep duration ($r = 0.46, p < 0.01$), habitual sleep efficiency ($r = 0.53, p < 0.001$) and global scores ($r = 0.59, p < 0.001$). Kennedy et al. (1991) confirmed these findings and Fawcett et al. (1990) identified insomnia as a predictor of suicide among patients with affective disorders.

The association between diminished life satisfaction and poor or insufficient sleep has also been reported in a few cross-sectional and retrospective studies, with others showing sleep satisfaction rather than sleep duration to predict greater quality of well-being. The Finnish Twin

Cohort study examined life satisfaction and sleep quality in 12964 individuals with a mean age of 33 years at baseline and 6 years later (Paunio et al. 2009). Those with poor sleep quality at baseline had a significantly elevated risk of later life dissatisfaction (OR= 2.1, 95% CI: 1.7, 2.7 from logistic regression on individuals), suggesting a predictive relation between poor sleep quality and subsequent incident life dissatisfaction but not the converse. In a cross-sectional study examining the association between poor sleep quality and day-time affect in 35 individuals with major depression, 25 with minor depression and 36 controls, Bower et al. (2010) showed poor sleep quality to predict lower ambulatory positive affect, even after accounting for diagnostic group. This is in keeping with the overwhelmingly negative affect reported by the NEB both in relation to eating and day-to-day living.

4.7.5. *Unanswered Questions*

This qualitative analysis of the interview data has revealed the influence of childhood obesity and traumatic life events on the development of NEB, the effect of poor sleep on the onset and maintenance of NEB, the compulsive nature of eating at night and chaotic day-time eating patterns, the negative emotions relating to eating and general day-to-day living experienced by NEB sufferers and the prevalence of conflictful relationships.

However, many questions remain unanswered:

- Are levels of childhood obesity and trauma the same in non-NEB individuals?
- What is the relationship between NEB and adult obesity?
- Do other obese individuals without NEB have similar issues with control and chaotic day time eating?
- Is poor sleep quality a problem for other obese individuals?
- Do the symptoms of other obesity related co-morbidity affect the sleep and eating patterns of other obese individuals without NEB in the same way?

It was decided therefore, to undertake a closer comparison with a matched control group of other study participants without NEB, to examine the degree to which characteristics identified in the NEB participants also feature in other obese individuals.

4.8. *Comparison with Non-NEB Matched Control Group*

4.8.1. *Rationale for Matching*

All participants in the original identification study were interviewed and examined for the presence of NEB. During this process, 51 obese individuals without NEB were identified. These individuals had been subjected to the same interview processes as the NEB individuals and were therefore considered an appropriate sample from which a matched control group could be identified. In addition, time constraints and limited study resources made it impractical to recruit further research participants. The four key variables included in the matching process were gender, BMI, work status and age.

4.8.2. *Gender*

The study population as a whole contained a higher proportion of males than the normal clinic population (43% versus 33%). The numbers of males in the NEB study group and non-NEB study group were similar (45% versus 42%). A quarter of the whole study group (25%) had Obstructive Sleep Apnoea (OSA) or symptoms suggestive of OSA, with 19% in the NEB group and 28% in the non-NEB group. OSA is known to be more common in obese males and may have accounted for the relatively high numbers in both groups due to its known association with disturbed sleep (Coughlin et al. 2004). Evidence shows that poor sleep is more problematic for females than males and early landmark NES studies suggested NES to be more common in females. It was important therefore to control for gender, to ensure that other characteristics associated with male or females were not influencing conclusions about the characteristics of NEB.

4.8.3. *BMI*

The mean BMI of the study group was 50.0 (10.7) kg/m² compared to 48.5 (11.7) kg/m² in the clinic population. The difference between the NEB group and non-NEB study group was not statistically significant, although the mean BMI of the NEB group was slightly higher (52.5 (11.7) kg/m² versus 48.4 (9.8) kg/m²). The NEB group reported a high level of physical dysfunction affecting their sleep and attributed this to a variety of physical causes, including obesity-related co-morbidity. The link between rising BMI and the risk of developing obesity-related co-morbidity such as OSA, type 2 diabetes and depression is well documented (Coughlin

et al. 2004;Dong, Sanchez, & Price 2004;Fogelholm et al. 2007). The debilitating effects of chronic pain also worsen with increasing BMI (Karp & Reynolds 2009). Despite the lack of difference in BMI between the two groups, in order to determine the effect of co-morbidity on NEB it was important to account for this in the matching process. Due to the small sample size, it was impossible to match every individual according to co-morbidity, and it was considered more realistic to establish a BMI cut-off at which point the physical consequences of co-morbidity are likely to affect Activities of Daily Living (ADL). Research evidence for an appropriate cut-off level is limited, with researchers tending to focus on the BMI levels at which co-morbidities develop rather than their subsequent impact. For example, once the BMI rises above 30 kg/m², the risk of developing type 2 diabetes increases exponentially (Chan et al. 1994), although it is less clear at which point diabetes disturbs eating and sleeping behaviour. NICE consider an individual with a BMI > 50 kg/m² to be sufficiently physically impaired to recommend bariatric surgery as a first line treatment and clinical experience suggests that most obese individuals appear to struggle with some aspects of ADL (Activities of Daily Living) once their BMI is > 50 kg/m². Thus a cut-off of 50 kg/m² was chosen on which to base stratification of BMI.

4.8.4. *Work Status*

It was important to control for work status as individuals in the NEB group were less likely to be working than non-NEB individuals (29% versus 54% p=0.03). For most individuals who work, the start of the working day tends to be at a standard time in the morning (7- 9.30 am). According to the Social Zeitgeber hypothesis of Ehlers Frank & Kupfer (1988), work has an effect on regulating social rhythms and anchoring bed and rising time. Individuals with NEB had chaotic and irregular day-time eating patterns and it is possible that working individuals with a propensity to night-eating behaviour may curtail the tendency to get up and eat if there is no opportunity to sleep in at morning time or catch up on sleep later in the day. The impact of work-related stress on sleeping patterns must also be considered, as individuals with NEB who do work may be influenced by different factors than those who are not in work.

4.8.5. *Age*

There was no difference in mean age between the NEB and non-NEB group in the initial analysis. The NEB group were slightly younger (mean 43.3yrs versus 46.0yrs). A larger

proportion of the NEB group (42%) were aged less than 35 years than the non-NEB group (10%). This may be partially a cohort effect related to the rapid rise in obesity over the past 20 years. Whilst it is possible that night-eating behaviour is more common in younger obese adults and age needs to be accounted for in determining the characteristics of NEB, its role in determining the interplay between obesity and NEB is less important than other factors such as the degree of obesity, its impact on physical and psychological functioning and work status.

4.8.6. *Matching Process*

Twenty eight subjects were matched in total and the breakdown of individuals matched per strata is displayed in Table 32. Gender, BMI and work status were prioritized over age. All 14 male NEB individuals and 17 female NEB individuals were matched for gender. One non-working NEB male with a BMI of 51 kg/m² was matched with a similar non-NEB male with a BMI of 46 kg/m². Although the cut-off for BMI was 50 kg/m², the two readings were considered close enough for the match to be acceptable. Matching individuals according to work status was more difficult, due to the low numbers in the NEB group who were working. Eight male NEB participants with a BMI >50 kg/m² were not working, as opposed to only 4 of the similar non-NEB group, thus 3 were not matched and the fourth was the individual matched with the lower BMI group described above. As five was considered a sufficient number of individuals to represent the BMI >50 kg/m² non-working group, it was not considered necessary or practical to identify and interview other individuals who were not part of the original study to perform further matching. Where possible, individuals in the individual NEB group were age-matched to within 5 years with individuals in the non-NEB group. Age matching was not successful for 8 NEB individuals, due to the high number of young individuals with NEB, particularly in the non-working, lighter female group. These 8 were all matched with an older individual, as age as a variable was considered to have less effect per se on physical functioning than BMI and the primary purpose of the analysis was to examine the effect of obesity on the NEB.

Table 32 - Table showing number of NEB individuals and non-NEB individuals who were matched according to gender, BMI, age and work status strata.

		<i>Male</i>			<i>Female</i>			
		<i>NEB (n=14)</i>	<i>Non NEB (n= 20)</i>	<i>Male matches</i>	<i>NEB (n=17)</i>	<i>Non NEB (n= 30)</i>	<i>Female matches</i>	<i>Total matches</i>
<i>BMI>50</i>	<i>Working</i>	2	6	2	2	3	2(2) ¹	4
	<i>Not working</i>	8	4	5(2) ¹	5	6	5(1) ¹	10
<i>BMI<50</i>	<i>Working</i>	2	4	2	3	14	3	5
	<i>Not working</i>	2	6	2	7	7	7(3) ¹	9
<i>Total</i>				11	17			28

¹ Numbers in brackets indicate the number of NEB individuals within that particular matched sub-group who were younger than their matched control by more than 5 years.

4.8.7. *Statistical Analysis*

Comparisons were made between the 28 NEB individuals and their non-NEB matched controls. Due to cost and time constraints, it was not possible to transcribe the interviews of the non-NEB interviews for line-by-line NVivo coding and analysis; thus the interviews were replayed and re-examined, concentrating on key areas of childhood obesity, traumatic life events, eating patterns, mood and sleep. Qualitative data obtained from this re-examination are presented as a narrative, or in tabulated form. Where appropriate, supporting evidence from quantitative data is also presented. Differences between the NEB group and non-NEB matched individuals were compared. The Wilcoxon Signed Ranks test was used to compare differences in EDE scores. This is a non-parametric test used on ordinal or ranked data to test the differences between medians. Differences in BDI and NEQ (2004) scores were compared using the Paired t test. This is used with scale data to compare the means of two variables. Differences between nominal data such as the presence of childhood obesity and traumatic life events were compared using the non-parametric McNemar test. This is applied to 2 x 2 contingency tables with a dichotomous trait

with matched pairs of subjects to determine whether the row and column marginal frequencies are equal. Multiple testing was justified as this was exploratory analysis and the current relationship between individual variables is unknown. Eating patterns are presented as a spread sheet in Appendix 9.

4.8.8. Results

4.8.8.1. Onset of Obesity and Significant Life Events

The non-NEB matched control group reported similar rates of childhood obesity to the matched NEB group (13/28 vs 17/28) and the individual differences between the NEB and matched control pairs was not statistically significant (Table 33). Childhood bullying as a result of obesity was a common theme throughout the interviews, with a larger number (n=6) perceiving this to have a major impact on their life in comparison to the matched NEB group (n=2). Fewer number of individuals in general reported traumatic life events to the matched NEB group (13/28 vs 18/28) although the individual differences between the NEB and matched control pairs was not statistically significant (Table 34). Whilst fewer in number, events were similar in nature, with death/absence of a significant individual featuring predominantly (Table 35).

Table 33 - Pairwise comparison of the numbers of individuals with childhood onset obesity in the matched NEB group and non-NEB matched control group.

<i>Non-NEB matched control group</i>			
<i>Matched NEB group</i>	<i>Yes</i>	<i>No</i>	<i>Total</i>
<i>Yes</i>	9	8	17
<i>No</i>	4	7	11
<i>Total(n)</i>	13	15	28

McNemar test p value = 0.25

Table 34 - Pairwise comparison of the numbers of individuals experiencing traumatic life events in the matched NEB group and non-NEB matched control group.

<i>Non-NEB matched control group</i>			
<i>Matched NEB group</i>	<i>Yes</i>	<i>No</i>	<i>Total (n)</i>
<i>Yes</i>	9	9	18
<i>No</i>	4	6	10
<i>Total (n)</i>	13	15	28

McNemar test p value = 0.16

For the individuals who developed obesity as an adult, there appeared to be a clear relationship between the onset of obesity and a traumatic life event for 7 individuals as opposed to developing obesity as a result of lifestyle changes. One individual recalls gaining weight after witnessing a murder as a young adult and another deliberately gained weight to make herself unattractive after being raped in her own home. For 4 individuals, the event was due to a physical cause such as an international standard sportsman having to end his career after injuring his ankle.

Table 35 - Categorisation of traumatic life events experienced by the matched non-NEB and non-NEB matched control groups and number of individual experiencing such events. Some individuals experienced more than one event.

<i>Traumatic event</i>	<i>Number of participants experiencing the event</i>	
	<i>Matched Non-NEB</i>	<i>Matched NEB</i>
Partner's agoraphobia / dementia, child with chronic illness	2	1
Parent's/partners alcoholism (own cannabis addiction)	1	2
Serious sexual assault (self,siblings)	1	3
Death/suicide of: parents/ grandparents siblings/partner/child/child in utero	7	4
Physical injury: back/ accidental burning/brain haemorrhage/ ankle, childhood accident, hospitalisation	4	4
Divorce(self/parents)/ absent parents	5	5
Childhood bullying	6	2
Premature retirement/off work	1	3
Witness to a murder	1	0
Tourette's age 11	1	0
Teenage pregnancy/motherhood	0	2
Maltreatment by family	0	4

4.8.8.2. Sleep

The matched non-NEB group reported similar difficulties and frustrations with sleep to the matched NEB group with high numbers of the matched control group describing difficulty falling asleep (n=17) despite being tired. However, the mean score of the NEQ (2004) sleep-related item was significantly higher for the matched NEB group (2.6 (1.5) vs 1.7(1.6), $p = 0.04$),

likewise significantly more of the matched NEB group reported poor sleep in comparison to their matched control on the BDI sleep-related item (Table 36).

Table 36 - Comparison of the NEQ and BDI sleep-related item scores between the matched NEB and non-NEB matched control group using paired t tests (A higher score in both the NEQ and BDI items indicates a greater degree of dysfunction).

	<i>Mean(SD)</i>		<i>P value</i>	<i>95% Confidence Interval of the difference (NEB-Non-NEB)</i>
	<i>Matched NEB n=28</i>	<i>Matched non-NEB n=28</i>		
<i>NEQ sleep item</i>				
Trouble getting to sleep	2.6 (1.5)	1.7 (1.6)	0.04	0.1, 1.8
<i>BDI sleep items</i>				
Poor sleep ¹	1.6(1.0)	1.0(1.1)	0.04	0.04, 1.2
Feel tired	1.5(1.0)	1.1(0.8)	0.07	-.04,1.0

¹ n=26.

Reasons for struggling to get to sleep were generally not specified, although one individual who had experienced the sudden death of both parents admitted to being frightened to go to sleep in case he didn't wake up and once staying awake for 2 and a half days. One individual was disturbed by intrusive thoughts of 'letting himself down' and used the radio to block these out. Two individuals blamed previous shift work for current sleep problems. Four individuals gave physical causes; two could not fall asleep if they felt hungry; two reported hip and leg pain and one reported previous breathing related difficulties which improved very recently after starting Continuous Positive Airway Pressure treatment for OSA. A similar variety of distraction techniques was performed by the non-NEB group individuals as with the matched NEB group to help with dropping off to sleep (Table 37) and a larger number (50%) reported sleeping with their partner (Table 38).

Table 37 – Categorisation of activities undertaken either before bed or during the night by the non- NEB matched control group (n=28) and the matched NEB group (n=28) and the number of individuals undertaking each activity. NB some individuals undertook several activities.

<i>Activity before sleep and during night</i>	<i>Number of participants undertaking the activity</i>	
	<i>Matched Non-NEB</i>	<i>Matched NEB</i>
Watch TV/DVD	3	16
Read/puzzles	3	7
Radio/Music	1	2
Drink alcohol	2	1
Paperwork	1	1
Walks around to stretch legs	1	0
Late night shopping	0	1
Computer	0	1

Table 38 - Categorisation of sleeping arrangements of the non-NEB matched control group and matched NEB group and number of individuals per sleeping arrangement (n=28).

<i>Sleeping arrangements</i>	<i>Number of individuals per sleeping arrangement</i>	
	<i>Matched Non-NEB</i>	<i>Matched NEB</i>
Sleeps with partner	14	8
Lives alone	8	7
Intermittent partners	0	4
Lodges with family: own room	2	0
Lives with parents: own room	3	4
Shares room with sister	0	1
Spare room (disturbs partner)	0	2
Not specified	1	2

Once asleep, similar levels of sleep disturbance were noted in the non-NEB control group as in the matched NEB group, with similar numbers citing either medical or breathing-related causes (15 versus 13) for waking up during the night (Table 39). Seven of the control group identified an ‘emotional’ reason for waking up, as opposed to only 4 of the matched NEB group.

Table 39 – Categorisation of reasons for current night-time awakenings in the non-NEB matched control group and matched NEB group and number of participants per reason for night-time awakening. N.B. some individuals in both groups give more than one reason for waking

<i>Reasons for night-time awakening</i>	<i>Number of individuals per reason for night-time awakening</i>	
	<i>Matched Non-NEB</i>	<i>Matched NEB</i>
Eating-related:		
Day-time restraint	0	4
Needs a drink	9	2
Feels peckish (does not eat)	1	0
Sleep-related:		
Day-time sleep	0	2
Not tired	0	1
Early to bed	0	1
Always 'tired'	3	0
Medical:		
Legs, pain, pressure , hot, restless legs	4	2
Back pain	2	0
Hip pain	1	0
Hiatus hernia	0	1
Diabetes > problems sleeping	0	1
Psoriasis- awake scratching and washing hands	0	1
ME	0	1
Dry mouth	0	2
Need for toilet	11	3
Too hot	1	0
Arthritis	1	0
Breathing:		
Sleep Apnoea and mask problems	3	5
Can't get my breath	0	1
Chronic obstructive Pulmonary Disease	0	1
Snoring	3	0
Emotional:		
Emotional stress	0	1
Bad dream, nightmare	1	1
Feeling unhappy, low, crying	1	1
Stress at work	0	1
Frightened, jittery, panic attacks	4	0
Worrying	1	0
External:		
Noises outside, light sleeper	1	2
Not sure why	0	4
Not specified	2	0
Not disturbed	6	0

Once awake, 11 individuals would get up to the toilet and another 11 individuals would go to the toilet and either go downstairs to make a drink or have a drink by the bedside. Two individuals

reported going downstairs to sit in a chair, but none reported eating overnight, whatever the initial reason for waking and getting out of bed. Generally, sleep was described to be of similar, poor quality to the matched NEB group with many individuals reporting restless and fitful sleep patterns and subsequent day-time fatigue. Five individuals reported falling asleep in the day, two struggled to get out of bed in the morning and one individual even fell asleep during the research interview.

4.8.8.3. Eating Behaviour

In comparison to the matched NEB group, the non-NEB control group appeared to have a less chaotic eating pattern. Meals appeared to be distributed in a more standard manner throughout the day, with less reported snack times and late eating (see spreadsheet in Appendix 9, row 2). Comparison of the NEQ (2004) eating-related items showed individuals with NEB to be more likely to report consuming higher amounts of food after suppertime than their matched control ($p < 0.01$) although differences in eating start time were not quite significant ($p = 0.07$) (Table 40). Comparison between the EDE global and subscale scores of the matched NEB individuals and their matched non-NEB controls showed no significant differences overall (Table 41). Significant individual differences were seen in the items of 'eating in secret' ($p = 0.03$) and 'avoidance of exposure' ($p = 0.04$) as were noted earlier in group differences. The 'dietary rules' item was also significant ($p = 0.03$), with NEB individuals less likely to report higher dietary restraint than their matched control. NEB individuals were also significantly more likely to have a higher fear of weight gain than their matched control ($p = 0.05$). Differences between the individuals on the 'fear of losing control' item were not significant, with less numbers of the matched NEB group reporting higher scores, although only 19 matched pairs were available for this item and problems with its interpretation have previously been discussed. Control-related items on the NEQ (2004) showed significant differences between matched pairs, with the matched NEB individuals more likely to report cravings to eat before bedtime ($p < 0.01$) and poor control over eating at bedtime ($p = 0.03$).

Twenty of the matched control group considered themselves to have good control over eating and the number of positive responses alluding to this during the interview per individual and the nature of responses are listed in Table 42. Although the majority of responses about

control were positive, they were sometimes qualified with remarks suggesting it is a temporary state, subject to change.

Table 40 - Pairwise comparison of the NEQ (2004) eating-related items between the matched NEB and non-NEB matched control group using paired t-tests. A higher score on all items indicates a greater degree of dysfunction.

<i>NEQ(2004) items (eating- related)</i>	<i>Mean(SD)</i>		<i>P value</i>	<i>95% Confidence Interval of the difference (NEB-Non-NEB)</i>
	<i>Matched NEB n=28</i>	<i>Matched non- NEB n=28</i>		
Morning hunger	3.0 (1.2)	3.0 (1.2)	1.0	-0.8, 0.8
Eat for the first time	1.4 (9.1)	0.9 (1.0)	0.07	-.03, 1.0
Cravings before bedtime	3.0 (1.3)	1.1 (1.3)	<0.001	1.2, 2.6
Control before bedtime	2.3 (1.4)	1.4 (1.3)	0.03	0.2, 1.7
Intake after supper time	2.0 (0.9)	0.8 (0.8)	<0.001	1.7, 5.8

Table 41 - Comparison of the EDE v12 subscale and global scores between the matched NEB and non-NEB matched control group using the Paired Sample Sign test (n=28 unless otherwise stated)¹.

	<i>Negative ranks(n)</i>	<i>Positive ranks (n)</i>	<i>Ties (n)</i>	<i>P value</i>
EDE global score	15	13	0	0.37
<i>Restraint subscale items:</i>				
Restraint over eating	11	12	5	0.36
Avoidance of eating	11	3	14	0.15
Empty stomach ²	6	3	18	0.11
Food avoidance	8	15	5	0.18
Dietary Rules ²	6	15	6	0.03
Restraint subscale score	10	16	2	0.32
<i>Eating Concern subscale items:</i>				
Preoccupation with food	16	7	5	0.28
Fear of losing control over eating ³	6	5	6	0.29
Social eating	15	10	3	0.36
Eating in secret	16	7	5	0.03
Guilt about eating	14	8	6	0.25
Eating Concern subscale score	17	10	1	0.20

Shape Concern subscale items:

Dissatisfaction with shape	13	8	7	0.20
Preoccupation with shape or weight	11	10	7	0.70
Importance of shape	12	13	3	0.46
Fear of weight gain	13	8	7	0.05
Discomfort at seeing body ²	15	5	7	0.31
Avoidance of exposure	20	6	2	0.04
Shape Concern subscale score	17	10	1	0.26

Weight Concern subscale items:

Dissatisfaction with weight	13	10	5	0.26
Desire to lose weight	6	5	17	0.92
Reaction to prescribed weighing ⁴	6	8	9	0.34
Importance of weight	11	12	5	0.41
Weight Concern subscale score	14	14	0	0.61

¹ Negative rank numbers indicate the numbers of non-NEB matched controls scoring lower than their NEB matched individual, ² n=27 ³ n=17, ⁴ n= 23.

Table 42 - Number of references to control over eating per individual in the non-NEB matched control group

<i>No. of individual references to good control over eating</i>	<i>No. of individuals (n=28)</i>	<i>Reported comments</i>
0	2	Not in control – never tried to be (n=1) Rarely in control(n=1)
1	6	Struggle to be in control (n=1) No portion control(n=1) Control off and on(n=1) Now controlled, but previous overeating(n=1) In control(n=1)
2	6	Control for past 2 months – now slipping a bit(n=1) Never out of control (n=1) Now controlled, but previous overeating(n=1) Normal control(n=1) Regular eater(n=1) Restricts diet(n=1)
3	11	Good control (n=3) Can say no (n=1) Occasional slip(n=1) Doesn't feel controlled but restricts a lot(n=1) Controlled now but not in past (n=3) No fear of losing control(n=1) 65% in control(n=1)
4	2	In control at moment(n=1) Good control (n=1)
5	1	Struggling to finish meals (n=1)

4.8.8.4. Low Mood

Comparison of the NEQ mood-related item scores of the NEB and non-NEB matched individuals showed no significant differences, although for 10 of the individual BDI items the difference was significant and the difference in total scores was significant. One further item of ‘no interest in others’ was approaching significance (Table 43).

Table 43 - Pairwise comparison of the BDI item scores and NEQ (2004) mood-related item scores between the matched NEB and non-NEB matched control group using paired t tests. A higher total score and higher score on each item indicates a greater degree of dysfunction.

<i>BDI items</i>	<i>Mean(SD)</i>		<i>P value</i>	<i>95% Confidence Interval of the difference (NEB-Non-NEB)</i>
	<i>Matched NEB n=28</i>	<i>Matched non-NEB n=28</i>		
<i>BDI total score</i>	26.04(10.49)	17.14(10.95)	0.001	3.94, 13.84
Feel sad	1.04 (0.96)	0.57 (0.69)	0.03	0.06, 0.87
No future ¹	1.22 (1.01)	0.48 (0.75)	0.001	0.34, 1.15
Feel failure ¹	1.52 (1.22)	0.85 (0.91)	0.02	1.20,1.24
Get satisfaction	1.57 (0.96)	1.04 (0.69)	0.01	0.12, 0.95
Feel guilty	1.43 (1.14)	1.07 (1.12)	0.17	-0.16, 0.88
Feel punished ¹	0.67 (1.07)	0.52 (1.09)	0.60	-0.43, 0.73
Disappointed with self	1.46 (0.74)	1.18 (1.06)	0.15	-0.11, 0.68
Blame self	1.64 (0.87)	1.00 (0.77)	0.003	0.25, 1.04
Harm self ¹	0.56 (0.58)	0.26 (0.45)	0.03	0.03, 0.56
Crying	1.18 (1.02)	0.39 (0.57)	<0.001	0.40, 0.17
Get irritated	1.04 (0.64)	0.75 (0.75)	0.16	-0.12, 0.69
No interest in others	0.89 (0.83)	0.54 (0.79)	0.07	-0.03, 0.74
Make decisions	0.86 (0.89)	0.46 (0.64)	0.04	0.02, 0.76
Feel ugly	1.93 (0.94)	1.54 (1.17)	0.15	-0.15, 0.93
Too much effort	1.68 (0.77)	1.46 (0.84)	0.33	-0.23, 0.65
Poor appetite	0.79 (0.88)	0.29 (0.66)	0.03	0.05, 0.95
Lost weight ²	0.81 (1.06)	0.88 (1.21)	0.80	-0.68, 0.58
Health worries	1.36 (0.95)	1.00 (0.77)	0.15	-0.14, 0.86
No interest in sex ²	1.73 (1.22)	0.88 (1.07)	0.01	0.22, 1.47
<i>NEQ items (mood related)</i>				
Feeling blue	2.21 (1.42)	1.61 (1.42)	0.15	-0.23,1.45
When is mood lower?	2.07 (1.65)	1.46 (1.60)	0.25	-0.46,1.68

¹ n=27, ² n=26,

4.8.9. *Discussion*

4.8.9.1. **Development of NEB**

All participants in this study were either obese or overweight with 17 (61%) of the matched NEB group reporting being obese as children together with 13 (46%) of the matched control group. No significant differences between the presence of childhood obesity and NEB were noted and for those who developed NEB as children, obesity appeared to predate the night-eating behaviour and resulted from parental attempts to restrain day-time eating. Accounts of obesity-related teasing, bullying and discrimination were very prevalent in both groups and only one individual reported developing NEB in response to this. The matched NEB group reported somewhat more significant life events than the non-NEB matched control group 18 (64%) versus 13 (46%), although the differences between matched pairs was not significant. In this study, traumatic events such as rape, witnessing a murder and sexual abuse appeared to have a clear influence on the onset of obesity for individuals in both groups, although this did not always result in the onset of NEB for individuals in the NEB group, suggesting the development of NEB in adults may be influenced by factors other than obesity.

4.8.9.2. **NEB and Control over Eating**

There was a clear distinction between the perceived dietary control of the NEB group and the non-NEB matched control group, with the latter group appearing to have a less chaotic eating pattern, more dietary restraint and less secretive eating. This loss of control is a persistent feature of eating disorders (ED) which involve overeating, such as Binge Eating Disorder (BED) and Bulimia (BU). In BED and BU, the volume of the overeating episode is traditionally considered crucial to the definition of a 'binge' (Colles, Dixon, & O'Brien 2007; Fairburn & Beglin 1990), yet sufferers themselves associate binging with restraint and subsequent loss of control, whether the amount overeaten is objectively large or small (Telch, Pratt, & Niego 1998). Persistent restraint is a negative prognostic indicator that predicts relapse following treatment of BN and BED (Safer et al. 2002). It is possible that maladaptive cognitions caused by high restraint, for example 'one chocolate bar will make me obese' may be the trigger behind the experience of loss of control. Latner et al. (2007) argue that it is this experience which is more important as a predictor of eating-related and general distress than the amount eaten during the overeating

episode itself. Hildebrandt & Latner (2006) describe this blurring between the intensity of the experience and the actual volume eaten as 'binge drift'.

4.8.9.3. NEB, Loss of Control and Depression

Goldschmidt et al. (2008), compared 96 adolescents with either binge eating, overeating with loss of control, overeating without loss of control and a control group with normal eating and compared their weight, shape concerns and depressive symptoms. No distinction was found between the binge eating and overeating groups reporting loss of control, with both having significantly higher scores than the control groups. Colles, Dixon & O'Brien (2008b) examined 180 bariatric surgery candidates and found those reporting severe emotional disturbance due to loss of control, regardless of BED or NES diagnosis, reported higher symptoms of depression ($p < 0.001$), appearance dissatisfaction ($p = 0.009$) and poorer mental health related quality of life ($p = 0.027$).

In this study both the matched NEB and matched non-NEB groups had mean BDI scores indicative of low/high moderate levels of depression (26.0 and 17.1 respectively), although the matched NEB group had a significantly higher total score and significantly higher scores on 11 of the individual items. The matched NEB group also reported greater perceived loss of control over eating than the matched non-NEB group suggesting a possible similar pathology between the NEB participants in this study and individuals with BED and BU, independent of the amounts eaten. In Colles Dixon & O'Brien's post-operative bariatric surgery follow-up study (2008a), uncontrolled eating and grazing were associated with poor post-operative weight loss and elevated psychological distress ($p = 0.008$ and $p < 0.001$ respectively). The use of validated tools to examine in more depth the perceived lack of control over eating in these individuals would allow for further insight into this finding.

4.8.9.4. NEB and Sleep Quality

Disturbed sleep was a distinctive feature of both the NEB and the matched control group in this study, with seventeen individuals in the control group (60%) and all subjects with NEB (100%) reporting disturbed sleep. No significant difference was reported between the NEB group and matched controls for the sleep items of the NEQ (2004), although significant differences were

noted on the BDI sleep-related items, suggesting individuals who were more depressed were likely to have worse quality sleep.

