ANOREXIA NERVOSA:
CONTROVERSY REGARDING DIAGNOSTIC CRITERIA AND
EVIDENCE FOR CONTROL-RELATED COGNITIVE AND METACOGNITIVE
MECHANISMS MAINTAINING THE DISORDER

SIEW PENG SOON

Submitted in total fulfilment of requirements of the degree of
Doctor of Philosophy

November 2012

Melbourne School of Psychological Sciences
The University of Melbourne

Produced on archival quality paper
ABSTRACT

Background. The study was divided into three sections. (i) The utility of current controversial physical diagnostic criteria for Anorexia Nervosa (AN), specifically, the weight threshold and amenorrhea. A literature review revealed no clinically relevant differences signified by the physical criteria; however, existing studies had not assessed the cognitive diagnostic criteria for AN and had excluded individuals on oral contraception. (ii) Perfectionism, self-esteem, control-related cognitions, and dysfunctional metacognitions have been proposed to maintain AN, yet empirical evidence is limited, nor have existing studies controlled for the potentially confounding effects of low weight, depression, and anxiety. (iii) The lack of efficacy in treating AN may reflect our limited understanding of the mechanisms underlying its maintenance and treatment, highlighting the need for model testing. Fairburn, Shafran, and Cooper (1999) proposed a cognitive model constructed on the individual’s need for control; however, empirical evidence for the model is limited. A potentially useful addition to a maintenance model is the untested higher level of cognition, metacognition and its dysfunctionality in AN.

Research Aims. (i) Test for differences between subgroups of AN patients signified by each physical criterion, and by oral contraceptive use, on clinically relevant factors including cognitive AN diagnostic criteria, AN maintenance factors, and general psychopathology. (ii) Assess differences between AN and dieting and non-dieting comparison groups in perfectionism, self-esteem, control-related cognitions, and metacognitions, whilst controlling for effects of low weight, depression, and anxiety. (iii) Test two control-centred maintenance models, Model A (cognitive level) and Model B (metacognitive level), in predicting the outcome variable, drive for thinness, in AN and non-eating disordered comparison groups.

Method. Participants were 110 female AN patients and 132 non-eating disordered females (66 dieting, 66 non-dieting) from the community, aged 18 to 45 years. Information was collected through self-report measures. (i) ANOVAs and t-tests were used for group comparisons. (ii) MANOVAs, MANCOVAs and ANCOVAs were used for group comparisons, with body mass index, depression, and anxiety as covariates. (iii) Path analyses were used to test Model A and Model B.

Results. (i) Measures were remarkably similar for all AN subgroups \((p > .05)\), with few statistically significant differences. (ii) Variables were rated as significantly
more severe in the AN group compared with the comparison groups (Bonferroni-adjusted \( p < .006 \)). Differences were of clinical significance, still present after controlling for low weight, depression, and anxiety. (iii) In the AN group only, overall fit of Models A and B were excellent, with key predicted pathways confirmed \( (p < .05) \).

**Conclusion.** Findings have implications for DSM-V development, confirming that the weight and amenorrhea criteria do not signify clinically relevant differences and should be applied with caution when used. Perfectionism, self-esteem, and control-related cognitions and metacognitions distinguished AN from the non-eating disordered comparison sample. Preliminary empirical evidence supported the two control-related maintenance models of drive for thinness in AN, one at the cognitive level as proposed in Fairburn et al.’s (1999) cognitive theory of AN, and the other a novel metacognitive model, both including perfectionism and self-esteem. Control-related cognitive and metacognitive variables are thus potential targets of AN treatment.
DECLARATION

This is to certify that

(i) the thesis comprises only my original work towards the PhD;
(ii) due acknowledgement has been made in the text to all other material used;
(iii) the thesis is fewer than 100,000 words in length, exclusive of tables, maps, bibliographies, appendices, and footnotes;
(iv) the research reported in the thesis was conducted in accordance with the principles for the ethical treatment of human subjects as approved for this research by the University of Melbourne Human Research Ethics Committee, the Melbourne Health Human Research Ethics Committee, and The Melbourne Clinic Research Ethics Committee.

_____________________________________________
Siew Peng Soon
ACKNOWLEDGEMENTS

First and foremost, I would like to express my deepest appreciation and gratitude towards my supervisor, Dr Nola Rushford, who not only provided me with exceptional academic guidance, generosity of her time, lighting-quick reviews of my endless (boring) drafts, invaluable insight from her vast knowledge, frequent bursts of brilliant suggestions, but for so much more than that. Her endless patience, encouragement, compassion, and concern for my personal happiness and well-being has nurtured me over the years, contributed to my increased confidence, and rendered this research experience a thoroughly enjoyable one. It has been an incredible honour to work with Nola, and I thank her for allowing my never-ending PhD candidature to repeatedly foil her retirement plans.

Also, I extend my sincere thanks to members of my PhD Academic Advisory Committee, Associate Professor Lisa Phillips, Dr Paul Dudgeon, and Professor Henry Jackson, whose suggestions and recommendations were instrumental to the fruition of this thesis. Their support prevented me from falling to pieces in dire and trying times, for which I am truly thankful.

I am also deeply grateful to a very special colleague, Dr Kim-Michelle Gilson, who dealt with my weekly thesis-related meltdowns with limitless positivity and love. She was my go-to person for discussing issues from structural equation modelling dramas to personal life difficulties—that says a lot about how much she was there for me! Kim brought laughter and fun into the research process, and it is my privilege to have found a friend like her.

Further, I would like to thank my colleagues Ms Dana Ben-Israel and Ms Susan Bullock, whose assistance in collecting the data for this thesis made the process infinitely more bearable. Their friendship and our shared experiences, traumatic and otherwise, have kept me going through periods of despair. Dana and Suzy are partly responsible for my intact mental health through getting me started on outdoor running, providing me with much needed stress-relief and fresh air.

I would not have succeeded without the generous and selfless support of my family. Dad, thank you for teaching me through example that it is possible to be enthusiastic about your work every day, and for instilling in me the importance of choosing a profession that you enjoy. (Although admittedly, I cannot fathom how you adore engineering the way you do; you are truly the original nerd in the family.) Your love and support has inspired me and given me the confidence to take risks and face
failure, and I am eternally grateful. Mum, your unwavering belief in my capabilities and push for me to achieve my full potential has given me faith in myself. Thank you for the little things that demonstrate your ceaseless love: always checking in on me despite being so far away, and being immensely interested in mundane details of my work and research. I feel truly cared for and wish I could give you back the lost nights of sleep that were spent worrying about me. (For all your stress, you will be able to say that you have a daughter who is a doctor, albeit a “fake” one who cannot tell the difference between ringworm and chemical burns, nor prescribe you medication at your convenience.) And of course, my gorgeous sisters, Ms Siewhui and Siewling Soon, I am grateful to you for your perpetual enthusiasm and wonderful companionship throughout this process (here is where I wish I could insert emoticons of hearts and kisses). Siewhui has been my resident cheerleader and a great source of comfort, and I am truly blessed to have her continually rooting for me despite being thousands of miles away. My adorable grandfather, who constantly drops unsubtle hints that he would like to see me graduate in his lifetime, also deserves a mention: 公公，我终于要博士毕业了！谢谢您从小照顾我，疼我，对我有这么多信心。我很感激您和阿妈，如果没有你们的爱，就不会有今天的我。To Ms Kong Sok Yean or Auntie Vivian, who is family to me, I would like to express my appreciation for teaching me that there is more to life than chasing after “success” in the meaningless rat race, thereby always putting things into perspective when I lose my way and am overwhelmed by the enormity of the task of this thesis. For extending your warmth, affection, and encouragement, and for treating me like a daughter, thank you.

Last but not least, I am greatly indebted to Ms Kexin Fong, who made immeasurable sacrifices by putting her career on hold and relocating halfway across the world to support me in the writing of this thesis. Without her ability to pre-empt my imminent episodes of hysterical panic or read my convoluted mind, her insistence that I occasionally have fun, her gift for making delicious meals appear magically at my desk when I forget to eat, and the million other day-to-day things she does to express her quiet but steadfast care, I would not have completed this thesis with my sanity intact. I would like to say so much more but words are inadequate. I thank her from the bottom of my heart, and am truly blessed to have her in my life.
# Table of Contents

LIST OF ABBREVIATIONS ........................................................................................................... x

LIST OF TABLES .......................................................................................................................... xii

LIST OF FIGURES ...................................................................................................................... xiv

CHAPTER 1: INTRODUCTION .................................................................................................. 1

CHAPTER 2: DIAGNOSTIC CATEGORIES & PREVALENCE OF EATING DISORDERS ...................... 3

1. ANOREXIA NERVOSA ........................................................................................................... 5
   1.1 Current diagnostic criteria 5
   1.2 Previous and proposed diagnostic criteria 6

2. BULIMIA NERVOSA ............................................................................................................. 7
   2.1 Current diagnostic criteria 7
   2.2 Previous and proposed diagnostic criteria 8

3. EATING DISORDER NOT OTHERWISE SPECIFIED ................................................................ 9
   3.1 Current diagnostic criteria 9
   3.2 Proposed diagnostic categories 10

4. THE PREVALENCE OF EATING DISORDERS ..................................................................... 11
   4.1 Treatment-seeking samples 11
   4.2 Community samples 13

5. IMPLICATIONS FOR THE PRESENT STUDY ..................................................................... 15

CHAPTER 3: ISSUES WITH THE WEIGHT & AMENORRHEA CRITERIA FOR ANOREXIA NERVOSA ............ 17

1. THE WEIGHT CRITERION .................................................................................................... 18
   1.1 Methods of calculating the weight cut-off 19
   1.2 Dissent on best method of determining if criterion is met 19
   1.3 Empirical evidence for clinical differences signified by the criterion 20
   1.4 Implications for the present study 26

2. THE AMENORRHEA CRITERION ......................................................................................... 27
   2.1 Rationale behind the criterion 28
   2.2 Inconsistencies between individuals in development of amenorrhea 29
   2.3 Empirical evidence for clinical differences signified by the criterion 30
   2.4 Use of the oral contraceptive pill in individuals with Anorexia Nervosa 39
   2.5 Implications for the present study 40

3. AIMS AND RESEARCH QUESTIONS .................................................................................. 42
   3.1 Aims 42
   3.2 Variables assessed in the present study 43
   3.3 Research question 1 44
CHAPTER 4: EXAMINATION OF RISK & MAINTENANCE FACTORS OF ANOREXIA NERVOSA

1. BRIEF OVERVIEW OF FACTORS ASSOCIATED WITH ANOREXIA NERVOSA

2. PERFECTIONISM
   2.1 Theoretical relationship with Anorexia Nervosa
   2.2 Empirical evidence for its association with Anorexia Nervosa

3. LOW SELF-ESTEEM
   3.1 Theoretical relationship with Anorexia Nervosa
   3.2 Empirical evidence for its association with Anorexia Nervosa

4. CONTROL-RELATED COGNITIONS
   4.1 Theoretical relationship with Anorexia Nervosa
   4.2 Empirical evidence for its association with Anorexia Nervosa

5. DYSFUNCTIONAL METACOGNITIONS
   5.1 Theoretical relationship with Anorexia Nervosa
   5.2 Empirical evidence for its association with Anorexia Nervosa

6. AIMS AND RESEARCH QUESTIONS
   6.1 Aims
   6.2 Research question 2

CHAPTER 5: TWO COGNITIVE MAINTENANCE MODELS OF ANOREXIA NERVOSA

1. EXISTING COGNITIVE THEORIES FOR ANOREXIA NERVOSA: BACKGROUND FOR MODEL A
   1.1 Garner and Bemis’ (1982) cognitive model
   1.2 Fairburn and colleagues’ (1999) cognitive model
   1.3 The basis of Model A
   1.4 Empirical evidence for cognitive models of Anorexia Nervosa
      1.4.1 Evidence for mechanisms involving perfectionism
      1.4.2 Evidence for mechanisms involving self-esteem
      1.4.3 Evidence for mechanisms involving control-related cognitions
      1.4.4 Indirect evidence from treatment trials of cognitive therapy
      1.4.5 Implications for the present study

2. ADVANCEMENTS IN COGNITIVE THEORY FOR ANOREXIA NERVOSA: BACKGROUND FOR MODEL B
   2.1 Enhanced cognitive theories
   2.2 The relevance of metacognitive theory
   2.3 The basis of Model B

3. MODEL TESTING
   3.1 Model A
   3.2 Model B

4. AIMS AND RESEARCH QUESTIONS
   4.1 Aims
   4.2 Research question 3
CHAPTER 6: METHOD ............................................................................................................. 136

1. PARTICIPANTS .................................................................................................................. 136

2. PROCEDURE ..................................................................................................................... 137

3. MEASURES ....................................................................................................................... 138
   3.1 Demographic and weight-related information ....................................................... 139
   3.2 Diagnostic criteria for Anorexia Nervosa .............................................................. 139
   3.3 Common eating disorder characteristics ............................................................... 141
   3.4 Control-related cognitions ..................................................................................... 142
   3.5 Dysfunctional metacognitions .............................................................................. 144
   3.6 General psychopathology ...................................................................................... 145

4. STATISTICAL ANALYSES ............................................................................................... 146
   4.1 Research question 1 ............................................................................................... 146
   4.2 Research question 2 ............................................................................................... 147
   4.3 Research question 3 ............................................................................................... 147

CHAPTER 7: RESULTS ........................................................................................................... 151

1. PRELIMINARY ANALYSES ............................................................................................... 151
   1.1 Missing data ........................................................................................................... 151
   1.2 Validation of newly created “General Need for Control” variable ......................... 151
   1.3 Combining patient groups: Inpatients, day patients, and outpatients ................ 153
   1.4 Differentiating comparison groups: Dieters and non-dieters ................................ 154
   1.5 Descriptive statistics ............................................................................................. 155

2. RESEARCH QUESTION 1 .................................................................................................... 157
   2.1 Analysis specific participants .............................................................................. 157
   2.2 Assumption testing ............................................................................................... 158
   2.3 Research question 1(a): Univariate ANOVA & post-hoc power analysis results .... 159
   2.4 Research question 1(b): Independent samples t-test results ................................ 161

3. RESEARCH QUESTION 2 .................................................................................................... 162
   3.1 Analysis specific participants .............................................................................. 162
   3.2 Assumption testing ............................................................................................... 162
   3.3 Research question 2(a): MANOVA results ............................................................ 162
   3.4 Research question 2(b): MANCOVA results ....................................................... 166
   3.5 Research question 2(c): ANCOVA results ........................................................... 166
   3.6 Research question 2(d): MANCOVA results ....................................................... 167

4. RESEARCH QUESTION 3 .................................................................................................... 168
   4.1 Analysis specific participants .............................................................................. 168
   4.2 Assumption testing ............................................................................................... 168
   4.3 Correlations between variables in Models A & B .................................................. 169
   4.4 Research question 3(a): Model A path analysis results ........................................ 172
      4.4.1 Anorexia Nervosa group ............................................................................... 172
      4.4.2 Non-eating disordered comparison group ..................................................... 173
      4.4.3 Conclusion ..................................................................................................... 174
   4.5 Research question 3(b): Model B path analysis results ........................................ 175
      4.5.1 Anorexia Nervosa group ............................................................................... 175
      4.5.2 Non-eating disordered comparison group ..................................................... 177
      4.5.3 Conclusion ..................................................................................................... 178
CHAPTER 8: DISCUSSION........................................................................................................ 179

1. RESEARCH QUESTION 1(A) .......................................................................................... 179
   1.1 Interpretation of findings ......................................................................................... 179
   1.2 Application of findings in context of previous literature ....................................... 180
   1.3 Implications: DSM-V, clinical and research .......................................................... 181
   1.4 Strengths, limitations, and future directions ......................................................... 184

2. RESEARCH QUESTION 1(B) .......................................................................................... 185
   2.1 Interpretation of findings ......................................................................................... 185
   2.2 Application of findings in context of previous literature ....................................... 186
   2.3 Implications: DSM-V, clinical and research .......................................................... 186
   2.4 Strengths, limitations, and future directions ......................................................... 187