Healey et al. (1981) attributes poor sleep to an 'anxious personality type', suggesting that a person predisposed to vulnerability to stress who experiences a stressful life event reacts with maladaptive coping mechanisms by internalizing their reaction, which in turn leads to emotional arousal and physical activation during sleep. The responses of 31 good sleepers and 31 poor sleepers to an unspecified sleep questionnaire and The Schedule of Recent Experience were compared. Poor sleepers had greater mean numbers of awakenings 2.1 vs 0.8 ($p < 0.001$) and 34.6% attributed the awakenings to psychological states as opposed to 14.3% of the good sleepers. Seventy four percent of the poor sleepers reported a major event related to the onset of their sleep problems and poor sleepers reported more undesirable events ($p < 0.001$) and nearly twice as many losses of significant others, on average (1.19 vs 0.52, $p < 0.01$). The poor sleepers viewed themselves less favourably than good sleepers in terms of personal characteristics, considering themselves to be 'bad' (59.3% vs 4.5%, $p < 0.01$) and 'weak' (65.2% versus 21.7%, $p < 0.01$). They also reported less frequently having a good time with their family as a child, less likely to feel good as a child and more eating problems as a child. This internalizing pattern of somatisation focused on eating and sleeping problems supports the psychological profile of poor sleepers as an anxiety-prone, obsessive-worrisome cognitive type. Yet if this theory was applicable to NEB, the NEB group would have reported more 'emotional' reasons for waking up, as opposed to the non-matched NEB group, when the opposite occurred.

Indeed Germain et al. (2004), dispute Healey's theory of internalization. They examined a sample of 368 (190 females) experiencing Post Traumatic Stress Disorder (PTSD) and looked at the effect of trauma characteristics, psychiatric co-morbidity, and patient characteristics on sleep quality. Subjects completed the Pittsburgh Sleep Quality Index and PTSD scale. Although male subjects were significantly older (45yrs versus 36yrs, $p < 0.001$), gender, age and psychiatric co-morbidity had no influence on sleep quality, with the degree of trauma alone influencing the degree of sleep quality, suggesting sleep is less affected by personality than external factors beyond the individual's control.

4.8.9.5. **NEB, Obesity and Co-morbidity**

With a wide range of physical causes for waking and poor sleep quality appearing so prevalent across both groups which were also characterized by obesity, it is difficult to determine the role of NEB in this relationship. Evidence of the relationship between poor sleep quality, obesity and obesity-related co-morbidities is growing, although relationships are often complex making it difficult to determine cause and effect. For example, poor sleep is associated with the metabolic syndrome which has a known relationship between Obstructive Sleep Apnoea and obesity (Coughlin et al. 2004). In a sample of 210 university volunteers (age 45.8 (6.0), 57% male) 30% were identified as poor sleepers and 20% as having the metabolic syndrome (Jennings et al. 2007). After adjusting for the independent effects of sex and age on the metabolic syndrome, logistic regression analysis using the PSQI mean global score only (4.6 (2.6)) showed that for every increase of 2.6 points on the PSQI, an individual was 1.4 times more likely to meet the criteria for metabolic syndrome ($p=0.02$). Unfortunately degree of obesity and co-morbidity were not reported in Jennings' study, making inference about causal relationships difficult.

4.8.9.6. **Limitations of the Matched Control Study**

Qualitative analysis of the NEB group and comparison with a matched non-NEB group suggests that NEB covers a broad spectrum of night-eating behaviours and develops at various stages in life. Its strongest associations appear to be between depression and perceived lack of control over eating, with the links to childhood obesity, significant life events and poor sleep quality appearing weaker in the general context of adult obesity. However, several limitations must be accounted for when interpreting these qualitative results. As a decision was taken to use individuals from the original study as matched controls, not all NEB individuals were matched successfully on all criteria, with age being the most difficult. As this was essentially exploratory analysis with data examined retrospectively, themes such as childhood obesity and trauma which appeared relevant after analysis of the NEB group interviews could not be developed and their characteristics explored further.

In-depth interview examination of traumatic life events requires trained psychology personnel in order to ensure participants are managed safely and this was beyond the remit of the skills of the researchers conducting this study. The high number of sleep problems identified in

both groups with varied reasons for waking up was surprising and was not fully anticipated when the original study was designed as little was known then about the relationship between obesity and sleep quality. Sleep does not feature strongly in the NESHI or the NEQ (2004), which were the main tools utilised to identify NEB, thus most of the sleep findings are based on qualitative accounts. It could also be argued that sleep problems have been overrepresented in both groups as some of the controls had poor sleep and came forward for this reason and the relationship between poor sleep, NEB and OSA is still unclear. Whilst the investigation to date has shed light on the characteristics of NEB, questions still remain as to its true prevalence in an obese UK population. Prevalence data from the initial study was based on a purposive sample with individuals encouraged to come forward if they had NES symptoms in order to develop a screening tool. It is possible therefore that the extent of NEB characteristics in this population has been under or overestimated. It was decided therefore to use validated tools to explore the relationship between OSA, sleep quality and NES in a more systematic way in order to answer the following questions:

4.8.10. *Unanswered Questions*

- What is the true prevalence of NEB in an obese population?
- How common a problem is poor sleep quality in the obese population?
- What is the role of obesity related co-morbidity, such as OSA, in the relationship between NEB and poor sleep?

5. PREVALENCE STUDY

5.1. *Rationale*

Since initiating the original identification study, Stunkard's NEQ(2004) (Colles, Dixon, & O'Brien 2007; Marshall et al. 2004) has undergone successful validation and reliability studies, allowing for prevalence data on night-eating to be collected quantitatively. Quantitative tools also exist to measure sleep quality and to screen for suspected Obstructive Sleep Apnoea. It was proposed therefore to invite consecutive patients attending the WMC to complete tools which screen for night-eating and OSA and measure sleep quality simultaneously. As well as collecting prevalence data, correlational analysis could then be performed to explore further the links between night-eating, sleep quality and obesity related co-morbidity in an obese population.

5.2. *Methodology*

5.2.3. *Screening for Obstructive Sleep Apnoea: The Epworth Sleepiness Scale*

Obstructive Sleep Apnoea requires a sleep study for diagnosis. This could involve full polysomnography (the gold standard) or a limited sleep study involving overnight oximetry (measures of respiratory effort or nasal airflow). However, daytime sleepiness is the main presenting feature of Obstructive Sleep Apnoea and The Epworth Sleepiness Scale (ESS) developed by Johns (1992) is considered a simple method for measuring the general level of daytime sleepiness or sleep propensity in adults. It has been used in more than 1500 publications and was developed using a different conceptual framework as a quicker alternative to other tools such as the Multiple Sleep Latency Test (MSLT) which looks at day-to-day changes and early onset of REM sleep which is needed to diagnose narcolepsy. The MSLT and the similar Maintenance of Wakefulness Test (MWT) are time consuming to deliver, requiring the placement of electrodes and for the subject to lie down whilst the speed of sleep onset is measured at 4 time points in a day.

The ESS is a one-off self administration tool, with individuals indicating retrospectively, on a scale of 0-3, the chance of dozing in 8 specific circumstances commonly met in daily life. The situations were chosen on a priori grounds to differ in their soporific nature (i.e ability to induce dozing). A score of 0 indicates no chance of dozing and 3 indicates a high chance

(subjects receive a total score ranging from 0-24). When tested on 104 students, item analysis had a Cronbach's alpha of 0.73 (Nguyen et al. 2006). Testing on a combined group of 54 subjects with OSA and 96 with other sleep disorders revealed a Cronbach's alpha of 0.88. Whilst both would be considered acceptable, the disparity may be as a result of non-normal distribution of some items or differences in the range of scores, with sleep disorder subject scores ranging from 0-24 vs 0-18 for the students.

Factor analysis in the same study identified an Eigenvalue of 3.95 for sleep disorder subjects and 2.07 for students. Normalised factor loadings ranged from 0.49 to 0.76 in all items for both groups apart from items 6 (0.25) and 8 (0.37) for students. As so few students dozed in these situations, very little variance was associated with those item scores. Johns concludes that the presence of one factor alone supports the use of a single total score, although the correlation co-efficient which measures strength of relationship and not agreement was used.

Nguyen et al.(2006), examined the clinical reproducibility of the tool in 142 individuals referred to a sleep clinic (76% male, mean age 44 (11)). Whilst they confirmed the 'sleepiness' factor, with an Eigenvalue of 6.2, 2 other factors, albeit minor and not clearly discernible from the main factor were also identified, which may be boredom or inattentiveness. Smith et al.(2008) performed confirmatory factor analysis on the ESS scores of 759 Australian subjects with OSA and concluded that Johns' original single factor structure did not fit the data. A re-specified single factor solution did and this improved fit was confirmed with a second confirmatory factor analysis.

Normative values were identified by Parkes et al.(1998). In a sample of 188 UK control subjects with normal sleep-wake habits, the mean score was 4.5 (3.3) with a range between 0-11. This concurs with Johns' mean score of 4.6 (2.8) and range of 0-10 for 72 Australian corporation workers (Johns & Hocking 1997). These scores are lower than those of 104 medical students (55 males, mean age 20.9 (2.8)) (Nguyen et al. 2006). Baseline mean was 7.6 (3.9) with a similar level of 7.6 (3.9) on 87 who retested 5 months later. The Pearson Correlation Coefficient between the pairs was 0.82 ($p < 0.001$). Although the medical student population was considered 'normal', student lifestyles often involve chaotic sleep patterns which may have accounted for the higher mean scores.

5.2.3.1. *Sensitivity and Specificity*

Despite the difference in normative values between studies, Johns found acceptable levels of sensitivity and specificity when he compared the performance of the ESS with the MSLT and MWT on 530 individuals, ages ranging from 17-68 suffering from narcolepsy (Johns 2000). Previously published data from several investigations were used to calculate the reference range of normal values for each test, defined by the mean ($\pm 2SD$) or by the 2.5 or 97.5 centiles. With a cut-off score > 10 the sensitivity of the ESS was 93.5% and specificity 100%. Above 10 is now generally accepted as an appropriate cut point for suspecting OSA. Sensitivity and specificity for the MSLT based on the standard cut-off of < 5 minutes was 80.9% and 89.8% respectively. The MWT had a higher sensitivity of 84.3% at the standard cut-off of < 12 minutes and higher specificity at 98.4%. Caution must be taken in interpreting these data as study individuals had narcolepsy and not OSA. The relatively low correlation between the MSLT scores and the ESS score ($r = -0.27$) suggests that these tools may not measure the same construct or be suitable for screening for OSA.

5.2.3.2. *Reproducibility and Reliability*

Ngygen et al. (2006) compared the scores of 142 patients (76% men, mean age 44 (11)) who were pre-screened for OSA in the community and their repeated scores in a specialist clinic (mean time difference of 71 (92) days). Both mean scores were remarkably similar (11.1(5.2) vs 11.2 (5.3), although Bland-Altman plot analyses demonstrated a wide scatter of values for ESS score differences, with a range of -8 to 8. Forty one percent of the study group had an individual score difference of 3 or more, and 23% of 5 or more, which is higher than in other studies which have examined non OSA subjects. The greatest variability was for question 7, which related to sitting quiet after lunch and the least variability was seen in questions 6 and 8, which relate to sitting and talking and sitting in a car. It is difficult to assess whether variability generally was due to lack of stability of the condition, measurement error, or poor reproducibility of the tool. Scoring positive on items 6 and 8, given their social unacceptability, is likely to reflect severe OSA, which is less likely to change in the time between assessments. It is possible that low variability may be normal in 'normative' studies or that larger group numbers may show greater

variability. Also, subjects in other studies may have known the reasons for testing and tried to match previous replies.

In a separate study comparing score differences of 56 individuals over a longer time period of 7 months, 18% changed OSA 'status' whilst waiting for treatment. Four individuals changed scores from above 10 to below 10 and 6 vice versa (Kaminska et al. 2010). Those improving their scores may have done so as a result of changes in their condition, which could be influenced by external factors such as weight loss and not be a reflection of the reliability of the test. Given that the majority of those changing scores, showed an apparent worsening of the condition, which would be anticipated given the long wait for treatment, it does suggest the tool has reasonable reproducibility.

The strongest evidence for the use of the ESS by individuals with OSA comes from studies assessing sensitivity to change, which consistently show reductions in scores when standard treatments for OSA are applied. Despite earlier results, when Ngygen himself compared the scores of 54 OSA patients at baseline and post-treatment (minimum of 3 months) he found a reduction in mean scores from 14.3 (3.6) to 7.4 (4.1) ($p < 0.001$) (Nguyen et al. 2006). Hardinge, Pitson & Stradling (1995) compared the baseline ESS scores of 50 individuals with OSA and after 2 months treatment with Continuous Positive Airway Pressure (CPAP). Subjects were 94% male, with a mean age of 50.2 years and mean weight of 110 kg. Mean scores reduced from 16.4 (3.7) to 7.0 (4.0) ($p = 0.0001$).

A second group of 25 individuals, 100% male, with a mean age of 50.9 years and weight of 96 kg saw a significant reduction in mean score from 15.2 (5.6) to 6.0 (3.6) after 1 year of treatment ($p = 0.0001$). Compliance testing in 12 of the subjects tested after 1 year was good, suggesting score reductions were due to the effectiveness of treatment. Similar results were found by Kaminska et al. (2010) who saw a reduction in mean scores from 12.4 (6.8) to 7.6 (5.0) ($p = 0.0001$) in 68 individuals treated for a median of 40 months.

5.2.3.3. Limitations

Despite its widespread use as a screening tool for OSA it is important to be aware of the limitations of the ESS. It does not distinguish between simple snoring and OSA as some snorers have Excessive Daytime Sleepiness with no known cause (Osman et al. 1999). Similarly, in a

random sample of 587 individuals without insomnia and 116 with insomnia in a community population, ROC analysis showed the ESS to differentiate poorly between the two groups (Sanford et al. 2006). Scores in the insomnia group were higher, but not necessarily predictive of insomnia. The tool is also not helpful in identifying mental disorders in sleep clinic patients (DeZee et al. 2006).

5.2.4. *Measuring Sleep Quality: The Pittsburgh Sleep Quality Index*

The Pittsburgh Sleep Quality Index (PSQI) was developed by Buysse (1988) with the stated aims of providing a reliable, valid and standardized measure of sleep quality, which could discriminate between good and poor sleepers. In addition, the index should be easy to interpret and provide a brief, clinically useful assessment of a variety of sleep disturbances that might affect quality. It has been used in 900 publications and assesses self-rated activity over the previous month in order to identify patterns of dysfunction. Nineteen items are grouped into 7 component scores and weighted equally on a 0-3 scale. Components are a combination of qualitative and quantitative items, empirical and clinical in origin rather than statistical and comprise: Subjective Sleep Quality, Latency, Duration, Habitual Sleep Efficiency, Sleep Disturbances, Use of Sleep Medication and Daytime Dysfunction. Component scores are summed to provide a global score ranging from 0-21. A high score indicates poor sleep quality.

5.2.4.1. *Factor Analysis*

Buysse concedes that the tool is lacking in factor structure but goes some way to addressing this in a study examining the PSQI scores of 52 'good' sleepers (mean age 59.9, males 40), 34 'poor' sleepers with major depressive disorder (mean age 50.9, males 25) and 62 'poor' sleepers with the disorders of initiating and maintaining sleep (DIMS) or disorders of excessive somnolence (DOES) (mean age 42.2 years, males 24) (Buysse et al. 1988). The overall group mean score was 7.4 (5.1). The component scores revealed a Cronbach's alpha of 0.83 suggesting they were all measuring the same construct. The largest component-total coefficients were for 'Habitual Sleep Efficiency' and 'Subjective Sleep Quality' (0.76 for each). The lowest, at 0.35 was for 'Sleep Disturbances' and may be due to the high number of items which make up this component and which may be susceptible to individual variation over time.

Cole et al. (2006) performed factor analysis on the scores of 67 elderly individuals (mean age 68.9, 55% female) who were currently depressed, 143 individuals in remission and 207 with no mental illness. The sample was split randomly, with exploratory factor analysis performed on one half and confirmatory factor analysis on the second half. The author concluded the best fit was found with the 2 factors identified by Buysse, but with a third factor of 'Daily Disturbances'. The relationship between each component score to its respective factor in this 3 factor model was significant and large, ranging from the standardized path coefficients of 0.43 (Sleeping Medication Use to Perceived Sleep Quality) to 0.91 (Habitual Sleep Efficiency to Sleep Efficiency). Correlations between the factors ranged from 0.42 (medium large effect) to 0.82 (very large effect). Of course this may not hold true in younger subjects with different clinical presentations. Smyth (2008) reports on the usefulness of the tool in evaluating sleep quality in older adults, but provides no data to support this.

5.2.4.2. *Validity, Sensitivity and Specificity*

The PSQI appeared to distinguish well between the different groups in Buysse's study. Using ANCOVA with age and sex as covariates, the adjusted mean score of the control group was 2.7 (1.7), the depressed group 11.1 (4.3), the DIMS 10.4 (4.6) and the DOES 6.5 (3.0). A cut-off score of 5 gave a sensitivity of 89.6% and specificity of 86.5%, with a group-wide kappa of 0.75 ($p < 0.001$). Sensitivity for individual groups, although high, was different with a sensitivity of 97% for the depressed group, 84.4% for the DIMS and 88% for the DOES. It was concluded that a PSQI score above 5 indicates serious problems in at least 2 component areas or moderate problems in more than 3. A later study by Backhaus et al. (2002) confirmed these sensitivity and specificity findings with a score above 5 indicating sleep disturbances in 80 insomnia subjects and 45 healthy subjects with a sensitivity of 98.7% and specificity of 84.4%. A score above 5 is now generally accepted as indicating poor quality sleep.

5.2.4.3. *Test-retest Reliability*

Ninety one of Buysse's 148 sample completed the PSQI twice, with no significant difference in global and component scores (Buysse et al. 2008). Scores were similar for each diagnostic group. The Pearson Product Moment Correlation Coefficient for global scores was 0.85, with individual component scores ranging from 0.65 to 0.85. Correlations for the controls

were generally lowest and highest for the DIMS, with both groups highest on the quantitative components measuring sleep duration and sleep latency, rather than the qualitative components which require a subjective assessment about sleep-related difficulties.

5.2.4.4. *Stability*

The stability of both the PSQI and the ESS were confirmed by Knutson et al. (2006a) when large numbers of black and white early middle-aged individuals completed both tools in the Cardia study. Six hundred and ten individuals completed the PSQI and 609 the ESS at baseline and one year later. No significant difference was found between sample means (PSQI 5.7 (3.1) vs 5.9 (3.1) and ESS 7.4 (4.3), vs 7.2 (4.2)). For both tools, a high within-subject reliability was noted with intra-class correlation coefficients above 0.80 for both tools. Backhaus et al. (2002) performed short term test-retest reliability studies on 80 individuals with primary insomnia. The test-retest interval ranged between 2 days and a few weeks. The overall PSQI global score Correlation Coefficient was good at 0.87. Validity testing using sleep logs and polysomnography in the same sample, show high correlations with sleep logs, but lower correlations with polysomnography data.

The PSQI appears useful in measuring sleep quality in a variety of populations. Carpenter & Andrykowski (1998) tested its use in chronic illness in a group of 155 bone marrow transplant patients, 56 renal transplant patients, 102 women with breast cancer and 159 with benign breast problems. Cronbach's alphas were 0.80 across groups and correlations between global and component scores were moderately high. Algul et al. (2009) used the tool with 124 obese individuals (32 with BMI 30-34.9 kg/m² (Class 1 obesity), and 92 with a BMI \geq 35 kg/m² (class 11 obesity)) and 106 healthy control subjects. The class 11 obesity group had a significantly worse PSQI global score than the control group (7.3 (4.1) vs 4.9 (3.6) $p < 0.001$)

5.2.5. *Screening for NES: The Night Eating Questionnaire (2008)*

Stunkard's original NESQ was conceived as a 4 point Likert scale with 9 items, including visual analogue scales. This was developed into a 14 item NEQ (2004) with a 5 point Likert scale and no visual analogue scales. Validation studies published in 2008 have confirmed the 2004 version of the NEQ as an acceptable measure of severity of NES with one important revision. Item 13 which explores awareness whilst night-eating is now excluded from scoring giving a

possible score range from 0-52. The rationale for excluding this item is based on the assumption that individuals without awareness are not suffering from NES, but a sleep-related disorder. For the purposes of the prevalence study, in order to distinguish between the study screening tool and the unvalidated NEQ (2004) used in the identification study, the validated NEQ will henceforth be described as the NEQ (2008).

Evidence for use of the NEQ (2008) was evaluated together from three separate NES studies (Allison et al. 2008b; Backhaus et al. 2002). Study 1 examined factor structure and internal consistency and included 1980 persons with self-diagnosed NES who completed the NEQ (2004) on the Internet. The mean score was 33.1 (7.5). Principal components analysis was used to generate four factors and a total Cronbach's alpha of 0.70. Yet only one individual factor had an acceptable alpha above 0.7: (nocturnal ingestions 0.94). Other alphas were evening hyperphagia 0.65 and morning anorexia 0.57. Factors 4 and 5 (mood and sleep) were combined into a single construct, with even then a low alpha of 0.30. The rationale for combining these two factors is unclear and it is hardly surprising that 2 factors measuring different constructs generate a poor alpha when combined together.

The second study in 81 outpatients diagnosed with NES found acceptable convergent validity of the NEQ (2004) with additional measures of night eating, disordered eating, sleep, mood, and stress. The mean score was 32.4 (6.8) and significantly higher in normal weight individuals as opposed to obese. The third study compared scores from obese bariatric surgery candidates with and without NES and found appropriate discriminant validity of the NEQ (2004). Of 184 individuals, 10.3% (19) were identified with NES. Mean scores were NES 26.2 (8.1) vs non-NES 16.0 (6.3). The positive predictive value (PPV) of the NEQ (2004) at a score of 25 or higher was low (40.7%), increasing to 72.7% at a score of 30 or greater. The negative predictive value (NPV) was high for cut scores of both 25 and 30 (95.2% and 94.0% respectively).

Reviewing the evidence as a whole, the authors conclude that the NEQ (2008) appears to be an efficient, valid measure of severity for NES. As previously discussed, a clear relationship exists between mood and sleep, although combining both concepts into a single construct does not appear logical. Presenting data from different NES studies together, given the diverse NES criteria on which studies are based is also questionable. NES criteria on which the first 2 studies

are based are not reported and the third study did not use the 2003 criteria. Despite these limitations, it is at present the only tool available to systematically identify potential NES sufferers although it falls far short of being a diagnostic tool. Its use by other researchers is becoming widespread. Other researchers have found similar cut points useful. Lundgren et al. (2006) examined scores in 399 psychiatric patients and found a PPV of 52% using a cut point of 25 and 68% with a cut point of 30. Re-examination of the data when item 13 was removed from the total score gave an increased PPV of 62% and 77%, respectively. Although Stunkard recommends excluding the awareness item when scoring the NEQ (2008), results from the identification and characterisation studies suggest considerable variability in the reporting of awareness of night eating by individuals with NEB. For this reason, the NEQ (2008) was delivered in this prevalence study with the item included, although the individual item score was excluded from the total score calculation in keeping with scoring guidelines.

5.3. *Ethical Approval*

Ethical approval to conduct the prevalence study was obtained from St Helen's and Knowsley Ethics Committee (ref. no. 04/Q1508/9) on 30th July 2009. Approval from the UCLan Research Ethics Committee (ref. no. CA 142) was obtained on 4th September 2009. Management approval ref. no. 04DE006 was obtained from the Hospital R&D committee on 23rd September 2009.

5.4. *Data Collection*

Immediately prior to the period of data collection for the prevalence study, the WMC expanded from one clinic a week to 6 clinics per week, with a mixture of approximately 12-14 new and follow-up patients per clinic. An anonymised screening tool (screening tool v 1.3 24/05/09) was devised which included the NEQ (2008), ESS and PSQ as well as height, weight, age and gender in order to include demographic characteristics in the analysis (Appendix 10). A patient information sheet (PIS) (version 1.4 24/05/09) was posted by JC to all WMC clinic attendees a week before their clinic appointment to explain the purpose of the study. The screening tool and the PIS were then handed to consecutive attendees on arrival at clinic by the clinic nurse. Permission was obtained from the nurse manager of the Hospital Outpatient Department and Professor W, the consultant in charge of the WMC. JC and another obesity

specialist nurse colleague placed copies in the clinic notes prior to the start of clinic to reduce disruption to staff. Participants were invited to either complete the tools whilst waiting for their appointment and return them to the clinic nurse, or return them by post in a pre-paid envelope. The majority were completed in clinic although a small number (approximately 5%) were returned by post.

As this was an anonymised process it was not possible to collect information on the number on patients who declined to participate, although anecdotally, clinic staff noted very few patients who declined. The initial collection period was planned for 1 month, but due to staff holidays and clinic cancellations, data were collected over a 5 week period and stopped once a total of 103 completed tools were returned. A further 31 tools were returned with incomplete data: either height, weight and gender (data collected on a separate page) were inadvertently omitted or non-specific sleep and wake times were given, rendering the PSQI data invalid.

5.5. *Statistical Analysis*

Analysis was based on 103 individuals for whom a complete data set exists (weight was not available for 2 individuals). SPSS (version 14) and Inter Cooled Stata (version 9) were used to perform data analysis. Data was generally presented as means (SD). Independent samples t tests were used to perform between-groups analyses of means on interval data and Mann Whitney U tests on ordinal data. Differences in gender were assessed using the binomial test. Frequencies (%), with chi-square analysis, were calculated for the proportions of individuals above and below screening tool cut points (ESS > 10, PSQI >5, NEQ \geq 25 (first cut point) NEQ \geq 30 (second cut point)). Fisher's exact test and bootstrapping were used on small sample sizes either approaching significance or just reaching significance (p-values between 0.01 and 0.1). Pearson Correlation Coefficients were calculated to examine potential linearity of relationships between individual screening tool scores.

5.6. *Results*

5.6.1. *Comparison between Gender*

The study group had a significantly higher representation of females than males (74 (72%) females vs 29 (28%) males, $p < 0.001$) (Table 44). Ages ranged from 17yrs to 69yrs with an average age of 44.4 yrs. Males were slightly younger (males mean 43 yrs: females 44.9yrs,

p=0.46). Body weight ranged from 82 kg to 219 kg and BMI ranged from 30.3 kg/m² to 76.5 kg/m². Both were significantly higher in males (mean weight 151.8 kg males vs 122.3 kg females, p= <0.001: mean BMI 50.1 kg/m² males vs BMI 45.7 kg/m² females, p= 0.04). The mean PSQI score was 9.1 (4.5) with males scoring very slightly higher (males 9.5 (4.7): females 9.0 (4.4), p= 0.63). The mean ESS score was 8.1 (5.6) with males scoring higher although the difference was not significant: (males 9.6 (6.5): females 7.5 (5.1), p= 0.12). The mean NEQ (2008) score was 16.4 (17.3) with a similar pattern of difference between males and females noted (males 17.6 (7.4): females 15.9 (7.3), p= 0.29). No significant differences between male and female individual item scores were seen on the PSQI, ESS or NEQ (2008) (data not shown).

Table 44 - Comparison of the characteristics and screening tool total scores between males and females of the whole study group. Differences reported as mean (SD) and using independent samples t tests unless otherwise stated (¶ Binomial test).

<i>Characteristic</i>	<i>Mean (SD)</i>			<i>P value</i>	<i>95% Confidence Interval of the difference (male-female)</i>
	<i>Total (n= 103)</i>	<i>Male (n=29)</i>	<i>Female (n= 74)</i>		
<i>Age (yrs)</i>	44.4 (11.9)	43.0 (12.9)	44.9 (11.5)	0.46	-7.1, 3.2
<i>Gender (%)¶</i>	-	28	72	<0.001	20%,40%
<i>BMI (kg/m²) (n=101)</i>	47.0 (9.8)	50.1 (9.5)	45.7 (9.7)	0.04	0.2, 8.6
<i>Wt (kg) (n=101)</i>	130.7 (29.9)	151.8 (27.9)	122.3 (26.4)	<0.001	17.8, 41.2
<i>PSQI total score</i>	9.1 (4.5)	9.5 (4.7)	9.0 (4.4)	0.63	-1.5,2.4
<i>ESS total score</i>	8.1 (5.6)	9.6 (6.5)	7.5 (5.1)	0.12	-.6, 4.8
<i>NEQ total score</i>	16.4 (7.3)	17.6 (7.4)	15.9 (7.3)	0.29	-1.5. 4.9

5.6.2. *The Night Eating Questionnaire (NEQ 2008)*

Individual item score means ranged from 0.3 (0.7) to 2.9 (1.2) (Table 45). Lowest mean scores were related to behaviour whilst eating at night and highest scores were seen in items reflecting low mood and morning anorexia. Mean scores from the NEQ (2004) administered verbally in the earlier identification study are also presented in Table 45.

Similar trends are seen between the two groups of scores, with high levels of morning anorexia and lower scores for items related to eating at night. The mean score in this prevalence study was 16.4 (7.3), excluding item 13 which is not counted in the scoring, with 15 individuals (14.6%) scoring ≥ 25 (first cut off) and 4 (3.9%) scoring ≥ 30 (second cut off). Whilst the verbally administered NEQ (2004) in the identification study had a similar mean total score of 18.9 (9.8) (excluding item 13), much larger numbers had scores above the two cut points, reflecting the divergent nature of the sample which included individuals very likely to have NES and individuals very unlikely to have it.

Table 45 – ¹ Mean (SD) NEQ (2008) individual item scores, mean (SD) total score (excluding item 13) and percent scoring ≥ 25 and ≥ 30 of the whole prevalence study group. ² Mean (SD) scores from the verbally administered NEQ (2004) used in the Identification study.

<i>Item number</i>	<i>NEQ item</i>	<i>Prevalence study Item score Mean (SD) (n=103)¹</i>	<i>Identification study Item score Mean (SD) (n=81)²</i>
1	Morning hunger	2.7 (1.2)	3.0 (1.1)
2	Eat for the first time	0.8 (0.8)	1.1 (0.9)
3	Cravings before bedtime	1.8 (1.5)	2.1 (1.6)
4	Control before bedtime	1.7 (1.3)	1.8 (1.4)
5	Intake after supertime	0.8 (0.8)	1.2 (1.0)
6	Feeling blue	2.1 (1.4)	1.9 (1.4)
7	When feel blue?	2.9 (1.2)	1.8 (1.6)
8	Trouble getting to sleep	1.7 (1.2)	2.0 (1.6)
9	Up at night	1.6 (1.5)	1.4 (1.7)
10	Cravings at night	0.6 (1.0)	1.0 (1.5)
11	Need to eat to sleep?	0.3 (0.7)	0.7 (1.4)
12	How often snack?	0.4 (0.7)	0.8 (1.4)
13	How aware when eating at night?	2.1 (1.7)	1.1 (1.7)
14	Control at night	1.5 (1.4)	0.8 (1.4)
Total score (excluding item 13)		16.4 (7.3)	18.9 (9.8)
Number with NEQ score ≥ 25 (%)		15 (14.6%)	23 (28.4%)
Number with NEQ score ≥ 30 (%)		4 (3.9%)	15 (18.5%)

5.6.3. *The Epworth Sleepiness Scale*

Individual mean item scores ranged from 0.3 (0.7) to 1.7 (1.0) with individuals most likely to doze whilst watching television and least likely to doze in a car whilst stopped in traffic

(Table 46). Although the mean total score was 8.1 (5.6), 32% (n=33) scored above 10, indicating suspected OSA.

Table 46 - Mean Epworth Sleepiness Scale individual item scores, mean (SD) total score and percent scoring above 10 for the whole study group.