3. RESEARCH QUESTIONS 2(A) TO (D) ........................................................................... 188
   3.1 Interpretation of findings ......................................................................................... 188
   3.2 Application of findings in context of previous literature ....................................... 190
   3.3 Clinical and research implications ....................................................................... 195
   3.4 Strengths, limitations, and future directions ......................................................... 195

4. RESEARCH QUESTIONS 3(A) AND (B) ....................................................................... 197
   4.1 Interpretation of findings ......................................................................................... 197
   4.2 Application of findings in context of previous literature ....................................... 199
   4.3 Clinical and research implications ....................................................................... 205
   4.4 Strengths, limitations, and future directions ......................................................... 206

5. GENERAL LIMITATIONS AND RECOMMENDATIONS FOR FUTURE RESEARCH ........ 208

6. CONCLUSION ................................................................................................................. 210
   6.1 Findings that clarify and extend current knowledge .............................................. 210
   6.2 Novel findings ....................................................................................................... 211
   6.3 Summary .............................................................................................................. 213

REFERENCES .................................................................................................................... 215

APPENDICES ..................................................................................................................... 242

APPENDIX A: DESCRIPTION OF TREATMENT MODALITIES ...................................... 242
APPENDIX B: PARTICIPANT INFORMATION & CONSENT FORM .................................. 243
APPENDIX C: “IS YOUR WEIGHT TOO LOW FOR GOOD HEALTH?” CHECKLIST ............. 250
APPENDIX D: DEMOGRAPHIC & WEIGHT-RELATED QUESTIONS .............................. 252
APPENDIX E: EATING DISORDER INVENTORY, 3rd EDITION ....................................... 253
APPENDIX F: ROSENBERG SELF-ESTEEM SCALE ....................................................... 257
APPENDIX G: METACOGNITIONS QUESTIONNAIRE, BRIEF VERSION ...................... 258
APPENDIX H: DEPRESSION ANXIETY STRESS SCALES .......................................... 260
**LIST OF ABBREVIATIONS**

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>β</td>
<td>Standardised path coefficient</td>
</tr>
<tr>
<td>η²</td>
<td>Eta-squared</td>
</tr>
<tr>
<td>ηp²</td>
<td>Partial eta-squared</td>
</tr>
<tr>
<td>χ²</td>
<td>Chi-square</td>
</tr>
<tr>
<td>AN</td>
<td>Anorexia Nervosa</td>
</tr>
<tr>
<td>ANCOVA</td>
<td>Analysis of covariance</td>
</tr>
<tr>
<td>ANOVA</td>
<td>Analysis of variance</td>
</tr>
<tr>
<td>APA</td>
<td>American Psychiatric Association</td>
</tr>
<tr>
<td>BED</td>
<td>Binge Eating Disorder</td>
</tr>
<tr>
<td>BMI</td>
<td>Body mass index</td>
</tr>
<tr>
<td>BN</td>
<td>Bulimia Nervosa</td>
</tr>
<tr>
<td>c.r.</td>
<td>Critical ratio</td>
</tr>
<tr>
<td>CBT</td>
<td>Cognitive behavioural therapy</td>
</tr>
<tr>
<td>CFI</td>
<td>Comparative fit index</td>
</tr>
<tr>
<td>CI</td>
<td>Confidence interval</td>
</tr>
<tr>
<td>DASS</td>
<td>Depression Anxiety Stress Scales</td>
</tr>
<tr>
<td>DSM</td>
<td>Diagnostic and Statistical Manual of Mental Disorders</td>
</tr>
<tr>
<td>DV</td>
<td>Dependent variable</td>
</tr>
<tr>
<td>ED</td>
<td>Eating disorder</td>
</tr>
<tr>
<td>EDE</td>
<td>Eating Disorder Examination</td>
</tr>
<tr>
<td>EDI</td>
<td>Eating Disorder Inventory</td>
</tr>
<tr>
<td>EDNOS</td>
<td>Eating Disorder Not Otherwise Specified</td>
</tr>
<tr>
<td>FIML</td>
<td>Full information maximum likelihood</td>
</tr>
<tr>
<td>ICD</td>
<td>International Statistical Classification of Diseases and Related Health Problems</td>
</tr>
<tr>
<td>IPT</td>
<td>Interpersonal therapy</td>
</tr>
<tr>
<td>MANCOVA</td>
<td>Multivariate analysis of covariance</td>
</tr>
<tr>
<td>MANOVA</td>
<td>Multivariate analysis of variance</td>
</tr>
<tr>
<td>MCQ-30</td>
<td>Metacognitions Questionnaire—Brief Version</td>
</tr>
<tr>
<td>NICE</td>
<td>The National Institute for Clinical Excellence</td>
</tr>
<tr>
<td>OCP</td>
<td>Oral contraceptive pill</td>
</tr>
<tr>
<td>RANZCP</td>
<td>The Royal Australian and New Zealand College of Psychiatrists</td>
</tr>
<tr>
<td>RMSEA</td>
<td>Root mean square error of approximation</td>
</tr>
<tr>
<td>RSES</td>
<td>Rosenberg Self-Esteem Scale</td>
</tr>
<tr>
<td>S-REF</td>
<td>Self-Regulatory Executive Function</td>
</tr>
<tr>
<td>SCID</td>
<td>Structured Clinical Interview for the DSM Disorders</td>
</tr>
<tr>
<td>Tukey HSD</td>
<td>Tukey’s honestly significant difference test</td>
</tr>
<tr>
<td>Short Code</td>
<td>Description</td>
</tr>
<tr>
<td>------------</td>
<td>------------------------------</td>
</tr>
<tr>
<td>UK</td>
<td>United Kingdom</td>
</tr>
<tr>
<td>US</td>
<td>United States</td>
</tr>
<tr>
<td>YSQ</td>
<td>Young Schema Questionnaire</td>
</tr>
</tbody>
</table>
# List of Tables

<table>
<thead>
<tr>
<th>Table</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1</td>
<td>DSM-IV-TR Diagnostic Criteria and Subtypes for Anorexia Nervosa</td>
<td>6</td>
</tr>
<tr>
<td>2.2</td>
<td>DSM-IV-TR Diagnostic Criteria and Subtypes for Bulimia Nervosa</td>
<td>8</td>
</tr>
<tr>
<td>2.3</td>
<td>ED Prevalence Studies in Adult Treatment-Seeking Samples using DSM-IV Criteria</td>
<td>13</td>
</tr>
<tr>
<td>2.4</td>
<td>ED Prevalence Studies in Community Samples using DSM-IV Criteria</td>
<td>15</td>
</tr>
<tr>
<td>3.1</td>
<td>Studies Comparing Full-syndrome with Subthreshold AN (Weight Criterion Not Met)</td>
<td>24</td>
</tr>
<tr>
<td>3.2</td>
<td>Studies Comparing Full-syndrome with Subthreshold AN (Amenorrhea Criterion Not Met)</td>
<td>36</td>
</tr>
<tr>
<td>3.3</td>
<td>Variables of Interest to Research Questions 1(a) and (b)</td>
<td>44</td>
</tr>
<tr>
<td>4.1</td>
<td>Factors Hypothesised to Lead to the Development and Maintenance of AN</td>
<td>47</td>
</tr>
<tr>
<td>4.2</td>
<td>Reviews of Studies Assessing Perfectionism in AN</td>
<td>53</td>
</tr>
<tr>
<td>4.3</td>
<td>Individual Studies Assessing Perfectionism in AN</td>
<td>56</td>
</tr>
<tr>
<td>4.4</td>
<td>Review of Studies Assessing Low Self-Esteem in AN</td>
<td>64</td>
</tr>
<tr>
<td>4.5</td>
<td>Individual Studies Assessing Low Self-Esteem in AN</td>
<td>67</td>
</tr>
<tr>
<td>4.6</td>
<td>Individual Studies Assessing Control-Related Cognitions in AN</td>
<td>77</td>
</tr>
<tr>
<td>4.7</td>
<td>Individual Cross-Sectional Studies Assessing Dysfunctional Metacognitions in AN</td>
<td>83</td>
</tr>
<tr>
<td>5.1</td>
<td>Studies Assessing Mechanisms of Perfectionism</td>
<td>101</td>
</tr>
<tr>
<td>5.2</td>
<td>Studies Assessing Mechanisms of Self-Esteem</td>
<td>108</td>
</tr>
<tr>
<td>5.3</td>
<td>Studies Assessing Mechanisms of Control-Related Cognitions</td>
<td>112</td>
</tr>
<tr>
<td>5.4</td>
<td>Treatment Trials for CBT in Outpatient AN Populations</td>
<td>115</td>
</tr>
<tr>
<td>5.5</td>
<td>Proposed Individual Pathways in Models A and B</td>
<td>135</td>
</tr>
<tr>
<td>5.6</td>
<td>Items Comprising the “General Need for Control” Variable</td>
<td>143</td>
</tr>
<tr>
<td>6.1</td>
<td>Subscales of the MCQ-30 and Examples of Relevant Items</td>
<td>145</td>
</tr>
<tr>
<td>7.1</td>
<td>Mean BMI and Scores on AN Diagnostic Criteria for Inpatients, Day Patients, and Outpatients</td>
<td>154</td>
</tr>
<tr>
<td>7.2</td>
<td>Mean Scores and ANOVA Results for BMI and AN Diagnostic Criteria for Dieting and Non-Dieting Comparison Groups</td>
<td>155</td>
</tr>
<tr>
<td>7.3</td>
<td>Mean Scores and Univariate ANOVA Results for Demographic and Clinical Characteristics of AN, Dieting and Non-Dieting Comparison Groups</td>
<td>156</td>
</tr>
<tr>
<td>7.4</td>
<td>Subgroups of AN Patients Meeting Full and Subthreshold AN Criteria, or on the OCP</td>
<td>157</td>
</tr>
<tr>
<td>7.5</td>
<td>Final Three Groups Used in Analyses for Research Question 1(a)</td>
<td>158</td>
</tr>
<tr>
<td>7.6</td>
<td>Mean Scores and Univariate ANOVA Results for Full Syndrome and Subthreshold AN Groups on Variables of Interest</td>
<td>160</td>
</tr>
</tbody>
</table>
7.7 Mean Scores and \( t \)-test Results for OCP and Non-OCP AN Groups on Variables of Interest 161

7.8 Mean Scores and Separate Univariate \( F \)-tests from MANOVA for AN, Dieting, and Non-Dieting Comparison Groups on Variables of Interest 165

7.9 Estimated Marginal Means (Controlling for BMI) and Separate Univariate \( F \)-tests from MANCOVA for AN, Dieting, and Non-Dieting Comparison Groups on Variables of Interest 166

7.10 Means and Estimated Marginal Means (Controlling for Depression) for AN, Dieting, and Non-Dieting Comparison Groups on Self-Esteem 167

7.11 Means and Estimated Marginal Means (Controlling for Depression and Anxiety) for AN, Dieting, and Non-Dieting Comparison Groups on Control-Related Metacognitive Variables 168

7.12 Means, Standard Deviations, and Correlations Between Variables in Models A and B for the AN Group 171

7.13 Means, Standard Deviations, and Correlations Between Variables in Models A and B for the Non-Eating Disordered Comparison Group 171
### LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.1</td>
<td>Schematic representation of the development section of Garner &amp; Bemis’ (1982) cognitive model of AN</td>
</tr>
<tr>
<td>5.2</td>
<td>Schematic representation of the maintenance section of Garner &amp; Bemis’ (1982) cognitive model of AN</td>
</tr>
<tr>
<td>5.3</td>
<td>Schematic representation of Fairburn et al.’s (1999) cognitive maintenance model of AN</td>
</tr>
<tr>
<td>5.4</td>
<td>Schematic representation of the metacognitive model of psychological disorder or the Self-Regulatory Executive Function model, adapted from Wells and Matthews’ (1996) diagram</td>
</tr>
<tr>
<td>5.5</td>
<td>Schematic representation of the Driving Mechanism section from Fairburn et al.’s (1999) cognitive maintenance model of AN</td>
</tr>
<tr>
<td>5.6</td>
<td>Schematic representation of Feedback Mechanism 1 from Fairburn et al.’s (1999) cognitive maintenance model of AN</td>
</tr>
<tr>
<td>5.7</td>
<td>Schematic representation of Model A</td>
</tr>
<tr>
<td>5.8</td>
<td>Schematic representation of Model B</td>
</tr>
<tr>
<td>6.1</td>
<td>Visual analogue scales measuring fear of gaining weight and self-worth invested in the body</td>
</tr>
<tr>
<td>6.2</td>
<td>Visual analogue scale measuring body image disturbance</td>
</tr>
<tr>
<td>6.3</td>
<td>Visual analogue scale measuring sense of control dependent on eating/weight</td>
</tr>
<tr>
<td>7.1</td>
<td>Standardised regression weights and variance explained by “General Need for Control” variable for each indicator variable</td>
</tr>
<tr>
<td>7.2</td>
<td>Error bars representing 95% confidence intervals for the mean perfectionism scores for AN, dieting, and non-dieting comparison groups</td>
</tr>
<tr>
<td>7.3</td>
<td>Proposed Model A assessed in AN sample, with standardised regression weights and variance explained for each endogenous variable</td>
</tr>
<tr>
<td>7.4</td>
<td>Revised Model A assessed in AN sample, with standardised regression weights and variance explained for each endogenous variable</td>
</tr>
<tr>
<td>7.5</td>
<td>Proposed Model A assessed in non-eating disordered comparison sample, with standardised regression weights and variance explained for each endogenous variable</td>
</tr>
<tr>
<td>7.6</td>
<td>Proposed Model B assessed in AN sample, with standardised regression weights and variance explained for each endogenous variable</td>
</tr>
<tr>
<td>7.7</td>
<td>Revised Model B assessed in AN sample, with standardised regression weights and variance explained for each endogenous variable</td>
</tr>
<tr>
<td>7.8</td>
<td>Proposed Model B assessed in non-eating disordered comparison sample, with standardised regression weights and variance explained for each endogenous variable</td>
</tr>
</tbody>
</table>
CHAPTER 1: INTRODUCTION

The thesis comprises three research topics. Research Question 1 examines the utility of current physical diagnostic criteria for Anorexia Nervosa (AN); Research Question 2 examines existing and novel maintenance factors for AN; and Research Question 3 involves the testing of control-related cognitive and metacognitive mechanisms through which the factors maintain the disorder. The following chapters provide the background, a review of existing literature, and the rationale for each topic. Then, the methods and procedure used for testing the three research questions are described and results provided. Finally, implications of the results, limitations of the research, and recommendations for future research are discussed.

Chapter 2 provides a summary of current, previous, and proposed future Diagnostic and Statistical Manual of Mental Disorders (DSM) criteria for eating disorders (EDs), including AN, Bulimia Nervosa (BN), and Eating Disorder Not Otherwise Specified (EDNOS). It examines the prevalence of EDs in clinical and non-clinical populations, concluding that the high prevalence of EDNOS, comprised largely of individuals with subthreshold AN, has implications for the present research project.

In Chapter 3, the controversial physical diagnostic criteria of AN, the weight and amenorrhea criteria, are examined in greater detail. A review of the criteria and their sometimes inconsistent development, difficulties with methods used to determine if the criteria are met, and clinical differences signified by the criteria is undertaken. Implications for the present research project are drawn. Further, use of the oral contraceptive pill (OCP) is discussed with regards to amenorrhea status. Research Questions 1(a) and (b) are developed to examine clinical differences signified by both physical criteria, and by OCP use, in individuals with AN.