ESS item no.	ESS item	Mean (SD) item score (n=103)
	How likely are you to doze off...	
1	sitting and reading?	1.3 (1.1)
2	watching TV?	1.7 (1.0)
3	sitting inactive in a public place (eg theatre, meeting)	0.7 (1.0)
4	as a passenger in a car?	1.0 (1.1)
5	lying down to rest in the afternoon?	1.9 (1.0)
6	sitting and talking to someone?	0.3 (0.7)
7	sitting after lunch?	1.0 (1.1)
8	in a car while stopped in traffic?	0.3 (0.7)
	Total score	8.1 (5.6)
	Number with ESS score > 10 (%)	33 (32 %)

5.6.4. *The Pittsburgh Sleep Quality Index*

Individual component mean scores ranged from 0.4 (0.9) to 1.7 (0.6), although only one item, ‘use of medication’ scored less than 1, on average (Table 47). Highest scores were noted for the ‘sleep latency’ and ‘sleep disturbances’ items. The mean score of 9.1(4.5) was much higher than the cut-off of 5, which indicates poor quality sleep, with a large number of individuals (76 (74%)) scoring above the cut-off.

Table 47 - Mean Pittsburgh Sleep Quality Index individual component scores, mean (SD) total score and percent scoring above 5 for the whole study group.

<i>Components (C) of the Pittsburgh Sleep Quality Index</i>	<i>Component score mean (SD) (n=103)</i>
C1 subjective sleep quality	1.5 (0.9)
C2 sleep latency	1.6 (1.1)
C3 sleep duration	1.3 (1.1)
C4 habitual sleep efficiency	1.2 (1.2)
C5 sleep disturbances	1.7 (0.6)
C6 use of sleeping medication	0.4 (0.9)
C7 daytime dysfunction	1.5 (0.9)
Total score	9.1 (4.5)
Number (%) with PSQI score > 5	76 (74%)

5.6.5. Comparison of Demographic Data according to Cut Points

A higher proportion of males were noted in the suspected OSA group as opposed to the non-OSA group (12 (36.4%) vs 17 (24.3%)) although the differences were not significant (p= 0.2) (Table 48). Differences in weight were significant (suspected OSA 140.8 (33.9 kg) vs non-OSA 125.9 (26.6) kg, p= 0.02) and differences in BMI were approaching significance (suspected OSA 50.8 (10.3) kg/m²: non-OSA 43.1 (8.0) kg/m², p= 0.09). Differences in age were not significant.

Table 48 - Comparison of characteristics between participants with suspected OSA and those without suspected OSA. Differences reported as means (SD) and using independent samples t tests. (¶ Chi square analysis)

<i>Characteristic</i>	<i>Suspected presence of OSA based on an ESS score >10 Mean (SD)</i>		<i>P value</i>	<i>95% Confidence Interval of the difference (OSA-non OSA)</i>
	<i>OSA n=33</i>	<i>Non OSA n=70</i>		
<i>Age (yrs)</i>	43.6 (9.6)	44.7 (12.8)	0.68	-3.9,6.0
<i>BMI (kg/m²)</i>	50.8 (10.3)	43.1 (8.0)	0.09	-1.5, 16.8
<i>Weight(kg)</i>	140.8(33.9)	125.9 (26.6)	0.02	-27.2,-2.6
<i>Number (%) of males ¶</i>	12 (36.4)	17 (24.3)	0.20	-7%, 31%

A similar proportion of males were noted in the poor sleep quality (PSQ) group as opposed to the good sleep quality (GSQ) group (21 (27.6 %) vs 8 (29.6%) p= 0.84) (Table 49). Differences in weight (PSQ 130.5 (30.6)kg: GSQ 131.5 (28.4) kg, BMI (PSQ 47.2 (10.1) kg/m²: GSQ 46.5 (9.3) kg/m² and age (PSQ 44.1 (11.8) years: GSQ 44.9 (12.4)years, were not significant.

Table 49 - Comparison of characteristics between participants with poor quality sleep and those with good quality sleep. Differences reported as means (SD) and using independent samples t tests. (¶ Chi square analysis)

<i>Characteristic</i>	<i>Poor sleep quality based on a PSQI score > 5 Mean (SD)</i>		<i>P value</i>	<i>95% Confidence Interval of the difference (Poor sleep – good sleep)</i>
	<i>Poor sleep quality n=76</i>	<i>Good sleep quality n=27</i>		
<i>Age (yrs)</i>	44.1 (11.7)	44.9 (12.4)	0.77	-4.5, 6.1
<i>Weight(kg)</i>	130.5 (30.6)	131.5 (28.4)	0.88	-12.5, 14.6
<i>BMI (kg/m²)</i>	47.2 (10.1)	46.5 (9.3)	0.77	-5.1, 3.8
<i>Number (%) of males ¶</i>	21 (27.6)	8 (29.6)	0.84	-20%, 18%

Based on a cut-off score of ≥ 25 on the NEQ (2008), males were over represented in the suspected NES group as opposed to the non-NES group (6 (40%) vs 23 (26.1%)) although this was not significant ($p= 0.27$) (Table 50). At a cut-off of ≥ 30 the reverse was noted, (1 (25%) vs 28 (28.3%)), although again this was not significant (Table 51). At both cut points, individuals with suspected NES were younger, with the difference more pronounced in the higher cut point group (suspected NES 32.3yrs (10.5) vs non NES 44.8yrs (11.7), $p= 0.04$). A higher mean BMI and weight was noted in the suspected NES group at the lower cut point, with weight approaching significance (suspected NES 142.8kg (30.1) vs non NES 128.6 kg (29.5), $p= 0.09$). Whilst weight remained higher in the suspected NES group at the higher cut point, BMI was lower, although neither difference was significant.

Table 50 - Comparison of characteristics between participants with suspected NES and those without suspected NES at a cut point of > 25 on the NEQ. Differences reported as means (SD) and using independent samples t tests. (¶ Chi square analysis).

<i>Characteristic</i>	<i>Suspected presence of NES based on an NEQ score ≥ 25</i>		<i>P value</i>	<i>95% Confidence Interval of the difference (NES-non-NES)</i>
	<i>Mean (SD)</i>			
	<i>NES n=15</i>	<i>Non-NES n=88</i>		
<i>Age (yrs)</i>	40.1 (10.5)	45.1 (12.0)	0.13	-1.5 ,11.6
<i>Weight(kg)</i>	142.8 (30.1)	128.6 (29.5)	0.09	-30.6, 2.2
<i>BMI (kg/m²)</i>	48.8 (7.7)	46.7 (10.2)	0.45	-7.6, 3.4
<i>Number (%) of males ¶</i>	6 (40.0)	23 (26.1)	0.27	-0.1, 0.4

Table 51 - Comparison of characteristics between participants with suspected NES and those without suspected NES at a cut point of > 30 on the NEQ. Differences reported as means (SD) and using independent samples t tests (¶ Chi square analysis).

<i>Characteristic</i>	<i>Suspected presence of NES based on a NEQ score ≥ 30</i>		<i>P value</i>	<i>95% Confidence Interval of the difference (NES-non NES)</i>
	<i>Mean (SD)</i>			
	<i>NES n=4</i>	<i>Non NES n=99</i>		
<i>Age (yrs)</i>	32.3 (10.5)	44.8 (11.7)	0.04	-24.4 , -0.8
<i>Weight(kg)</i>	138.7 (30.9)	130.4 (30.0)	0.59	-22.1, 38.6
<i>BMI (kg/m²)</i>	45.8 (9.1)	47.0 (9.9)	0.81	-11.1, 8.8
<i>Number (%) of males ¶</i>	1 (25)	28 (28.3)	0.87	-0.5, 0.4

5.6.6. Correlations between Tools

Pearson's correlation between the PSQI total scores and NEQ (2008) total scores was quite strong ($0.55, p < 0.001$) (Figure 11). The correlation remained strong even when controlling for ESS ($0.51, p < 0.001$). Comparison of the proportion of participants with a PSQI > 5 and NEQ ≥ 25 was not significant with 13 positive for both ($p = 0.34$) (Table 52). A similar trend was seen with the PSQI and NEQ scores ≥ 30 although 13 of the 15 individuals with suspected NES at the 25 and above cut point had a PSQI > 5 and all 4 individuals with suspected NES at the higher cut point had a PSQI > 5 (Table 53).

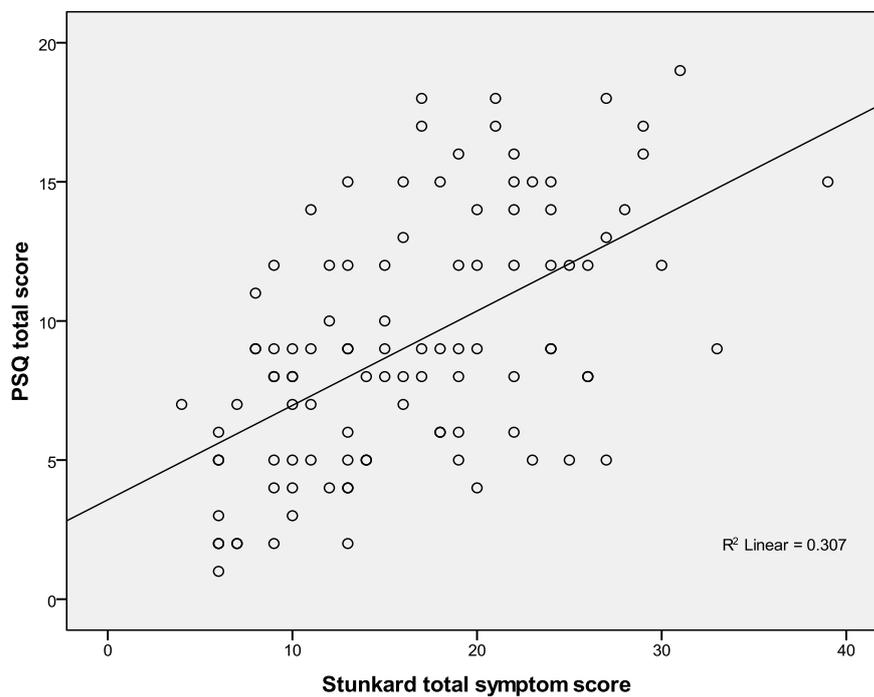


Figure 11 - Relationship between NEQ total score and PSQI total score for all study participants (n=103).

Table 52 - Comparison of the proportion of participants in the study group with a PSQI score > 5 and NEQ >25. Significance tests performed using Fisher's Exact Test (p= 0.34).

		<i>PSQI cut points</i>		
		<i>PSQI > 5 n (%)</i>	<i>PSQI ≤ 5 n (%)</i>	<i>Total n (%)</i>
<i>NEQ cut point ≥25</i>	<i>NEQ ≥25 n (%)</i>	13 (13.3)	2 (86.7)	15(15)
	<i>NEQ < 25 n (%)</i>	63 (71.6)	25 (28.4)	88(85)
<i>Total (n)</i>		76	27	103

Table 53 - Comparison of the proportion of participants in the study group with a PSQI score > 5 and NEQ >30. Significance tests performed using Fisher's exact test (p= 0.57).

		<i>PSQI cut points</i>		
		<i>PSQI >5 n (%)</i>	<i>PSQI ≤ 5 n (%)</i>	<i>Total n (%)</i>
<i>NEQ cut point ≥30</i>	<i>NEQ ≥30 n (%)</i>	4 (3.0)	0 (0)	4 (4)
	<i>NEQ < 30 n (%)</i>	72 (72.7)	27 (26.2)	99 (96)
<i>Total (n)</i>		76	27	103

A weaker correlation was seen between the PSQI and ESS total scores (0.34, p = 0.001) (Figure 12). This correlation reduced when controlling for the NEQ (2008) (0.23, p = 0.019).

Comparison of the proportion of individuals with suspected OSA and poor sleep quality was approaching significance with 25.8% (n=25) of the study group positive for both (p = 0.07) (Table 55).

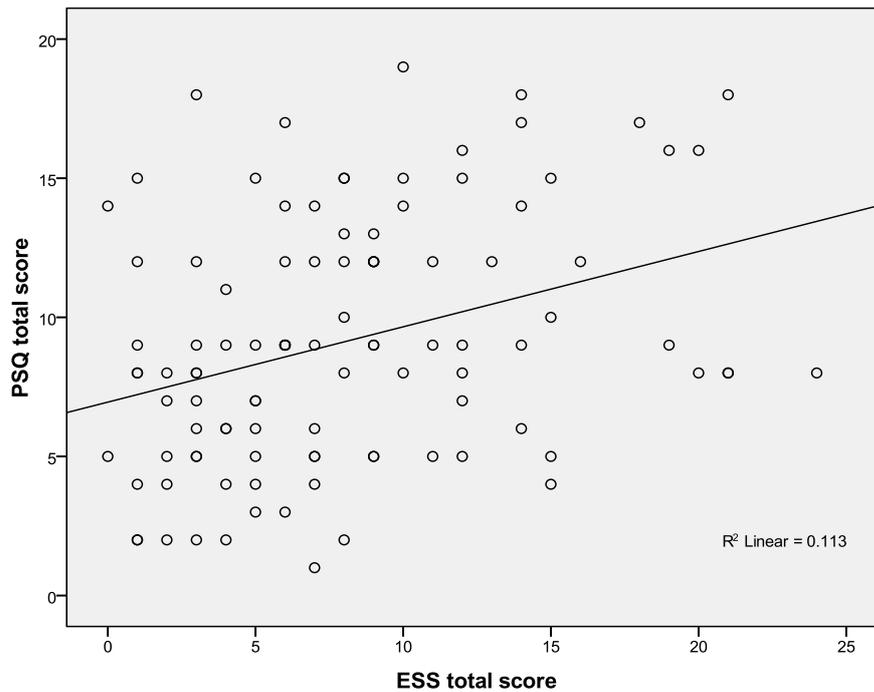


Figure 12 - Relationship between ESS total score and PSQI total score for all study participants (n=103).

Table 54 - Comparison of the proportion of participants in the study group with an ESS score >10 and PSQI >5. Significance tests performed using Fisher’s exact test (p = 0.09).

		<i>PSQI cut points</i>		
		<i>PSQI >5 n (%)</i>	<i>PSQI ≤5 n (%)</i>	<i>Total n (%)</i>
<i>ESS cut points</i>	<i>ESS < 10 n (%)</i>	51 (67.1)	23 (85.2)	51(49.5)
	<i>ESS > 10 n (%)</i>	25 (32.9)	4 (14.8)	52(51.5)
<i>Total (n)</i>		76	27	103

A similar weak correlation was seen between the NEQ (2008) and ESS total scores (0.27 p = 0.006) (Figure 13). The correlation was no longer significant when controlling for the PSQI. Comparison of the proportion of participants scoring > 10 on the ESS and ≥ 25 on the NEQ (2008) was not significant (p=0.19) with more individuals with suspected NES scoring < 10 on the ESS (n=9) as opposed to >10 (n= 6) (Table 55). A similar trend was seen in participants with a NEQ score ≥ 30 with 3 individuals with suspected NES scoring < 10 on the ESS and 1 scoring >10 (Table 56).

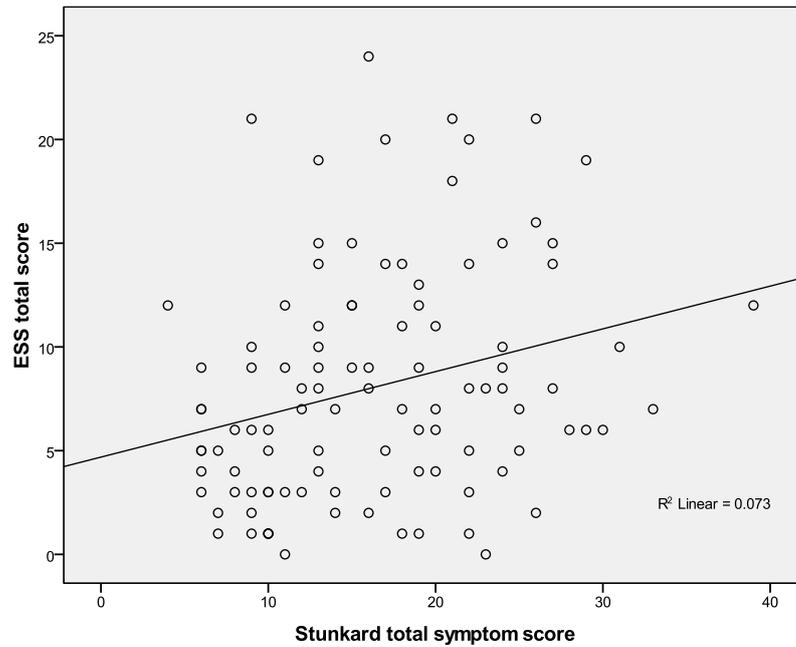


Figure 13 - Relationship between NEQ total score and ESS total score for all study participants (n=103).

Table 55 - Comparison of the proportion of participants in the study group with an ESS score > 10 and < 10 and NEQ ≥ 25 and < 25. Significance tests performed using Fishers exact test (p= 0.35).

		<i>ESS cut points</i>		
		<i>ESS > 10 n (%)</i>	<i>ESS < 10 n (%)</i>	<i>Total n (%)</i>
<i>NEQ cut point ≥ 25</i>	<i>NEQ ≥ 25</i>	6 (20.7)	9 (12.2)	15 (15)
	<i>NEQ < 25</i>	23 (79.3)	65 (87.8)	88 (85)
	<i>Total</i>	29	74	103

Table 56 - Comparison of the proportion of participants in the study group with an ESS score >10 and NEQ >30. Significance tests performed using Fisher's exact test (p= 1.0).

		<i>ESS cut points</i>		
		<i>ESS > 10 n (%)</i>	<i>ESS < n (%)</i>	<i>Total n (%)</i>
<i>NEQ cut point >30</i>	<i>NEQ >30 n (%)</i>	1 (3.4)	3 (4.1)	4(4)
	<i>NEQ < 30 n (%)</i>	28(96.6)	71 (95.9)	99 (96)
	<i>Total (n)</i>	29	74	103

5.7. *Discussion*

5.7.1. *Demographics: Age/Gender/BMI*

The mean age of this study group was 44.5 (12.2) years and is similar to the mean age of the first study group (44.6 (11.6) years) and the earlier clinic audit population (44.4 (11.9) years). A similar representation of males was seen in this group as in the audit population (28% vs 33%), confirming the unexpectedly high representation of males in the identification study (43%). Differences between groups were also noted in BMI. The mean BMI of 47.0 (9.8) kg/m² in this group, compares to the audit BMI of 48.5 (9.9) kg/m², both of which are lower than the mean BMI in the identification study (50.0 (10.7) kg/m²). The mean BMI in this study was significantly higher in males and although higher in males in the first study the difference was not significant. (52.4 kg/m² vs 48.1 kg/m² p= 0.09).

5.7.2. *The Epworth Sleepiness Scale*

ESS scores were wide ranging (0-24). The mean score of 8.1(5.6), is slightly higher than the mean score of 7.5 (4.8) noted by Algul et al.(2009) in a class II obesity group, although Algul's group was exclusively male (n=92), without co-morbidity and had a lower mean BMI (38.9 (3.1) kg/m²). As expected, the study mean score was higher than the mean score of 4.5 (3.3) identified by Parkes (1998) and 4.6 (2.8) by Johns & Hocking (1997) in healthy controls, but lower than 11.1(5.2) identified by Ngygen (2006) in an OSA population. No gender differences were noted between mean scores, or individuals scoring above the cut point of 10 (32%). This compares with 30.4% of Algul's participants scoring above 10 and 27% of the identification study group diagnosed with, or under investigation for OSA, of whom 68% were male. In this study, only 36.4% of those with a score above 10 were male, although there was a higher proportion of males in this group than in the group with scores of 10 or less. No correlation between ESS and age was noted although a positive correlation was seen between BMI and ESS (r = 0.23, p = 0.02), with those scoring above 10 having a higher mean BMI (50.8 vs 43.1 kg/m² p = 0.09).

This relationship between body weight and OSA is a consistent finding in other OSA studies (Coughlin et al. 2004). Higher item scores were noted for situations in which dozing

would be considered more socially acceptable such as watching TV, with lower item scores for situations where individuals might make more effort to keep alert, such as in conversation or whilst driving. Admission of dozing whilst driving can have serious affects on driving eligibility and employment, encouraging individuals to mediate their response to these items. Other researchers have found similar trends in item scores (Algul et al. 2009;Nguyen et al. 2006).

5.7.3. *The Pittsburgh Sleep Quality Index*

PSQI scores ranged from 1-19 reflecting a variety of sleep experiences although an overwhelming 74% had a score above 5 reflecting poor sleep quality. This reflects the findings in the matched control study where 100% of the NEB group and 60% of the non-NEB group self-reported poor quality sleep. Sixty five percent of Algul's class II obesity group also reported scores above 5. Mean scores in this study group as opposed to Algul's population were higher (mean 9.1 (4.5) vs 7.3 (4.1)), probably as a result of higher BMI and greater co-morbidity. As expected, the mean score was higher than in a healthy control population (2.7(1.7)), though similar to a depressed outpatient population (11.1 (4.3)). No gender, weight or age differences were seen in this study between individuals with scores above or below the cut point and no correlation was seen between age and PSQI total score, or BMI and PSQI total score. This conflicts with the positive correlation between BMI and PSQI ($r= 0.28$ $p<0.01$) noted by Algul but the difference in mean BMI between the two studies may account for this (47.0 (9.8) kg/m^2 vs 38.9 (3.1) kg/m^2).

Differences in item scores were in keeping with results from other studies (Backhaus et al. 2002;Buysse et al. 1988;Nguyen et al. 2006).The lowest score for the use of sleep medication probably reflects the increasing reluctance of General Practitioners to prescribe night sedation. High items scores for sleep disturbances and subjective sleep quality are in keeping with the qualitative results of the matched control study where individuals with and without NEB reported many physical and psychological reasons for disturbed sleep.

5.7.4. *The Night Eating Questionnaire (2008)*

Verbal administration of the NEQ (2004) during the diagnostic interviews in the identification study gave mean scores of 28.3 (8.0) for the NEB group and 13.0 (5.3) for the non-NEB group. The mean score of 16.4 (7.3) in this study (range: 4-39) is in keeping with the mean

score of 16.0 described by Allison et al. (Allison et al. 2008b) in a non-NES bariatric surgery group and much lower than the mean scores identified in NES populations (33.1 (self-diagnosis group), 32.4 (medically diagnosed group) and 26.2 (bariatric surgery group with NES) (Allison et al. 2008b; Backhaus et al. 2002; Nguyen et al. 2006). Percentages with suspected NES according to the cut points of ≥ 25 and ≥ 30 were lower than individuals screening positive on the ESS and PSQI ($\geq 25 = 14.6\%$, $\geq 30 = 3.9\%$). The higher numbers at the lower cut-point reflects the findings of the diagnostic interview for the identification study that a significant number of individuals may experience some symptoms of NES, but those experiencing all symptoms are comparatively rare. More females were noted in the suspected NES groups at both cut points, although, proportionally, more men were noted in the NES group at the lower cut score, than the upper cut score. However, small numbers in the higher cut score group in particular make it difficult to interpret these findings meaningfully. Whilst the sensitivity of the tool is increased at the higher cut score, given the broad range of NEB characteristics identified in both the identification study and the characterisation study, it would seem logical to use the lower cut score when screening in a clinical setting. Identifying a few false positive cases would seem preferable to excluding the majority of individuals with mild to moderate night-eating who may benefit from treatment. Those suspected of NES at both cut points were significantly younger than those with scores below the cut points. A negative correlation between age in years and NEQ (2008) total score was noted ($r = -0.25$, $p = 0.01$). This relationship with age was reflected in the (non significant) differences in age between the NEB and non-NEB group in the first study (mean age 42.3 years vs 46 years). No correlation was noted between BMI and NEQ (2008) total scores, although the mean BMI was higher in the NES group at the lower cut point, reflecting non significant differences in BMI between the NEB and non-NEB group in the first study (mean BMI (52.5 kg/m² vs 48.4 kg/m²)).

As would be expected, low scores were noted on NEQ (2008) items describing night-eating behaviour as the majority of individuals did not report night-eating. Moderately high mean scores were noted on the items related to control over eating (lack of control after evening meal, 1.7(1.3); lack of control when eating at night, 1.5 (1.4)). This is in keeping with qualitative reports describing a lack of control when night-eating during diagnostic interviews. Given the

high mean BDI scores in both the NEB and non-NEB group in the identification study (24.8 (10.9) vs 17.7 (10.8)) and high levels of depression reported consistently in both NES and obese groups, the mean mood-related item scores appeared lower than anticipated.

5.7.5. *Limitations of the Prevalence Study*

Results from the prevalence data in this study must be viewed cautiously, and differentiation must be made between conclusions based on screening as opposed to diagnostic tools. Allison et al. (2008b) advise caution in using the NEQ (2008) as a screening tool, suggesting its best use is as a measure of NES symptom severity. The ESS also measures day-time sleepiness, a symptom of OSA as opposed to a diagnostic pre-requisite. This study sample included individuals new to the clinic as well as follow-up patients who may already be receiving treatment for OSA, thus levels of daytime sleepiness may be under-estimated. Whilst the PSQI contains some objective measures of sleep, such as sleep duration and sleep latency, it is overwhelmingly a subjective sleep quality measurement that may not necessarily reflect objective experiences.

5.7.6. *Relationship between OSA, Night Eating and Sleep Quality*

A relatively strong positive correlation was seen between the PSQI and NEQ (2008) total scores suggesting a relationship between night-eating and poor sleep quality. This relationship did not diminish even when controlling for day-time sleepiness. A significant, though weaker relationship was seen between poor sleep quality and day-time sleepiness, which was weaker still when controlling for night-eating. The weak correlation between night-eating and day-time sleep disappeared when controlling for poor sleep quality. This suggests that day time sleepiness, a symptom of OSA may be a separate construct having no influence over night-eating and its relationship with poor sleep quality. Night-eating has a limited impact on the relationship between poor sleep quality and day-time sleepiness and poor sleep quality has a strong influence on the weak relationship between night-eating and day time sleep.

5.7.7. *Conclusion*

To conclude, levels of suspected NES identified in the prevalence study appeared similar to that noted in the identification study. Suspected NES was far more common at the lower cut point, than the higher cut point, suggesting greater numbers of individuals with mild to moderate

difficulty and fewer cases of severe night-eating. The high levels of poor sleep quality confirm the findings of the characteristics study, that sleep problems are widespread in a morbidly obese population and this needs to be taken into account when obesity is being treated. Day-time sleepiness, whilst relatively common, has little impact on the relationship between night-eating and sleep quality, confirming that NES and OSA are not closely related constructs.

6. GENERAL DISCUSSION OF THESIS

6.1. *Chapter Overview*

This chapter is presented in 3 sections. Section 1 summarises the key findings of the identification, characterisation and prevalence studies and discusses these findings in the light of current thinking in NES. Section 2 discusses the emergent study design, strengths and weakness of the tools used in this programme of studies and their applicability to NES research in obese populations. Section 3 discusses the implications of study findings for current practice and future research and concludes with some aspects of self-reflection.

6.2. *Section 1 : Key Findings*

6.2.1. *Identification Study*

The 2003 criteria for defining NES were found to be too restrictive and excluded many individuals with some night-eating behaviours. A broader Night Eating Behaviour (NEB) group was identified, which included individuals eating less than 50% of their calorie intake after the evening meal, individuals who stay up very late to eat before going to bed, or sleep in the day and eat at night. Comparison with non-night eaters showed night-eaters (NEB) to have higher BDI depression scores, less likely to be working and to have greater perceived lack of control over eating. A greater proportion of the NEB group were aged less than 35 years. A greater variability was noted in reported sleep duration in the NEB group and significant numbers reported sleep-related co-morbidity. The presence of low level eating disorder pathology was noted in both groups. Due to limitations with the potential screening tool and doubts as to the appropriateness of the 2003 criteria for identifying NES, it was not possible to estimate NES prevalence at this stage and further developmental work on the tool was halted in favour of a more qualitative exploration of NEB characteristics and their inter-relationship from the participants' perspectives.

6.2.2. *Characterization Study*

Thematic analysis of the NEB group interviews revealed self-reports of 12 individuals developing NEB as children or teenagers and 18 as adults. There appeared to be a link between NEB and obesity, but the link between significant life events and NEB was less clear. NEB individuals described eating behaviour which was compulsive, chaotic and uncontrolled and in

the context of negative emotions, low mood and conflictful relationships. Sleep was disturbed by lots of physical factors as well as the need to eat. Comparison with a matched control group found similar levels of childhood obesity and life events. High numbers of the non-NEB group also reported poor sleep quality which was influenced by a variety of physical and emotional factors. The non-NEB group reported much greater perceived control over eating. To summarise, NEB appeared to cover a broad spectrum of behaviours, developing at various ages and to be associated with perceived lack of control over eating and depression. NEB is also associated with childhood obesity, significant life events and poor sleep quality, although these features are also present in morbidly obese individuals without NEB.

6.2.3. *Prevalence Study*

Results from the prevalence study showed suspected NES in 14.6% (n=15) using the cut score of >25, and 3.9% (n=4) using a cut score of >30 based on Stunkard's validated NEQ (2008) (Allison et al. 2008b). OSA was suspected in 32% (n= 33) of the participants. Poor sleep quality was widespread and reported by 74% (n= 76) of the clinic population. Correlational analysis showed day-time sleepiness, a symptom of OSA, to be a separate construct, which even when present, appeared to have little impact on the relationship between night-eating and poor sleep quality. When night-eating was present, this had limited impact on the relationship between poor sleep quality and day-time sleepiness and the relationship between night-eating and day-time sleepiness itself was weak. Thus, whilst poor sleep quality appeared to have a strong influence on both night-eating and day-time sleepiness in turn, there is little evidence to suggest the concepts of night-eating and day-time sleepiness are closely related.

6.2.4. *Current Thinking in NES*

These results are applicable to an obesity clinic population and were based on the criteria proposed by Stunkard in 2003 for NES. As previously discussed, the restrictive nature of the criteria was acknowledged in 2008 by Stunkard's team when more relaxed criteria based on 2 core features were proposed, namely that at least 25% of food intake should be consumed after the evening meal and/or at least 2 episodes of nocturnal eating per week. (Allison et al. 2008a). However, the research community continued to debate the criteria and acknowledged that continual confusion surrounding the criteria was preventing research moving forward. As a result,

the First International Night Eating Symposium was held by the International NES Working Group in 2008 and revised diagnostic criteria, specifically for research purposes were proposed in 2010 (Allison et al. 2010b). The 2010 criteria are listed in Table 57 and compared to the 2003 criteria. Symposium participants also called for more research studies which systematically examined the co-occurrence of symptoms defined in the criteria in order to calculate conditional probabilities and identify subtypes (Striegel-Moore et al. 2008).

Table 57 - Comparison between the 2003 and 2010 criteria for NES

2003 criteria (All need to be met)	2010 criteria (All of A-F need to be met)	
<p>Evening hyperphagia. At least 50% of the daily caloric intake is consumed in snacks after last evening meal.</p> <p>Consumption of high calorie snacks during awakenings on frequent occasions</p>	A	<p>Core criterion: Daily pattern of eating demonstrates a significantly increased intake in the evening and/or night-time, as manifested by one or both of the following:</p> <ol style="list-style-type: none"> 1. At least 25% of food intake is consumed after the evening meal. 2. At least 2 episodes of nocturnal eating per week.
	B	<p>Core criterion: Awareness and recall of evening and nocturnal episodes are present</p>
<p>Morning anorexia, even if subject eats breakfast</p> <p>Awakenings at least once a night, at least 3 nights a week</p>	C	<p>Core descriptors: The clinical picture is characterised by at least three of the following features</p> <ol style="list-style-type: none"> 1. Lack of desire to eat in the morning and/or breakfast is omitted on four or more mornings per week. 2. Presence of a strong urge to eat between dinner and sleep onset and/or during the night. 3. Presence of a belief that one must eat in order to initiate or return to sleep 4. Sleep onset and or/sleep maintenance insomnia are present four or more nights per week. 5. Mood is frequently depressed and/or mood worsens in the evening
	D	<p>Core criterion: The disorder associated with significant distress and/or impairment in functioning</p>
<p>The pattern occurs for a period of at least 3 months</p>	E	<p>Core criterion: The disordered pattern of eating has been maintained for at least 3 months</p>
<p>Exclusion of other eating disorders</p>	F	<p>Core criterion: The disorder is not secondary to substance abuse or dependence, medical disorder, medication or another psychiatric disorder.</p>

The 2010 proposed research criteria are discussed in turn below in light of the findings of this programme of studies in a morbidly obese population.

6.2.4.1. *Core Criterion A*

‘The daily pattern of eating demonstrates a significantly increased intake in the evening and/or night-time, as manifested by one or both of the following:

- **At least 25% of food intake is consumed after the evening meal.**
- **At least 2 episodes of nocturnal eating per week.’**

Findings from the identification study, in which 14 individuals reported evening hyperphagia but at a level less than 50% support the reduction of the proportion of calories eaten after the evening meal from 50% to 25% and this reduced volume now seems widely accepted. However, the definition of the ‘evening meal’ continues to lack clarity, especially given cultural differences. Lundgren et al. (2011) propose it should be taken to be the first food eaten after 5pm, which could be a ‘main meal’ or ‘snack’ if no main meal is eaten. If no food is eaten up to 8pm, then any food after 8pm is classed as ‘after the evening meal’. In the identification study, the definition of ‘evening meal’ was based on Stunkard’s definition in the NESHI and clarification was given in the prevalence study to interpret ‘supper’ to mean ‘after the evening meal’. Without reporting the definition explicitly in individual studies, comparisons between studies based on this criterion are meaningless, particularly in obese populations in whom evening hyperphagia and late evening eating are common. Although in the identification study a significant difference in the amount of food eaten after the evening meal was reported in the NEB group, individuals who ate late but stopped eating and went to bed before midnight were excluded, as morning anorexia followed by evening hyperphagia is such a common feature in morbid obesity. The point at which ‘evening overeating’ becomes ‘night-eating behaviour’ still continues to be difficult to define in a morbidly obese group, although the 2010 criteria do at least attempt to account for individuals who stay up very late to eat. The reduction in the criteria from 3 nocturnal ingestions a week to 2 reflects current proposed changes to reduce from 3 to 2 the number of days a week required for binge eating behaviour to be classed as BED. If this reduction were applied to the identification study, one further individual would have been

classed as full NES as opposed to sub-threshold NES. Whilst this change is, no doubt, intended to make the criteria more inclusive, in practical terms it has little value as, even if NEB was not very frequent, treatment strategies would still seek to address it, to prevent escalation of the problem and further weight gain.

6.2.4.2. *Core Criterion B*

‘Awareness and recall of evening and nocturnal episodes are present’

This criterion was not included in the 2003 criteria and is usually not reported in papers, although it is assumed in the literature that NES individuals have full awareness of their behaviour, otherwise they would be classed as Sleep Related Eating Disorder (Devlin 2007; Howell, Schenck, & Crow 2008). This suggests a dichotomous classification where awareness is completely present or absent. This experience was not reflected in the identification or characterisation studies, where the degree of awareness varied considerably both between and within individual reports, although distress and behaviour associated with other parasomnia such as sleep walking were not reported. Some individuals gave conflicting reports at various stages in the interview while others supported Allison’s findings that individuals often report lack of awareness initially, but can recall events with further probing (Allison et al. 2010b). Whilst an individual’s reluctance to admit to an apparently shameful and secretive behaviour may have an effect on initial reporting, measures for defining and reporting awareness in order to distinguish NES from other sleep disorders need to be more robust. It might be helpful to include items in the NESHI which help obesity and ED specialists to distinguish NES from other sleep disorders.

6.2.4.3. *C Core Descriptors*

‘The clinical picture is characterised by at least three of the following features:

The 2010 criteria propose that 3 of the following 5 descriptors are required for a diagnosis of NES, although all but one appear common in obese populations regardless of NES diagnosis.

1. ‘Lack of desire to eat in the morning and/or breakfast is omitted on 4 or more mornings per week’.

Striegel Moore’s typology study (Striegel-Moore et al. 2008) noted a strong negative linear relationship between night-eating and morning anorexia and this criterion has been a consistent

feature of all versions of NES criteria. However, this relationship is also true of morning anorexia and chronic obesity (Huang et al. 2010) and its relevance as a distinguishing feature of NES in a morbidly obese population remains doubtful. No difference was noted between the NEB group and the non-NEB group on the morning anorexia item of the NEQ (2004). Seventy three percent of the identification study sample reported not eating breakfast and the true rate was probably higher, as several of those eating breakfast were probably doing so reluctantly in order to comply with dietetic advice. Whilst this descriptor is likely to be helpful as a distinguishing feature in normal weight individuals it is of little use in a morbidly obese population.

2. 'Presence of a strong urge to eat between dinner and sleep onset and/or during the night'.

In the identification study, a significant difference was noted between the NEB and non-NEB group for the items relating to eating after the evening meal and the urge to eat at night. Given the substantial variability in evening and night-eating in this obese population it is possible that the cognitions and behaviours driving the urge to eat are more relevant than the timings of meals/snacks and amounts eaten. Individuals with NEB reported greater loss of control during these eating episodes in particular and during eating episodes generally. As previously discussed, perceived loss of control is a classic feature of many ED sufferers who report having less personal control over outcomes in the world and a tendency to attribute the cause of 'bad' life events to aspects of themselves. Future areas of research need to examine whether NEB individuals share this distinctive emotional make-up and are characterized by an external locus of control and poor self efficacy, making them less able to control their eating patterns and whether this is a distinctive feature of NEB as opposed to morbid obesity.

3. 'Presence of a belief that one must eat in order to initiate or return to sleep'

Evidence from the characterisation study showed that obese individuals often wake up at night and get out of bed for a variety of reasons, mainly to go to the toilet. Getting up for a drink is very common and yet only some individuals will go on to eat before returning to bed, describing a strong compulsion to do so. Clearly, obese individuals are often presented with opportunities to eat at night and yet only some individuals will feel this compulsion. In an obese population,

this appears to be the one key distinguishing feature that separates night-eaters from non-night-eaters and adds weight to the argument that obese NEB sufferers either have different cognitions and emotions to obese individuals without NEB, or that they all have similar cognitions, but some individuals find restraint at high risk times easier than others. The psychological profile of poor sleepers having anxiety-prone, obsessive-worrisome personalities did not seem particularly applicable to this NEB group, who reported less emotional reasons for waking up, compared to the non-NEB group. A greater understanding of the NEB 'personality type', above and beyond the obese 'personality type' may help to answer these questions.

4. Sleep onset and or/sleep maintenance insomnia are present four or more nights per week.

The requirement for sleep disturbance to be present on 4 or more nights a week, but night-eating to be present on 2 or more nights seems incongruous in a syndrome where the emphasis is on night-eating. Whilst it suggests an attempt to rectify the lack of focus on sleep disturbance in the 2003 criteria, it raises the question as to whether, in an obese population at least, the focus of the syndrome has been on the wrong behaviour.

Physical impairment caused by morbid obesity and obesity related co-morbidity had a large effect on the sleep of individuals in this programme of studies. Eighty percent of individuals in the characterisation study reported poor sleep quality, although this was no doubt influenced by the screening question relating to poor sleep included in the flyer. Yet a similar level (74%) of reported poor sleep quality was replicated in the prevalence study. As previously discussed, sleep disturbance can be both a cause and effect of obesity and is a predominant feature of mental illness, in particular depression (Monti & Monti 2005). The correlation between night-eating and poor sleep quality was quite strong (0.55, $p < 0.001$), but comparison of the numbers of individuals identified with potential NES (14.6%) in the prevalence study, as opposed to the numbers with poor sleep quality (74%), indicates that the impaired sleep is by far the more common behaviour. It would seem clinically more useful, therefore, to propose that an 'Impaired Sleep Syndrome' would be a more appropriate title for the cluster of behaviours found in obesity populations. It may be more helpful to identify poor sleepers first and treat those within that group who also eat at night as a sub-group needing specialist support. Evidence from

the characterisation study suggests NEB may develop through a variety of mechanisms, such as strict parental control over eating or in response to life events and that with time, the extent to which NEB and obesity are responsible for the causation and maintenance of each other becomes less clear. Therefore, within the night-eating subgroup identified by 'Impaired Sleep Syndrome' it may be appropriate to identify further subgroups requiring differing treatments, depending on each individual's NEB-obesity pathway.

5. 'Mood is frequently depressed and/or mood worsens in the evening'

Whilst low mood is undoubtedly a feature of NES, as it currently stands, this descriptor could be applied to most obese populations, with obese individuals often describing overeating in the evening as a result of low mood as a main contributor to their obesity. In the identification study, the NEB group scored higher on the BDI than the non-NEB group, although both groups had high numbers of individuals with cut scores indicating moderate or severe depression (NEB 68% and non-NEB 42%). These findings compare with other morbidly obese populations (Dixon, Dixon, & O'Brien 2003). Lundgren et al. (2010) used the NEQ (2008) to examine NES in 68 individuals (60% female, age 43.9 (10.4)) with serious mental illness who were also obese (mean BMI 37.2(8.1) kg/m²). The mean (SD) NEQ score of 19.0 (7.3), was similar to the mean score of the identification study (20.7 (6.6)) although higher numbers reported evening hyperphagia of $\geq 50\%$ and 3 or more nocturnal ingestions a week (25% vs 10%).

Striegel-Moore et al. (2010) found similar high levels of depression in night-eaters when developing a screening tool for BED in the BEST study. In 285 females with BED, 14.4% reported at least 1 nocturnal eating episode in the past 28 days. These individuals reporting at least one night-eating episode were more likely to be non-white ($p=0.02$), married ($p=0.003$) and have slightly higher BMIs than other study participants although the difference was not significant (29.6 (8.0) kg/m² vs 31.7(8.8) kg/m²). After adjusting for the effects of binge eating, a significant difference was noted in BDI scores between the night-eaters and non-night-eaters (20.9 (12.1) vs 14.0 (10.1), $p < 0.001$). The mean BMI of this study's participants was lower than in the identification study and Lundgren's study and is reflected in a lower mean BDI score for the non-night eaters. Despite the high BDI scores in both the NEB and non-NEB group in the identification study, those with NEB were significantly more depressed, suggesting NEB is more

likely a feature of severe depression than mild/moderate depression. Evidence from the obesity-NEB pathway analysis showed negative affect to be a dominant feature throughout, yet it is surprising that despite all the evidence linking low mood to NES that this is still not considered a core criterion.

6.2.4.4. *Core Criterion D*

‘The disorder is associated with significant distress and/or impairment in functioning’

Distress and impairment cover a very broad remit and definitions can be problematic and differ between individuals. With regard to distress, Leskela et al.(2004) differentiate between stressful life events and life stress and consider an individuals’ ability to cope with the stress to be influenced by the proximity of the event, the impact of environmental influences and other factors and psychosocial variables such as personality and social support. The criterion itself does not distinguish between whether an individual is currently feeling distressed or whether the night-eating developed as a result of a stressful event which has long passed. Tzischinsky & Latzer (2004) found that the timing of sexual abuse coincided with the onset of night-eating in BU and BED adolescent subjects. Sassaroli et al.(2009) examined the daily and nightly anxiety levels of NES and BED adult subjects and found current night anxiety to be more prevalent in the NES group. In the characterisation study, this feature of distress during night-eating was only reported by two individuals with more individuals reporting mixed feelings after the eating episode such as shame and guilt and relief at being able to sleep. It is possible that in an obese population, night-eating has become a chronic, habitual behaviour with the distress of influencing events long passed. Opportunities to explore this further were limited as the characterisation study relied on analysis of previously collected data. Further research using tools to measure life events such as the Bedford Life Events and Difficulties Schedule (LEDS) may be helpful to explore this further in obese NES populations (Brown & Harris 1978).

Allison et al.(2010b) describe distress related to night-eating as ‘distress about weight gain, shame of eating and inability to stay within a prescribed calorie deficit’. However, these emotions were reported equally in the characterisation study by both night-eaters and non-night eaters and are all common features of morbid obesity. Likewise, problems with physical functioning were common experiences in both groups depending on the degree of obesity,

disturbed sleep, depression and chronic pain. Objective measures of functioning such as the ability to hold down employment may be clinically more useful in this population. NEB individuals in this study were less likely to work, indicating a high degree of dysfunction. Whether this contributed to the onset and maintenance of NEB or was a consequence of NEB was not determined in this study.

6.2.4.5. *Core Criterion E*

‘The disordered pattern of eating has been maintained for at least 3 months.’

This criterion brings NES in line with the criteria for BED and is an attempt to differentiate between an acute event, such as a bereavement, temporarily disrupting eating and sleep patterns and a more chronic problem, worthy of therapeutic endeavour. In the identification study, only one individual reported night eating behaviour lasting less than 3 months with the mean duration being 9.3 (12.7) years. This suggests that in a morbidly obese population at least, this criterion is probably not relevant as night-eating is more likely to be a chronic behaviour.

6.2.4.6. *Core Criterion F*

‘The disorder is not *secondary* to substance abuse or dependence, medical disorder, medication or another psychiatric disorder.’

The 2003 criteria excluded other ED from the diagnosis of NES as all early evidence at the time suggested NES was a different construct. Current thinking now suggests more work needs to be undertaken to identify the relationship between NES and other ED and for this reason, this particular criterion has been reworded and now includes a major shift in emphasis. According to the 2010 criteria, individuals with NES and sub-threshold symptoms of other ED would be classed as NES, but individuals with other ED and some sub-threshold symptoms of NES would not be classed as NES. However, categorising individuals into ED groups can be problematic. In the identification study there was general agreement between raters on the presence of NEB, with more disagreement centred on the presence of other ED. Considerable low-level eating disorder pathology and cross-over with sub-threshold groups was noted throughout the sample. De Zwann et al.(2006) had similar findings. Lundgren et al.(2011) examined the prevalence of NES based on the 2010 criteria in an ED population (Mean BMI kg/m² 21.4 (8.5)) of 68 individuals with AN (n=32), BN (n=32) and EDNOS (n=4). Twenty five percent met all of the

2010 criteria with a mean NEQ (2008) total score of 21.1(8.6). Forty two percent reported evening hyperphagia and 23% reported eating two or more times in the night. It is proposed that if the NES and other ED concur, the NES should 'defer' to the other ED and the night-eating aspects be considered as symptom dimensions. Whilst this might be a reasonable assumption based on Lundgren's normal/underweight population, identifying ED in morbidly obese groups is not so clear cut. As previously discussed, evening hyperphagia is a common construct in individuals with morbid obesity, NES and BED and assessing an objectively large amount, relative to BMI can be difficult. Distinguishing between overeating and 'binging' can be a grey area in obese individuals. More emphasis is often placed on the cognitions and behaviours surrounding the hyperphagia than the volume eaten and the issue of control is often the central distinguishing factor determining whether the individual is 'overeating' or 'binging'.

In the characterisation study, night-eaters reported more chaotic eating patterns generally, in comparison to the non-night eaters and feelings of lack of control, compulsion and guilt after night-eating episodes. These emotions are all characteristics of BED, thus adding to the growing body of evidence that NES and other ED are similar and not separate constructs. Root et al.(2010), in the Swedish Twin Study of Adults, looked for a genetic link between Night Eating (NE) and Binge Eating (BE). NE was defined as awakenings with food intake at least once a week and 25% or more of food intake after the last evening meal. Males were more likely to report NE behaviours and moderate heritability for NE was found in both males and females. The genetic correlation between BE and NE in females (0.66) indicated a substantial, but not complete overlap in genetic factors. In obese individuals at least, further work is required to identify whether night-eaters are different from individuals with other ED in how they perceive their control over eating and whether this differs between the day and night. Focus needs to shift away from the behaviours which, in obese populations, are influenced by many other physical factors and explore further the cognitions and emotions affecting the night-eating.

Criterion F also suggests a diagnosis of NES is not appropriate if it is secondary to other medical conditions and yet applying this criterion to a morbidly obese population is fraught with difficulty. The complex relationship between NES, obesity, poor sleep, obesity-related co-morbidity and depression means that most individuals showing night-eating behaviour would not

be classed as having NES. For example an individual who developed obesity, a subsequent co-morbidity such as OSA or depression and then NES would not be considered to have NES, whilst an individual developing obesity and subsequent OSA or depression as a result of NES would be. They both require treatment for the same problems, regardless of the pathogenesis.

6.2.5. *Summary of Criteria Data*

Stunkard's team continue to argue that more work needs to be done to conceptualise NES. Further investigation is required on its relationship with traumatic life events, and psychiatric co-morbidity, the age of onset of NES, course of NES over time, and its effect on circadian rhythm. Yet NES has been under considerable scrutiny for the past 20 years without much progress being made. Results from this current programme of studies indicate that in obese populations at least, impaired sleep is a bigger disruptive factor on the day-to-day functioning of the majority of individuals and this is the problem which ought to be prioritised. In this population, a shift of focus is required towards identifying behaviours which would be more appropriately entitled 'Impaired Sleep Syndrome', of which night-eaters may be a sub-group often experiencing severely depression. Results also suggest that night-eaters exhibit too many features of other ED for this relationship to be ignored and that previous guidance to separate NES from other ED was probably counter-productive and a hindrance to the characterisation of NES.

6.3. *Section 2: Study Design and Study Tools*

6.3.1. *Emergent Design*

When considering the overall results of this thesis, it is important to acknowledge the extent to which the design of the programme of studies emerged in response to initial findings and developed over time. At the outset, there was no reason to question the individual components of NES based on the 2003 criteria and it was anticipated that once individuals with NES had been identified, the thesis would develop by exploring treatment options for these individuals. The development of a screening tool was primarily to assist with identification. However, during the identification phase, it became apparent that the range of experiences described in the diagnostic interviews was not reflected fully in the diagnostic criteria, suggesting NES may not be sufficiently conceptualised at that stage. Although not originally planned, it was decided in

the second phase of study to analyse retrospectively this rich data set using a qualitative approach in order to contribute to the conceptualisation process and inform the future direction of research. The key relationship between poor sleep quality, obesity and night eating emerged as a result of this process. It is important however, to acknowledge the limitations of retrospective qualitative data analysis and accept that the structured interview design may have prevented the identification and exploration of other potentially relevant themes. The final phase of the programme of studies was designed once the conceptualisation phase was completed, with the purpose of exploring the findings of the thematic analysis in a systematic manner using validated tools.

6.3.2. *The Night Eating Syndrome History and Inventory*

Stunkard's team advise using the NESHI to explore the characteristics of NES, yet despite its widespread use it has never been validated and is likely to require major revision in order to accommodate the revised criteria. In its current form it contains several omissions which have limited potential findings in this programme of studies. There are no items relating to the characterisation of poor sleep and lack of clarity over some key diagnostic areas such as defining 'supper' and 'degree of awareness' can lead to inconsistencies in interpretation.

Additionally, in keeping with the NESHI guidelines, estimates of calorie intake were based on self-report during discussion with the interviewer. Lack of study resources prevented intake being estimated from food diaries although good agreement was seen between the two raters, one of whom was a dietitian, in identifying cases of NEB. Allison et al.(2010b) are currently calling for more precise methods to identify calorie intake and yet, in an obese population at least, it would seem less important to estimate the exact volume of food eaten at various times and more important to identify chaotic eating patterns and lack of perceived control over eating.

6.3.3. *The Night Eating Questionnaire (2008)*

The NEQ (2008) is the only screening tool available for identifying NES in various populations. In the prevalence study it was helpful in contextualising the proportion of individuals with night-eating within an obese population and it is noteworthy that the two cut

points identified similar levels of potential NES and sub-threshold NES as in the identification study. It is important, however to appreciate the difference between screening tools and diagnostic tools. Moreover, Allison et al. (2008b) advised caution in using the NEQ (2008) as either a screening or diagnostic tool, suggesting its best use is as a measure of NES symptom severity. This may be because the authors recognise the weaknesses in its original construction and are underplaying its applicability to NES populations. Concerns still remain as to its discriminant validity in an obese population, given that 3 of the 4 factors identified through principal components analysis, namely evening hyperphagia, morning anorexia, low mood and disturbed sleep, also feature strongly in obese populations (Allison et al. 2008b). Given that Stunkard's team has now revised the criteria and core criterion such as 'distress and impairment of functioning' have been added, it would seem timely to revisit the NEQ (2008) and question whether the same tool is appropriate for an underweight teenager and a morbidly obese adult.

6.3.4. *The Eating Disorder Examination (version 12)*

The EDE is used as a gold standard for identifying eating disorders and is used commonly by NES researchers in varied populations. It is designed to concentrate on the four key attitudes and behaviours of Eating Concern, Restraint, Weight Concern and Shape Concern (Fairburn & Cooper 1993). General, mood-related constructs such as anxiety and depression, or psychological constructs such as perfectionism, are deliberately omitted because, although common in patients with ED, they are not specific to these disorders and satisfactory measures already exist to measure these constructs. Self-report versions of the EDE are now in common use, based on the same premise and constructs as the verbal EDE. It is questionable whether the EDE is an appropriate tool for use in an obese population. Whilst useful for its diagnostic value in identifying ED, results from its subscales may have limited value as they were not constructed with obese populations in mind.

Whilst some key attitudes and behaviours such as the Eating Concern and Restraint subscales may be applicable to an obese population, it is reasonable to assume that morbid obesity would distort or inflate scores on the Weight Concern and Shape Concern subscales as it would not be dysfunctional for an individual with morbid obesity to be preoccupied with their shape and weight. In this study, no significant between-group differences were seen in the

subscale scores, although the NEB group reported a lower dietary restraint subscale score which approached significance. It is difficult to say whether the lack of difference between the two groups was due to a lack of difference between the characteristics of the NEB and non-NEB groups or whether morbid obesity was limiting the appropriateness of the tool. Certainly, many individuals could not answer the item relating to 'fear of losing control' on the grounds that they had already considered themselves to have lost control.

Studies examining the discriminant validity of the EDE between subjects with ED and 'normal' controls have shown mixed results (Cooper, Cooper, & Fairburn 1989; Rosen et al. 1990; Wilson & Smith 1989). Cooper and Fairburn's study, showed a significant difference on all the EDE items, with the least significant being for food avoidance ($z = 2.73$, $p = 0.006$) although differences in BMI were not reported. In a smaller study, Wilson & Smith (1989) compared 15 female patients with BN with 15 'restrained' women without an ED who have similar thoughts on fear of weight gain, body image and frequency of thoughts about food. Differences in baseline mean weight were not significant, although the BN group was heavier (139.2 kg versus 121.7 kg). BMI is not reported, although the absolute weights suggest a heavier population and are in keeping with the weights in the identification study (155.3 kg (NEB) vs 138.3 kg (non-NEB)). Significant differences were seen between both groups for all subscales apart from Restraint, as would be expected, in contrast to scores on the Eating Disorder Inventory (EDI), which showed differences between groups on the Bulimia items only. Rosen et al. (1990) tested further the incremental discriminant validity of the EDE, comparing the Weight Concern and Shape Concern subscale scores of 20 BN sufferers and 29 'restrained' female psychology students with the self-report Body Shape Questionnaire (BSQ). Using multivariate discriminant function analysis to examine both subscales and the BSQ as a predictor of group membership, they concluded that the two EDE subscales did not contribute anything above and beyond the negative body image attitudes assessed by the BSQ. These findings differ from Wilson's study, although the EDI measures body part dissatisfaction only, whereas the BSQ has a wider remit, with similar constructs to the Weight and Shape Concern items of the EDE. Of course, if the BSQ and WC and SC items perform similarly, this calls into question the main 'added value' of the EDE.

6.3.5. *The Beck Depression Inventory*

The BDI-1A was used for this study in keeping with other NES researchers at the time, although its appropriateness as a tool in future NES research must be considered carefully. Firstly, several versions of the tool exist and there appears to be general confusion surrounding versions in later NES papers, with Allison et al. referring to the BDI-II in two papers in 2007, although still using the scoring system and reference for the BDI-1A in one of the papers (Allison et al. 2007a; Allison et al. 2007b). No reference is given by O'Reardon et al. (2006), although an earlier paper uses the BDI-II (2004) and Pawlow, O'Neil & Malcolm (2003) refer to the BDI as a 20-item tool with a 0-60 score! Similar confusion exists in the obesity field, with researchers appearing to use the BDI-1A but referencing the BDI-I (Chen et al. 2007; Dixon, Dixon, & O'Brien 2003; Munoz et al. 2007). Researchers in other fields generally seem unaware of the differing versions. When Beck, Steer & Garbin (1988) attempted a meta-analysis of studies focusing on the psychometric properties of the BDI they concluded there was no feasible way of determining which version had been used from the descriptions presented in the majority of studies and had to consider them together. This must be a major consideration when interpreting results of any studies using this tool.

Further caution must be taken when interpreting results from studies using version II. Whilst both the BDI-1A and BDI-II report similar performances, it is important to remember that some items are different. Beck et al. (1996) compared the BDI-1A and BDI-II in 140 psychiatric out-patients with a mixture of mood, anxiety and adjustment disorders and found high internal consistency of both (0.89 and 0.91 respectively). Item total correlations of the 18 similar items showed equal performance, with anhedonia scoring highest on both versions. Point-biserial correlations of age, sex, and ethnicity showed no significant association with either version. However, researchers generally report total scores and the mean BDI-II score was 2 points higher ($p < 0.001$). This was mainly attributed to the rewording of the sleep and appetite items which now allow for increases as well as decreases in appetite and sleep. Steer & Beck (1985) had originally argued these increases had been left out deliberately, as the occurrence of loss of appetite and sleep disturbance was so high in severely depressed patients (72% and 87% respectively), and as increased appetite and sleep occur so frequently in normal individuals, a

high number of false positives would result. Indeed as the same cut-off is used, his rewording detected more potentially depressed individuals, as 14% were hungrier and 20% sleepier.

Changes in appetite and sleep items will clearly impact on scores in an obese population. Using the BDI-IA, as opposed to the BDI-II with its altered sleep and appetite items, may have underestimated the levels of depression in this study, although this may be mitigated by this being a morbidly obese population with sleep related co-morbidity, factors which might result in over-scoring on these items. Future NES research using the BDI needs to take into account the version used and the degree of obesity in participants.

One further concern is its apparent lack of discriminant validity with regard to anxiety. Although anxiety was not particularly reported as an important feature at night in the characterisation study, it features as a core criterion in Stunkard's 2010 criteria. When comparing a depressed population with an anxious population, Steer et al. (1986) found significantly higher mean BDI scores in the depressed group (26.37 versus 14.46). However, in a study of 130 psychiatric inpatients, anxiety and depression were both measured by self-ratings and observer ratings and analysis of multitrait-multimethod matrices by confirmatory factor analysis failed to confirm the discriminant validity of the BDI with respect to anxiety (Richter et al. 1998).

To a certain extent this may be the result of trans-diagnostic symptoms confounding the findings as some symptoms of depression are also features of other illnesses. Cognitive-affective items such as performance impairment, for example, are also features of alcoholism. One would also expect somatic items to feature highly in obese subjects (Munoz et al. 2007) found a mean BDI score of 16.47 in 257 pre-operative bariatric surgery patients (mean BMI 53.2 kg/m²). When the 21 items were subject to exploratory maximum likelihood factor analysis, there was strong loading onto the 2 factors previously identified, but seven of the top 10 most endorsed items related to somatic complaints. The mean of the somatic items 9.3 (4.0) was significantly higher than for cognitive/affective items 8.6 (6.2) $t = -2.2, p=0.03$ (Munoz et al. 2007). It is possible therefore that the somatic items may artificially inflate the score in obese populations.

6.3.6. *The Epworth Sleepiness Scale*

The ESS cannot be viewed as a diagnostic tool as it measures day-time sleepiness, a symptom of OSA as opposed to a diagnostic pre-requisite. Although it is acknowledged to be a reliable and valid tool for identifying OSA, in obese populations with high levels of negative affect, identifying the causes of day-time sleepiness can be problematic. De Zee et al.(2006), found that the ESS was not helpful in identifying mental disorders and day-time sleepiness due to depression, with no relationship identified between the ESS scores and Primary Care Evaluation of Mental Disorders (PRIMED) scores in 165 individuals referred to a sleep clinic. In obesity clinical practice, disturbed sleep and day-time sleepiness are such common features that preliminary identification of OSA often needs to rely on other symptoms, such as snoring and reported apnoeas (long periods between breaths) in order to generate a referral to a sleep specialist.

The prevalence study sample included individuals new to the clinic as well as follow-up patients who may already be receiving treatment for OSA, thus levels of day-time sleepiness may have been under-estimated. Results would have been strengthened if individuals suspected of OSA were followed up to confirm diagnosis and levels of those already diagnosed and receiving treatment were also identified.

6.3.7. *The Pittsburgh Sleep Quality Index*

Whilst the PSQI contains some objective measures of sleep, such as sleep duration and sleep latency, it is overwhelmingly a subjective sleep quality measurement that may not necessarily reflect objective experiences. Tworoger et al.(2005) measured sleep quality in 73 women, aged 20-40 years, with BMI less than 30kg/m². Subjective and objective sleep measures were combined. Participants completed the PSQI and wore an actigraph monitor over 5 nights. 30% reported a PSQI above 5 although no association was found between the PSQI scores and objective actigraph data. An overwhelming majority of individuals in the prevalence study reported poor sleep, although the PSQI scoring mechanism combines both objective components, such as total sleep time and subjective components such as self-reported sleep quality, to give a total score. To ensure that the PSQI reflects both the objective and subjective experiences equally in obese populations, it may be helpful to undertake further analysis on the

PSQI data from the prevalence study to identify if the qualitative and quantitative components of each individuals' scores 'match' and reflect a similar experience. Despite the author's view that the PSQI is a user-friendly tool, the scoring system is complex, unlike the ESS and this is a major drawback to its widespread use in clinical practice.