Chapter 4 details the proposed risk and maintenance factors of AN in existing literature, focusing on perfectionism, low self-esteem, control-related cognitions, and dysfunctional metacognitions. For each factor, the proposed theoretical relationship with AN and existing empirical evidence for them are reviewed. Research Questions 2(a) to (d) are developed to examine differences between AN, dieting, and non-dieting comparison groups, taking into account potential confounding factors of low weight, depression, and anxiety.
In Chapter 5, forming the basis for proposed Model A, two classic cognitive theories of AN by Garner and Bemis (1982) and Fairburn, Shafran, and Cooper (1999) are described, and the scant empirical evidence for these models reviewed. Then, to set the groundwork for Model B, newer enhanced cognitive theories and the relevance of metacognitive theory for AN are discussed. The rationale for development of Models A and B, proposed maintenance mechanisms for AN based on issues of control at the cognitive and metacognitive level respectively, is described. Research Questions 3(a) and (b) are developed to test Models A and B in AN and non-eating disordered comparison groups.

In Chapter 6, the methods for the research project are documented, including descriptions of participant groups, the procedure for data collection, self-report measures utilised, and statistical analyses applied to each research question.

Chapter 7 provides results from statistical analyses. They include preliminary analyses pertaining to missing data, validation of a newly created measure, the combination or differentiation of subgroups, and descriptive statistics. Then, results for each research question are provided, comprising a description of analysis-specific participant groups, testing of assumptions for statistical analyses, and findings pertaining to each research question.

Finally, the discussion of findings is presented in Chapter 8. For each research question, findings are interpreted in the context of previous literature, clinical and research implications are deliberated, and strengths, limitations, and future directions considered. General limitations of the research project as a whole are stated with recommendations for future research. To conclude, findings are briefly categorised into being an extension of current knowledge, or novel.
CHAPTER 2: DIAGNOSTIC CATEGORIES & PREVALENCE OF EATING DISORDERS

There has been considerable debate regarding diagnostic categories for eating disorders, particularly diagnostic criteria for AN. In the most recent text revision of the DSM (DSM-IV-TR; American Psychiatric Association, 2002) on which the present research is based, EDs have been classified into two specific diagnoses—AN and BN, differentiated on physical weight, behavioural symptoms, and psychopathological constructs. Disorders of eating that do not meet the full criteria for AN or BN are classified as EDNOS. The key concern is that the majority of patients being treated in clinical settings for EDs do not meet full criteria for AN or BN, presenting with “partial syndrome”, “subthreshold”, or “subclinical” disorders, and are resultantly classified under the residual EDNOS category, conveying very little about their symptoms or illness severity. This is more common for diagnoses of subthreshold AN than BN.

Due to these issues in the existing DSM diagnostic classification, some recent trends have favoured other methods of classification. For example, it has been suggested that EDs exist on a continuum of severity rather than discrete categories (Polivy & Herman, 1987; Shisslak, Crago, & Estews, 1995), that EDs are one syndrome with a spectrum of different manifestations (VanderHam, Meulman, VanStrien, & vanEngeland, 1997), and that a single “transdiagnostic” category of EDs should be used in place of distinct disorders (Fairburn & Walsh, 2002, Fairburn & Bohn, 2005). These suggestions have been proposed because of apparent similarities between EDs in cognition and symptoms, however it is also important to remember that even apparently similar behaviour may be maintained by very different cognitions, which may have different implications for treatment (Cooper, 2005). Existing empirical evidence suggested that treatment outcomes of AN and BN were starkly different, with psychological treatment for BN considerably more effective than for AN (for a review see Hay, Bacaltchuk, Stefano, & Kashyap, 2009). A large proportion of AN patients developed chronic disabilities with only about a third in remission after one year of different specialist treatments (Hay, Bacaltchuk, Claudino, Ben-Tovim, & Yong, 2003; Treasure & Schmidt, 2005), and there has been no conclusive evidence that any psychological therapy is effective in the treatment of AN (for reviews see Wilson, Grilo, & Vitousek, 2007; Agras & Robinson, 2008; Bulik, Berkman, Brownley, Sedway, & Lohr, 2007), providing indirect evidence that AN is clinically distinct from BN. There is robust evidence, including genetics, epidemiology, response to psychotropic drugs and
neuroimaging suggesting that AN should be considered a distinct and separate condition (Birmingham, Touyz, & Harbottle, 2009; Clinton, Button, Norring, & Palmer, 2004; Keel et al., 2004; for a review, see Collier & Treasure, 2004). Interestingly, a recent study assessed the concept of a transdiagnostic theory of EDs (i.e., that AN and BN are part of one disease with a common causality) and the prudence of combining AN, BN, and EDNOS into a single diagnosis (Birmingham et al., 2009). The study found that the hypothesis that AN and BN were a single disorder of a common cause was not supported by Hill’s (1965) Criteria of Causation and the transdiagnostic theory did not meet the minimal conditions needed to establish a causal relationship. Whilst these newer models of EDs have highlighted the overlap and similarities between diagnostic groups, the alternative classification systems are lacking in empirical validation and have not been adequately developed for research or clinical purposes.

Although the DSM classification is imperfect, the purpose of clinical classification is to aid clinical work and provide guidance regarding differences in treatment response and prognosis (Dilling, 2000). Transdiagnostic theories would change diagnostic criteria, which would result in a change in the specificity and sensitivity of diagnosis (First et al., 2004), making it impossible to draw historical comparisons of EDs, compare treatment efficacy to historical controls, or estimate sample size for clinical trials (Birmingham et al., 2009). Also, if BN and EDNOS were merged with AN, the morbidity and mortality rate of AN would be homogenised and drop considerably, de-emphasising the seriousness of AN and possibly resulting in a reduction of funding for treatment in this medically complicated group (Birmingham et al., 2009). Hence, whilst these proposed alternative classification systems are noteworthy, they have not been empirically supported or adequately developed, and further discussion is beyond the scope of the present study. The DSM categorical approach, which is a more appropriate fit to the present-day medical model (Szmukler, 1985), will be applied with caution and flexibility to the present study. Maintaining the EDs as separate diagnoses will allow for comparison of epidemiology and treatment interventions over time.

This chapter aims to give an overview of current DSM-IV-TR diagnostic criteria for AN (Section 1), BN (Section 2), and EDNOS (Section 3), and compare these with previous versions of the DSM and proposed DSM-V diagnostic criteria for each ED category. Then, the prevalence of EDs in clinical and community samples will be examined (Section 4) and implications for the present study discussed (Section 5).
1. ANOREXIA NERVOSA

1.1 CURRENT DIAGNOSTIC CRITERIA

The diagnostic criteria for AN as defined in the DSM-IV-TR (APA, 2002) are shown in Table 2.1. They consist of three components: behavioural, psychological, and physical criteria. Behaviourally, AN is characterised by a relentless pursuit of thinness and refusal to maintain a normal body weight involving extreme restriction of food, usually accompanied by excessive exercise, and/or purging and binge-eating. The presence or absence of purging/binge-eating defines the subtypes of AN (Table 2.1). Psychologically, AN is characterised by dysfunctional thoughts and beliefs about food, eating, weight and shape, including an intense fear of gaining weight, body image disturbance in which the body is perceived as larger than reality, undue influence of weight or shape on self-esteem, and denial of the seriousness of low weight. The two physical criteria are being clinically underweight and in females, the absence of regular menses or amenorrhea.

The dysfunctional psychological characteristics of AN, fear of gaining weight (Criterion B) and body image disturbance (Criterion C), have been found to characterise many patients with this disorder in North America, Western Europe, and Australia (Mitchell, Cook-Myers, & Wonderlich, 2005). However, the physical diagnostic criteria of being underweight (Criterion A) and amenorrhea (Criterion D) have been contentious. According to the DSM-IV-TR, should an individual make extreme efforts to lose weight and meet all psychological criteria of AN, except that despite significant weight loss, weight is still in the normal range, or she has regular menses, the individual would be classified into EDNOS (APA, 2002). This is problematic as the EDNOS diagnosis has many clinical and research implications. The controversy surrounding the weight and amenorrhea criteria for diagnosis of AN are examined in detail in Chapter 3.
<table>
<thead>
<tr>
<th>Diagnostic Criteria</th>
<th>Subtypes</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Refusal to maintain body weight at or above a minimally normal weight for age and height;</td>
<td><strong>Restricting Type:</strong> during the current episode of Anorexia Nervosa, the person has not regularly engaged in binge-eating or purging behaviour (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas)</td>
</tr>
<tr>
<td>B. Intense fear of gaining weight or becoming fat, even though underweight;</td>
<td><strong>Binge-Eating/Purging Type:</strong> during the current episode of Anorexia Nervosa, the person has regularly engaged in binge-eating or purging behaviour (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas)</td>
</tr>
<tr>
<td>C. Disturbance in the way in which one’s body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight;</td>
<td></td>
</tr>
<tr>
<td>D. In postmenarcheal females, amenorrhea, i.e., the absence of at least three consecutive menstrual cycles.</td>
<td></td>
</tr>
</tbody>
</table>


### 1.2 Previous and Proposed Diagnostic Criteria

To highlight the arbitrary nature of the weight and amenorrhea criteria, one only has to look at previous versions of AN diagnostic criteria. In the DSM-III (APA, 1980), the weight criterion was a weight loss of at least 25% of original body weight. Along with major changes made to many diagnostic categories, this was changed in the DSM-III-R (APA, 1987) and DSM-IV (APA, 1994), to a weight loss of at least 15% below expected weight (or < 85% of ideal weight). Little change was made in the DSM-IV-TR (2002) criterion. Likewise, amenorrhea was not required for diagnosis of AN in DSM-III. It was introduced in DSM-III-R and continued as a requirement in DSM-IV and DSM-IV-TR.

Looking forward to the DSM-V, proposed guidelines indicate that there will be no substantial change to the present psychological criteria (Criteria B and C) of AN (APA, 2012). The weight criterion (Criterion A) has been proposed to be reworded to focus on restricting behaviours:

Restriction of energy intake relative to requirements leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health. Significantly low weight is defined as a weight that is less than minimally normal, or, for children and adolescents, less than that minimally expected (APA, 2012).
The DSM-V Eating Disorders Work Group has highlighted that it is not desirable to set a specific numerical standard for weight for AN, as such standards are at least somewhat arbitrary, and the judgment of whether weight is inappropriately low is best made by the clinician in light of all relevant information including age, sex, developmental trajectory, and physical health (APA, 2012). In DSM-V, it is also proposed that the current amenorrhea criterion (Criterion D) be eliminated as there are individuals who exhibit all other AN symptoms but report at least some menstrual activity, and the criterion cannot be applied to females who are pre-menarchal, post-menopausal, or on the OCP, and males (APA, 2012).

2. **BULIMIA NERVOSA**

2.1 **CURRENT DIAGNOSTIC CRITERIA**

To appreciate the full picture of ED diagnostic categories, BN will be briefly discussed although it is not a focus of the present study. Table 2.2 presents the DSM-IV-TR (APA, 2002) diagnostic criteria for BN. Behaviourally, the disorder is characterised by repeated episodes of binge eating, followed by inappropriate compensatory behaviours such as purging, laxative use, or excessive exercise. There is a frequency criterion for binging and purging, requiring these behaviours to take place at least twice a week for three months to qualify for diagnosis. Psychologically, BN is characterised by the undue influence of weight or shape on self-esteem. There is no weight criterion for BN. The disorder is also divided into two subtypes: purging or non-purging type.

Whilst there is little disagreement over the behavioural (Criteria A and B) and psychological criteria (Criterion D) for BN, the minimum frequency criterion of binging (Criterion C) has been a matter of contention. If an individual were to meet all behavioural and psychological criteria of BN, but binge less than twice a week, a diagnosis of EDNOS would be given. Individuals who fall into the subthreshold BN group are diagnosed as EDNOS often because they do not fulfil the frequency criterion (Herzog, Hopkins, & Burns, 1993), with one study reporting that this occurred in more than a third of individuals with subthreshold BN (Turner & Bryant-Waugh, 2004). Studies also found that this requirement was often not met in children and adolescents, who may nevertheless have been engaging in dangerous bingeing and purging behaviours (Striegel-Moore et al., 2000; Ricciardelli, Williams, & Kiernan, 1999).
Table 2.2

*DSM-IV-TR (APA, 2002) Diagnostic Criteria and Subtypes for Bulimia Nervosa*

<table>
<thead>
<tr>
<th>Diagnostic Criteria</th>
<th>Subtypes</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Recurrent episodes of binge eating. An episode of binge eating is characterised by both of the following: (1) eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances (2) a sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating);</td>
<td><strong>Purging Type:</strong> during the current episode of Bulimia Nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas</td>
</tr>
<tr>
<td>B. Recurrent inappropriate compensatory behaviour in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise;</td>
<td><strong>Nonpurging Type:</strong> during the current episode of Bulimia Nervosa, the person has used other inappropriate compensatory behaviours, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas</td>
</tr>
<tr>
<td>C. The binge eating and inappropriate compensatory behaviours both occur, on average, at least twice a week for 3 months;</td>
<td></td>
</tr>
<tr>
<td>D. Self-evaluation is unduly influenced by body shape and weight;</td>
<td></td>
</tr>
<tr>
<td>E. The disturbance does not occur exclusively during episodes of Anorexia Nervosa.</td>
<td></td>
</tr>
</tbody>
</table>


Although some researchers have argued that a lower minimum frequency of bingeing and purging would be more appropriate (Wilson & Eldredge, 1991; Fairburn & Bohn, 2005; Garfinkel, Kennedy, & Kaplan, 1995), there is some empirical evidence supporting the existing BN frequency criterion. These studies paint a more severe clinical picture in individuals who met the frequency criterion compared to those who did not, in ED symptomatology (Turner & Bryant-Waugh, 2004), psychological distress (Bunnell, Shenker, Nussbaum, Jacobson, & Cooper, 1990) and impairment in functioning (Garfinkel, Kennedy, et al., 1995). However, there have also been studies with contrasting results, reporting that individuals with a binge frequency of once per week had similar clinical characteristics to those meeting the current criterion (for a review see Wilson & Sysko, 2009).

2.2 **Previous and Proposed Diagnostic Criteria**

Whilst the DSM-IV criteria for BN did not change substantially from the DSM-III-R, it is expected in DSM-V to bring about some changes. No alterations are proposed for the behavioural and psychological criteria of BN (Criteria A, B, and D). However, in line with existing empirical evidence, the frequency of bingeing (Criterion
C) is proposed to be reduced to once a week, instead of twice, for three months (APA, 2012). Also, deletion of subtypes has been recommended for the DSM-V as the present non-purging subtype is difficult to identify and define, and closely resembles the newly proposed diagnostic category of Binge Eating Disorder (BED; APA, 2012). A comprehensive analysis of BN diagnostic criteria is beyond the scope of this study, however it is reassuring to note that diagnosing BN is less problematic than AN, and subthreshold BN is less frequently diagnosed as EDNOS than subthreshold AN (Bunnell et al., 1990; Dancyger & Garfinkel, 1995). The present study and remaining chapters will focus on AN, for which diagnosis is plagued with controversy and inconsistency.

3. EATING DISORDER NOT OTHERWISE SPECIFIED

3.1 CURRENT DIAGNOSTIC CRITERIA

The EDNOS category is reserved for EDs of clinical severity that do not meet diagnostic criteria for either AN or BN, making it a residual category with no inclusion or positive criteria. Rather, diagnosis is made by exclusion of AN or BN. Whilst the description of EDNOS in the DSM-IV-TR (APA, 2002) encompasses other atypical eating disorders such as repeatedly chewing and spitting food without swallowing, these descriptions are poorly characterised, with the exception of BED. Empirical evidence suggests that a substantial proportion of individuals diagnosed with EDNOS have subthreshold AN or BN rather than an atypical ED (Watson & Andersen, 2003; McIntosh et al., 2004). This is especially so for AN, with studies revealing that in both clinical populations (Bunnell et al., 1990; Dalle Grave & Calugi, 2007) and non-clinical populations (Dancyger & Garfinkel, 1995), most EDNOS cases are subthreshold forms of AN.

EDNOS is a common eating disorder treated by clinicians. In fact, it has been firmly established as the most common ED diagnosis made in outpatient settings (Fairburn & Bohn, 2005) and in a substantial proportion of inpatients (Dalle Grave & Calugi, 2007). In the past, EDNOS was largely ignored by researchers and there were no studies regarding its treatment. Two reasons suggested for this neglect has been that “not otherwise specified” or NOS diagnoses are in general rarely studied (Pincus, Davis, & McQueen, 1999), and the absence of positive diagnostic criteria makes it difficult to delineate these disorders (Fairburn & Bohn, 2005). In recent years,
Researchers have begun to recognise the prominence of EDNOS in treatment settings, with some newer studies intentionally designed to have broad inclusion criteria, encompassing individuals with EDNOS as well as those who meet criteria for AN and BN (e.g., Fairburn et al., 2009; Byrne, Fursland, Allen, & Watson, 2011).

Whilst individuals with EDNOS are sometimes thought to be less severely unwell than those with AN or BN, there is evidence that this may not be accurate. A study of 147 ED patients found that those with EDNOS were of similar weight and menstrual status as those with BN, and had similar levels of ED behaviours (e.g., restrictive eating, strenuous exercise, self-induced vomiting, laxative abuse) and ED psychopathology (e.g., fear of weight gain, body image disturbance, undue influence of weight or shape on self-evaluation) as those with AN and BN (Button, Benson, Nollett, & Palmer, 2005). EDNOS patients also had a similar number of clinic appointments and length of contact with the service as AN and BN patients, accounting for about half of all outpatient appointments and inpatient days (Button et al., 2005). Evidently, the DSM-IV-TR diagnostic categorisation has a major nosologic problem as it defines EDNOS as a residual category, although it is the most commonly diagnosed ED (Dalle Grave & Calugi, 2007) and is no less severe than AN and BN in ED behaviours, ED psychopathology, and service utilisation (Button et al., 2005).

3.2 Proposed Diagnostic Categories

In the revisions for ED categories in the DSM-V, some new ED categories have been proposed. BED, previously classified under EDNOS, will have a separate diagnostic category (APA, 2012). Additionally, ED presentations previously classified under ‘Childhood Disorders’ have been proposed to be moved to the ED category, including Pica (persistent eating of non-food substances), Rumination Disorder (repeated regurgitation of food), and Avoidant/Restrictive Food Intake Disorder (eating or feeding disturbance resulting in failure to meet nutritional or energy needs). The DSM-V Eating Disorders Work Group has proposed renaming EDNOS as ‘Feeding and Eating Conditions Not Elsewhere Classified’, which will encompass various eating problems that meet clinical significance including subthreshold AN, BN, and BED, purging disorder, night eating syndrome, and a residual category ‘Other Feeding or Eating Condition Not Elsewhere Classified’ (APA, 2012).
4. The Prevalence of Eating Disorders

Before looking at existing prevalence studies, it is important to note that whilst the prevalence of BN and EDNOS remained the same between DSM-III-R and IV, since there were no changes in their diagnostic criteria, there has been a substantial difference in prevalence of AN between both versions of the DSM. One study found that there was 100% agreement for BN and EDNOS diagnoses between DSM-III-R and IV, but only a 79% agreement for AN, with a kappa value (measure of inter-rater agreement) of only .485 (Sunday et al., 2001). To avoid incorrectly generalising between the older and newer classification systems, only research using DSM-IV or newer DSM-IV-TR criteria will be examined. Little change was made in the DSM-IV-TR, with the main modifications being additional text sections to provide more information. Hence, earlier prevalence studies that used DSM-III or DSM-III-R criteria (Bunnell et al., 1990; Hall & Hay, 1991; Clinton & Glant, 1992; Herzog et al., 1993; Mitrany, 1992; Button & Whitehouse, 1981; Dancyger & Garfinkel, 1995; Johnson-Sabine, Wood, Patton, Mann, & Wakeling, 1988; Walters & Kendler, 1995; Bushnell, Wells, Hornblow, Oakley-Browne, & Joyce, 1990; Kendler et al., 1991; Garfinkel, Lin, et al., 1995) will not be discussed in the following sections.

4.1 Treatment-Seeking Samples

In treatment-seeking samples, EDNOS was generally found to be the most common ED diagnosis made. Looking first at outpatients, prevalence studies reported that more than half of those presenting to outpatient services in Western countries were diagnosed with EDNOS (Fairburn et al., 2007; Martin, Williamson, & Thaw; 2000; Mitchell et al., 2007; Turner & Bryant-Waugh; 2004), with an average prevalence of 60.0% (Fairburn & Bohn, 2005). Table 2.3 shows the prevalence figures from four adult outpatient studies with sizable samples. In comparison, under 10% of outpatients were diagnosed with AN and about a third diagnosed with BN, according to studies that used stringent Eating Disorder Examination (EDE; Fairburn & Cooper, 1993) assessment procedures (Fairburn et al., 2007; Mitchell et al., 2007; Turner & Bryant-Waugh, 2004). Martin et al.’s (2000) findings used other assessment methods and were slightly inconsistent, reporting a higher proportion of outpatients being diagnosed with AN (19.4%). However, this was still a substantially smaller proportion compared to those diagnosed with EDNOS (57.7%) in the same study. It is evident that in outpatient samples, EDNOS was consistently found to be the most common ED diagnosis.
Three studies included inpatient samples (Table 2.3). In general, inpatient settings tend to include the most severe ED cases that require hospitalisation and a higher level of medical intervention due to extreme emaciation. However, admission criteria to inpatient wards can vary across different services, resulting in discrepant prevalence rates of EDs in inpatients and outpatients. Of the three studies that included inpatient samples, two studies combined inpatients and outpatients (Button et al., 2005; Sunday et al., 2001). Clear interpretations from these studies cannot be made without making generalisations across inpatients and outpatients, as the proportions of inpatients and outpatients in the samples were not clearly delineated. To obtain a clearer picture of the prevalence of inpatients, Dalle Grave and Calugi (2007) only included inpatients in their study using the stringent EDE assessment procedure. As indicated in Table 2.3, unsurprisingly, a large proportion of inpatients were diagnosed with AN (41.9%). Interestingly, there was a similar proportion of inpatients diagnosed with EDNOS (40.3%), with BN being the smallest group (17.8%). This study provides evidence that EDNOS is also a common diagnosis in inpatients, albeit having a lower prevalence compared to outpatients.
<table>
<thead>
<tr>
<th>Authors</th>
<th>N</th>
<th>AN (%)</th>
<th>BN (%)</th>
<th>EDNOS (%)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fairburn et al.</td>
<td>170</td>
<td>4.7</td>
<td>35.3</td>
<td>60</td>
<td>At two UK ED clinics, participants aged 16 to 65 years. Did not include patients with BMI (&lt; 16.0 \text{ kg/m}^2). Diagnosis made using EDE(^1).</td>
</tr>
<tr>
<td>Martin et al.</td>
<td>175</td>
<td>19.4</td>
<td>22.9</td>
<td>57.7</td>
<td>From US archival records, participants all female, mean age = 24.8 years. Diagnosis made using IDED(^2).</td>
</tr>
<tr>
<td>Mitchell et al.</td>
<td>687</td>
<td>9</td>
<td>32.3</td>
<td>58.7</td>
<td>At four US clinics, participants 93% female, mean age = 29.5 years (SD = 11.3). Diagnosis made using EDE-Q(^1).</td>
</tr>
<tr>
<td>Turner &amp; Bryant-Waugh (2004)</td>
<td>200</td>
<td>5.5</td>
<td>22.5</td>
<td>67</td>
<td>At UK ED service, participants 94% female. Diagnosis made using EDE(^1).</td>
</tr>
<tr>
<td>Button et al.</td>
<td>147</td>
<td>6.8</td>
<td>36.7</td>
<td>43</td>
<td>At UK ED service, participants 96% female, mean age = 26.3 years. Diagnosis made using clinical interview.</td>
</tr>
<tr>
<td>Sunday et al.</td>
<td>288</td>
<td>8.3</td>
<td>59.7</td>
<td>27.1</td>
<td>In US, participants all female, mean age = 29.2 years (SD = 8.7). Diagnosis made using SCID(^3).</td>
</tr>
<tr>
<td>Dalle Grave &amp; Calugi (2007)</td>
<td>186</td>
<td>41.9</td>
<td>17.8</td>
<td>40.3</td>
<td>At ED ward in Italy, participants 93% female, mean age = 26.0 years. Diagnosis made using EDE(^1).</td>
</tr>
</tbody>
</table>

Note. ED—Eating disorder; DSM-IV—Diagnostic and Statistical Manual of Mental Disorders, 4\(^{th}\) Edition; AN—Anorexia Nervosa; BN—Bulimia Nervosa; EDNOS—Eating Disorder Not Otherwise Specified; BMI—Body mass index; US—United States; UK—United Kingdom.

1 Eating Disorder Examination (EDE; Fairburn & Cooper, 1993; EDE-Q; Fairburn & Beglin, 1994)
2 Interview for the Diagnosis of Eating Disorders IV (IDED; Kutlesic, Williamson, Gleaves, Barbin, & Murphy-Eberenz, 1998)
3 Structured Clinical Interview for DSM-IV Disorders (SCID; First, Gibbon, Spitzer, & Williams, 1995)

### 4.2 COMMUNITY SAMPLES

Accurate prevalence rates of population level EDs, particularly EDNOS, and their interpretation in the community is unclear, complicated by the secrecy usually associated with ED behaviours (Machado, Machado, Gonçalves, & Hoek, 2007; Polivy & Herman, 2002). This is evidenced by an examination of medical records of 39 women who chose not to participate in a survey on EDs, indicating that those with eating or weight problems and previous or current EDs (43.6%) were overrepresented in this population (Beglin & Fairburn, 1992). The authors concluded that estimates of population level prevalence of EDs were likely to be underestimates, and obtaining accurate prevalence figures of EDs in community samples may be impossible due to individuals who choose not to participate. Additionally, EDNOS in particular is difficult
to identify; its lack of positive criteria for diagnosis results in no agreed way of
determining who meets criteria (Fairburn & Bohn, 2005). Figures quoted tended to be
for the prevalence of participants with ED features (e.g., Hay, 1998), with no check
being made that these features resulted in clinically significant distress or impairment,
an essential requirement for the diagnosis of EDNOS (Fairburn & Bohn, 2005). The
neglect of EDNOS in population level research is apparent, with prevalence studies
frequently excluding EDNOS, focusing instead on AN, BN, and BED (e.g., Hay, 1998;
Hudson, Hiripi, Pope, & Kessler, 2007; Bushnell et al., 1990; Oakley-Browne, Wells,
Scott, & McGee, 2006; Preti et al., 2009; Striegel-Moore et al., 2003; Taylor, Caldwell,
Baser, Faison, & Jackson, 2007; Alegria et al., 2007; Nicdao, Hong, & Takeuchi, 2007).
In some prevalence studies, individuals with EDNOS are included, but only as an
extension of AN or BN, that is, those who do not meet strict criteria for AN or BN are
included as “broad”, “partial syndrome”, or “subthreshold” forms of these disorders
(e.g., Keski-Rahkonen et al., 2007, 2008; Lewinsohn, Striegel-Moore, & Seeley, 2000).
For a comprehensive review of epidemiological studies for EDs, please refer to Wade,
Keski-Rahkonen, and Hudson (2011).

The present review focuses on four studies that looked at population level data
using DSM-IV criteria, and assessing AN, BN, and EDNOS. The studies are
summarised in Table 2.4. The results from these studies, which only included females,
showed proportions of EDs similar to that of clinical studies. EDNOS formed the
largest proportion of EDs in the population for both lifetime prevalence (Ghaderi &
Scott, 1999; Favaro, Ferrara, & Santonastaso, 2003) and point prevalence (Bulik,
Sullivan, & Kendler, 2000; Gauvin, Steiger, & Brodeur, 2009), and was two to eighteen
times as common as AN. The population prevalence of BN was in between that of
EDNOS and AN (Ghaderi & Scott, 1999; Bulik et al., 2000; Favaro et al., 2003; Gauvin
et al., 2009). These studies suggest that EDNOS was also the most prevalent ED in
population level community studies.
### Table 2.4

**ED Prevalence Studies in Community Samples using DSM-IV (APA, 1994) Criteria**

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>AN (%)</th>
<th>BN (%)</th>
<th>EDNOS (%)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bulik, Sullivan, &amp; Kendler (2000)</td>
<td>2163</td>
<td>0.6</td>
<td>1.9</td>
<td>5.8</td>
<td>Point prevalence of females in the US from a population-based registry, aged 17 to 55 years, mean age = 30.1 years. Diagnosis made using SCID1.</td>
</tr>
<tr>
<td>Favaro, Ferrara, &amp; Santonastaso (2003)</td>
<td>934</td>
<td>2.0</td>
<td>4.6</td>
<td>6.4</td>
<td>Lifetime prevalence of females in two areas in Italy, aged 18 to 25 years. Diagnosis made using SCID1.</td>
</tr>
<tr>
<td>Gauvin, Steiger, &amp; Brodeur (2009)</td>
<td>1501</td>
<td>0</td>
<td>0.6</td>
<td>18.5</td>
<td>Point prevalence of females from Canada, aged 20 to 40 years. Diagnosis made using EDE-Q2.</td>
</tr>
</tbody>
</table>

Note: ED—Eating disorder; DSM-IV—Diagnostic and Statistical Manual of Mental Disorders, 4th Edition; AN—Anorexia Nervosa; BN—Bulimia Nervosa; EDNOS—Eating Disorder Not Otherwise Specified; US—United States.

1 Structured Clinical Interview for DSM-IV Disorders (SCID; First et al., 1995)
2 Eating Disorder Examination—Questionnaire version (EDE-Q; Fairburn & Beglin, 1994)

5. **Implications for the Present Study**

Sections 1 to 3 described the current DSM-IV-TR diagnostic criteria for EDs, which have changed over time and will continue to evolve with future versions of the DSM. Some of these diagnostic criteria are controversial, with the physical criteria of weight and amenorrhea for AN being particularly problematic. Prevalence data summarised in Section 4 suggested that EDNOS was the most prevalent ED in clinical and non-clinical samples from various Western countries, followed by BN, with AN having the smallest prevalence, except in inpatient settings.

Although AN and BN are the most well-known and best described EDs, most individuals with eating problems do not meet full diagnostic criteria for these disorders, falling instead into the residual category of EDNOS (Machado et al., 2007). Ironically, although most common, EDNOS is the most neglected, least studied group of ED patients. The usefulness of a classification that excludes so many clinical cases from major diagnostic groups has been questioned (Beumont, Al-Alami, & Touyz, 1988). As discussed, individuals diagnosed with EDNOS are most commonly subthreshold forms of AN (p. 9). Of those with subthreshold AN, a substantial proportion do not meet full diagnosis because they fail to meet the physical criteria of AN—being underweight or
having amenorrhea (Watson & Andersen, 2003; McIntosh et al., 2004). Due to the clinical and research implications of the EDNOS diagnosis, further evaluation of the weight and amenorrhea criteria for AN is crucial, and will be examined in the next chapter.
**CHAPTER 3: ISSUES WITH THE WEIGHT & AMENORRHEA CRITERIA FOR ANOREXIA NERVOSA**

Chapter 2 indicated that EDNOS was the most common ED diagnosis and occurred more commonly in subthreshold AN than subthreshold BN. Of individuals with subthreshold AN, at least one third failed to meet full diagnosis because they did not meet the weight criterion (Turner & Bryant-Waugh, 2004), and another third because they did not have amenorrhea (Gendall et al., 2006), making the physical criteria of AN the criteria that subthreshold AN patients diagnosed with EDNOS most commonly failed to meet (Cachelin & Maher, 1998).

Longitudinal studies found that at one to four years follow up, 79% of individuals with subthreshold EDs continued to have ED symptomatology, and approximately 30-46% progressed to full syndrome EDs (Herzog, Hopkins, & Burns, 1993; Striegel-Moore, Silberstein, Frensch, & Rodin, 1989; Yager, Landsverk, & Edelstein, 1987). The highly persistent ED symptomatology and low rates of recovery in subthreshold EDs, which commonly develop into full syndrome EDs, signify that the current diagnostic criteria may be too restrictive. The crossover to full syndrome EDs suggests that the subthreshold diagnosis may be an artefact of the timing of the assessment or interview, with individuals either in a phase of relative “health” or in the process of developing a full disorder (Herzog et al., 1993).