6.3.8. *Thematic Analysis*

Due to the retrospective nature of the data analysis, caution must be taken in interpreting the results of the characterisation study. Applicability of the findings would have been strengthened if the data collection had followed a traditional Grounded Theory approach from the beginning, allowing the emergent themes to be truly grounded in the data. In particular, themes such as the effects of significant life events, or work status on NEB could not be developed fully although these remain interesting areas for further exploration. To mitigate the limitations of the thematic analysis of the NEB interviews, as large a sample size as possible was chosen with 30 of the 31 NEB participant interviews included in the analysis (one interviewee declined to be taped). Participants who were suspected of having some features of night-eating were invited for interview to ensure interviewees had some experience of the central phenomenon being explored. In addition, maximum variation sampling was used by including those with NES and sub-threshold NES. This was to allow for diversity in emergent categories as exploring various conditions makes a theory denser and increases its explanatory power: comparisons give categories specificity. As Wimsatt (1981) states,

'verification of ideas and categories is achieved by searching for negative and qualifying evidence and the full range of variation in the phenomena under scrutiny'.

The use of the 3 stage coding process to analyse the data is justified in the characterisation study, as all Grounded Theorists stress that its techniques and procedures are tools, not directives and they make allowances for some procedural flexibility in the face of the inevitable contingencies of research (Charmaz 2009; Glaser & Strauss 1967; Strauss & Corbin 2008). Due to a lack of resources, it was not possible to perform detailed analysis of the non-NEB group transcripts and conclusions from the matched control study may have been strengthened if this had been undertaken.

6.4. *Section 3: Implications for Future Research and Current Practice*

6.4.1. *Generalisation of Findings*

Participants in this programme of studies were recruited from a standard UK obesity clinic population, reflecting a typically morbidly obese population. Thus prevalence estimates discussed in this study may not reflect estimates in other populations. It is important to acknowledge that these were individuals coming forward for treatment and may have included some individuals wanting bariatric surgery. It is possible that they were more motivated to change and had different psychological characteristics than similar individuals in the general population. In addition, all individuals participating in the identification study were white Caucasian. Whilst this reflected the limited ethnic mix of the clinic population, this may not be true of other UK obesity clinic populations and differs from similar US NES populations (Allison et al. 2007b; Lundgren et al. 2006). Thus caution must be taken in applying these findings to other ethnic groups, especially as cultural differences may influence the timing and structure of meal times. Similarly, 'standard' evening meal and sleep/wake times differ considerably between countries, with some Mediterranean countries, for example sleeping in the day and eating late at night as part of the usual daily routine. However, in the context of obesity, deviation from normal routines is less relevant than the level of impairment caused by the behaviours such as night eating and poor sleep quality.

The use of different analytical approaches may have some impact on the generalisation of findings. In the prevalence study, a quantitative approach using correlational analysis was taken, resulting in findings which are likely to be applicable to other morbidly obese populations. Retrospective thematic analysis of interview data however, is more context-specific. Thus key findings in the characterisation study, such as the effect of parental control over childhood overeating on the development of NEB, may be true in a general sense although the strength of the relationship and its relative importance may vary between populations and would require further investigation before its true impact on NEB is determined.

6.4.2. *Future Research.*

Individuals with NEB in the identification and characterisation studies were characterised by poor sleep, severe depression, chaotic eating patterns and lack of work.

Individuals without NEB had similar poor sleep patterns and low mood. Whilst the diagnostic debate about NEB continues, it may be useful to move the focus away from night-eating onto poor sleep quality. A treatment option based on Social Rhythm Theory may help individuals with poor sleep and also have an impact on night-eating. According to the Social Rhythm Theory, socializing with other individuals is an important feature of daily living as meeting and eating with others tends to be at standard times and has the effect of regulating social rhythms and anchoring bed and rising time (Carney et al. 2006). Thus work, eating and sleep patterns act as external social cues which anchor our daily routine and are known as 'zeitgebers' (German word meaning time-giver). These cues have an effect on the suprachiasmatic nucleus (SCN), the internal pacemaker in the anterior hypothalamus and entrain the human and circadian rhythms' free running cycle (Grandin, Alloy, & Abramson 2006). Thus the primary path of Ehler's social zeitgeber theory (Ehlers, Frank, & Kupfer 1988) consists of a chain of events in which instability of social rhythms can lead to dysregulation of the SCN and a resulting instability in specific biological rhythms. For example, Stetler, Dickerson & Miller (2004), identified that the circadian rhythms of cortisol could be influenced by social activities involving other people but that the influence was lessened if the activities were non social. Morin, Rodrigue & Ivers (Morin, Rodrigue, & Ivers 2003) showed a similar relationship with sleep. Individuals with a relatively delayed sleep-wake cycle or variable rise time were more likely to be poor sleepers. Poor sleepers have greater schedule variability in the timing of social rhythms and decreased activity levels, especially in those activities involving social interaction. This latter effect is no longer present when controlling for mood, suggesting an effect that may be mood-mediated. Others report similar findings (Brown et al. 1996). Thase & Trivedi (2002) and Stetler, Dickerson & Miller (2004), describe the attenuation theory which suggests depressive episodes arise as a consequence of life events disturbing social zeitgebers and that a biological abnormality in depressed individuals' pacemakers may make it difficult for them to entrain to their social environments, perhaps explaining the observed desynchronicity of circadian rhythms in depressed individuals. There is consistent evidence that depressed individuals find social interactions to be less enjoyable and less intimate compared to non-depressed individuals (Nezlek, Hampton, & Shean 2000). These individuals score lower on The Social Rhythm Metric

developed by Monk et al. (1990) and score higher than normal controls on the Social Rhythm Disruption Rating Scales developed by Frank et al. (1999) and which measures the degree of disruption in ones sleep-wake cycle brought about by a life event (Haynes et al. 2006).

NES is seen as a disorder of circadian rhythm dysfunction. All individuals in the identification study with NEB were characterised by disruption in all common ‘zeitgebers’, not only sleep, and were more depressed than other obese individuals without NEB. It is possible that individuals with NEB would benefit from treatment which promotes daily routines and socialisation and results in good sleep such as Interpersonal and Social Rhythm therapy (Frank, Swartz, & Kupfer 2000). Its focus is on helping individuals identify and maintain the regular routines of everyday life, including sleep and solve interpersonal issues and problems that may impact on these routines. Longitudinal studies examining the temporal associations of ‘zeitgebers’ such as work, mealtimes, sleep, mood, and biological and circadian rhythms in obese individuals may benefit all individuals with poor sleep quality, regardless of whether their disturbed sleep involves night eating behaviour or not.

6.4.3. *Implications for Practice*

Since commencing this programme of studies, awareness of NEB in the WMC has grown and initial assessments performed by both clinicians and dietitians routinely include questions related to night-eating. Capturing night-eating in food diaries is also now common practice. However within UK obesity clinics generally, awareness of NEB remains poor and is likely to do so until agreement is reached on the diagnostic criteria and a definitive screening tool is agreed. Awareness of the significant impact of poor sleep quality on obese populations is growing and there is a need for simple user-friendly tools to help clinicians distinguish between physical and psychological causes of poor sleep in obese groups. One proposed solution is to establish joint sleep and obesity assessment clinics, which would allow clinicians to share expertise and make quicker diagnoses in this specialist group.

Findings from the identification and characterisation studies indicate that NEB pathways are complex, resulting in varying levels of physical and psychosocial dysfunction, which differ between individuals. Treatment options need to reflect this diversity. Whilst negative affect

appears as a presenting feature for most NEB individuals, intensive psychological therapy may only be available for individuals who report extremely poor dietary control and associated psychological distress. For the majority of obese individuals with NEB and other related comorbidity, a variety of interventions may be appropriate, including support from Occupational Therapists to improve physical functioning at night and input from psychiatric nurses, specialist nurses and dietitians trained in cognitive behavioural therapy to help entrain social rhythms. As a minimum, there is a need for all therapists associated with obesity management to undergo training in the management of psychosocial function.

6.5. *Personal Reflection*

Although the undertaking of a thesis is a process of discovery, it is also an opportunity for self-reflection. Schon (1983) describes ‘the cognitive examination of experience’ as an essential component of continuous learning. Despite feeling considerable pride at developing my own initial research idea to a fruitful conclusion, reflecting back over the process as a whole, I recognise that I have found it challenging to deliver a thesis on a part-time basis in a timely manner. At the start of the process I did not appreciate the considerable time required for indirect research activity, such as progress reports and ethical applications and often found it challenging to balance time spent on these activities with direct research activity and full-time employment, despite study support from my employer. A tendency to agonise over detail at the expense of the bigger picture and a writing style that was initially very wordy and non-scientific, contributed to the challenges of time management.

As a novice researcher with no experience of the contingencies of research, I did not anticipate fully how much I would need to question the findings of other experienced researchers in my field of study. The findings in the identification study were not what I expected based on the research literature, although my clinical experience reassured me that the participants’ reports of their experiences were accurate. For some time I struggled to ‘make the pieces in the jig-saw fit’. With the support of my supervisory team, I was eventually able to consider that the jig-saw pieces may be correct, but that the ‘picture on the lid’ may be different and this gave me the confidence to develop the thesis in an alternative direction using different methodologies. As a result, I have learned to be more adaptable and to trust my clinical instincts earlier. I am also

aware of the importance of being open to a variety of methodologies when designing future studies in order to choose the most appropriate method to answer the question as opposed to adapting a question to fit a preferred methodology.

Extensive reading and interpretation of the research findings of others in the field has helped me develop my analytical skills as I have learnt to look beyond the simple results of a study, and to understand the wider agendas and collaborations of research groups. With regard to my individual learning style in particular, I have recognised that if I am struggling to understand an abstract concept, once I seek out a practical application it usually makes more sense and that I benefit particularly from learning in small groups such as tutorials and supervisory meetings.

6.6. *Conclusion*

This is the first study in the UK to investigate NES in a morbidly obese population. Despite a lack of consensus in the research community as to the characteristics of NES, investigation in this study has revealed NEB to be characterised by eating behaviour which is compulsive, chaotic and uncontrolled and in the context of negative emotions, low mood and conflictful relationships. It shares features of other eating disorders and future research is required to explore this relationship further. A substantial percentage of individuals developed NEB in childhood as a result of parental restrictions on food intake and this must be taken into account when treatments for childhood obesity are considered. An unexpected finding of this study was the large numbers of morbidly obese individuals experiencing poor sleep quality, of which individuals with NEB appeared to be a sub-group primarily experiencing more severe depression. Social Rhythm Therapy may be a possible treatment for individuals with impaired sleep, whether symptoms of NEB are present or not.

Appendix 1. The Eating Disorder Examination

RESTRAINT OVER EATING (Restraint subscale)

***Over the past 4 weeks have you been consciously trying to restrict what you eat, whether or not you have succeeded?**

Has this been to influence your shape or weight?

0 No attempt at restraint

1

2 Attempted to exercise restraint on less than half the days

3

4 Attempted to exercise restraint on more than half the days

5

6 Attempted to exercise restraint every day. []

Rate the number of days on which the subject has consciously attempted to restrict his or her food intake, whether or not he or she has succeeded.

The restraint should have been intended to influence shape, weight, or body composition, although this may not have been the sole or main reason.

It should have consisted of planned attempts at restriction, rather than spur-of-the-moment attempts such as the decision to resist a second helping.

Appendix 2. The Night Eating Syndrome History and Inventory (NESHI)

To be discussed verbally with subject

(Some areas are covered in the description of a 'typical day' at the start of the EDE)

- How hungry are you usually in the morning?
- What time do you typically get up each day?
- When do you usually eat for the first time?
- (If breakfast is eaten...). What do you typically eat for breakfast?
- When do you eat lunch? What do you typically eat?
- Do you snack between lunch and your evening meal? If yes, how much?
- When do you eat your dinner/evening meal? What do you typically eat?
- Do you have cravings or urges to eat snacks after supper, but before bedtime?
- If yes, what do you typically eat?
- How much control do you have over your eating between supper and bed time?
- How much of your daily food intake do you consume *after* suppertime?(Based on description of typical day and responses to above questions, make an educated estimate with the patient about their % of caloric intake after their evening meal.)
- Are you currently feeling blue or down in the dumps?
- When you are feeling blue, when is your mood lowest?
- How often do you have trouble getting to sleep?
- When do you usually go to bed? What is that like for you?
- Other than only to use the bathroom, how often would you get up at least once in the middle of the night?
- Do you have cravings or urges to eat snacks when you wake up at night?
- Do you need to eat in order to get back to sleep when you awake at night?
- When you get up in the middle of the night, how often do you snack?
- How much do you typically eat?
- When you snack in the middle of the night, how aware are you of what you are eating?
- How much control do you have over your eating while you are up at night?
- How long have your current difficulties with night eating been going on?

If night eating is suspected proceed to the next section.

DESCRIPTION OF NIGHT EATING

1. What foods do you prefer to eat when you eat at night? (*Circle all that apply*)
 - Meat
 - Ice cream/dairy products
 - Bread/toast
 - Pasta
 - Sweets/chocolate
 - Fruit/veg
 - Cereal
 - Crisps
 - Are these foods you eat regularly during the day? Y N
2. Do you ever make yourself sick after night eating ? Y N
3. On a scale of 0 to 100, with 0 being ‘no control’ and 100 being ‘total control’, how would you rate the amount of control you have over your night eating episodes?
4. At what age did you first begin night eating?
(Fill in exact age on blank if known and mark category)
 - a. before 12
 - b. between 12-18
 - c. between 18-30
 - d. between 30-55
 - e. 55 and over
5. When did you notice your night eating in relation to your weight?
 - a. overweight before began night eating
 - b. normal weight before began night eating
 - c. underweight before began night eating
6. How often did you diet before night eating began?
 - a. never
 - b. once every couple of years
 - c. once or twice a year
 - d. once every 3 months or more
7. Before you began night eating did you (circle all that apply)
 - a. stay up late at night regularly
 - b. work a night shift
 - c. endure a stressful event; if yes, identify
8. Does your night eating come and go?
 - a. Yes
 - b. No

What affects it?

9. Is your night eating consistent throughout the week?
 - a. same throughout the week
 - b. less on weekends/days off
 - c. more on weekends/days off

10. Does anyone in your family have symptoms of night eating?

11. Are you
 - a. married
 - b. divorced (was this associated with night eating (y/n)
 - c. co-habiting
 - d. living alone
 - e. living with roommates

12. Do you have a bed partner? Y/N.

If so, does your night eating affect your sleeping arrangement?
(Does your partner sleep in another bed because of the night eating?) Y/N

13. What strategies have you tried to stop night eating and were they successful?

14. Have you tried any over the counter remedies?

15. Any prescription medications?

WEIGHT AND DIET HISTORY

1. What has been your lowest adult weight (not due to illness, which you have maintained for at least 1 year)?

.....wt.....age.....maintained for.....years

2. What is the heaviest you have weighed? How old were you?

3. How much did you weigh
 - a. when you left school?
 - b. early twenties?
 - c. married?
 - d. other notable times?

4. Were you overweight as a child? At what age?

5. How do you remember being overweight at this time (pictures, clothes size, others telling you)?

6. What was your birth weight?

7. Diet History - Age and type of diet

Appendix 3. The Beck Depression Inventory

This questionnaire consists of 21 groups of statements. After reading each group of statements carefully, circle the number (0,1,2,3) next to the one statement in each group which best describes the way you have been feeling the past week, including today. If several statements within a group seem to apply equally well, circle each one. Be sure to read all the statements in each group before making your choice.

1. 0 I do not feel sad.
 1 I feel sad.
 2 I am sad all the time and I can't snap out of it.
 3 I am so sad or unhappy that I can't stand it.

2. 0 I am not particularly discouraged about the future
 1 I feel discouraged about the future.
 2 I feel I have nothing to look forward to.
 3 I feel that the future is hopeless and that things can't improve.

3. 0 I do not feel like a failure.
 1 I feel I have failed more than the average person
 2 As I look back on my life, all I can see is a lot of failures.
 3 I feel I am a complete failure as a person.

4. 0 I get as much satisfaction out of things as I used to
 1 I don't enjoy things the way I used to.
 2 I don't get real satisfaction out of anything anymore
 3 I am dissatisfied or bored with everything.

5. 0 I don't feel particularly guilty.
 1 I feel guilty a good part of the time.
 2 I feel quite guilty most of the time.
 3 I feel guilty all of the time.

6. 0 I don't feel I am being punished.
 1 I feel I may be punished.
 2 I expect to be punished.
 3 I feel I am being punished.

7. 0 I don't feel disappointed in myself.
 1 I am disappointed in myself.

- 2 I am disgusted with myself.
 3 I hate myself.
- 8.** 0 I don't feel I am any worse than anybody else
 1 I am critical of myself for my weaknesses or mistakes.
 2 I blame myself all the time for my faults.
 3 I blame myself for everything bad that happens.
- 9.** 0 I don't have any thoughts of killing myself.
 1 I have thoughts of killing myself, I would not carry them out.
 2 I would like to kill myself.
 3 I would kill myself if I had the chance.
- 10.** 0 I don't cry any more than usual.
 1 I cry more now than I used to.
 2 I cry all the time now.
 3 I used to be able to cry, but now I can't cry even though I want to.
- 11.** 0 I am no more irritated now than I ever am.
 1 I get annoyed or irritated more easily than I used to.
 2 I feel irritated all the time now.
 3 I don't get irritated at all by the things that used to irritate me.
- 12.** 0 I have not lost interest in other people.
 1 I am less interested in other people than I used to be
 2 I have lost most of my interest in other people.
 3 I have lost all of my interest in other people.
- 13.** 0 I make decisions about as well as I ever could.
 1 I put off making decisions more than I used to.
 2 I have greater difficulty in making decisions than before.
 3 I can't make decisions at all anymore.
- 14.** 0 I don't feel I look any worse than I used to.
 1 I am worried that I am looking old or unattractive.
 2 I feel that there are permanent changes in my appearance that make me look

- 3 unattractive.
○ 3 I believe that I look ugly.
15. ○ 0 I can work about as well as before
○ 1 It takes an extra effort to get started at doing something.
○ 2 I have to push myself very hard to do anything.
○ 3 I can't do any work at all.
16. ○ 0 I can sleep as well as usual.
○ 1 I don't sleep as well as I used to.
○ 2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
○ 3 I wake up several hours earlier than I used to and cannot get back to sleep.
17. ○ 0 I don't get more tired than usual.
○ 1 I get tired more easily than I used to.
○ 2 I get tired from doing almost anything.
○ 3 I am too tired to do anything.
18. ○ 0 My appetite is no worse than usual.
○ 1 My appetite is not as good as it used to be.
○ 2 My appetite is much worse now.
○ 3 I have no appetite at all anymore.
19. ○ 0 I haven't lost much weight, if any, lately.
○ 1 I have lost more than 5 pounds.
○ 2 I have lost more than 10 pounds
○ 3 I have lost more than 15 pounds.

I am purposely trying to lose weight by eating less. Yes__ No_

20. ○ 0 I am no more worried about my health than usual.
○ 1 I am worried about physical problems such as aches and pains; or upset stomach; or constipation.
○ 2 I am very worried about physical problems and it's hard to think of much else.
○ 3 I am so worried about my physical problems that I can't think about anything else.
21. ○ 0 I have not noticed any recent change in my interest of sex.
○ 1 I am less interested in sex than I used to be.
○ 2 I am much less interested in sex now.
○ 3 I have lost interest in sex completely.

Appendix 4. NES Study Screening Tool

Please answer each question by circling the number that best describes your behaviour over the past 3 months

Q1 How hungry are you usually in the morning?

0	1	2	3	4
Not at all	A little	Somewhat	Moderately	Very

Q2 When do you usually eat for the first time?

0	1	2	3	4
Before 9am	9.01-12am	12.01-3pm	3.01pm-6pm	After 6.01pm

Q3 Do you find it difficult to eat breakfast?

0	1	2	3	4
Not at all	A little	Somewhat	Moderately	Very

Q4 Would you say that food dominates your life?

0	1	2	3	4
Never	Sometimes	Half the time	Usually	Always

Q5 How much of your daily food intake do you consume after suppertime?

0	1	2	3	4
0%	1-25%	26-50%	51-75%	75-100%

Q6 How often do you have trouble getting to sleep?

0	1	2	3	4
Never	Sometimes	Half the time	Usually	Always

Q7 Apart from when you just get up to go the toilet, how often do you get up at least once in the middle of the night?

0	1	2	3	4
Never	Less than once a week	Once a week	More than once a week	Every night

Q8 When you get up in the middle of the night, how often do you snack?

0	1	2	3	4
Never	Sometimes	Half the time	Usually	Always

Q9 Do you ever eat large amounts of food in a short period of time (larger than most people would eat during a similar time and under similar circumstances)?

0	1	2	3	4
Never	Rarely	Occasionally	often	Nearly all the time

Q10 Have you recently lost more than 1 stone in a 3 month period? (please circle yes or no)

Yes

No

Q11 Do you worry you have lost control over how much you eat?

0	1	2	3	4
Never	Sometimes	Half the time	Usually	Always

Q12 Do you have urges to eat snacks after supper, but before bedtime?

0	1	2	3	4
Never	Sometimes	Half the time	Usually	Always

Q13 Do you ever make yourself sick because you feel uncomfortably full?

0	1	2	3	4
Never	Sometimes	Half the time	Usually	Always

Q14 If you snack in the middle of the night, how aware are you of your eating?

0	1	2	3	4
Unaware	Mostly unaware	Partially aware	Mostly aware	Completely aware

Tick here if you don't snack in the middle of the night

Q15 Do you ever feel low in mood?

0	1	2	3	4
Never	Sometimes	Half the time	Usually	Always

Do not answer the next question if you are never low in mood.

Q16 When you are feeling low in mood, is your mood better in the?

0	1	2	3	4
Early morning	Late morning	Afternoon	Early evening	Late evening/nighttime

Q17 How long have you experienced your current pattern of eating and sleeping?

0	1	2	3	4
Less than 3 months	Less than 6 months	Less than 9 months	Less than 12 months	More than 12 months

In order to help us with our statistical analysis, we would be grateful if you could provide the following details:

Male/Female

Age

Height

Weight

If you have any concerns about your response to the questionnaire

Are you currently employed? Yes/no

NES Questionnaire

Are you a student ? Yes/no

If you are employed, what is the name of your job?

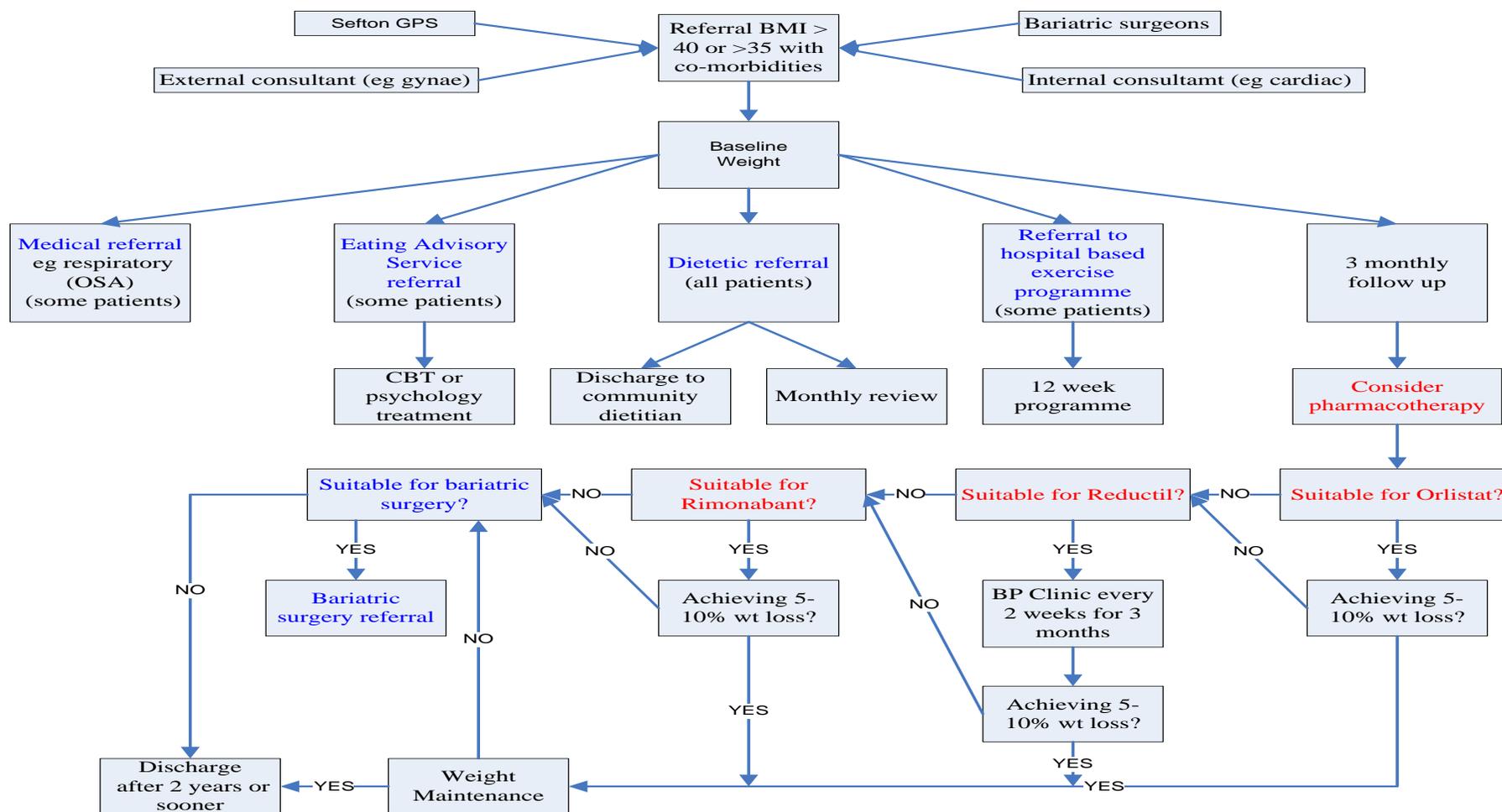
If you are employed, do you work part time? Yes/no

Please describe as carefully as possible the work that you do?

Name (optional)

Contact number (optional)

Appendix 5. Weight Management Clinic Pathway



Appendix 6. Matching of Sub-NES Group Characteristics with 2003 NES Criteria

Table 58 - Matching of sub-threshold NES group characteristics with the 2003 NES criteria

<i>Case no</i>	<i>Night Eating Syndrome Criteria</i>					<i>≥3 months</i>	<i>Absence of other ED</i>	<i>No of positive criteria</i>	<i>Other ED</i>	<i>Notes</i>
	<i>Am anorexia</i>	<i>Evening hyperphagia</i>	<i>Night awakenings</i>	<i>Eat during awakenings</i>						
66	0	0	1	0	1	1	3		Wakes often, eats <x1 week	
17	0	0	1	1	1	1	4			
39	0	0	1	1	1	1	4			
51	0	0	1	1	1	1	4			
23	0	1	1	1	1	1	5	Sub BU		
52	1	0	0	0	1	1	3		Eats until bedtime 1-5am	
73	1	0	0	0	1	1	4		Eats at 2am x3 week	
62	1	0	0	1	1	1	4		Sleeps in day, wakes to eat	
80	1	0	0	1	1	1	4		Sleeps in day, wakes to eat	
7	1	0	1	1	1	1	5			
8	1	0	1	1	1	1	5	Sub BED		
16	1	0	1	1	1	1	5			
47	1	0	1	1	1	1	5			
58	1	0	1	1	1	1	5			
65	1	0	1	1	1	1	5			
29	1	1	0	0	1	1	4		Eats until bedtime at 4am	
49	1	1	0	0	1	1	4		Eats until bedtime at 3am	
55	1	1	0	0	1	1	4		Eats until bedtime at 1.30am	
68	1	1	0	0	1	1	4		Eats until bedtime at 6am	

9	1	1	0	1	1	1	5		wakes x2week to eat (bed 3am)
27	1	1	0	1	1	1	5	Sub BU	Wakes x1 week, always eats
26	1	1	1	0	0	1	4		Eats until bedtime at 3 am
20	1	1	1	0	1	1	5	Sub BU	high calorie drinks at night
11	1	1	1	1	1	0	5	BU	
n (%)	19 (79)	10 (41)	14(58)	15 (62)	23 (96)	23 (96)			
positive									

<i>NES criteria</i>	
critterion	0 = negative for the criterion, 1= positive
1.	Morning anorexia, even if subject eats breakfast
2.	Evening hyperphagia: >50% kcal consumed in snacks after the last evening meal
3.	Awakenings at least once a night, at least 3 nights a week.
4.	Consumption of high calorie snacks during awakenings on frequent occasions
5.	Pattern occurs for a period of at least 3 months
6.	Absence of other eating disorders

Appendix 7. List of NVivo Codes

Name Activity	Name activity at night cooking day to day exercise need to be occupied physical functioning sex shopping social life work and study	
Barriers	Name blame self blame someone or something else procrastination resignation sabotage by others sabotage by self	
Change	Name accept help can't be bothered motivating factors motivation negative thinking planning positive thinking recognise need to change take responsibility third person	
Control	Name compulsion control after evening meal and before bed control over eating dietary rules empty stomach food avoidance yes no	Name how often avoided types avoided
Eating at night	laxatives and diuretics restraint restraint for eight hours thinking of food between meals	Name
	Name duration of night eating behaviour get up and eat yes no	Name

Eating patterns

late eating before bed yes no

affects other family members
aware of eating
craving to eat
current trigger
frequency
how long after bed time
initial trigger
location
need to eat to return to sleep
time of eating

Name
drink after midnight
eat after midnight
trigger

Name
drink four

Name
time and frequency
type

drink one

Name
time and frequency
type

drink three

Name
time and frequency
type

drink two

Name
time and frequency
type

eat in public
eat with family
Eating breakfast
meal four

Name
time and frequency
type

meal one

Name
time and frequency
type

meal three

Name
time and frequency
type

meal two

		Name time and frequency type
	Proportion of kcals after last evening meal secret eating snacking yes no	
		Name after meal four after meal one after meal three after meal two before meal one type of food variability of snacking
Episodes of overeating	variability of routine Name amount awareness of amount awareness of bingeing binging emotion during overeating end emotion end trigger event frequency location overeating episodes yes no rapid eating trigger emotion trigger event vomit	
Family and friends	Name key players living arrangements relationship with helpers relationships with family and friends	Name concern conflict disgust fear intimidation pressure pride resentment respect sense of loss shame support
Food emotions	Name addiction bored with food comfort cravings disconnected emptiness	

enjoyment
Entitlement

feel need to eat
feeling deprived
greedy
guilt
habit
hunger
hunger in the morning
obsessed with food
respect for food
satisfied
struggle
temptation
too tired to eat
torment

General emotions

Name
agrophobia
annoyance
anxiety
bitter
content
crutch
delusion
devastated
distraction
emotional distance
emotional stress
freedom
frustration
get bored
grief
lazy
lonely
low mood
relaxed
shame
shock
tired
unhappy
upset

Personality

Name
abraasive
all or nothing
competitive
determined
laugh at self
odd one out
rebel
self confidence
self harm
self image
self judgement

Name
misbehaving

Sleep

self loathing

Name
attempts to change sleep pattern
location
partner
patterns

Name
bed sleep time
duration of sleep
pattern
get up in night
Type

get wake up time
length of sleep
sleep in day
wake during sleep

Weight

quality of sleep

Name
attitude to weight

Name
body parts
concern over
weight gain
desire to lose
weight
easy for others
judged by others
like anorexia
obsession with
weight
other obese
people
shape or weight
weighing self

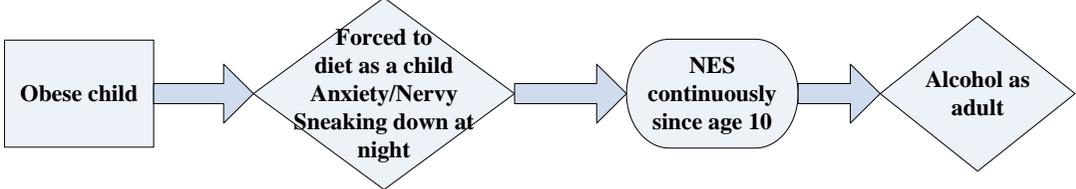
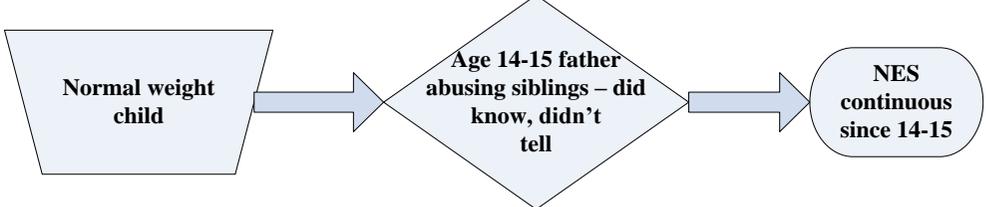
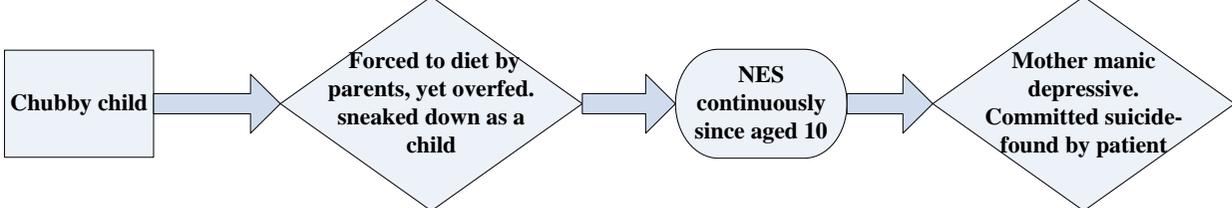
weight history

Name
changes in weight
family weight
key events
previous weight
loss attempts

Appendix 8. Pathway of Obesity, NEB and Life Events for each NEB Participant.