For individuals who do not meet full criteria for AN, the EDNOS diagnosis has clinical and research implications. Clinically, it communicates little diagnostic information, gives a false sense that the severity of an individual’s illness is less extreme, hinders early detection of individuals who are severely unwell, complicates the process of seeking reimbursement for treatment from insurance companies in some countries, and impedes treatment administration, as there are no treatment guidelines for EDNOS. In research, where subthreshold AN or BN patients (the majority of individuals presenting at ED services) have been excluded from studies, findings are not representative of the treatment-seeking population and cannot be generalised to them.

As mentioned earlier (p. 5), the utility of the weight threshold and amenorrhea criteria have been sources of fervent debate (Mitchell et al., 2005). Researchers have suggested that the number of people diagnosed with EDNOS would be substantially reduced if both these physical criteria, which were established in a somewhat arbitrary and non-empirical manner (Barlow, 1991), were relaxed. This chapter will examine the
weight criterion (Section 1) and amenorrhea criterion (Section 2) and discuss issues surrounding the criteria, empirical evidence for the clinical utility of the criteria, and implications for the present study. Then, aims and research questions to assess the clinical utility of both the criteria in the present study will be discussed (Section 3).

1. THE WEIGHT CRITERION

The refusal to maintain body weight at a minimally normal weight for age and height is required for diagnosis of AN. The DSM-IV-TR provides a “suggested guideline”, recommending that this criterion be assessed by an individual being at a weight that is less than 85% of what is considered normal for age and height according to published norms, or the stricter guideline of a body mass index (BMI; weight in kg/[height in m]^2) of ≤ 17.5 kg/m^2, as recommended by the International Statistical Classification of Diseases and Related Health Problems—10th Revision (ICD-10; World Health Organisation, 1992). One study found that a substantial 31% of individuals diagnosed with EDNOS missed a diagnosis of AN as they did not meet the weight criterion of BMI ≤ 17.5 kg/m^2 (Turner & Bryant-Waugh, 2004). The arbitrary nature of the cut-off is reflected in the changes made to the weight criterion over the years. In the DSM-III, a stricter 25% weight loss (or < 75% of normal weight for height and age) was required, and in proposed changes for the DSM-V, no specific cut-off is defined, with low weight defined as “weight that is less than minimally normal” (APA, 2012).

Although only intended to be a guide, the weight criterion for diagnosis of AN is typically strictly adhered to by investigators in recruitment of ED patients for research studies, by third-party insurance companies in determining reimbursement for treatment in some countries, by clinicians in making diagnoses and forming treatment plans, and to calculate AN prevalence in epidemiological studies that inform the public about the disorder (Walters & Kendler, 1995; Garfinkel, Lin, Goering, & Spegg, 1996). Whilst the ever-present citation of the 85% weight criterion creates a false sense of consensus, there is considerable controversy over the generally accepted cut-offs for underweight children, adolescents, and adults (Hamer, 1991; Hebebrand, Wehmeier, & Remschmidt, 2000; Llewellyn-Jones & Abraham, 1984; Oehlschlagel-Akiyoshi, Malewski, & Mahon, 1999; Selzer, Bowes, & Patton, 1995). The key questions to be addressed in the following sections are: how comparable or consistent are the methods of calculating the weight cut-off, is there a consensus amongst researchers on which method to use, and are there any meaningful psychological differences signified by the weight threshold?
1.1 METHODS OF CALCULATING THE WEIGHT CUT-OFF

Using published norms and tables. The DSM-IV-TR recommends using published norms and tables to determine 85% of ideal weight, providing the Metropolitan Life Insurance Company charts (1959) as one such example. The benefit of using published norms is that the tables have been adjusted for age and gender, which is imperative because the typical onset of AN occurs in adolescence and early adulthood, during which weight to height ratios change dramatically. However, comparison between different published tables is tricky as some vary in weight recommendations for older adults whereas others do not, some provide weight ranges rather than point estimates, and some provide clothed weights and others unclothed (Thomas, Roberto, & Brownell, 2009). Furthermore, due to an increase in obesity at the population level over time, average body weights are seeing an upward trend, increasing the weight criterion and encumbering comparison between studies over time. As researchers use a variety of different tables to determine underweight status (Thomas et al., 2009), it is difficult to make accurate comparisons between different studies.

Using body mass index cut-offs. For adults, in line with the ICD-10, the DSM-IV-TR recommends using a BMI cut-off of ≤ 17.5 kg/m². Whilst BMI has been promoted as the best index of under-nutrition due to its objectivity, easy calculation, and correlation with other physiological and psychological sequelae of AN (Beumont et al., 1988; Selzer et al., 1995), its use by clinicians and researchers in determining underweight status is far from consistent. There has been dispute over the appropriate cut-off, as the somewhat arbitrary 17.5 kg/m² has not been scientifically established (Mitchell et al., 2005), and there have been no available data providing definitive evidence for any specific cut-off weight. Additionally, BMI is not adjusted for age and sex, a problem when weight to height ratios change as they do throughout childhood, adolescence and early adulthood. To illustrate, a study found that with increasing age, the proportion of individuals with BMI ≤ 17.5 kg/m² dropped from 57% at 10 years of age to below 1% at 35 years of age in German female population (Hebebrand et al., 1996). Hence, the BMI cut-off should be applied with caution in younger people and more research is required to determine what BMI cut-off would have most clinical utility for AN diagnosis.

1.2 DISSENT ON BEST METHOD OF DETERMINING IF CRITERION IS MET

There is a lack of consensus regarding the weight criterion amongst clinicians in the field, with some arguing that the weight threshold should be adjusted upwards
(Watson & Andersen, 2003; Garfinkel, Kennedy, et al., 1995; Andersen, Bowers, & Watson, 2001; McIntosh et al., 2004), and others suggesting lower BMI cut-offs of 16 kg/m² (Beumont et al., 1988) and 15 kg/m² (Llewellyn-Jones & Abraham, 1984). To determine if researchers had a preferred method of calculating underweight status, Thomas et al. (2009) evaluated 99 AN studies that focused on comparing AN to subthreshold AN, or conducted AN treatment efficacy trials. They found that a majority of studies (76.8%) did not describe their method for assessing underweight participants. Of the remaining studies that did, 10 distinct methods were used in calculating underweight status, with no preference emerging (Thomas et al., 2009). Even when published tables or BMI cut-offs were employed, there was little consistency. The tables included the Metropolitan Life Insurance Company tables (1959), the Fogarty Table of Desirable Weights (Bray, 1975), and the Department of Health, Education, and Welfare norms (1979), for example.

For BMI, the cut-points utilised included 16.5, 17.5, 18.0 and 18.4 kg/m² (Thomas et al., 2009). The differences in the weight cut-off across studies gave a weight range of 7 kg for females and 11 kg for males, causing statistically significant differences in weight across the groups of individuals who were classified as underweight and therefore eligible for the diagnosis of AN (Thomas et al., 2009). It is possible that there are similar variations in methods used to determine underweight status of patients in clinical settings, consequently leading to substantial differences in the number of patients who are eligible for the diagnosis of AN in treatment-seeking individuals. Evidently, methods of calculating underweight status require revision for consistency and comparability.

1.3 Empirical evidence for clinical differences signified by the criterion

In deliberating about the 85% weight criterion, it is critical to consider that the main purpose of diagnostic classification is to aid clinicians in making appropriate treatment decisions. The critical point is whether there are meaningful clinical differences in individuals with AN who meet the weight criterion and those who do not. Studies assessing psychological differences will now be reviewed.

Description of studies. Four empirical studies were identified and are summarised in Table 3.1. The four studies were conducted in inpatient and outpatient treatment-seeking samples. One study used a BMI cut-off of < 17.5 kg/m² (McIntosh et al., 2004), two studies used the threshold of < 85% of ideal weight (Watson &
Anderson, 2003; Williamson, Gleaves, & Savin, 1992), and the remaining study did not report the method used to determine underweight status (Bunnell et al., 1990). A fifth study identified in the literature search (Santonastaso et al., 2009) was not included in this review because it included subthreshold AN patients who did not meet the weight, amenorrhea, or fear of gaining weight criteria, and the study provided overall analyses of variance and chi-square tests results, rather than pairwise group comparisons.

Limitations of studies. Limitations common to the four studies were that whilst group differences were assessed for some symptoms of AN (namely, the behavioural symptom of drive for thinness through dieting, excessive exercise, purging or using laxatives/diuretics), other clinically relevant cognitive diagnostic criteria of AN were neglected. They included body image disturbance, which was assessed in only two studies (McIntosh et al., 2004; Watson & Andersen, 2003), and fear of gaining weight and self-worth invested in the body, which were neglected by all four studies. Another flaw common to all studies was the use of a cross-sectional design that differentiated patients based on presenting symptoms, making it impossible to determine the course of illness or potential shifts across groups. The studies also utilised treatment-seeking samples, resulting in inherent sampling biases as individuals with AN not actively seeking treatment were not included. Two studies had small sample sizes for the full syndrome AN group (n = 12, Bunnell et al., 1990; n = 14, Williamson et al., 1992) and the subthreshold AN group (n = 15, Williamson et al., 1992), limiting the power with which differences between groups could be detected and the extent to which findings could be generalised. The more lenient selection criteria for groups in McIntosh et al.’s (2004) and Watson and Andersen’s (2003) studies, by not requiring amenorrhea for inclusion in the study, also made it difficult to draw conclusions about differences based purely on weight. To control for the large number of comparisons, one study utilised Tukey’s honestly significant difference (Tukey HSD) procedure to control for familywise error rate (Williamson et al., 1992), two studies set alpha at a more stringent level of $p < .01$ for significance (McIntosh et al., 2004; Watson & Andersen, 2003), while the remaining study did not discuss or provide values from data analysis (Bunnell et al., 1990). Despite these shortcomings, there were few differences in results between the studies (Table 3.1), with all four yielding consistent results across outpatient and inpatient samples using a range of interview and self-report measures. Their results will now be discussed.
Physical measures. Aside from differences in BMI and body fat by virtue of the grouping criteria, there were no significant differences between the underweight and non-underweight groups in physical measures, including highest and lowest premorbid weights (Watson & Andersen, 2003), blood pressure, heart rate, body temperature, and current or previous amenorrhea (McIntosh et al., 2004). Whilst differences were found in degree of bone density, which was reduced in the underweight group, this was unsurprising and was, to some degree, a self-fulfilling prophecy, as bone density is related to absolute weight, with lower weights resulting in lower bone mineral density regardless of psychiatric illness (Watson & Andersen, 2003). Overall, the studies did not provide evidence of physical differences in groups defined by the weight criterion.

Demographics and early experiences. One study found that the underweight AN group had higher full-scale intelligence and less physical or sexual abuse compared to the non-underweight AN group (Watson & Andersen, 2003). These findings need to be replicated before conclusive statements about demographics or early experiences can be made.

ED history. Most studies reported no significant differences between groups in ED history including age of onset of AN (Watson & Andersen, 2003), duration of AN, highest frequency of ED behaviours, and subtype membership (McIntosh et al., 2004). Only Watson and Andersen (2003) reported differences, with the EDNOS group having a significantly shorter length of illness, possibly because of later recognition of subthreshold AN due to their less emaciated state. Overall, the studies suggested that there were no differences between the underweight and non-underweight groups in ED history.

ED psychopathology. All four studies unanimously found no differences between the underweight and non-underweight AN groups in the AN psychopathology of body image dissatisfaction/disturbance and drive for thinness (Bunnell et al., 1990; McIntosh et al., 2004; Watson & Andersen, 2003; Williamson et al., 1992). However, as highlighted earlier, other clinically important AN diagnostic criteria of fear of gaining weight or self-worth invested in the body were not assessed by existing studies.

ED behaviours. With the exception of excessive exercising, which was higher in the underweight group (Watson & Andersen, 2003) both groups had the same frequencies of ED behaviours of restrictive eating, binge eating, self-induced vomiting
(McIntosh et al., 2004; Watson & Andersen, 2003; Williamson et al., 1992) and misuse of laxatives or diuretics (McIntosh et al., 2004).

**Common ED characteristics.** Common ED characteristics were similar across both groups, with no significant differences found in feelings following bingeing (Williamson et al., 1992), ineffectiveness, perfectionism, maturity fears (Bunnell et al., 1990; McIntosh et al., 2004), interpersonal distrust, interoceptive awareness, social insecurity, impulse regulation, and asceticism (McIntosh et al., 2004). There was one difference reported, that the non-underweight group had greater discrepancy between reported current and ideal weight, compared to the underweight group (McIntosh et al., 2004). However, this was possibly due to their higher current weight, as both groups reported similar ideal body size (Williamson et al., 1992).

**Psychiatric comorbidities.** Assessment of psychiatric comorbidities identified no differences between underweight and non-underweight groups in the number and intensity of other clinical symptoms, psychological distress (Bunnell et al., 1990; McIntosh et al., 2004), depression (Bunnell et al., 1990; Watson & Andersen, 2003), alcohol or drug abuse (Watson & Andersen, 2003), self-harm, or measures of global functioning (McIntosh et al., 2004).

**Service utilisation.** Only one study examined service utilisation, concluding that there were no differences between underweight and non-underweight groups in the type of service used (majority outpatient) and proportion of each group involuntarily treated (15%; Watson & Anderson, 2003). The same study also reported that the subthreshold AN group had shorter and fewer hospitalisations than those with full syndrome AN (Watson & Andersen, 2003). Investigators suggested that this was because individuals with subthreshold AN were less emaciated on admission, resulting in shorter hospitalisations as less weight restoration was required. Importantly, as discussed earlier (p. 22), the same study found that both underweight and non-underweight groups were similar in highest and lowest premorbid weights (Watson & Andersen, 2003), indicating that the higher weights of the subthreshold AN group on admission were likely to be an artefact of the time of assessment. Overall, there was little evidence of differences in service utilisation signified by the weight criterion.

**Conclusion.** The studies provided compelling evidence that there were no clinically important differences between individuals with AN regardless of whether they were strictly underweight at the time of assessment. However, differences between
underweight and non-underweight subthreshold AN patients on the cognitive diagnostic
criteria of fear of gaining weight and self-worth invested in the body were not assessed.
That aside, both groups of AN patients had comparable physical measures, ED
characteristics, service utilisation, comorbid psychopathology, distress, and secondary
impairment of psychosocial functioning. These findings were confirmed in a meta-
analysis which concluded that there were no differences found in studies comparing full
syndrome AN and subthreshold AN (meeting all AN criteria but weight) groups in
eating pathology ($d = -0.02, p = .93$), and comparing AN and EDNOS (including
subthreshold AN) groups in general psychopathology ($d = 0.02, p = .68$) and physical
health ($d = 0.14, p = .40$; Thomas, Vartanian, & Brownell, 2009). The findings from this
literature review challenge the existing diagnostic cut-off of 85% of ideal weight or
BMI $\leq 17.5$ kg/m$^2$, demonstrating that an absolute weight cut-off or weight as a
proportion of height may be unhelpful in diagnosing AN and may exclude patients with
clinically significant illnesses.

Table 3.1

| Studies Comparing Full-syndrome with Subthreshold AN (Weight Criterion Not Met) |
|-----------------|-----------------|-----------------|
| Participants    | Measures        | Outcomes        |
| Bunnell et al. (1990) | 33 outpatients at US clinic:   |
|                 | 12 full syndrome AN   |
|                 | 21 subthreshold AN (weight criterion not met) |
| Mean age = 16.6 years ($SD = 2.5$). Method for determining underweight not reported. | **Interview:** DSED$^1$ | **No significant differences in:** |
|                 | **Self-report measures:** EAT$^2$, EDI$^1$, BDI$^1$, SCL-90-R$^2$, BSQ$^4$, BSI$^7$ | **ED psychopathology:** body image dissatisfaction, drive for thinness |
|                 | **Common ED characteristics:** ineffectiveness, perfectionism, maturity fears, interoceptive awareness |
|                 | **Comorbid psychopathology:** depression, psychological distress, number/intensity of clinical symptoms |
Table 3.1 (continued)

<table>
<thead>
<tr>
<th>Participants</th>
<th>Measures</th>
<th>Outcomes</th>
</tr>
</thead>
</table>
| 56 female outpatients at New Zealand clinic:  
  - 33 full syndrome AN  
  - 23 subthreshold AN (weight criterion not met)  
  Aged 17-40 years. Full AN group BMI <17.5 kg/m², subthreshold AN group BMI 17.5–19 kg/m². Amenorrhoea not required for inclusion in study. | **Interviews:** SCID⁵, EDE⁹, HRSD¹⁰  
**Self-report measures:** BIAQ¹¹, BSQ⁸, TFEQ¹², EAT², SCL-90-R³  
**Differences:** Full syndrome AN group:  
  • Less discrepancy between current and ideal weight  
**No significant differences in:**  
  • **Physical measures:** weight history, blood pressure, heart rate, body temperature, current/previous amenorrhoea  
  • **ED history:** Highest frequency of ED behaviours, age of onset, duration of AN, subtype membership  
  • **ED psychopathology:** body image disturbance, drive for thinness, body dissatisfaction  
  • **Common ED characteristics:** ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, maturity fears, social insecurity, impulse regulation, asceticism  
  • **ED behaviours:** restrictive eating, bingeing, purging, overeating, misuse of laxatives/diuretics  
  • **Comorbid psychopathology:** psychological distress, global functioning, self-harm, number/intensity of clinical symptoms |  |
| 297 female inpatients at US hospitals:  
  - 230 full syndrome AN  
  - 67 subthreshold AN (weight and/or amenorrhoea criterion not met)  
  Full AN group < 85% of ideal weight, subthreshold group included 39 patients who were >85% of ideal weight. Subthreshold group collapsed across absence of weight and/or amenorrhoea criteria. | **Chart review of:** Interviews by health professionals  
**Self-report measures:** EAT-26², EDI-2², BDI⁴  
**Other assessment:** WAIS-R¹³  
**Differences:** Full syndrome AN group:  
  • **Demographics:** higher full-scale intelligence  
  • **Physical measures:** lower bone mineral density  
  • **ED history:** longer duration of AN, higher lifetime excessive exercise  
  • **Service utilisation:** higher number and longer duration of previous hospitalisations, more weight restored at end of treatment  
  • **Early experiences:** lower physical/sexual abuse  
**No significant differences in:**  
  • **ED history:** age of onset, highest/lowest weight  
  • **ED psychopathology:** drive for thinness, body image disturbance  
  • **ED behaviours:** restrictive eating, bingeing, purging  
  • **Comorbid psychopathology:** depression, alcohol/drug abuse  
  • **Service utilisation:** service type (majority outpatient), proportion of involuntarily treated (15% of each group) |  |
| 29 female inpatients and outpatients at US clinic:  
  - 14 full syndrome AN  
  - 15 subthreshold AN (weight criterion not met)  
  Full AN group < 85% of ideal weight according to Metropolitan Life Insurance Company norms (1983). | **Interview:** IDED¹⁴  
**Self-report measures:** EDI³, EAT², BULIT³, BIA⁶⁶  
**No significant differences in:**  
  • **ED psychopathology:** drive for thinness, body dissatisfaction  
  • **Common ED characteristics:** feelings following bingeing, ideal body size preference  
  • **ED behaviours:** restrictive eating, bingeing, purging |  |

Note. AN—Anorexia Nervosa; ED—Eating disorder; BMI—Body mass index; US—United States.

¹ Diagnostic Schedule for Eating Disorders (DSED; Johnson, 1984)
1.4 IMPLICATIONS FOR THE PRESENT STUDY

As discussed, no clear method to assess the weight criterion exists. Based on differences in opinion between researchers, a variety of approaches have been used to determine underweight status, leading to substantial differences between patients eligible for the diagnosis of AN. The literature review revealed that there was no evidence for the clinical utility of the weight criterion, as underweight and non-underweight subthreshold AN patients reported similar eating pathology, illness severity, psychiatric comorbidities, distress, secondary impairment in functioning, and were comparable on physical measures and service utilisation. However, an examination of differences between underweight and non-underweight AN patients on the cognitive diagnostic criteria of AN, the fear of gaining weight and self-worth invested in the body, has been neglected in existing studies. An assessment of the cognitive diagnostic criteria is needed to determine if these psychological features are manifestations of a state of emaciation or core features of the disorder regardless of weight status.

More recently, the DSM-V Eating Disorders Work Group highlighted that it was not desirable to set a specific numerical standard for weight for AN, as such standards were at least somewhat arbitrary, and the judgment of whether weight was inappropriately low was best made by the clinician in light of all relevant information, including age, sex, developmental trajectory, and physical health (APA, 2012). Broadening the criteria for patient samples in research rather than conforming to narrow DSM-IV-TR criteria will allow for better understanding of the course of illness,
predictors of treatment response, outcome, and differential treatment needs (Agras et al., 2004), providing information for treatment selection based on patient factors in this heterogeneous group. At present, until further research is available to determine appropriate percentage or weight ranges, existing empirical evidence suggests that it would be unwise to exclude individuals based on the strict weight cut-off. The rigid 85% weight criterion or 17.5 kg/m² cut point should not be interpreted as absolute, as they detract from the essence of Criterion A for diagnosis of AN: the drive for thinness, achieved through restricting food intake, fasting, excessive exercising, purging and misuse of laxatives or diuretics. As Beumont, Garner, and Touyz (1994) argued, the disorder should be conceptualised as a “dieting” disorder, with emphasis placed on the preoccupation with weight loss because, regardless of whether patients are overweight, underweight, or at normal weight at the time of the assessment, they often had varied or will vary across all three states. Fixating on a specific numerical weight cut point distracts from the relentless, intentional weight loss that is the crux of AN and the main culprit of devastating physical health consequences. It can occur at any weight.

In light of the existing empirical evidence, patients who failed to meet the weight criterion were not excluded from the present study. Given the relatively small sample sizes and the neglect of cognitive diagnostic criteria of AN in existing studies, the present study aimed to assess cognitive and psychological differences in underweight and non-underweight AN groups with a larger treatment-seeking sample. Aims and research questions are detailed in Section 3 (p. 42-44).

2. THE AMENORRHEA CRITERION

The DSM-IV-TR requires the presence of amenorrhea, or the absence of naturally occurring menstrual cycles, for at least three months for diagnosis of AN. This criterion has been the subject of fervent debate (Bulik et al., 2000; Cachelin & Maher, 1998; Garfinkel et al., 1996; Meyer, von Holtzapfel, Deffner, Engel, & Klick, 1986; Walters & Kendler, 1995), as its role in AN is not clear (Cachelin & Maher, 1998). The amenorrhea criterion can be difficult to assess as it depends largely on patients’ self-report of menstrual status, with little or no independent confirmation by clinicians. Also, it cannot be applied to pre-menarchal females, to females using hormone replacements such as OCPs, to post-menopausal females, or to males. Amenorrhea is found to be the criterion that is most commonly not present in women with subthreshold AN in the treatment-seeking population (Herzog et al., 1993). In outpatient samples,
one third of those who meet all other criteria for AN (including having a BMI of < 17.5 kg/m²) fail to meet diagnosis because they do not have amenorrhea (Gendall et al., 2006). In inpatient populations, this is also common, with about 25% of underweight ED patients not having amenorrhea (Roberto, Steinglass, Mayer, Attia, & Walsh, 2008; Dalle Grave, Calugi, & Marchesini, 2008).

The arbitrary nature of the amenorrhea criterion is reflected in the changes to diagnostic classifications over the years. In the DSM-III, amenorrhea was not required for diagnosis of AN, and in proposed changes for the DSM-V, amenorrhea has been removed from the diagnostic criteria for AN (APA, 2012). In determining the clinical utility of this criterion, the key questions that will be addressed in the following sections are: what is the rationale behind the amenorrhea criterion, do individuals develop amenorrhea consistently, are there meaningful psychological differences signified by the amenorrhea criterion, and what is the relevance of OCP use in individuals with AN?

2.1 RATIONALE BEHIND THE CRITERION

Amenorrhea was initially suggested as a criterion in early medical writings because it was believed that AN was associated with an impairment of hypothalamic functioning, for which amenorrhea is a symptom (Russell, 1970). The benefits of the criterion are that it is clear and objective, providing a marker of abnormal physiology and serving as an indicator of physical health status and biological abnormalities that may provide information about etiology of the illness and possible development of biological treatments (Attia & Roberto, 2009). However, whilst some argue that amenorrhea is associated with hypothalamic disturbance in AN, other psychiatric illnesses such as schizophrenia and depression are also associated with hypothalamic abnormalities which are not included in its diagnostic criteria (Garfinkel et al., 1996).

Although amenorrhea has been associated with a range of other biological markers such as lower bone mineral density, systolic blood pressure, pulse rate, and dopaminergic activity (Gendall et al., 2006), findings on bone health and biological differences between amenorrheic and non-amenorrheic AN individuals are mixed, making it difficult for conclusive inferences (for a review, please refer to Attia & Roberto, 2009). These biological differences are beyond the scope of the present study, which is focused on psychological factors. Importantly, the effects are reversible, with normal menstrual function resuming following nutritional rehabilitation and weight gain (Copeland, Sacks, & Herzog, 1995).
Some studies have linked amenorrhea with weight loss, reduced body fat, and low BMI, although a specified required percentage of body fat or a weight cut-off for normal menses has not been supported by empirical evidence (Golden & Carlson, 2008). Other studies reported that behaviours influencing energy balance (e.g., exercise or binge-eating) were associated with amenorrhea independent of BMI, concluding that amenorrhea occurred as a consequence of relative energy deficits (Gendall et al., 2006; Dalle Grave et al., 2008). Researchers have also suggested that the menstrual status of AN patients varied across the course of their illness due to fluctuations in weight, and was a function of the timing of the assessment rather than of improvement in psychiatric status (Copeland et al., 1995; Cachelin & Maher, 1998). Following from these studies, it appears that differences between amenorrheic and non-amenorrheic AN individuals reflect nutritional states, relative energy balance, and timing of the assessment, and occurs secondary to these factors. Unless amenorrhea is associated with clinically meaningful AN psychopathology variables or treatment outcomes, there is little or no clinical utility in differentiating AN individuals who do and do not menstruate.

2.2 **INCONSISTENCIES BETWEEN INDIVIDUALS IN DEVELOPMENT OF AMENORRHEA**

Despite being generally associated with weight loss and exercise, there have been many inconsistencies amongst individuals in the development of amenorrhea. Amenorrhea does not discriminate between full syndrome and subthreshold AN, occurring in obese women on low calorie diets although they had higher body weights that were sufficient for menstruation (Russell & Beardwood, 1970). Amenorrhea has also been shown to persist after weight restoration in some underweight individuals (Golden et al., 1997; Falk & Halmi, 1982; Brambilla et al., 2003). Studies indicated that amenorrhea occurred in about 20% of women before substantial weight loss (Theander, 1970; Halmi, 1974; Hurd, Palumbo, & Gharib, 1977), possibly due to distress the individual was experiencing early in their illness (Garfinkel et al., 1996). On the other hand, emaciated women with all behavioural and psychological symptoms of AN, including being underweight, may continue to menstruate (Thaw, Williamson, & Martin, 2001; Dalle Grave & Calugi, 2007), as may women with lower percentages of body fat (Golden & Carlson, 2008).

Furthermore, amenorrhea is not exclusive to AN and can occur in normal-weight women who engage in dietary restraint, individuals with BN, psychiatric inpatients, women experiencing psychological stress, or women who exercise strenuously, such as athletes (Watson & Andersen, 2003; Golden & Carlson, 2008). Taken together, the
findings indicated that whilst reduced weight and relative energy deficits were associated with amenorrhea, other factors such as timing of the assessment also contributed to its presence. Amenorrhea can occur in a variety of circumstances and its development is inconsistent between females before, during, or after recovery from AN. Hence, implementing it as a criterion for AN does not increase diagnostic specificity.

2.3 Empirical Evidence for Clinical Differences Signified by the Criterion

In considering the utility of the amenorrhea criterion, it is worth considering that the main purpose of clinical diagnoses is to guide clinicians in making suitable treatment decisions. The key issue is whether there are meaningful clinical differences in individuals with AN who are amenorrheic and those who are not. Studies assessing psychological differences will now be reviewed.

Description of studies. Seven empirical studies were identified and summarised in Table 3.2. Of the seven studies, two were of community samples (Dellava, Thornton, Lichtenstein, Pedersen, & Bulik, 2011; Garfinkel et al., 1996) and the other five of inpatient and outpatient treatment-seeking samples. With the exception of Cachelin and Maher (1998), who did not report the method used to determine amenorrhea status, the remaining studies determined this through clinical interview, including the EDE interview (Fairburn & Cooper, 1993) and the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders-IV (SCID-IV; First et al., 1995). Exclusion criteria for these studies included using hormone replacement therapy such as OCPs (Dalle Grave et al., 2008; Gendall et al., 2006; Roberto et al., 2008), being pregnant, breastfeeding, a history of gynaecological procedures (Dalle Grave et al., 2008; Gendall et al., 2006), being post-menopausal, or never having menstruated (Dalle Grave et al., 2008). Whilst some studies assessed biological variables (e.g., blood pressure, serum T4, leptin; Gendall et al., 2006) and bone density (Watson & Andersen, 2003), findings for these variables were not included in the review as they were beyond its scope. An eighth study was not included in the review because it included subthreshold AN patients who did not meet the weight, amenorrhea, or fear of gaining weight criteria, and provided results from overall analyses of variance and chi-square tests, rather than pairwise group comparisons (Santonastaso et al., 2009).

Limitations of studies. The common limitations of the studies were in determining amenorrhea status through self-report in interviews or computer-based assessments, some conducted by lay people, with no independent confirmation of
amenorrhea. The seven studies also mainly assessed behavioural symptoms of AN (i.e., the drive for thinness through dieting, excessive exercise, purging or using laxatives/diuretics), largely neglecting other AN criteria. With the exception of one study that assessed body image disturbance (Watson & Andersen, 2003), none of the other studies examined other clinically important cognitive AN diagnostic criteria of fear of gaining weight or self-worth invested in the body. The seven studies also used cross-sectional designs which differentiated patients based on presenting symptoms, making it impossible to determine the course of illness or potential shifts across groups. Three studies used relatively small sample sizes in the subthreshold AN group (n = 12, Cachelin & Maher, 1998; n = 16, Dalle Grave et al., 2008; n = 14, Gendall et al., 2006) limiting the extent to which findings could be generalised and the power with which differences between groups could be detected. Gendall et al. (2006) and Watson and Andersen’s (2003) studies did not require the weight criterion to be met for inclusion. Their more lenient selection criteria for groups made it difficult for conclusions to be drawn based purely on differences in menstrual status. Also, Dellava et al. (2011) did not have information pertaining to OCP use, leading to the possibility that women in the non-menstrual group were menstruating because of the use of these medications, and obscuring additional differences between groups that may have been found. To control statistically for the large number of comparisons, one study used a Bonferroni-adjusted alpha level of p < .004 (Roberto et al., 2008) for significance and two studies set alpha at a more stringent level of p < .01 for significance (Watson & Andersen, 2003; Dalle Grave et al., 2008). The remaining four studies did not control statistically for the large number of comparisons (Cachelin & Maher, 1998; Dellava et al., 2011; Gendall et al., 2006; Garfinkel et al., 1996). Despite these shortcomings, most results from all seven studies were in keeping with each other (Table 3.2), with similar findings across community, outpatient and inpatient samples, in a variety of interview and self-report measures. The findings will now be examined.