Table 59 - Partial NES Developing in Childhood and Teenage Years

Participant Number	Individual Participant Pathway	Co-morbidity
7	<pre> graph LR A[Obesity from aged 10.] --> B[Good childhood, No trauma identified. Sneaked down at night as a child] B --> C(NES continuously since age 11-12) </pre>	Type 2 diabetes Depression
8	<pre> graph LR A[Biggish child] --> B{Depression age 13 (mother planned to abort)} B --> C(Age 16, chronic pain led to poor sleep) C --> D{ME age 19} D --> E(NES continuously since 19) E --> F[Subsequent weight gain] </pre>	Depression
9	<pre> graph LR A[Chubby child] --> B{Bullied age 13 due to Weight. Parents divorce - didn't talk about it} B --> C(Poor sleep At school, wake every hour at night - teachers say sleeping in day) C --> D(NEB -Eating at night) D --> E{Current stress debts} </pre>	

Participant Number	Individual Participant Pathway	Co-morbidity
17	 <pre> graph LR A[Obese child] --> B{Forced to diet as a child Anxiety/Nervy Sneaking down at night} B --> C((NES continuously since age 10)) C --> D{Alcohol as adult} </pre>	Depression OSA
20	 <pre> graph LR A[/Normal weight child/] --> B{Age 14-15 father abusing siblings - did know, didn't tell} B --> C((NES continuous since 14-15)) </pre>	Depression
65	 <pre> graph LR A[Chubby child] --> B{Forced to diet by parents, yet overfed. sneaked down as a child} B --> C((NES continuously since aged 10)) C --> D{Mother manic depressive. Committed suicide - found by patient} </pre>	

Participant Number	Individual Participant Pathway	Co-morbidity
68	<pre> graph LR A[Always had a weight problem] --> B{Cannabis addiction} B --> C(('Never a morning person at school', All night sessions on cannabis)) C --> D[Munchies and weight gain] D --> E{Depression age 18} E --> F((NES continuously since college)) </pre>	Depression

Table 60 - Full NES Developing in Childhood and Teenage Years

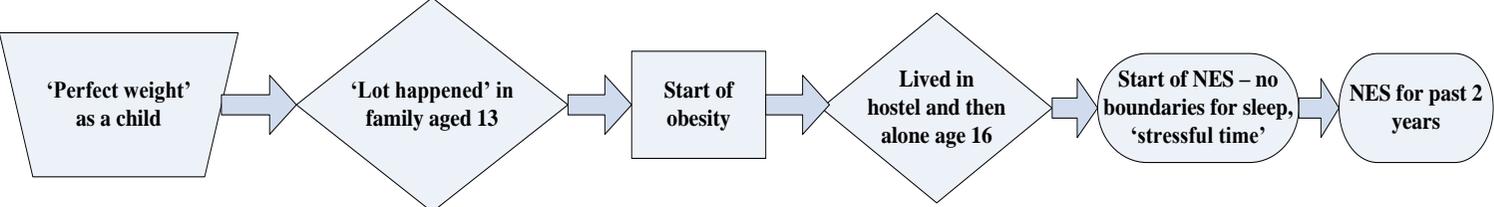
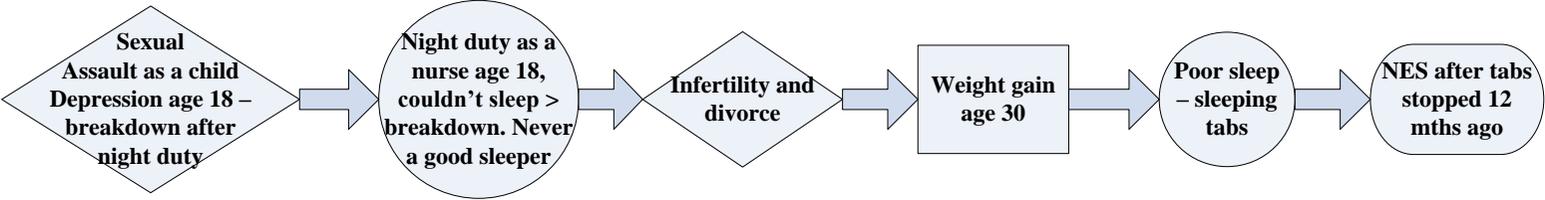
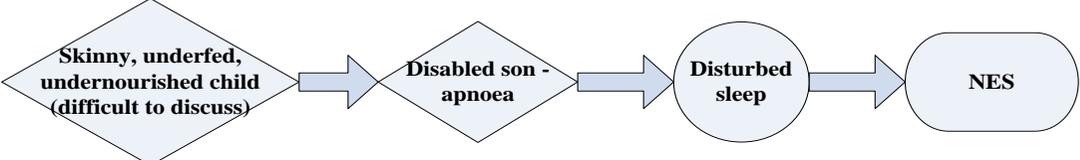
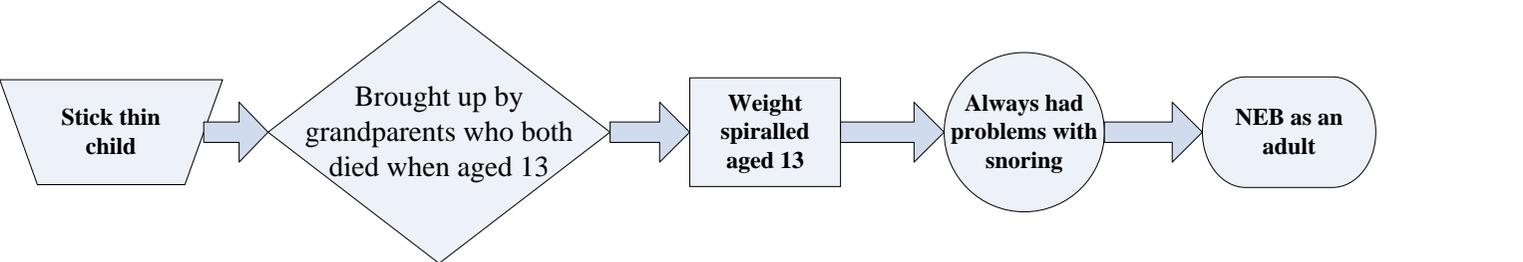
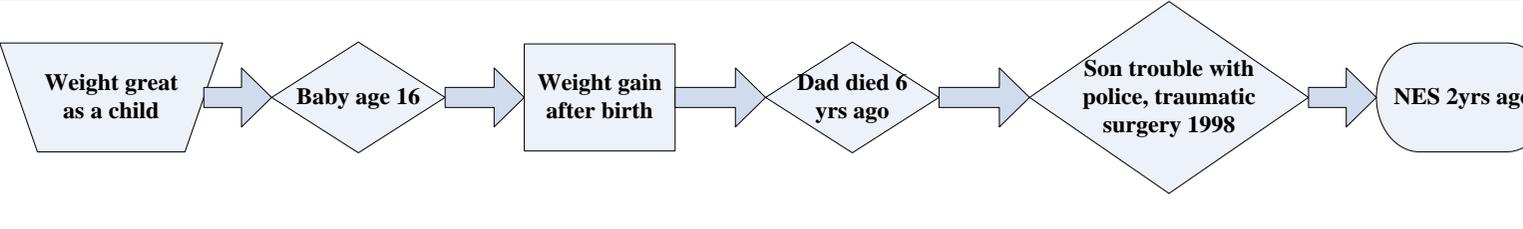
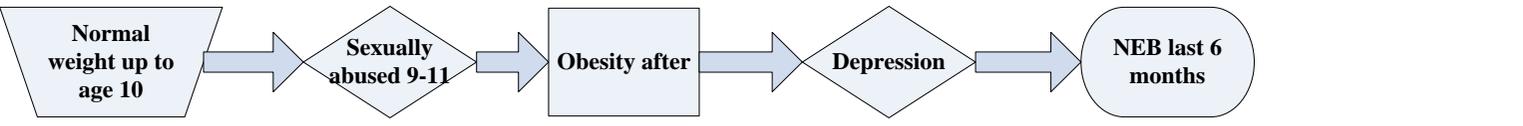
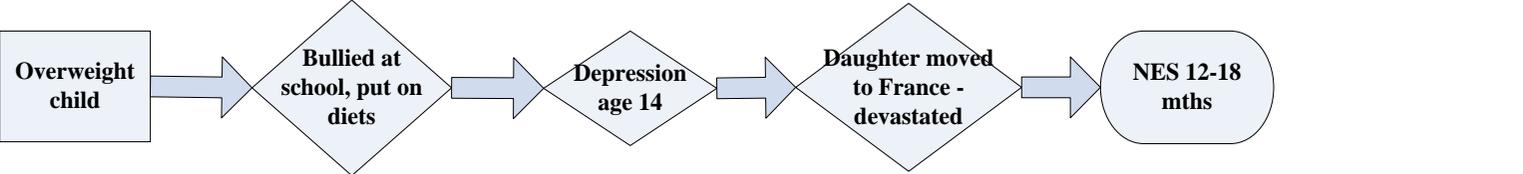
Participant Number	Individual Participant Pathway	Co-morbidity
10	 <pre> graph LR A[Hospitalised age 8 due to weight] --> B[More weight gain in teenage yrs.] B --> C{Absent mum. Conflict from father for eating} C --> D((Poor sleep in chair downstairs)) D --> E([NES as teenager]) </pre>	
40	 <pre> graph LR A[/'Perfect weight' as a child/] --> B{ 'Lot happened' in family aged 13 } B --> C[Start of obesity] C --> D{ Lived in hostel and then alone age 16 } D --> E([Start of NES - no boundaries for sleep, 'stressful time']) E --> F([NES for past 2 years]) </pre>	

Table 61 - Partial NES Developing in Adult Years

Participant Number	Individual Participant Pathway	Co-morbidity
16	 <pre> graph LR A{Sexual Assault as a child} --> B((Depression age 18 - breakdown after night duty)) B --> C((Night duty as a nurse age 18, couldn't sleep > breakdown. Never a good sleeper)) C --> D{Infertility and divorce} D --> E[Weight gain age 30] E --> F((Poor sleep - sleeping tabs)) F --> G([NES after tabs stopped 12 mths ago]) </pre>	Depression
23	 <pre> graph LR A{Skinny, underfed, undernourished child (difficult to discuss)} --> B{Disabled son - apnoea} B --> C((Disturbed sleep)) C --> D([NES]) </pre>	

26	 <pre> graph LR A[Stick thin child] --> B{Brought up by grandparents who both died when aged 13} B --> C[Weight spiralled aged 13] C --> D((Always had problems with snoring)) D --> E([NEB as an adult]) </pre>	OSA
27	 <pre> graph LR A[Weight great as a child] --> B{Baby age 16} B --> C[Weight gain after birth] C --> D{Dad died 6 yrs ago} D --> E{Son trouble with police, traumatic surgery 1998} E --> F([NES 2yrs ago]) </pre>	Depression
29	 <pre> graph LR A[Normal weight up to age 10] --> B{Sexually abused 9-11} B --> C[Obesity after] C --> D{Depression} D --> E([NES last 6 months]) </pre>	Type 2 diabetes OSA Depression
39	 <pre> graph LR A[Overweight child] --> B{Bullied at school, put on diets} B --> C{Depression age 14} C --> D{Daughter moved to France - devastated} D --> E([NES 12-18 mths]) </pre>	Depression

47	<pre> graph LR A[Obesity as a child] --> B((Poor sleep as a child)) B --> C{Bed wetter, given tablets to sleep 'lighter'} C --> D{Divorce} D --> E((NES after divorce in 20s to 30s)) </pre>	Type 2 diabetes
49	<pre> graph LR A[/Normal weight/] --> B{Broken leg age 17} B --> C[Weight gain after age 22] C --> D{Wife alcoholic also drink problem} D --> E{Stopped work, loved job} E --> F((NES past 2 years)) </pre>	Type 2 diabetes Depression
51	<pre> graph LR A[/Normal weight/] --> B{Pregnancy age 20} B --> C{Night feeds} C --> D[Weight gain after] D --> E((NES for 6-7 yrs)) </pre>	
52	<pre> graph LR A[/Skinny when younger/] --> B{SVT as a teenager and Thyroidectomy} B --> C{Depression} C --> D{Off work} D --> E((NES since stopping work)) </pre>	Depression

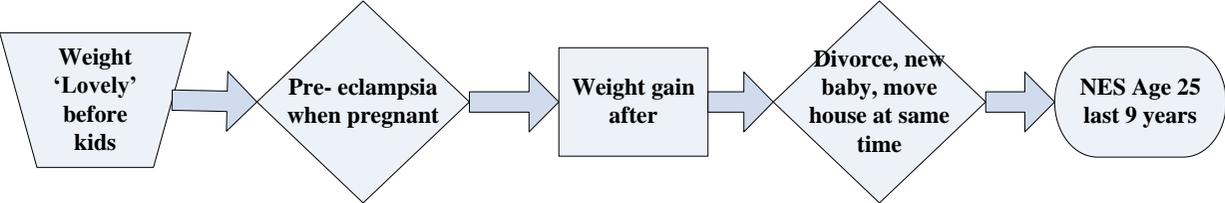
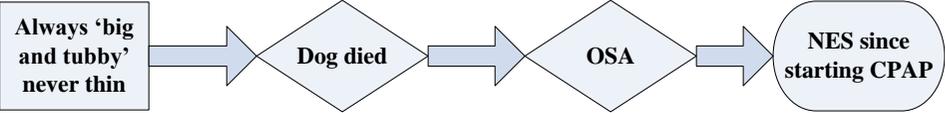
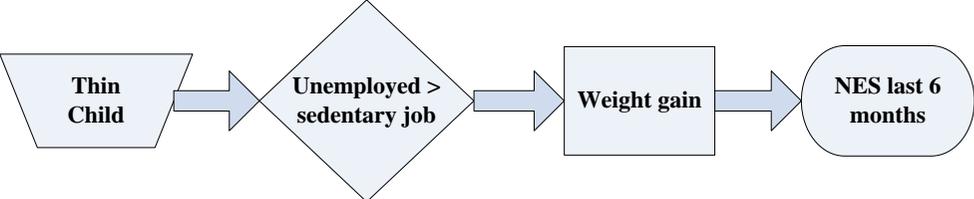
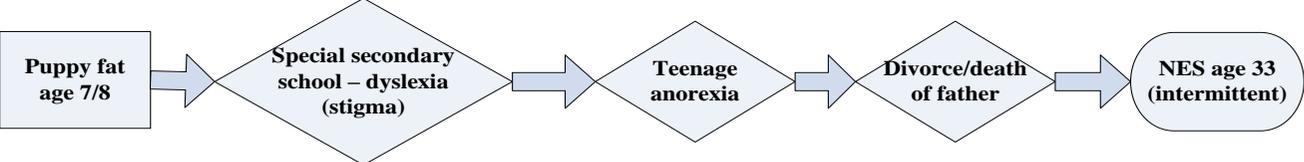
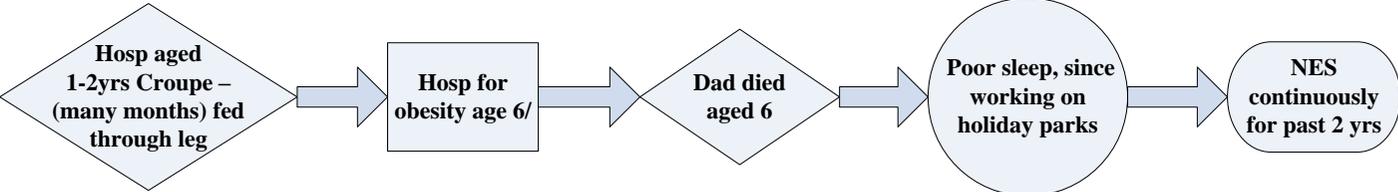
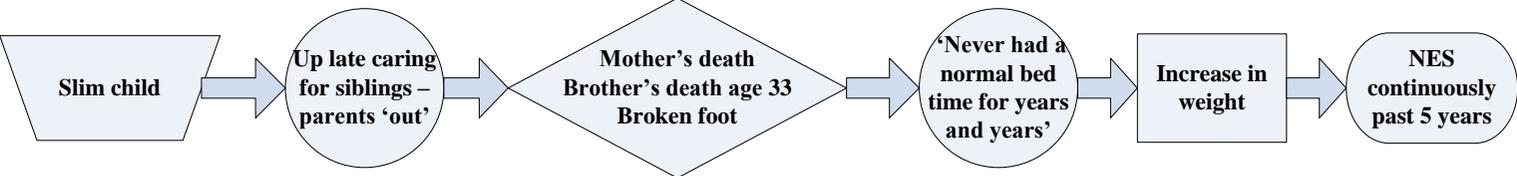
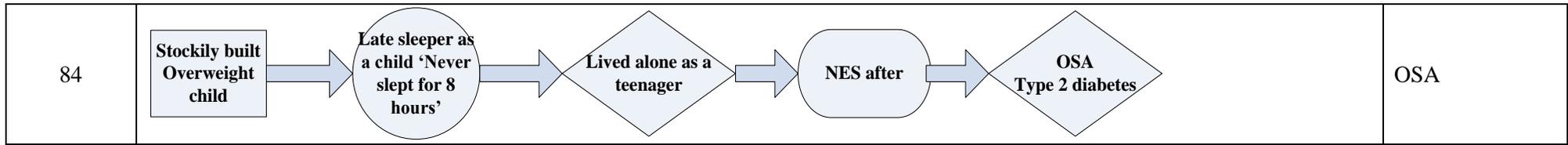
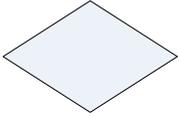
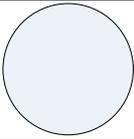
55		Depression
58		Type 2 diabetes OSA
62		Type 2 diabetes OSA Depression
66		Type 2 diabetes OSA

Table 62 - Full NES Developing in Adult Years

Participant Number	Individual Participant Pathway	Co-morbidity
4	 <pre> graph LR A[Puppy fat age 7/8] --> B{Special secondary school - dyslexia (stigma)} B --> C{Teenage anorexia} C --> D{Divorce/death of father} D --> E([NES age 33 (intermittent)]) </pre>	Depression
15	 <pre> graph LR A{Hosp aged 1-2yrs Croup - (many months) fed through leg} --> B[Hosp for obesity age 6/] B --> C{Dad died aged 6} C --> D((Poor sleep, since working on holiday parks)) D --> E([NES continuously for past 2 yrs]) </pre>	
24	 <pre> graph LR A[/Slim child/] --> B((Up late caring for siblings - parents 'out')) B --> C{Mother's death Brother's death age 33 Broken foot} C --> D(('Never had a normal bed time for years and years')) D --> E[Increase in weight] E --> F([NES continuously past 5 years]) </pre>	Depression



KEY	Symbol	Description
		Weight
		Life events
		Life narrative
		Sleep
		Night eating

Appendix 9. Sleep and Eating Patterns of NEB Group and Matched Controls

Characteristics of the NEB group.

The sleep and eating patterns of each individual in the NEB group (n=31) are presented pictorially on the top row of the spreadsheet which is included as a separate A3 sheet at the end of this thesis. Sleep and individual meal times are given individual colour codes. Trend lines are included for 7.0 am in the morning and 10.30 pm in the evening to help distinguish between day and night.

Characteristics of the matched control group

The second row on the spreadsheet contains the individual data for each of the 28 matched controls. Data for each control participant is displayed directly under the data of the NEB individual with whom the control was matched.

Appendix 10. The Prevalence Study Questionnaire

Instructions: The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

During the past month,

1. When have you usually gone to bed? _____
2. How long (in minutes) has it taken you to fall asleep each night? _____
3. When have you usually got up in the morning? _____
4. How many hours of actual sleep do you get at night? (This may be different than the number of hours you spend in bed) _____

Please tick the appropriate box below

5. During the past month, how often have you had trouble sleeping because you...	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
a. Cannot get to sleep within 30 minutes				
b. Wake up in the middle of the night or early morning				
c. Have to get up to use the bathroom				
d. Cannot breathe comfortably				
e. Cough or snore loudly				
f. Feel too cold				
g. Feel too hot				
h. Have bad dreams				
i. Have pain				
j. Other reason(s), please describe, including how often you have had trouble sleeping because of this reason(s):				
	Very good	Fairly good	Fairly bad	Very bad
6. During the past month, how would you rate your sleep quality overall?				

	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
7. During the past month, how often have you taken medicine (prescribed or “over the counter”) to help you sleep?				
8. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				

	No problem at all	Only a very slight problem	Somewhat of a problem	A very big problem
9. During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?				

These are optional questions and are not added into the score

10. Do you have a bed partner or roommate? **Please tick the appropriate answer**

- No bed partner or roommate _____
 Partner/roommate in other room _____
 Partner in same room, but not same bed _____
 Partner in same bed _____

Please tick the appropriate box below if you are able to answer

If you have a roommate or bed partner, ask him/her how often in the past month you have had...	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
(a) Loud snoring				
(b) Long pauses between breaths while asleep				
(c) Legs twitching or jerking while you sleep				
(d) Episodes of disorientation or confusion during sleep				
(e) Other restlessness while you sleep: please describe				

Sleep Questionnaire – The Epworth Sleepiness Scale

How likely are you to doze off or fall asleep during the following situations, in contrast to just feeling tired?	Would never doze/sleep	Slight chance of this happening	Moderate chance of this happening	High chance of this happening
a. Sitting and reading.				
b. Watching television.				
c. Sitting inactive in a public place, eg theatre, cinema, meeting.				
d. As a passenger in a car for an hour without a break.				
e. Lying down to rest in the afternoon when circumstances permit.				
f. Sitting and talking to someone.				
g. Sitting quietly after lunch without alcohol.				
h. In a car, while stopped for a few minutes in traffic.				

Directions: Please circle ONE answer for each question.

How hungry are you usually in the morning?

0	1	2	3	4
Not at all	A little	Somewhat	Moderately	Very

When do you usually eat for the first time?

0	1	2	3	4
Before 9am	9.01-12am	12.01-3pm	3.01pm-6pm	After 6.01pm

Do you have cravings or urges to eat snacks after supper, but before bedtime?

0	1	2	3	4
Not at all	A little	Somewhat	Very much so	Extremely so

‘supper’ is referring to your last evening meal.

How much control do you have over your eating between supper and bed time?

0	1	2	3	4
None at all	A little	Some	Very much	Complete

How much of your daily food intake do you consume *after* suppertime?

0	1	2	3	4
0% None	1-25% up to a quarter	26-50% about half	51-75% More than half	76-100% almost all

Are you currently feeling blue or down in the dumps?

0	1	2	3	4
Not at all	A little	Somewhat	Very much so	Extremely so

When you are feeling blue, is your mood lower in the

0	1	2	3	4
Early morning	Late Morning	Afternoon	Early evening	Late evening /night-time

How often do you have trouble getting to sleep?

0	1	2	3	4
Never	Sometimes	About half the time	Usually	Always

Other than only to use the bathroom, how often do you get up at least once in the middle of the night?

0	1	2	3	4
Never	Less than once a week	About once a week	More than once a week	Every night

If 0 on question 9, PLEASE STOP HERE

Do you have cravings or urges to eat snacks when you wake up at night?

0	1	2	3	4
Not at all	A little	Somewhat	Very much so	Extremely so

Do you need to eat in order to get back to sleep when you awake at night?

0	1	2	3	4
Not at all	A little	Somewhat	Very much so	Extremely so

When you get up in the middle of the night, how often do you snack?

0	1	2	3	4
Never	Sometimes	About half the time	Usually	Always

If 0 on question 12, PLEASE STOP HERE

When you snack in the middle of the night, how aware are you of what you are eating?

0	1	2	3	4
Not at all	A little	Somewhat	Very much so	Completely

How much control do you have over your eating while you are up at night?

0	1	2	3	4
None at all	A little	Some	Very much	Complete

How long have your current difficulties with night eating been going on?

.....months years

In order to help us with our statistical analysis, we would be grateful if you could provide the following details:

Male/Female

Age

Height (if known)

Weight (if known)

List of References

References

- Adami, G. F., Meneghelli, A., & Scopinaro, N. 1999, "Night eating and binge eating disorder in obese patients", *Int.J.Eat.Disord.*, vol. 25, no. 3, pp. 335-338.
- Adami, G. F., Campostano, A., Marinari, G. M., Ravera, G., & Scopinaro, N. 2002, "Night eating in obesity: a descriptive study", *Nutrition*, vol. 18, no. 7-8, pp. 587-589.
- Agargun, M. Y., Kara, H., & Solmaz, M. 1997, "Subjective sleep quality and suicidality in patients with major depression", *J.Psychiatr.Res.*, vol. 31, no. 3, pp. 377-381.
- Agras, W. S., Hammer, L. D., McNicholas, F., & Kraemer, H. C. 2004, "Risk factors for childhood overweight: a prospective study from birth to 9.5 years", *J.Pediatr.*, vol. 145, no. 1, pp. 20-25.
- Ajzen, I. 2002, "Perceived Behavioural Control, Self Efficacy, Locus of Control and the Theory of Planned Behaviour", *Journal of Applied Social Psychology*, vol. 32, no. 4, pp. 665-683.
- Algul, A., Ates, M. A., Semiz, U. B., Basoglu, C., Ebrinc, S., Gecici, O., Gulsun, M., Kardesoglu, E., & Cetin, M. 2009, "Evaluation of general psychopathology, subjective sleep quality, and health-related quality of life in patients with obesity", *Int.J.Psychiatry Med.*, vol. 39, no. 3, pp. 297-312.
- Allen, R. P. 2000, "Article reviewed: Behavioral and neuroendocrine characteristics of the night-eating syndrome", *Sleep Med.*, vol. 1, no. 1, pp. 67-68.
- Allison, K. C., Stunkard, A., & Thier, S. L. 2004, *Overcoming Night Eating Syndrome* New Harbinger Publications Inc, Oakland.
- Allison, K. Diagnosing Night Eating Syndrome. 2004. Personal Communication.
- Allison, K. C., Ahima, R. S., O'Reardon, J. P., Dinges, D. F., Sharma, V., Cummings, D. E., Heo, M., Martino, N. S., & Stunkard, A. J. 2005a, "Neuroendocrine profiles associated with energy intake, sleep, and stress in the night eating syndrome", *J.Clin.Endocrinol.Metab*, vol. 90, no. 11, pp. 6214-6217.
- Allison, K. C., Grilo, C. M., Masheb, R. M., & Stunkard, A. J. 2005b, "Binge eating disorder and night eating syndrome: a comparative study of disordered eating", *J.Consult Clin.Psychol.*, vol. 73, no. 6, pp. 1107-1115.
- Allison, K. C., Wadden, T. A., Sarwer, D. B., Fabricatore, A. N., Crerand, C. E., Gibbons, L. M., Stack, R. M., Stunkard, A. J., & Williams, N. N. 2006, "Night eating syndrome and binge eating disorder among persons seeking bariatric surgery: prevalence and related features", *Obesity.(Silver.Spring)*, vol. 14 Suppl 2, pp. 77S-82S.
- Allison, K. C., Crow, S. J., Reeves, R. R., West, D. S., Foreyt, J. P., Dilillo, V. G., Wadden, T. A., Jeffery, R. W., Van, D. B., & Stunkard, A. J. 2007a, "Binge eating disorder and night eating syndrome in adults with type 2 diabetes", *Obesity.(Silver.Spring)*, vol. 15, no. 5, pp. 1287-1293.

Allison, K. C., Grilo, C. M., Masheb, R. M., & Stunkard, A. J. 2007b, "High self-reported rates of neglect and emotional abuse, by persons with binge eating disorder and night eating syndrome", *Behav.Res.Ther.*, vol. 45, no. 12, pp. 2874-2883.

Allison, K. C., Engel, S. G., Crosby, R. D., de, Z. M., O'Reardon, J. P., Wonderlich, S. A., Mitchell, J. E., Smith, W. D., Wadden, T. A., & Stunkard, A. J. 2008a, "Evaluation of diagnostic criteria for night eating syndrome using item response theory analysis", *Eat.Behav.*, vol. 9, no. 4, pp. 398-407.

Allison, K. C., Lundgren, J. D., O'Reardon, J. P., Martino, N. S., Sarwer, D. B., Wadden, T. A., Crosby, R. D., Engel, S. G., & Stunkard, A. J. 2008b, "The Night Eating Questionnaire (NEQ): psychometric properties of a measure of severity of the Night Eating Syndrome", *Eat.Behav.*, vol. 9, no. 1, pp. 62-72.

Allison, K. C., Lundgren, J. D., Moore, R. H., O'Reardon, J. P., & Stunkard, A. J. 2010a, "Cognitive behavior therapy for night eating syndrome: a pilot study", *Am.J Psychother.*, vol. 64, no. 1, pp. 91-106.

Allison, K. C., Lundgren, J. D., O'Reardon, J. P., Geliebter, A., Gluck, M. E., Vinai, P., Mitchell, J. E., Schenck, C. H., Howell, M. J., Crow, S. J., Engel, S., Latzer, Y., Tzischinsky, O., Mahowald, M. W., & Stunkard, A. J. 2010b, "Proposed diagnostic criteria for night eating syndrome", *Int.J Eat.Disord.*, vol. 43, no. 3, pp. 241-247.

Andersen, G. S., Stunkard, A. J., Sorensen, T. I., Petersen, L., & Heitmann, B. L. 2004, "Night eating and weight change in middle-aged men and women", *Int.J.Obes.Relat Metab Disord.*, vol. 28, no. 10, pp. 1338-1343.

Aronoff, N. J., Geliebter, A., & Zammit, G. 2001, "Gender and body mass index as related to the night-eating syndrome in obese outpatients", *J.Am.Diet.Assoc.*, vol. 101, no. 1, pp. 102-104.

Atlantis, E. & Baker, M. 2008, "Obesity effects on depression: systematic review of epidemiological studies", *Int.J.Obes.(Lond)*, vol. 32, no. 6, pp. 881-891.

Ayas, N. T., White, D. P., Al-Delaimy, W. K., Manson, J. E., Stampfer, M. J., Speizer, F. E., Patel, S., & Hu, F. B. 2003a, "A prospective study of self-reported sleep duration and incident diabetes in women", *Diabetes Care*, vol. 26, no. 2, pp. 380-384.

Ayas, N. T., White, D. P., Manson, J. E., Stampfer, M. J., Speizer, F. E., Malhotra, A., & Hu, F. B. 2003b, "A prospective study of sleep duration and coronary heart disease in women", *Arch.Intern.Med.*, vol. 163, no. 2, pp. 205-209.

Backhaus, J., Junghanns, K., Broocks, A., Riemann, D., & Hohagen, F. 2002, "Test-retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia", *J.Psychosom.Res.*, vol. 53, no. 3, pp. 737-740.

Bass, J. & Turek, F. W. 2005, "Sleepless in America: a pathway to obesity and the metabolic syndrome?", *Arch.Intern.Med.*, vol. 165, no. 1, pp. 15-16.

Bastiaens, L., Riccardi, K., & Sakhrani, D. 2002, "The RAFFT as a screening tool for adult substance use disorders", *Am.J Drug Alcohol Abuse*, vol. 28, no. 4, pp. 681-691.

Bazeley, P. 2008, "Working with Data," in *Qualitative Data Analysis*, Sage, London, pp. 59-98.

- Beck, A. T., Ward, C. H., Mendleson, M., Mock, J., & Erbaugh, J. 1961, "An inventory for measuring depression", *Arch.Gen.Psychiatry*, vol. 4, pp. 561-571.
- Beck, Rush, Shaw, & Emery 1979, *Cognitive Therapy of Depression* Guilford Press, New York.
- Beck, A. T. & Steer, R. A. 1984, "Internal consistencies of the original and revised Beck Depression Inventory", *J.Clin.Psychol.*, vol. 40, no. 6, pp. 1365-1367.
- Beck, A. T., Steer, R. A., & Garbin, M. G. 1988, "Psychometric Properties of the Beck Depression Inventory: Twenty-five years of evaluation.", *Clinical Psychology Review*, vol. 8, pp. 77-100.
- Beck, Steer, & Brown 1996, *Beck Depression Inventory – II manual* Harcourt Brace, San Antonio TX.
- Beck, A. T., Steer, R. A., Ball, R., & Ranieri, W. 1996, "Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients", *J.Pers.Assess.*, vol. 67, no. 3, pp. 588-597.
- Beumont, P. J., Kopec-Schrader, E. M., Talbot, P., & Touyz, S. W. 1993, "Measuring the specific psychopathology of eating disorder patients", *Aust.N.Z.J.Psychiatry*, vol. 27, no. 3, pp. 506-511.
- Birketvedt, G. S., Florholmen, J., Sundsfjord, J., Osterud, B., Dinges, D., Bilker, W., & Stunkard, A. 1999, "Behavioral and neuroendocrine characteristics of the night-eating syndrome", *JAMA*, vol. 282, no. 7, pp. 657-663.
- Birketvedt, G. S., Sundsfjord, J., & Florholmen, J. R. 2002, "Hypothalamic-pituitary-adrenal axis in the night eating syndrome", *Am.J.Physiol Endocrinol.Metab*, vol. 282, no. 2, p. E366-E369.
- Bixler, E. O., Vgontzas, A. N., Lin, H. M., Calhoun, S. L., Vela-Bueno, A., & Kales, A. 2005, "Excessive daytime sleepiness in a general population sample: the role of sleep apnea, age, obesity, diabetes, and depression", *J.Clin.Endocrinol.Metab*, vol. 90, no. 8, pp. 4510-4515.
- Blaine, B. 2008, "Does depression cause obesity?: A meta-analysis of longitudinal studies of depression and weight control", *J.Health Psychol.*, vol. 13, no. 8, pp. 1190-1197.
- Blashfield, R. K., Sprock, J., & Fuller, A. K. 1990, "Suggested guidelines for including or excluding categories in the DSM-IV", *Compr.Psychiatry*, vol. 31, no. 1, pp. 15-19.
- Blundell, J. E. 2000, "What foods do people habitually eat? A dilemma for nutrition, an enigma for psychology", *Am.J.Clin.Nutr.*, vol. 71, no. 1, pp. 3-5.
- Boseck, J. J., Engel, S. G., Allison, K. C., Crosby, R. D., Mitchell, J. E., & de, Z. M. 2007, "The application of ecological momentary assessment to the study of night eating", *Int.J.Eat.Disord.*, vol. 40, no. 3, pp. 271-276.
- Boston, R. C., Moate, P. J., Allison, K. C., Lundgren, J. D., & Stunkard, A. J. 2008, "Modeling circadian rhythms of food intake by means of parametric deconvolution: results from studies of the night eating syndrome", *Am.J.Clin.Nutr.*, vol. 87, no. 6, pp. 1672-1677.

- Bower, B., Bylisma, L. M., Morris, B. H., & Rottenberg, J. 2010, "Poor reported sleep quality predicts low positive affect in daily life among healthy and mood-disordered persons", *J.Sleep Res.*, vol. 19, no. 2, pp. 323-332.
- Broman, J. E., Lundh, L. G., & Hetta, J. 1996, "Insufficient sleep in the general population", *Neurophysiol.Clin.*, vol. 26, no. 1, pp. 30-39.
- Brown & Harris 1978, *Social Origins of Depression: A study of Psychiatric Disorder in Women*.
- Brown, L. F., Reynolds, C. F., III, Monk, T. H., Prigerson, H. G., Dew, M. A., Houck, P. R., Mazumdar, S., Buysse, D. J., Hoch, C. C., & Kupfer, D. J. 1996, "Social rhythm stability following late-life spousal bereavement: associations with depression and sleep impairment", *Psychiatry Res*, vol. 62, no. 2, pp. 161-169.
- Brylinsky, J. & Moore, J. 1994, "The Identification of Body Build Stereotypes in Young Children", *J Res Pers*, vol. 28, pp. 170-181.
- Buchsbaum, D. G., Buchanan, R. G., Centor, R. M., Schnoll, S. H., & Lawton, M. J. 1991, "Screening for alcohol abuse using CAGE scores and likelihood ratios", *Ann.Intern.Med.*, vol. 115, no. 10, pp. 774-777.
- Busetto, L., Segato, G., De, L. M., De, M. F., Foletto, M., Vianello, M., Valeri, M., Favretti, F., & Enzi, G. 2005, "Weight loss and postoperative complications in morbidly obese patients with binge eating disorder treated by laparoscopic adjustable gastric banding", *Obes.Surg.*, vol. 15, no. 2, pp. 195-201.
- Buysse, D., Reynolds, C., Monk, T., Berman, S., & Kupfer, D. 1988, "The Pittsburgh Sleep Quality Index: A New Instrument for Psychiatric Practice and Research", *Psychiatry Research*, vol. 28, pp. 193-213.
- Buysse, D. J., Hall, M. L., Strollo, P. J., Kamarck, T. W., Owens, J., Lee, L., Reis, S. E., & Matthews, K. A. 2008, "Relationships between the Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), and clinical/polysomnographic measures in a community sample", *J.Clin.Sleep Med.*, vol. 4, no. 6, pp. 563-571.
- Cambell, M. M. B. P. M. F. S. J. 1984, "A factorail analysis of BDI scores", *Journal of Clinical Psychology*, vol. 40, pp. 992-996.
- Caracelli & Greene 1993, "Data Analysis Strategies for Mixed-Method Evaluation Designs", *Educational Evaluation and Policy Analysis*, vol. 15, no. 1, pp. 195-207.
- Carney, C. E., Edinger, J. D., Meyer, B., Lindman, L., & Istre, T. 2006, "Daily activities and sleep quality in college students", *Chronobiol.Int.*, vol. 23, no. 3, pp. 623-637.
- Carpenter, J. S. & Andrykowski, M. A. 1998, "Psychometric evaluation of the Pittsburgh Sleep Quality Index", *J.Psychosom.Res.*, vol. 45, no. 1 Spec No, pp. 5-13.
- Cavanaugh, S., Clark, D. C., & Gibbons, R. D. 1983, "Diagnosing depression in the hospitalized medically ill", *Psychosomatics*, vol. 24, no. 9, pp. 809-815.
- Ceru-Bjork, C., Andersson, I., & Rossner, S. 2001, "Night eating and nocturnal eating- two different or similar syndromes among obese patients?", *Int.J.Obes.Relat Metab Disord.*, vol. 25, no. 3, pp. 365-372.

- Chan, J. M., Rimm, E. B., Colditz, G. A., Stampfer, M. J., & Willett, W. C. 1994, "Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men", *Diabetes Care*, vol. 17, no. 9, pp. 961-969.
- Chaput, J. P., Drapeau, V., Hetherington, M., Lemieux, S., Provencher, V., & Tremblay, A. 2005, "Psychobiological impact of a progressive weight loss program in obese men", *Physiol Behav.*, vol. 86, no. 1-2, pp. 224-232.
- Charmaz, K. 2009, "Reconstructing Theory in Grounded Theory Studies," in *Constructing Grounded Theory: A practical guide through Qualitative Analysis*, Sage, Thousand Oaks, CA, pp. 123-150.
- Chen, E. Y., Bocchieri-Ricciardi, L. E., Munoz, D., Fischer, S., Katterman, S., Roehrig, M., Dymek-Valentine, M., Alverdy, J. C., & le, G. D. 2007, "Depressed mood in class III obesity predicted by weight-related stigma", *Obes.Surg.*, vol. 17, no. 5, pp. 669-671.
- Cizza, G., Skarulis, M., & Mignot, E. 2005, "A link between short sleep and obesity: building the evidence for causation", *Sleep*, vol. 28, no. 10, pp. 1217-1220.
- Coffey, A. & Atkinson, P. 1996, *Making sense of qualitative data: Complimentary research strategies* Sage, Thousand Oaks, CA.
- Cohrs, S. 2008, "Sleep Disturbances in Patients with Schizophrenia : Impact and Effect of Antipsychotics", *CNS.Drugs*, vol. 22, no. 11, pp. 939-962.
- Cole, J. C., Motivala, S. J., Buysse, D. J., Oxman, M. N., Levin, M. J., & Irwin, M. R. 2006, "Validation of a 3-factor scoring model for the Pittsburgh sleep quality index in older adults", *Sleep*, vol. 29, no. 1, pp. 112-116.
- Colles, S. L. & Dixon, J. B. 2006, "Night eating syndrome: impact on bariatric surgery", *Obes.Surg.*, vol. 16, no. 7, pp. 811-820.
- Colles, S. L., Dixon, J. B., & O'Brien, P. E. 2007, "Night eating syndrome and nocturnal snacking: association with obesity, binge eating and psychological distress", *Int.J.Obes.(Lond)*, vol. 31, no. 11, pp. 1722-1730.
- Colles, S. L., Dixon, J. B., & O'Brien, P. E. 2008a, "Loss of control is central to psychological disturbance associated with binge eating disorder", *Obesity.(Silver.Spring)*, vol. 16, no. 3, pp. 608-614.
- Colles, S. L., Dixon, J. B., & O'Brien, P. E. 2008b, "Grazing and Loss of Control Related to Eating: Two High-risk Factors Following Bariatric Surgery", *Obesity.(Silver.Spring)*, vol. 16, no. 3, pp. 615-622.
- Cooper, Z. & Fairburn, C. G. 1987, "The Eating Disorder Examination: A Semi-structured Interview for the Assessment of the Specific Psychopathology of Eating Disorders", *Int.J.Eat.Disord.*, vol. 6, no. 1, pp. 1-8.
- Cooper, Z., Cooper, P. J., & Fairburn, C. G. 1989, "The validity of the eating disorder examination and its subscales", *Br.J.Psychiatry*, vol. 154, pp. 807-812.
- Corbin, J. & Strauss, A. 1990, "Grounded theory method: procedures, canons and evaluative procedures", *Qualitative Sociology*, vol. 13, no. 1, pp. 3-21.

- Coughlin, S. R., Mawdsley, L., Mugarza, J. A., Calverley, P. M., & Wilding, J. P. 2004, "Obstructive sleep apnoea is independently associated with an increased prevalence of metabolic syndrome", *Eur.Heart J.*, vol. 25, no. 9, pp. 735-741.
- Creswell, J. 2003, *Research Design:Qualitative, quantitative and mixed methods approaches*, 2nd edn, Sage, Thousand Oaks.
- Creswell, J., Plano, & Clark, V. 2007, *Designing and Conducting Mixed Methods Research* Sage, Thousand Oaks, CA.
- D'Arrigo, T. 2007, "Snack attack. Night-eating syndrome is no joke", *Diabetes Forecast.*, vol. 60, no. 10, p. 20.
- Dalgleish, T., Tchanturia, K., Serpell, L., Hems, S., de Silva, P., & Treasure, J. 2001, "Perceived control over events in the world in patients with eating disorders: a preliminary study", *Personality and Individual Differences*, vol. 31, no. 3, pp. 453-460.
- de Zwaan, M., Roerig, D. B., Crosby, R. D., Karaz, S., & Mitchell, J. E. 2006, "Nighttime eating: a descriptive study", *Int.J.Eat.Disord.*, vol. 39, no. 3, pp. 224-232.
- Devlin, M. J., Goldfein, J. A., Flancbaum, L., Bessler, M., & Eisenstadt, R. 2004, "Surgical management of obese patients with eating disorders: a survey of current practice", *Obes.Surg.*, vol. 14, no. 9, pp. 1252-1257.
- Devlin, M. J. 2007, "Is there a place for obesity in DSM-V?", *Int.J.Eat.Disord.*, vol. 40 Suppl, p. S83-S88.
- DeZee, K. J., Jackson, J. L., Hatzigeorgiou, C., & Kristo, D. 2006, "The Epworth sleepiness scale: relationship to sleep and mental disorders in a sleep clinic", *Sleep Med.*, vol. 7, no. 4, pp. 327-332.
- Dixon, J. B., Dixon, M. E., & O'Brien, P. E. 2003, "Depression in association with severe obesity: changes with weight loss", *Arch.Intern.Med.*, vol. 163, no. 17, pp. 2058-2065.
- Dong, C., Sanchez, L. E., & Price, R. A. 2004, "Relationship of obesity to depression: a family-based study", *Int.J.Obes.Relat Metab Disord.*, vol. 28, no. 6, pp. 790-795.
- Drevets, W. C. 2001, "Neuroimaging and neuropathological studies of depression: implications for the cognitive-emotional features of mood disorders", *Curr.Opin.Neurobiol.*, vol. 11, no. 2, pp. 240-249.
- Ehlers, C. L., Frank, E., & Kupfer, D. J. 1988, "Social zeitgebers and biological rhythms. A unified approach to understanding the etiology of depression", *Arch.Gen.Psychiatry*, vol. 45, no. 10, pp. 948-952.
- Fairburn, C. G. & Beglin, S. J. 1990, "Studies of the epidemiology of bulimia nervosa", *Am.J.Psychiatry*, vol. 147, no. 4, pp. 401-408.
- Fairburn & Cooper 1993, "The Eating Disorder Examination.," in *Binge Eating: Nature, assessment and treatment*, Fairburn CG & Wilson GT, eds., Guildford Press, New York, pp. 317-360.

- Fawcett, J., Scheftner, W. A., Fogg, L., Clark, D. C., Young, M. A., Hedeker, D., & Gibbons, R. 1990, "Time-related predictors of suicide in major affective disorder", *Am.J Psychiatry*, vol. 147, no. 9, pp. 1189-1194.
- Finer, N., Bloom, S. R., Frost, G. S., Banks, L. M., & Griffiths, J. 2000, "Sibutramine is effective for weight loss and diabetic control in obesity with type 2 diabetes: a randomised, double-blind, placebo-controlled study", *Diabetes Obes.Metab*, vol. 2, no. 2, pp. 105-112.
- Fogelholm, M., Kronholm, E., Kukkonen-Harjula, K., Partonen, T., Partinen, M., & Harma, M. 2007, "Sleep-related disturbances and physical inactivity are independently associated with obesity in adults", *Int.J.Obes.(Lond)*, vol. 31, no. 11, pp. 1713-1721.
- Frank, E., Swartz, H. A., Mallinger, A. G., Thase, M. E., Weaver, E. V., & Kupfer, D. J. 1999, "Adjunctive psychotherapy for bipolar disorder: effects of changing treatment modality", *J Abnorm.Psychol.*, vol. 108, no. 4, pp. 579-587.
- Frank, E., Swartz, H. A., & Kupfer, D. J. 2000, "Interpersonal and social rhythm therapy: managing the chaos of bipolar disorder", *Biol.Psychiatry*, vol. 48, no. 6, pp. 593-604.
- Friedman, S., Even, C., Dardennes, R., & Guelfi, J. D. 2004, "Light therapy, nonseasonal depression, and night eating syndrome", *Can.J.Psychiatry*, vol. 49, no. 11, p. 790.
- Gangwisch, J. E., Malaspina, D., Boden-Albala, B., & Heymsfield, S. B. 2005, "Inadequate sleep as a risk factor for obesity: analyses of the NHANES I", *Sleep*, vol. 28, no. 10, pp. 1289-1296.
- Garner, D. M. & Garfinkel, P. E. 1979, "The Eating Attitudes Test: an index of the symptoms of anorexia nervosa", *Psychol.Med.*, vol. 9, no. 2, pp. 273-279.
- Garriguet, D. 2008, "Under-reporting of energy intake in the Canadian Community Health Survey", *Health Rep.*, vol. 19, no. 4, pp. 37-45.
- Geliebter, A. 2002, "New developments in binge eating disorder and the night eating syndrome", *Appetite*, vol. 39, no. 2, pp. 175-177.
- Germain, A., Buysse, D. J., Shear, M. K., Fayyad, R., & Austin, C. 2004, "Clinical correlates of poor sleep quality in posttraumatic stress disorder", *J.Trauma Stress.*, vol. 17, no. 6, pp. 477-484.
- Glaser, B. & Strauss, A. 1967, *The Discovery of Grounded Theory: strategies for qualitative research* Aldine Transaction, New Brunswick.
- Gluck, M. E., Geliebter, A., & Satov, T. 2001, "Night eating syndrome is associated with depression, low self-esteem, reduced daytime hunger, and less weight loss in obese outpatients", *Obes.Res.*, vol. 9, no. 4, pp. 264-267.
- Goldschmidt, A. B., Jones, M., Manwaring, J. L., Luce, K. H., Osborne, M. I., Cuning, D., Taylor, K. L., Doyle, A. C., Wilfley, D. E., & Taylor, C. B. 2008, "The clinical significance of loss of control over eating in overweight adolescents", *Int.J.Eat.Disord.*, vol. 41, no. 2, pp. 153-158.

- Goris, A. H., Westerterp-Plantenga, M. S., & Westerterp, K. R. 2000, "Undereating and underrecording of habitual food intake in obese men: selective underreporting of fat intake", *Am.J.Clin.Nutr.*, vol. 71, no. 1, pp. 130-134.
- Gotlib, I. H. 1984, "Depression and general psychopathology in university students", *J.Abnorm.Psychol.*, vol. 93, no. 1, pp. 19-30.
- Grandin, L. D., Alloy, L. B., & Abramson, L. Y. 2006, "The social zeitgeber theory, circadian rhythms, and mood disorders: review and evaluation", *Clin.Psychol.Rev.*, vol. 26, no. 6, pp. 679-694.
- Greeno, C. G., Wing, R. R., & Marcus, M. D. 1995, "Nocturnal eating in binge eating disorder and matched-weight controls", *Int.J.Eat.Disord.*, vol. 18, no. 4, pp. 343-349.
- Grilo, C. M., Masheb, R. M., & Wilson, G. T. 2001, "A comparison of different methods for assessing the features of eating disorders in patients with binge eating disorder", *J.Consult Clin.Psychol.*, vol. 69, no. 2, pp. 317-322.
- Grilo, C. M. & Masheb, R. M. 2004, "Night-time eating in men and women with binge eating disorder", *Behav.Res.Ther.*, vol. 42, no. 4, pp. 397-407.
- Guba E & Lincoln Y 1981, *Effective evaluation* Jossey- Bass, San Francisco.
- Gupta, N. K., Mueller, W. H., Chan, W., & Meininger, J. C. 2002, "Is obesity associated with poor sleep quality in adolescents?", *Am.J.Hum.Biol.*, vol. 14, no. 6, pp. 762-768.
- Hardinge, F. M., Pitson, D. J., & Stradling, J. R. 1995, "Use of the Epworth Sleepiness Scale to demonstrate response to treatment with nasal continuous positive airways pressure in patients with obstructive sleep apnoea", *Respir.Med.*, vol. 89, no. 9, pp. 617-620.
- Hasler, G., Buysse, D. J., Klaghofer, R., Gamma, A., Ajdacic, V., Eich, D., Ressler, W., & Angst, J. 2004, "The association between short sleep duration and obesity in young adults: a 13-year prospective study", *Sleep*, vol. 27, no. 4, pp. 661-666.
- Hay, P. & Fairburn, C. 1998, "The validity of the DSM-IV scheme for classifying bulimic eating disorders", *Int.J.Eat.Disord.*, vol. 23, no. 1, pp. 7-15.
- Haynes, P. L., McQuaid, J. R., ncoli-Israel, S., & Martin, J. L. 2006, "Disrupting life events and the sleep-wake cycle in depression", *Psychol.Med*, vol. 36, no. 10, pp. 1363-1373.
- Healey, E. S., Kales, A., Monroe, L. J., Bixler, E. O., Chamberlin, K., & Soldatos, C. R. 1981, "Onset of insomnia: role of life-stress events", *Psychosom.Med.*, vol. 43, no. 5, pp. 439-451.
- Henderson, M. & Freeman, C. P. 1987, "A self-rating scale for bulimia. The 'BITE'", *Br.J.Psychiatry*, vol. 150, pp. 18-24.
- Hildebrandt, T. & Latner, J. 2006, "Effect of self-monitoring on binge eating: treatment response or 'binge drift'?", *European Eating Disorders Review*, vol. 14, no. 1, pp. 17-22.
- Hollander, P. A., Elbein, S. C., Hirsch, I. B., Kelley, D., McGill, J., Taylor, T., Weiss, S. R., Crockett, S. E., Kaplan, R. A., Comstock, J., Lucas, C. P., Lodewick, P. A.,

- Canovatchel, W., Chung, J., & Hauptman, J. 1998, "Role of orlistat in the treatment of obese patients with type 2 diabetes. A 1-year randomized double-blind study", *Diabetes Care*, vol. 21, no. 8, pp. 1288-1294.
- Howell, M. J., Schenck, C. H., & Crow, S. J. 2008, "A review of nighttime eating disorders", *Sleep Med.Rev.*
- Huang, C. J., Hu, H. T., Fan, Y. C., Liao, Y. M., & Tsai, P. S. 2010, "Associations of breakfast skipping with obesity and health-related quality of life: evidence from a national survey in Taiwan", *Int.J.Obes.(Lond)*, vol. 34, no. 4, pp. 720-725.
- Hughes, E. 1971, *The Sociological Eye: selected papers* Aldine- Atherton, Chicago.
- Jacobi, C., Abascal, L., & Taylor, C. B. 2004, "Screening for eating disorders and high-risk behavior: caution", *Int.J.Eat.Disord.*, vol. 36, no. 3, pp. 280-295.
- Jennings, J. R., Muldoon, M. F., Hall, M., Buysse, D. J., & Manuck, S. B. 2007, "Self-reported sleep quality is associated with the metabolic syndrome", *Sleep*, vol. 30, no. 2, pp. 219-223.
- Johns, M. & Hocking, B. 1997, "Daytime sleepiness and sleep habits of Australian workers", *Sleep*, vol. 20, no. 10, pp. 844-849.
- Johns, M. W. 1992, "Reliability and factor analysis of the Epworth Sleepiness Scale", *Sleep*, vol. 15, no. 4, pp. 376-381.
- Johns, M. W. 2000, "Sensitivity and specificity of the multiple sleep latency test (MSLT), the maintenance of wakefulness test and the epworth sleepiness scale: failure of the MSLT as a gold standard", *J.Sleep Res.*, vol. 9, no. 1, pp. 5-11.
- Kalarchian, M. A., Wilson, G. T., Brolin, R. E., & Bradley, L. 2000, "Assessment of eating disorders in bariatric surgery candidates: self-report questionnaire versus interview", *Int.J.Eat.Disord.*, vol. 28, no. 4, pp. 465-469.
- Kaminska, M., Jobin, V., Mayer, P., Amyot, R., Perraton-Brillon, M., & Bellemare, F. 2010, "The Epworth Sleepiness Scale: Self-administration versus administration by the physician, and validation of a French version", *Can.Respir.J.*, vol. 17, no. 2, pp. 27-34.
- Karp, J. F., Scott, J., Houck, P., Reynolds, C. F., III, Kupfer, D. J., & Frank, E. 2005, "Pain predicts longer time to remission during treatment of recurrent depression", *J Clin.Psychiatry*, vol. 66, no. 5, pp. 591-597.
- Karp, J. & Reynolds, C. 2009, "Depression,Pain and Aging", *Focus*, vol. 7, no. 1, pp. 17-27.
- Kennedy, G. J., Kelman, H. R., & Thomas, C. 1991, "Persistence and remission of depressive symptoms in late life", *Am.J.Psychiatry*, vol. 148, no. 2, pp. 174-178.
- Keski-Rahkonen, A., Sihvola, E., Raevuori, A., Kaukoranta, J., Bulik, C. M., Hoek, H. W., Rissanen, A., & Kaprio, J. 2006, "Reliability of self-reported eating disorders: Optimizing population screening", *Int.J.Eat.Disord.*, vol. 39, no. 8, pp. 754-762.
- Knutson, K. L., Rathouz, P. J., Yan, L. L., Liu, K., & Lauderdale, D. S. 2006a, "Stability of the Pittsburgh Sleep Quality Index and the Epworth Sleepiness Questionnaires over 1

year in early middle-aged adults: the CARDIA study", *Sleep*, vol. 29, no. 11, pp. 1503-1506.

Knutson, K. L., Ryden, A. M., Mander, B. A., & Van, C. E. 2006b, "Role of sleep duration and quality in the risk and severity of type 2 diabetes mellitus", *Arch.Intern.Med.*, vol. 166, no. 16, pp. 1768-1774.

Kohatsu, N. D., Tsai, R., Young, T., Vangilder, R., Burmeister, L. F., Stromquist, A. M., & Merchant, J. A. 2006, "Sleep duration and body mass index in a rural population", *Arch.Intern.Med.*, vol. 166, no. 16, pp. 1701-1705.

Kretsch, M. J., Fong, A. K., & Green, M. W. 1999, "Behavioral and body size correlates of energy intake underreporting by obese and normal-weight women", *J Am Diet.Assoc.*, vol. 99, no. 3, pp. 300-306.

Kuldau, J. & Rand, C. 1986, "The night eating syndrome in the morbidly obese", *Int.J.Eat.Disord.*, vol. 5, pp. 143-148.

Lamerz, A., Kuepper-Nybelen, J., Bruning, N., Wehle, C., Trost-Brinkhues, G., Brenner, H., Hebebrand, J., & Herpertz-Dahlmann, B. 2005, "Prevalence of obesity, binge eating, and night eating in a cross-sectional field survey of 6-year-old children and their parents in a German urban population", *J.Child Psychol.Psychiatry*, vol. 46, no. 4, pp. 385-393.

Latner, J. D., Wetzler, S., Goodman, E. R., & Glinski, J. 2004, "Gastric bypass in a low-income, inner-city population: eating disturbances and weight loss", *Obes.Res.*, vol. 12, no. 6, pp. 956-961.

Latner, J. D., Hildebrandt, T., Rosewall, J. K., Chisholm, A. M., & Hayashi, K. 2007, "Loss of control over eating reflects eating disturbances and general psychopathology", *Behav.Res.Ther.*, vol. 45, no. 9, pp. 2203-2211.

Leskela, U. S., Melartin, T. K., Lestela-Mielonen, P. S., Rytsala, H. J., Sokero, T. P., Heikkinen, M. E., & Isometsa, E. T. 2004, "Life events, social support, and onset of major depressive episode in Finnish patients", *J Nerv.Ment.Dis.*, vol. 192, no. 5, pp. 373-381.

Lichtman, S. W., Pisarska, K., Berman, E. R., Pestone, M., Dowling, H., Offenbacher, E., Weisel, H., Heshka, S., Matthews, D. E., & Heymsfield, S. B. 1992, "Discrepancy between self-reported and actual caloric intake and exercise in obese subjects", *N.Engl.J.Med.*, vol. 327, no. 27, pp. 1893-1898.

Lightfoot, S. L. & Oliver, J. M. 1985, "The Beck Inventory: psychometric properties in university students", *J.Pers.Assess.*, vol. 49, no. 4, pp. 434-436.

Linton, S. J. 2004, "Does work stress predict insomnia? A prospective study", *Br.J Health Psychol.*, vol. 9, no. Pt 2, pp. 127-136.

Lundgren, J. D., Allison, K. C., Crow, S., O'Reardon, J. P., Berg, K. C., Galbraith, J., Martino, N. S., & Stunkard, A. J. 2006, "Prevalence of the night eating syndrome in a psychiatric population", *Am.J.Psychiatry*, vol. 163, no. 1, pp. 156-158.