**Physical measures.** Although both groups were below the 85% ideal weight threshold, the amenorrheic group had lower current weight (Cachelin & Maher, 1998) and lower BMI (Dalle Grave et al., 2008; Gendall et al., 2006; Roberto et al., 2008) compared to the non-menstrual group. Most studies found no significant differences in highest or lowest premorbid weight (Garfinkel et al., 1996; Gendall et al., 2006; Watson & Andersen, 2003), with the exception of Dellava et al. (2011), who reported that the amenorrheic group had lower lifetime minimum weight than the non-menstrual group (Dellava et al., 2011). However, this study was the only one that
included non-interview data collection methods, with a majority of respondents using computer-administered self-report (72%) compared to telephone interview (28%), possibly impacting results through loss of precision and detail provided by interview-based assessments (Dellava et al., 2011).

Demographics. With the exception of full-scale intelligence which was higher in the amenorrheic AN group compared to the non-amenorrheic AN group (Watson & Andersen, 2003), most studies found no significant differences in demographics including age (Garfinkel et al., 1996; Gendall et al., 2006; Roberto et al., 2008), socioeconomic status (Cachelin & Maher, 1998), and education level (Dalle Grave et al., 2008). Only one study had inconsistent findings, reporting that the amenorrheic group was older (Dellava et al., 2011). In general, the studies do not provide consistent evidence for differences between the amenorrheic and non-amenorrheic groups on demographics.

Family history and early experiences. Reports on early experiences and family history were inconsistent. Some studies found no differences between groups in parental psychopathology, parental marital status, and parental disharmony (Garfinkel et al., 1996), and other studies reported that the amenorrheic group had less physical or sexual abuse (Watson & Andersen, 2003), less family control, more recreational family experiences and a higher level of family expressiveness (Cachelin & Maher, 1998). Until there is more empirical evidence available, conclusive inferences cannot be made about the association of menstrual status with family history and early experiences.

ED history. Whilst most studies found no differences between the amenorrheic and non-amenorrheic AN groups for age of onset and duration of AN, two had the following discrepant results: compared to the amenorrheic AN group, the non-amenorrheic group had a later age of onset (Gendall et al., 2006) and shorter duration of the disorder (Watson & Andersen, 2003). These differences could possibly be due to later onset or later recognition and diagnosis of the disorder due to the less emaciated states of the non-amenorrheic group (Watson & Andersen, 2003).

ED psychopathology. Findings on core AN psychopathology were consistent across all studies, with no differences found between amenorrheic and non-amenorrheic groups in body image disturbance/distortion, body size overestimation, drive for thinness (Cachelin & Maher, 1998; Watson & Andersen, 2003), and eating, weight, or shape concerns (Dalle Grave et al., 2008; Gendall et al., 2006; Roberto et al., 2008).
ED behaviours. Both groups were similar on the presence of ED behaviours including dietary restraint, binging, purging, and laxative or diuretic misuse across all studies. The presence of excessive exercise was found to be similar in both groups in the community (Dellava et al., 2011); however in treatment-seeking samples, it was higher in the amenorrheic group (Dalle Grave et al., 2008; Watson & Andersen, 2003). Looking more closely at the frequency of ED behaviours, the amenorrheic group reported lower frequency of binging and vomiting (Dalle Grave et al., 2008), and higher frequency of excessive exercise (Gendall et al., 2006). Following from these findings, it is unsurprising that Roberto et al. (2008) found that the amenorrheic group had significantly more restricting subtype compared to the non-amenorrheic group ($\chi^2(1) = 15.662, p < .001$). Although Cachelin and Maher (1998) found no differences in subtypes between the groups, their sample size was small compared to Roberto et al.’s (2008) study (refer to Table 3.2), giving less power to find differences between groups. Overall, the studies provided no evidence for differences in the presence of ED behaviours, with the exception of excessive exercise, which was higher in the amenorrheic group. When examining the frequency of ED behaviours, the amenorrheic group had less binging and vomiting and more exercise behaviours, and likewise, more restricting subtype, compared to the non-amenorrheic group.

Common ED characteristics. No differences were found between the amenorrheic and non-amenorrheic groups on ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, maturity fears, and having an external locus on control (Cachelin & Maher, 1998). The presence or absence of menstruation was not accompanied by any difference in assessed ED characteristics.

Psychiatric comorbidities. Six studies found no differences in comorbid psychopathology, with amenorrheic and non-amenorrheic groups reporting similar levels of depression, bipolar disorder, anxiety, alcohol or drug abuse/dependence, and general psychopathology such as hypochondriasis, hysteria, paranoia and schizophrenia. The exception was Dellava et al.’s (2011) study of twins in a community sample, which found lower rates of major depression and phobias in the amenorrheic group compared to the non-amenorrheic group. However, the researchers acknowledged that their failure to account for the non-independence of the data for the major depression scale (due to the inclusion of both members of twin pairs in the analyses) could have resulted in false positive findings. Overall, the studies did not provide consistent evidence that
amenorrhea status was accompanied by differences in the severity of general psychopathology.

*Personality.* Findings on personality were mixed, with two studies reporting no difference in self-directedness, concern over mistakes, personal standards, doubts about actions, neuroticism, extraversion (Dellava et al., 2011), novelty seeking, harm avoidance, reward dependence, persistence, self-directedness, cooperativeness, and self-transcendence (Dalle Grave et al. 2008). The exception was Gendall et al. (2006), who reported that the amenorrheic group had higher persistence and lower novelty seeking compared to the non-amenorrheic group.

*Service utilisation.* The presence or absence of menstruation did not influence service utilisation, with both groups having similar type of service used (majority outpatient), proportion of each group involuntarily treated (15%; Watson & Andersen, 2003), years in treatment (Cachelin & Maher, 1998), number of previous hospitalisations (Dalle Grave et al., 2008; Roberto et al., 2008), and procedures and therapeutic strategies used in treatment (Dalle Grave et al., 2008).

*Treatment outcome.* One study reported that the amenorrheic group had more weight restored at the end of treatment, as well as longer and more previous hospitalisations (Watson & Andersen, 2003), however, results may have been biased by the variable duration of treatment in the study. Dalle Grave et al. (2008) assessed patients following a fixed 20-week manualised cognitive behavioural therapy (CBT) treatment, reporting that the amenorrheic and non-amenorrheic AN groups did not differ in discharge BMI, dropout rate, time to drop out, and ED and general psychopathology at the end of treatment.

*Conclusion.* The studies provided evidence that there were few statistically significant differences between individuals with AN based on the presence or absence of amenorrhea, as both groups were comparable on demographics, ED psychopathology, ED behaviours, common ED characteristics, service utilisation, treatment outcome and psychiatric comorbidities. The findings were confirmed in a meta-analysis which concluded that no differences were found in studies comparing full syndrome AN and subthreshold AN (meeting all criteria except amenorrhea) groups in eating pathology ($d = 0.20, p = .32$), and comparing AN and EDNOS groups (including subthreshold AN) in general psychopathology ($d = 0.02, p = .68$) and physical health ($d = 0.14, p = .40$; Thomas et al., 2009). However, it is important to note that differences in
amenorrheic and non-amenorrheic subthreshold AN patients on the important cognitive diagnostic criteria of fear of gaining weight and self-worth invested in the body have been neglected in the existing literature.

Whilst there were few differences found in community samples (Dellava et al., 2011; Garfinkel et al., 1996), some differences were found between the amenorrheic and non-amenorrheic groups in clinical samples. Notably, the amenorrheic group had lower current weight and BMI, lower frequency of bingeing and vomiting, higher frequency of excessive exercise, and were more commonly restricting subtype, compared to the non-amenorrheic group. Taken together, the findings support the theory that menstruation is associated with higher BMIs and relative energy surpluses (e.g., increased calorie consumption through binge eating, or lowered energy expenditure from less excessive exercise). Interestingly, in addition to looking at differences between the groups, Dalle Grave et al. (2008) also conducted a logistic regression analysis, revealing that the sole predictors of amenorrhea in underweight AN patients were baseline BMI (OR = 2.70, 95% CI [1.38-5.29], p = 0.004) and intense exercise (OR = 0.93, 95% CI [0.86-0.99], p = 0.037), with psychopathological variables not associated with presence or absence of amenorrhea. Further support was provided by Pinheiro et al. (2007) and Abraham, Pettigrew, Boyd, Russell, and Taylor’s (2005) studies, which were not included in the present review as their samples included patients with AN, BN, and EDNOS but were undifferentiated by ED diagnosis. In Pinheiro et al.’s study, the amenorrheic ED group had significantly lower highest/lowest lifetime BMI, lower frequency of bingeing and vomiting, higher frequency of exercising and laxative use, and greater caloric restriction compared to the non-amenorrheic group. In Abraham et al.’s study, the best predictors of amenorrhea at admission for individuals with EDs were a current BMI of ≤ 18.0 kg/m² and having rules for exercising. Taken together, these findings on weight, ED behaviours, and subtype membership corroborate the theory that amenorrhea is a function of lower BMI and relative energy or nutritional deficits, rather than a core feature of AN. Amenorrhea appears to be secondary to weight loss and nutritional status, which are already required for diagnosis of AN, and does not discriminate between ED diagnoses.

Overall, these findings challenged the utility of the existing amenorrhea criterion, providing evidence that individuals in the non-amenorrheic subthreshold AN group were considerably underweight, displayed similar high levels of ED symptomatology and general psychopathology, obtained the same types of treatment.
and amounts of intensive healthcare resources, and had similar treatment outcomes compared to the amenorrheic AN group. Most differences between the groups led to the consistent conclusion that amenorrhea resulted from lower BMI and relative energy or nutritional deficits regardless of ED diagnosis, rather than being a defining feature of AN. The amenorrhea criterion could exclude patients with clinically significant illnesses. Indeed, these studies provided preliminary evidence that individuals diagnosed with EDNOS because they failed to meet the amenorrhea criterion had a greater age of onset (Gendall et al., 2006) and shorter duration of the disorder (Dalle Grave et al., 2008) compared to the amenorrheic AN group, possibly due to later recognition and diagnosis of the disorder. The clinical utility of the amenorrhea criterion is questionable, and the studies in this review support its exclusion from the diagnostic criteria of AN.

### Table 3.2

**Studies Comparing Full-syndrome with Subthreshold AN (Amenorrhea Criterion Not Met)**

<table>
<thead>
<tr>
<th>Participants</th>
<th>Measures</th>
<th>Outcomes</th>
</tr>
</thead>
</table>
| 52 female inpatients & outpatients at US hospitals: | **Interviews:** MMPI-2, IMP² | **Differences: Full syndrome AN group:**
| | **Self-report measures:** BDQ³, EDI⁴, FES⁵, I-E Scale⁶, BDI⁷ | - Demographics & ED history: younger age, lower weight
| | | - Family environment: less family control, more recreational family experiences, higher family expressiveness
| | | **No significant differences in:**
| | | - Demographics & ED history: socioeconomic status, age of onset, subtype membership
| | | - ED psychopathology: body image disturbance, drive for thinness, body dissatisfaction, body size overestimation, eating concerns
| | | - Common ED characteristics: ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, maturity fears, external locus of control
| | | - ED behaviours: bingeing, purging
| | | - Comorbid psychopathology: depression, general psychopathology
| | | - Service utilisation: years in ED treatment
<p>| 40 full syndrome AN | | |
| 12 subthreshold AN (amenorrhea criterion not met) | | |
| Mean age = 23.8 years. All participants &lt; 85% of ideal weight based on Metropolitan Life Insurance Company tables (1959). Average weight = 73.5% of ideal weight. | | |</p>
<table>
<thead>
<tr>
<th>Participants</th>
<th>Measures</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dalle Grave et al. (2008)</strong>&lt;br&gt;73 female inpatients at Italian hospital:&lt;br&gt;• 57 full syndrome AN&lt;br&gt;• 16 subthreshold AN (amenorrhea criterion not met)&lt;br&gt;Aged 12-50 years. All participants BMI ≤ 17.5 kg/m².</td>
<td><strong>Interview:</strong> EDE³&lt;br&gt;<strong>Self-report measures:</strong> BDI⁷, STAI⁹, TCI¹⁰</td>
<td><strong>Differences: Full syndrome AN group:</strong>&lt;br&gt;• Demographics &amp; ED history: lower BMI, shorter duration of AN&lt;br&gt;• ED psychopathology: higher overall severity of ED, higher shape/weight concerns&lt;br&gt;• ED behaviours: lower bingeing and purging, higher intense exercise&lt;br&gt;No significant differences in:&lt;br&gt;• Demographics &amp; family history: level of education, age of onset, premorbid BMI, parental marital status&lt;br&gt;• ED psychopathology: eating concerns&lt;br&gt;• ED behaviours: restrictive eating, laxative misuse&lt;br&gt;• Comorbid psychopathology: depression, anxiety&lt;br&gt;• Personality: novelty seeking, harm avoidance, reward dependence, persistence, self-directedness, cooperativeness, self-transcendence&lt;br&gt;• Service utilisation: number of previous inpatient admissions&lt;br&gt;• Treatment outcome (following 20-week inpatient CBT): dropout rate, time-to-dropout, final BMI, ED psychopathology, general psychopathology</td>
</tr>
<tr>
<td><strong>Dellava et al. (2011)</strong>&lt;br&gt;473 female twins from Swedish twin registry:&lt;br&gt;• 92 full syndrome AN&lt;br&gt;• 71 subthreshold AN (amenorrhea criterion not met)&lt;br&gt;Aged 20-47 years. All participants BMI &lt; 17.55 kg/m². No information on OCP use available.</td>
<td><strong>Telephone interview or computer-administered:</strong> SCID-IV¹¹&lt;br&gt;<strong>Self-report measures:</strong> TCI¹⁰, FMPS¹², EPI¹³</td>
<td><strong>Differences: Full syndrome AN group:</strong>&lt;br&gt;• Demographics &amp; ED history: older, lower lifetime lowest BMI&lt;br&gt;• Comorbid psychopathology: lower major depression, phobias&lt;br&gt;No significant differences in:&lt;br&gt;• ED history: lifetime highest BMI&lt;br&gt;• ED behaviours: fasting, bingeing, purging, laxative/diuretic misuse, excessive exercise, diet pill use&lt;br&gt;• Comorbid psychopathology: generalised anxiety disorder, panic disorder, obsessive compulsive disorder, alcohol/drug use&lt;br&gt;• Personality: Self-directedness, concern over mistakes, personal standards, doubts about actions, neuroticism, extraversion</td>
</tr>
<tr>
<td>Participants</td>
<td>Measures</td>
<td>Outcomes</td>
</tr>
<tr>
<td>--------------</td>
<td>----------</td>
<td>----------</td>
</tr>
</tbody>
</table>
| Gendall et al. (2006) | Interviews: SCID-III-R<sup>11</sup>, HRSD<sup>14</sup>, EDE<sup>8</sup>. Self-report measures: TCI<sup>10</sup> | Differences: Full syndrome AN group:  
- Demographics & ED history: lower BMI, lower age of onset, higher frequency of exercise, lower binge frequency  
- Personality: lower novelty-seeking, higher persistence  
No significant differences in:  
- Demographics & ED history: age, weight, body fat, lowest/highest BMI, duration of AN  
- ED psychopathology: eating, weight & shape concerns  
- ED behaviours: restrictive eating, purging, laxative misuse, fasting  
- Comorbid psychopathology: depression, anxiety, bipolar disorder, alcohol abuse/dependence, global assessment of functioning  
- Personality: harm avoidance, reward dependence |
| Garfinkel et al. (1996) | Interview: CIDI<sup>15</sup> | No significant differences in:  
- Demographics & ED history: age, age of onset, maximum/minimum weight, percentage weight loss  
- ED behaviours: bingeing, purging  
- Comorbid psychopathology: current/lifetime depression, anxiety, alcohol dependence  
- Family history & early experiences: detention early in life, sexual abuse, parental psychopathology, parental suicide attempts, parental unemployment/hospitalisation, parental disharmony |
| Roberto et al. (2008) | Chart review of: Interviews by health professionals, EDE<sup>8</sup>, SCID-IV<sup>11</sup>. Self-report measures: BDI<sup>7</sup>, BAI<sup>16</sup> | Differences: Full syndrome AN group:  
- ED history: lower lifetime/admission BMI, more restricting subtype  
No significant differences in:  
- Demographics & ED history: age, duration of AN, discharge BMI  
- ED psychopathology: eating, weight & shape concerns  
- ED behaviours: restrictive eating  
- Comorbid psychopathology: depression, anxiety  
- Service utilisation: number of previous hospitalisations |
Table 3.2 (continued)