Lundgren, J. D., Allison, K. C., & Stunkard, A. J. 2006, "Familial aggregation in the night eating syndrome", *Int.J.Eat.Disord.*

- Lundgren, J. D., Allison, K. C., O'Reardon, J. P., & Stunkard, A. J. 2008a, "A descriptive study of non-obese persons with night eating syndrome and a weight-matched comparison group", *Eat.Behav.*, vol. 9, no. 3, pp. 343-351.
- Lundgren, J. D., Newberg, A. B., Allison, K. C., Wintering, N. A., Ploessl, K., & Stunkard, A. J. 2008b, "123I-ADAM SPECT imaging of serotonin transporter binding in patients with night eating syndrome: a preliminary report", *Psychiatry Res.*, vol. 162, no. 3, pp. 214-220.
- Lundgren, J. D., Rempfer, M. V., Brown, C. E., Goetz, J., & Hamera, E. 2010, "The prevalence of night eating syndrome and binge eating disorder among overweight and obese individuals with serious mental illness", *Psychiatry Res*, vol. 175, no. 3, pp. 233-236.
- Lundgren, J. D., McCune, A., Spreser, C., Harkins, P., Zolton, L., & Mandal, K. 2011, "Night eating patterns of individuals with eating disorders: Implications for conceptualizing the night eating syndrome", *Psychiatry Res*, vol. 186, no. 1, pp. 103-108.
- Manni, R., Ratti, M. T., & Tartara, A. 1997, "Nocturnal eating: prevalence and features in 120 insomniac referrals", *Sleep*, vol. 20, no. 9, pp. 734-738.
- Marshall, H. M., Allison, K. C., O'Reardon, J. P., Birketvedt, G., & Stunkard, A. J. 2004, "Night eating syndrome among nonobese persons", *Int.J.Eat.Disord.*, vol. 35, no. 2, pp. 217-222.
- Mazzeo, S. E., Saunders, R., & Mitchell, K. S. 2006, "Gender and binge eating among bariatric surgery candidates", *Eat.Behav.*, vol. 7, no. 1, pp. 47-52.
- McElroy, S. L., Hudson, J. I., Capece, J. A., Beyers, K., Fisher, A. C., & Rosenthal, N. R. 2007, "Topiramate for the treatment of binge eating disorder associated with obesity: a placebo-controlled study", *Biol.Psychiatry*, vol. 61, no. 9, pp. 1039-1048.
- Mellinger, G. D., Balter, M. B., & Uhlenhuth, E. H. 1985, "Insomnia and its treatment. Prevalence and correlates", *Arch.Gen.Psychiatry*, vol. 42, no. 3, pp. 225-232.
- Miyaoka, T., Yasukawa, R., Tsubouchi, K., Miura, S., Shimizu, Y., Sukegawa, T., Maeda, T., Mizuno, S., Kameda, A., Uegaki, J., Inagaki, T., & Horiguchi, J. 2003, "Successful treatment of nocturnal eating/drinking syndrome with selective serotonin reuptake inhibitors", *Int.Clin.Psychopharmacol.*, vol. 18, no. 3, pp. 175-177.
- Monk, T. H., Flaherty, J. F., Frank, E., Hoskinson, K., & Kupfer, D. J. 1990, "The Social Rhythm Metric. An instrument to quantify the daily rhythms of life", *J Nerv.Ment.Dis.*, vol. 178, no. 2, pp. 120-126.
- Monti, J. M. & Monti, D. 2005, "Sleep disturbance in schizophrenia", *Int.Rev.Psychiatry*, vol. 17, no. 4, pp. 247-253.
- Morgan, D. L. 1998, "Practical strategies for combining qualitative and quantitative methods: applications to health research", *Qual.Health Res.*, vol. 8, no. 3, pp. 362-376.
- Morgan, J. F., Reid, F., & Lacey, J. H. 1999, "The SCOFF questionnaire: assessment of a new screening tool for eating disorders", *BMJ*, vol. 319, no. 7223, pp. 1467-1468.

- Morin, C. M., Rodrigue, S., & Ivers, H. 2003, "Role of stress, arousal, and coping skills in primary insomnia", *Psychosom.Med.*, vol. 65, no. 2, pp. 259-267.
- Morse, J. M. 1991, "Approaches to qualitative-quantitative methodological triangulation", *Nurs.Res.*, vol. 40, no. 2, pp. 120-123.
- Morse, S. A., Ciechanowski, P. S., Katon, W. J., & Hirsch, I. B. 2006, "Isn't this just bedtime snacking? The potential adverse effects of night-eating symptoms on treatment adherence and outcomes in patients with diabetes", *Diabetes Care*, vol. 29, no. 8, pp. 1800-1804.
- Mullington, J. M., Chan, J. L., Van Dongen, H. P., Szuba, M. P., Samaras, J., Price, N. J., Meier-Ewert, H. K., Dinges, D. F., & Mantzoros, C. S. 2003, "Sleep loss reduces diurnal rhythm amplitude of leptin in healthy men", *J.Neuroendocrinol.*, vol. 15, no. 9, pp. 851-854.
- Munoz, D. J., Chen, E., Fischer, S., Roehrig, M., Sanchez-Johnson, L., Alverdy, J., Dymek-Valentine, M., & Ie, G. D. 2007, "Considerations for the use of the Beck Depression Inventory in the assessment of weight-loss surgery seeking patients", *Obes.Surg.*, vol. 17, no. 8, pp. 1097-1101.
- Napolitano, M. A., Head, S., Babyak, M. A., & Blumenthal, J. A. 2001, "Binge eating disorder and night eating syndrome: psychological and behavioral characteristics", *Int.J.Eat.Disord.*, vol. 30, no. 2, pp. 193-203.
- Neumark-Sztainer, D., Story, M., & Faibisch, L. 1998, "Perceived stigmatization among overweight African-American and Caucasian adolescent girls", *J.Adolesc.Health*, vol. 23, no. 5, pp. 264-270.
- Nezlek, J. B., Hampton, C. P., & Shean, G. D. 2000, "Clinical depression and day-to-day social interaction in a community sample", *J Abnorm.Psychol.*, vol. 109, no. 1, pp. 11-19.
- Nguyen, A. T., Baltzan, M. A., Small, D., Wolkove, N., Guillon, S., & Palayew, M. 2006, "Clinical reproducibility of the Epworth Sleepiness Scale", *J.Clin.Sleep Med.*, vol. 2, no. 2, pp. 170-174.
- Nordin, M., Knutsson, A., Sundbom, E., & Stegmayr, B. 2005, "Psychosocial factors, gender, and sleep", *J.Occup.Health Psychol.*, vol. 10, no. 1, pp. 54-63.
- Nowicki, S. J. & Hopper, A. E. 1974, "Locus of control correlates in an alcoholic population", *J.Consult Clin.Psychol.*, vol. 42, no. 5, p. 735.
- O'Brien, B. 2005, "Night eating syndrome and Gotu Kola", *Ir.Med.J.*, vol. 98, no. 10, pp. 250-251.
- O'Reardon, J. P., Ringel, B. L., Dinges, D. F., Allison, K. C., Rogers, N. L., Martino, N. S., & Stunkard, A. J. 2004, "Circadian eating and sleeping patterns in the night eating syndrome", *Obes.Res.*, vol. 12, no. 11, pp. 1789-1796.
- O'Reardon, J. P., Stunkard, A. J., & Allison, K. C. 2004, "Clinical trial of sertraline in the treatment of night eating syndrome", *Int.J.Eat.Disord.*, vol. 35, no. 1, pp. 16-26.
- O'Reardon, J. P., Peshek, A., & Allison, K. C. 2005, "Night eating syndrome : diagnosis, epidemiology and management", *CNS.Drugs*, vol. 19, no. 12, pp. 997-1008.

- O'Reardon, J. P., Allison, K. C., Martino, N. S., Lundgren, J. D., Heo, M., & Stunkard, A. J. 2006, "A randomized, placebo-controlled trial of sertraline in the treatment of night eating syndrome", *Am.J.Psychiatry*, vol. 163, no. 5, pp. 893-898.
- Offord, D. R., Kraemer, H. C., Kazdin, A. E., Jensen, P. S., & Harrington, R. 1998, "Lowering the burden of suffering from child psychiatric disorder: trade-offs among clinical, targeted, and universal interventions", *J.Am.Acad.Child Adolesc.Psychiatry*, vol. 37, no. 7, pp. 686-694.
- Osman, E. Z., Osborne, J., Hill, P. D., & Lee, B. W. 1999, "The Epworth Sleepiness Scale: can it be used for sleep apnoea screening among snorers?", *Clin.Otolaryngol.Allied Sci.*, vol. 24, no. 3, pp. 239-241.
- Ota, A., Masue, T., Yasuda, N., Tsutsumi, A., Mino, Y., & Ohara, H. 2005, "Association between psychosocial job characteristics and insomnia: an investigation using two relevant job stress models--the demand-control-support (DCS) model and the effort-reward imbalance (ERI) model", *Sleep Med.*, vol. 6, no. 4, pp. 353-358.
- Padez, C., Mourao, I., Moreira, P., & Rosado, V. 2005, "Prevalence and risk factors for overweight and obesity in Portuguese children", *Acta Paediatr.*, vol. 94, no. 11, pp. 1550-1557.
- Park, R. 1967, *On social control and collective behaviour* University of Chicago Press, Chicago.
- Parkes, J. D., Chen, S. Y., Clift, S. J., Dahlitz, M. J., & Dunn, G. 1998, "The clinical diagnosis of the narcoleptic syndrome", *J Sleep Res*, vol. 7, no. 1, pp. 41-52.
- Patel, S. R., Blackwell, T., Redline, S., ncoli-Israel, S., Cauley, J. A., Hillier, T. A., Lewis, C. E., Orwoll, E. S., Stefanick, M. L., Taylor, B. C., Yaffe, K., & Stone, K. L. 2008, "The association between sleep duration and obesity in older adults", *Int.J.Obes.(Lond)*, vol. 32, no. 12, pp. 1825-1834.
- Paunio, T., Korhonen, T., Hublin, C., Partinen, M., Kivimaki, M., Koskenvuo, M., & Kaprio, J. 2009, "Longitudinal study on poor sleep and life dissatisfaction in a nationwide cohort of twins", *Am.J.Epidemiol.*, vol. 169, no. 2, pp. 206-213.
- Pawlow, L. A., O'Neil, P. M., & Malcolm, R. J. 2003, "Night eating syndrome: effects of brief relaxation training on stress, mood, hunger, and eating patterns", *Int.J.Obes.Relat Metab Disord.*, vol. 27, no. 8, pp. 970-978.
- Pierce, J. W. & Wardle, J. 1997, "Cause and effect beliefs and self-esteem of overweight children", *J.Child Psychol.Psychiatry*, vol. 38, no. 6, pp. 645-650.
- Powers, P. S., Perez, A., Boyd, F., & Rosemurgy, A. 1999, "Eating pathology before and after bariatric surgery: a prospective study", *Int.J.Eat.Disord.*, vol. 25, no. 3, pp. 293-300.
- Qin, L. Q., Li, J., Wang, Y., Wang, J., Xu, J. Y., & Kaneko, T. 2003, "The effects of nocturnal life on endocrine circadian patterns in healthy adults", *Life Sci.*, vol. 73, no. 19, pp. 2467-2475.
- Rand, C. S., Macgregor, A. M., & Stunkard, A. J. 1997, "The night eating syndrome in the general population and among postoperative obesity surgery patients", *Int.J.Eat.Disord.*, vol. 22, no. 1, pp. 65-69.

- Rand, C. & Kulda, J. 1986, "Eating patterns in normal weight individuals :bulimia, restrained eating and the night eating syndrome.", *Int.J.Eat.Disord.*, vol. 5, pp. 75-84.
- Rand, C. & Kulda, J. 1993, "Morbid Obesity: a comparison between a general population and among postoperative obesity surgery patients", *Int.J.Eat.Disord.*, vol. 17, pp. 657-661.
- Reilly, J. J., Armstrong, J., Dorosty, A. R., Emmett, P. M., Ness, A., Rogers, I., Steer, C., & Sherriff, A. 2005, "Early life risk factors for obesity in childhood: cohort study", *BMJ*, vol. 330, no. 7504, p. 1357.
- Rejeski, W. J., Lang, W., Neiberg, R. H., Van, D. B., Foster, G. D., Maciejewski, M. L., Rubin, R., & Williamson, D. F. 2006, "Correlates of health-related quality of life in overweight and obese adults with type 2 diabetes", *Obesity.(Silver.Spring)*, vol. 14, no. 5, pp. 870-883.
- Rennie, K. L., Coward, A., & Jebb, S. A. 2007, "Estimating under-reporting of energy intake in dietary surveys using an individualised method", *Br.J Nutr.*, vol. 97, no. 6, pp. 1169-1176.
- Resta, O., Foschino Barbaro, M. P., Bonfitto, P., Giliberti, T., Depalo, A., Pannacciulli, N., & De, P. G. 2003, "Low sleep quality and daytime sleepiness in obese patients without obstructive sleep apnoea syndrome", *J.Intern.Med.*, vol. 253, no. 5, pp. 536-543.
- Ricca, V., Mannucci, E., Moretti, S., Di, B. M., Zucchi, T., Cabras, P. L., & Rotella, C. M. 2000, "Screening for binge eating disorder in obese outpatients", *Compr.Psychiatry*, vol. 41, no. 2, pp. 111-115.
- Richter, P., Werner, J., Heerlein, A., Kraus, A., & Sauer, H. 1998, "On the validity of the Beck Depression Inventory. A review", *Psychopathology*, vol. 31, no. 3, pp. 160-168.
- Rogers, N. L., Dinges, D. F., Allison, K. C., Maislin, G., Martino, N., O'Reardon, J. P., & Stunkard, A. J. 2006, "Assessment of sleep in women with night eating syndrome", *Sleep*, vol. 29, no. 6, pp. 814-819.
- Rolland-Cachera, M. F., Deheeger, M., Maillot, M., & Bellisle, F. 2006, "Early adiposity rebound: causes and consequences for obesity in children and adults", *Int.J.Obes.(Lond)*, vol. 30 Suppl 4, p. S11-S17.
- Root, T. L., Thornton, L. M., Lindroos, A. K., Stunkard, A. J., Lichtenstein, P., Pedersen, N. L., Rasmussen, F., & Bulik, C. M. 2010, "Shared and unique genetic and environmental influences on binge eating and night eating: a Swedish twin study", *Eat.Behav.*, vol. 11, no. 2, pp. 92-98.
- Rosen, J. C., Vara, L., Wendt, S., & Leitenberg, H. 1990, "Validity studies of the Eating Disorder Examination", *Int.J.Eat.Disord.*, vol. 9, pp. 519-528.
- Rosenghan, M. C., Uhr, M., Schussler, P., & Steiger, A. 2005, "Elevated plasma ghrelin levels in night-eating syndrome", *Am.J.Psychiatry*, vol. 162, no. 4, p. 813.
- Rosmond, R. 2004, "Obesity and depression: same disease, different names?", *Med.Hypotheses*, vol. 62, no. 6, pp. 976-979.

- Rossmann, G. & Wilson, B. 1985, "Numbers and words: Combining quantitative and qualitative methods in a single, large-scale evaluation study.", *Evaluation Review*, vol. 9, no. 5, pp. 627-643.
- Rotter, J. B. 1966, "Generalized expectancies for internal versus external control of reinforcement", *Psychol.Monogr.*, vol. 80, no. 1, pp. 1-28.
- Safer, D. L., Lively, T. J., Telch, C. F., & Agras, W. S. 2002, "Predictors of relapse following successful dialectical behavior therapy for binge eating disorder", *Int.J.Eat.Disord.*, vol. 32, no. 2, pp. 155-163.
- Sanford, S. D., Lichstein, K. L., Durrence, H. H., Riedel, B. W., Taylor, D. J., & Bush, A. J. 2006, "The influence of age, gender, ethnicity, and insomnia on Epworth sleepiness scores: a normative US population", *Sleep Med.*, vol. 7, no. 4, pp. 319-326.
- Sassaroli, S., Ruggiero, G. M., Vinai, P., Cardetti, S., Carpegna, G., Ferrato, N., Vallauri, P., Masante, D., Scarone, S., Bertelli, S., Bidone, R., Busetto, L., & Sampietro, S. 2009, "Daily and nightly anxiety among patients affected by night eating syndrome and binge eating disorder", *Eat.Disord.*, vol. 17, no. 2, pp. 140-145.
- Schatzman, L. 1991, "Dimensional analysis: notes on an alternative approach to the grounding of theory in qualitative research," in *Social organisations and social process: essays in honour of Anselm Strauss*, Maines DR, ed., Aldine de Gruyter, New York.
- Schenck, C. H. & Mahowald, M. W. 1994, "Review of nocturnal sleep-related eating disorders", *Int.J.Eat.Disord.*, vol. 15, no. 4, pp. 343-356.
- Schon, D. 1983, *The Reflective Practice, How Professionals think in Action* Basic Books, United States.
- Silber, M. H., ncoti-Israel, S., Bonnet, M. H., Chokroverty, S., Grigg-Damberger, M. M., Hirshkowitz, M., Kapen, S., Keenan, S. A., Kryger, M. H., Penzel, T., Pressman, M. R., & Iber, C. 2007, "The visual scoring of sleep in adults", *J.Clin.Sleep Med.*, vol. 3, no. 2, pp. 121-131.
- Smith, S. S., Oei, T. P., Douglas, J. A., Brown, I., Jorgensen, G., & Andrews, J. 2008, "Confirmatory factor analysis of the Epworth Sleepiness Scale (ESS) in patients with obstructive sleep apnoea", *Sleep Med.*, vol. 9, no. 7, pp. 739-744.
- Smyth, C. A. 2008, "Evaluating sleep quality in older adults: the Pittsburgh Sleep Quality Index can be used to detect sleep disturbances or deficits", *Am.J.Nurs.*, vol. 108, no. 5, pp. 42-50.
- Spaggiari, M. C., Granella, F., Parrino, L., Marchesi, C., Melli, I., & Terzano, M. G. 1994, "Nocturnal eating syndrome in adults", *Sleep*, vol. 17, no. 4, pp. 339-344.
- Spiegel, K., Leproult, R., & Van, C. E. 1999, "Impact of sleep debt on metabolic and endocrine function", *Lancet*, vol. 354, no. 9188, pp. 1435-1439.
- Spiegel, K., Knutson, K., Leproult, R., Tasali, E., & Van, C. E. 2005, "Sleep loss: a novel risk factor for insulin resistance and Type 2 diabetes", *J.Appl.Physiol*, vol. 99, no. 5, pp. 2008-2019

- Spitzer, R. L., Devlin M, Walsh BT, Hasin D, Wing R, Marcus M, Stunkard A, Wadden T, Yanovski S, Agras S, Mitchell J, & Nonas C 2006, "Binge Eating Disorder: A multisite field trial of the diagnosis criteria", *Int.J.Eat.Disord.*, vol. 11, pp. 191-203.
- Spitzer, R. L., Yanovski, S., Wadden, T., Wing, R., Marcus, M. D., Stunkard, A., Devlin, M., Mitchell, J., Hasin, D., & Horne, R. L. 1993, "Binge eating disorder: its further validation in a multisite study", *Int.J.Eat.Disord.*, vol. 13, no. 2, pp. 137-153.
- Steer, R. A. & Beck, A. T. 1985, "Modifying the Beck Depression Inventory: reply to Vredenburg, Krames, and Flett", *Psychol.Rep.*, vol. 57, no. 2, pp. 625-626.
- Steer, R. A., Beck, A. T., Riskind, J. H., & Brown, G. 1986, "Differentiation of depressive disorders from generalized anxiety by the Beck Depression Inventory", *J.Clin.Psychol.*, vol. 42, no. 3, pp. 475-478.
- Stetler, C., Dickerson, S. S., & Miller, G. E. 2004, "Uncoupling of social zeitgebers and diurnal cortisol secretion in clinical depression", *Psychoneuroendocrinology*, vol. 29, no. 10, pp. 1250-1259.
- Stettler, N., Zemel, B. S., Kumanyika, S., & Stallings, V. A. 2002, "Infant weight gain and childhood overweight status in a multicenter, cohort study", *Pediatrics*, vol. 109, no. 2, pp. 194-199.
- Storch, E. A., Roberti, J. W., & Roth, D. A. 2004, "Factor structure, concurrent validity, and internal consistency of the Beck Depression Inventory-Second Edition in a sample of college students", *Depress.Anxiety.*, vol. 19, no. 3, pp. 187-189.
- Strauss, A. 1978, *Negotiations: Varieties, contexts, processes and social order* Jossey-Bass, San Francisco.
- Strauss, A. 1993, *Continual Permutations of Action* Aldine de Gruyter, New York.
- Strauss, A. & Corbin, J. 2008, "Practical considerations," in *Basics of Qualitative Research*, Knight V & Connelly S, eds., Sage, Thousand Oaks, CA, pp. 19-43.
- Strauss, R. S. 2000, "Childhood obesity and self-esteem", *Pediatrics*, vol. 105, no. 1, p. e15.
- Striegel-Moore, R. H., Thompson, D., Franko, D. L., Barton, B., Affenito, S., Schreiber, G. B., & Daniels, S. R. 2004, "Definitions of night eating in adolescent girls", *Obes.Res.*, vol. 12, no. 8, pp. 1311-1321.
- Striegel-Moore, R. H., Dohm, F. A., Hook, J. M., Schreiber, G. B., Crawford, P. B., & Daniels, S. R. 2005, "Night eating syndrome in young adult women: Prevalence and correlates", *Int.J.Eat.Disord.*, vol. 37, no. 3, pp. 200-206.
- Striegel-Moore, R. H., Franko, D. L., May, A., Ach, E., Thompson, D., & Hook, J. M. 2006a, "Should night eating syndrome be included in the DSM?", *Int.J.Eat.Disord.*, vol. 39, no. 7, pp. 544-549.
- Striegel-Moore, R. H., Franko, D. L., Thompson, D., Affenito, S., & Kraemer, H. C. 2006b, "Night eating: prevalence and demographic correlates", *Obesity.(Silver.Spring)*, vol. 14, no. 1, pp. 139-147.

- Striegel-Moore, R. H., Franko, D. L., Thompson, D., Affenito, S., May, A., & Kraemer, H. C. 2008, "Exploring the typology of night eating syndrome", *Int.J.Eat.Disord.*
- Striegel-Moore, R. H., Franko, D. L., & Garcia, J. 2009, "The validity and clinical utility of night eating syndrome", *Int.J.Eat.Disord.*, vol. 42, no. 8, pp. 720-738.
- Striegel-Moore, R. H., Rosselli, F., Wilson, G. T., Perrin, N., Harvey, K., & DeBar, L. 2010, "Nocturnal eating: association with binge eating, obesity, and psychological distress", *Int.J.Eat.Disord.*, vol. 43, no. 6, pp. 520-526.
- Stunkard, A., Grace, & Wolff 1955, "The night-eating syndrome; a pattern of food intake among certain obese patients", *Am J Med*, vol. 19, no. 1, pp. 78-86.
- Stunkard, A., Berkowitz, R., Wadden, T., Tanrikut, C., Reiss, E., & Young, L. 1996, "Binge eating disorder and the night-eating syndrome", *Int.J.Obes.Relat Metab Disord.*, vol. 20, no. 1, pp. 1-6.
- Stunkard, A. J. & Allison, K. C. 2003, "Two forms of disordered eating in obesity: binge eating and night eating", *Int.J.Obes.Relat Metab Disord.*, vol. 27, no. 1, pp. 1-12.
- Stunkard, A., Allison, K., & Lundgren, J. 2008, "Issues for DSM-V: night eating syndrome", *Am.J.Psychiatry*, vol. 165, no. 4, p. 424.
- Stunkard, A. & Lu, X. Y. 2010, "Rapid changes in night eating: considering mechanisms", *Eat.Weight.Disord.*, vol. 15, no. 1-2, p. e2-e8.
- Taheri, S., Lin, L., Austin, D., Young, T., & Mignot, E. 2004, "Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index", *PLoS.Med.*, vol. 1, no. 3, p. e62.
- Taheri, S. 2006, "The link between short sleep duration and obesity: we should recommend more sleep to prevent obesity", *Arch.Dis.Child*, vol. 91, no. 11, pp. 881-884.
- Takeda, E., Terao, J., Nakaya, Y., Miyamoto, K., Baba, Y., Chuman, H., Kaji, R., Ohmori, T., & Rokutan, K. 2004, "Stress control and human nutrition", *J.Med.Invest*, vol. 51, no. 3-4, pp. 139-145.
- Tanaka-Matsumi, J. & Kameoka, V. A. 1986, "Reliabilities and concurrent validities of popular self-report measures of depression, anxiety, and social desirability", *J.Consult Clin.Psychol.*, vol. 54, no. 3, pp. 328-333.
- Tashakkori & Teddlle 1998, *Mixed Methodology: Combining Qualitative and Quantitative Approaches* Sage.
- Telch, C. F., Pratt, E. M., & Niego, S. H. 1998, "Obese women with binge eating disorder define the term binge", *Int.J.Eat.Disord.*, vol. 24, no. 3, pp. 313-317.
- Thase, M. E. & Trivedi, M. 2002, "Optimizing treatment outcomes for patients with depression and generalized anxiety disorder", *Psychopharmacol.Bull.*, vol. 36 Suppl 2, pp. 93-102.
- Thomas, W. 1966, *On social control and collective behaviour* University of Chicago Press, Chicago.

- Tucker, P., Masters, B., & Nawar, O. 2004, "Topiramate in the treatment of comorbid night eating syndrome and PTSD: a case study", *Eat.Disord.*, vol. 12, no. 1, pp. 75-78.
- Turner, J. A. & Romano, J. M. 1984, "Self-report screening measures for depression in chronic pain patients", *J.Clin.Psychol.*, vol. 40, no. 4, pp. 909-913.
- Two-roger, S. S., Davis, S., Vitiello, M. V., Lentz, M. J., & McTiernan, A. 2005, "Factors associated with objective (actigraphic) and subjective sleep quality in young adult women", *J.Psychosom.Res.*, vol. 59, no. 1, pp. 11-19.
- Tzischinsky O & Latzer Y 2004, "Nocturnal eating: prevalence features and night sleep among binge eating disorder and bulimia nervosa patients in Israel", *European Eating Disorders Review*, vol. 12, no. 2, pp. 101-109.
- Vander Wal, J. S., Waller, S. M., Klurfeld, D. M., McBurney, M. I., & Dhurandhar, N. V. 2005, "Night eating syndrome: evaluation of two screening instruments", *Eat.Behav.*, vol. 6, no. 1, pp. 63-73.
- Vetrugno, R., Manconi, M., Ferini-Strambi, L., Provini, F., Plazzi, G., & Montagna, P. 2006, "Nocturnal eating: sleep-related eating disorder or night eating syndrome? A videopolysomnographic study", *Sleep*, vol. 29, no. 7, pp. 949-954.
- Vinai, P., Allison, K. C., Cardetti, S., Carpegna, G., Ferrato, N., Masante, D., Vallauri, P., Ruggiero, G. M., & Sassaroli, S. 2008, "Psychopathology and treatment of night eating syndrome: a review", *Eat.Weight.Disord.*, vol. 13, no. 2, pp. 54-63.
- Vioque, J., Torres, A., & Quiles, J. 2000, "Time spent watching television, sleep duration and obesity in adults living in Valencia, Spain", *Int.J.Obes.Relat Metab Disord.*, vol. 24, no. 12, pp. 1683-1688.
- von Kries R., Toschke, A. M., Wurmser, H., Sauerwald, T., & Koletzko, B. 2002, "Reduced risk for overweight and obesity in 5- and 6-y-old children by duration of sleep--a cross-sectional study", *Int.J.Obes.Relat Metab Disord.*, vol. 26, no. 5, pp. 710-716.
- Vorona, R. D., Winn, M. P., Babineau, T. W., Eng, B. P., Feldman, H. R., & Ware, J. C. 2005, "Overweight and obese patients in a primary care population report less sleep than patients with a normal body mass index", *Arch.Intern.Med.*, vol. 165, no. 1, pp. 25-30.
- Wadden T & Foster, G. 2001, *Weight and lifestyle Inventory* University of Pennsylvania, Philadelphia.
- Wadden, T. A. & Phelan, S. 2002, "Behavioural assessment of the obese patient. Appendix 10.1 Section K. Eating Patterns 111.," in *Handbook of obesity treatment*, Wadden TA & Stunkard AJ, eds., Guildford: New York, pp. 219-220.
- Wakefield, J. C. 1997, "Diagnosing DSM-IV--Part I: DSM-IV and the concept of disorder", *Behav.Res.Ther.*, vol. 35, no. 7, pp. 633-649.
- Wallace, W. J., Sheslow, D., & Hassink, S. 1993, "Obesity in children: a risk for depression", *Ann.N.Y.Acad.Sci.*, vol. 699, pp. 301-303.
- Waller, G. 1998, "Perceived control in eating disorders: relationship with reported sexual abuse", *Int.J.Eat.Disord.*, vol. 23, no. 2, pp. 213-216.

- Whisman, M. A., Perez, J. E., & Ramel, W. 2000, "Factor structure of the Beck Depression Inventory-Second Edition (BDI-II) in a student sample", *J.Clin.Psychol.*, vol. 56, no. 4, pp. 545-551.
- Whitaker, R. C., Wright, J. A., Pepe, M. S., Seidel, K. D., & Dietz, W. H. 1997, "Predicting obesity in young adulthood from childhood and parental obesity", *N.Engl.J Med.*, vol. 337, no. 13, pp. 869-873.
- Wilding, J., Van, G. L., Rissanen, A., Vercruyse, F., & Fitchet, M. 2004, "A randomized double-blind placebo-controlled study of the long-term efficacy and safety of topiramate in the treatment of obese subjects", *Int.J.Obes.Relat Metab Disord.*, vol. 28, no. 11, pp. 1399-1410.
- Wilfley, D. E., Schwartz, M. B., Spurrell, E. B., & Fairburn, C. G. 1997, "Assessing the specific psychopathology of binge eating disorder patients: interview or self-report?", *Behav.Res.Ther.*, vol. 35, no. 12, pp. 1151-1159.
- Wilson, G. T. & Smith, D. 1989, "Assessment of bulimia nervosa", *Int.J.Eat.Disord.*, vol. 8, pp. 173-179.
- Wimsatt, W. 1981, "Robustness, Reliability and Overdetermination," in *Scientific Enquiry and the Social Sciences*, Brewer MB & Collins BE, eds., Jossey- Basss, San Francisco, pp. 124-163.
- Winkelman, J. W., Herzog, D. B., & Fava, M. 1999, "The prevalence of sleep-related eating disorder in psychiatric and non-psychiatric populations", *Psychol.Med.*, vol. 29, no. 6, pp. 1461-1466.
- Winkelman, J. W. 2003, "Treatment of nocturnal eating syndrome and sleep-related eating disorder with topiramate", *Sleep Med.*, vol. 4, no. 3, pp. 243-246.
- Wolk, R., Gami, A. S., Garcia-Touchard, A., & Somers, V. K. 2005, "Sleep and cardiovascular disease", *Curr.Probl.Cardiol.*, vol. 30, no. 12, pp. 625-662.
- World Health Organisation 2003, *Diet, Nutrition and the Prevention of Chronic Diseases. Report of a joint WHO/FAO expert consultation.*
- Yaggi, H. K., Araujo, A. B., & McKinlay, J. B. 2006, "Sleep duration as a risk factor for the development of type 2 diabetes", *Diabetes Care*, vol. 29, no. 3, pp. 657-661.
- Youngstedt, S. D. & Kripke, D. F. 2004, "Long sleep and mortality: rationale for sleep restriction", *Sleep Med.Rev.*, vol. 8, no. 3, pp. 159-174.