<table>
<thead>
<tr>
<th>Participants</th>
<th>Measures</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>297 female inpatients at US hospitals:</td>
<td><strong>Chart review</strong> of: Interviews by health professionals</td>
<td><strong>Differences, Full AN group:</strong></td>
</tr>
<tr>
<td>• 230 full syndrome AN</td>
<td></td>
<td>• <strong>Demographics:</strong> higher full-scale intelligence</td>
</tr>
<tr>
<td>• 67 subthreshold AN (weight and/or amenorrhea criterion not met)</td>
<td><strong>Self-report measures:</strong> EAT-26(^1), EDI-2(^2), BDI(^3)</td>
<td>• <strong>ED history:</strong> longer duration of AN, higher lifetime excessive exercise</td>
</tr>
<tr>
<td>Subthreshold group included 18 with amenorrhea, 49 with irregular menses. 28 of subthreshold group met all criteria except amenorrhea. Subthreshold group collapsed across absence of weight and/or amenorrhea criteria.</td>
<td><strong>Other assessment:</strong> WAIS-R(^4)</td>
<td>• <strong>Service utilisation &amp; treatment outcomes:</strong> higher number and longer duration of previous hospitalisations, more weight restored at end of treatment</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• <strong>Early experiences:</strong> lower physical/sexual abuse</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>No significant differences in:</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• <strong>ED history:</strong> age of onset, highest/lowest weight</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• <strong>ED psychopathology:</strong> drive for thinness, body image disturbance</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• <strong>ED behaviours:</strong> restrictive eating, bingeing, purging</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• <strong>Comorbid psychopathology:</strong> depression, alcohol/drug abuse</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• <strong>Service utilisation:</strong> service type (majority outpatient), proportion of involuntarily treated (15% of each group)</td>
</tr>
</tbody>
</table>

*Note. AN—Anorexia Nervosa; ED—Eating disorder; BMI—Body mass index; OCP—Oral contraceptive pill; CBT—Cognitive behavioural therapy; US—United States.*

1. Minnesota Multiphasic Personality Inventory-2 (MMPI-2; Hathaway & McKinley, 1989)
2. Image Marking Procedure (IMP; Askevold, 1975)
3. Body Distortion Questionnaire (BDQ; Slade & Russell, 1973)
4. Eating Disorder Inventory (EDI; Garner & Olmsted, 1984; EDI-2; Garner, 1991)
5. Family Environment Scale (FES; Moos & Moos, 1994)
6. Internal-External Locus of Control Scale (I-E Scale; Rotter, 1966)
7. Beck Depression Inventory (BDI; Beck et al., 1961)
8. Eating Disorder Examination (EDE; Fairburn & Cooper, 1993)
9. State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970)
10. Temperament and Character Inventory (TCI; Cloninger, Svrakic, Przybeck, & Wetzel, 1994)
11. Structured Clinical Interview for DSM Disorders (SCID-III-R; Spitzer et al., 1992; SCID-IV; First et al., 1995)
12. Frost Multidimensional Perfectionism Scale (FMPS; Frost, Marten, Lahart, & Rosenblate, 1990)
13. Eysenck Personality Inventory (EPI; Schapiro et al., 2001)
15. World Health Organisation Composite International Diagnostic Interview (CIDI; World Health Organisation, 1990)
16. Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988)
17. Eating Attitudes Test (EAT-26; Garner et al., 1982)
18. Wechsler Adult Intelligence Scale, Revised (WAIS-R; Wechsler, 1981)

2.4 **Use of the oral contraceptive pill in individuals with Anorexia Nervosa**

Whilst it is common for individuals with AN to be infertile due to severely low weight, negating the need for birth control, women with AN may still be prescribed with OCPs for various non-contraceptive purposes. Population level studies have shown
that almost 60% of women use OCPs for non-contraceptive purposes, including the reduction of severe menstrual pain, prevention of menstruation-related migraines, or the treatment of acne, hirsutism, or endometriosis (Jones, 2011).

In the case of women with AN, over 90% of this population demonstrate osteopenia, and almost 40% demonstrate osteoporosis at one or more skeletal sites (Grinspoon, Thomas, Miller, Herzog, & Klibanski, 2002). Osteopenia and osteoporosis occur in individuals with AN due to low weight (Klibanski, Biller, Schoenfeld, Herzog, & Saxe, 1995; Hotta, Shibasaki, Sato, & Demura, 1998), which results in inadequate bone growth and development during adolescence, and premature bone loss if the illness continues into adulthood (Biller et al., 1989). Whilst recent research has shown that the most effective treatment of osteoporosis in AN is early detection and weight restoration, with oestrogen or OCP administration having little effect (for a review, refer to Mehler & MacKenzie, 2009), OCP administration in some underweight patients may result in improved bone density (Klibanski et al., 1995), and may augment the effects of other medical interventions in improving bone density when used in a combined treatment strategy (Grinspoon et al., 2002). Hence, women with AN are sometimes prescribed oestrogen, usually in the form of OCPs, with the objective of protecting bone density (Rigotti, Nussbaum, Herzog, & Neer, 1984).

Clinical observation of the patients seen at ED services in metropolitan areas in Australia suggest that a substantial proportion of patients are using the OCP, which is frequently prescribed and available at low cost under the Medicare system in Australia. Descriptive statistics for the proportion of AN and non-eating disordered comparison group participants in the present study using the OCP are provided in the Method chapter (p. 136-137).

2.5 **Implications for the Present Study**

The rationale behind the amenorrhea criterion is unclear, and amenorrhea does not occur consistently in individuals. It is not a phenomenon exclusive to AN and cannot be applied to males, and females who are pre-menarchal, post-menopausal, or on OCPs. The available literature suggested that the presence of amenorrhea did not usefully distinguish a category of low weight patients, as it occurred secondary to weight loss and relative energy deficits, having no effect on clinically meaningful psychopathological variables including eating pathology, illness severity, psychiatric comorbidities, distress, service utilisation and treatment outcome. However, the existing
literature did not assess group differences in amenorrheic and non-amenorrheic individuals with AN on the cognitive diagnostic criteria of AN, fear of gaining weight and self-worth invested in the body. Also, most studies in the literature review excluded AN participants on the OCP. Only Roberto et al. (2008) reported the proportion of their patient sample using the OCP, which was 7.1%. Although this was not a large proportion of patients in their United States (US) sample, clinical observation of the patients seen at ED services in metropolitan areas in Australia suggest that a more substantial proportion of patients are using the OCP. However, little is known about how AN patients on the OCP fare on clinically important psychological factors compared to amenorrheic and non-amenorrheic AN groups.

Many researchers have supported exclusion of the amenorrhea criterion in the diagnosis of AN (Mitchell et al., 2005; Fairburn & Bohn, 2005; Dalle Grave et al., 2008; Roberto et al., 2008; Garfinkel et al., 1996). Accordingly, the Eating Disorders Work Group has recommended deletion of the amenorrhea criterion for DSM-V (APA, 2012). The designation of EDNOS for individuals who fail to meet the amenorrhea criterion poses a problem because we lack treatment guidelines for the heterogeneous category of EDNOS, and it may give patients the false impression of not having a disorder of clinical severity, or the opportunity to deny the presence of eating problems (Dalle Grave et al., 2008; Attia & Roberto, 2009). Eliminating the amenorrhea criterion could increase the chances of receiving appropriate treatment for women who share many features of AN but continue to have some menstrual activity (Attia & Roberto, 2009). Rather than conforming to narrow DSM-IV-TR criteria, the use of clinically meaningful diagnoses allows for better understanding of the course of illness, predictors of treatment response, outcome, and differential treatment needs (Agras et al., 2004).

In light of the existing empirical evidence, patients who failed to meet the amenorrhea criterion were not excluded from the present study. Given the relatively small sample sizes and the neglect of cognitive diagnostic criteria of AN in existing studies, the present study aimed to assess cognitive and psychological differences in amenorrheic and non-amenorrheic AN groups using a larger treatment-seeking sample. Further, the present study also aimed to explore a new area that has been previously neglected, comparing AN patients on the OCP to those who are not. Aims and research questions are detailed in Section 3 (p. 42-44).
3. **AIMS AND RESEARCH QUESTIONS**

3.1 **AIMS**

The present study intended to extend previously discussed findings on clinically significant differences between full syndrome AN and subthreshold AN patients who did not meet the weight criterion (BMI ≤ 17.5 kg/m²) and/or amenorrhea criterion, with a larger AN sample. Patients who demonstrated a drive for thinness as determined through clinical interview by their treating clinician and met all other cognitive diagnostic criteria of AN were included in the study, then divided into subgroups based on whether they met the weight and/or amenorrhea criteria, or were using the OCP.

In determining the variables to be examined in the group comparisons, the present study took into account that studies examined in the earlier literature reviews consistently found no differences between the full syndrome and subthreshold AN groups on the following factors: demographics (Garfinkel et al., 1996; Gendall et al., 2006; Roberto et al., 2008; Cachelin & Maher, 1998; Dalle Grave et al., 2008), physical measures (Watson & Andersen, 2003; McIntosh et al., 2004), ED behaviours (McIntosh et al., 2004; Williamson et al., 1992; Cachelin & Maher, 1998; Dalle Grave et al., 2008; Dellava et al., 2011; Garfinkel et al., 1996; Gendall et al., 2006; Roberto et al., 2008; Watson & Andersen, 2003), ED history (Watson & Andersen, 2003; McIntosh et al., 2004), common ED characteristics (Williamson et al., 1992; Bunnell et al., 1990; McIntosh et al., 2004; Cachelin & Maher, 1998), service utilisation (Watson & Andersen, 2003; Dalle Grave et al., 2008; Roberto et al., 2008), and psychiatric comorbidities (Bunnell et al., 1990; McIntosh et al., 2004, Watson & Andersen, 2003; Garfinkel et al., 1996; Cachelin and Maher, 1998; Dalle Grave et al., 2008; Gendall et al., 2006; Roberto et al., 2008). Hence, these variables were not further examined.

In line with the focus of the present study on cognitive and psychological factors, the first aim was to compare the full syndrome and subthreshold AN groups, adding to the literature by examining the cognitive AN diagnostic criteria that have been neglected in existing studies, and examining novel clinically relevant factors that are critical in the maintenance of AN. The factors included in the present study are described in the next section. The second aim was to address the lack of knowledge about AN patients on the OCP, as this group was clinically observed to be a substantial proportion of patients seen at ED services in metropolitan Australia, and often excluded from studies. AN patients on the OCP and those who were not were compared on the same variables, described in the following section.
3.2 VARIABLES ASSESSED IN THE PRESENT STUDY

The relevant factors can be divided into five categories and are summarised in Table 3.3.

**AN diagnostic criteria.** The AN diagnostic criteria include the drive for thinness (Criterion A) and body image disturbance (Criterion C), for which no differences have been found when comparing underweight and non-underweight AN groups (Bunnell et al., 1990; McIntosh et al., 2004; Watson & Andersen, 2003; Williamson et al., 1992), as well as amenorrheic and non-amenorrheic AN groups (Cachelin & Maher, 1998; Watson & Andersen, 2003). The present study will add to the literature by also assessing the two cognitive diagnostic criteria of AN that have been wholly neglected, the fear of gaining weight (Criterion B) and self-worth invested in the body (Criterion C). They are clinically important as they embody the severity of illness, and affect treatment planning and treatment outcome.

**Common ED characteristics.** Whilst a number of common ED characteristics (e.g., perfectionism, ineffectiveness, maturity fears, interpersonal distrust) have been examined in existing studies, with no differences found between the underweight and non-underweight AN groups (Bunnell et al., 1990; McIntosh et al., 2004), as well as between amenorrheic and non-amenorrheic groups (Cachelin & Maher, 1998), only two ED characteristics of relevance to the present study will be assessed: perfectionism, which has been assessed in several existing studies included in the earlier literature reviews, and self-esteem, which has not. The basis for selection of both these criteria are discussed in detail in Chapters 4 and 5.

**Control-related cognitions.** Whilst control-related factors have not been examined in existing studies, they have been described richly in theoretical accounts of AN by original ED theorists (Bruch, 1973; Garfinkel & Garner, 1982; Slade, 1982) and more recently by Fairburn, Shafran, et al. (1999). Of particular interest to the present study are the general need for control and the sense of control being dependent on eating/weight. The basis for selection of both these control-related cognitions are discussed in detail in Chapters 4 and 5.

**Dysfunctional metacognitions.** The five domains of dysfunctional metacognitions, positive beliefs about worry, negative beliefs about the uncontrollability/danger of thoughts, the need to control thoughts, cognitive confidence, and cognitive self-consciousness, have not been previously examined in comparisons of
the subgroups of the present study. However, there has been recent interest in the AN field regarding the relevance of metacognitions, or the individual’s thoughts and beliefs about their thoughts, in maintaining AN (Cooper, Grocutt, Deepak, & Bailey, 2007; Woolrich, Cooper, Turner, 2008; McDermott & Rushford, 2011). The relevance of dysfunctional metacognitions to AN is examined in detail in Chapters 4 and 5.

General psychopathology. Existing studies have assessed specific psychiatric comorbidities (e.g., bipolar disorder, alcohol or drug abuse, self-harm), finding no differences in underweight and non-underweight AN groups (Bunnell et al., 1990; McIntosh et al., 2004, Watson & Andersen, 2003) and amenorrheic and non-amenorrheic AN groups (Cachelin & Maher, 1998; Dalle Grave et al., 2008; Garfinkel et al., 1996; Gendall et al., 2006; Roberto et al., 2008; Watson & Andersen, 2003). The present study focused on general psychopathology, namely depression, anxiety, and stress, which are clinically important indicators of reduced well-being.

3.3 Research Question 1

Research Question 1(a). Are there differences between the groups with full syndrome AN and subthreshold AN (not meeting diagnostic criteria for weight [BMI <17.5 kg/m²] and/or amenorrhea) on the variables of interest (Table 3.3)?

Research Question 1(b). Are there differences between AN patients on the OCP and those who are not, on the variables of interest (Table 3.3)?

Table 3.3
Variables of Interest to Research Questions 1(a) and (b)

<table>
<thead>
<tr>
<th>AN diagnostic criteria</th>
<th>Common ED characteristics</th>
<th>Control-related cognitions</th>
<th>Dysfunctional metacognitions</th>
<th>General psychopathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Fear of gaining weight</td>
<td>• Perfectionism</td>
<td>• General need for control</td>
<td>• Positive beliefs about worry</td>
<td></td>
</tr>
<tr>
<td>• Self-worth invested in the body</td>
<td>• Self-esteem</td>
<td>• Sense of control dependent on eating/weight</td>
<td>• Negative beliefs about the uncontrollability/danger of thoughts</td>
<td></td>
</tr>
<tr>
<td>• Body image disturbance</td>
<td></td>
<td></td>
<td>• Need to control thoughts</td>
<td></td>
</tr>
<tr>
<td>• Drive for thinness</td>
<td></td>
<td></td>
<td>• Cognitive confidence</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Cognitive self-consciousness</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Depression</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Anxiety</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Stress</td>
<td></td>
</tr>
</tbody>
</table>

Note. AN—Anorexia Nervosa; ED—Eating disorder.
Chapter 3 compared individuals who met full criteria for AN with those who met all criteria except weight and amenorrhea, on a number of factors that have been proposed to be associated with AN. The factors included measures of AN symptomatology, common ED characteristics including perfectionism and low self-esteem, control-related cognitions, dysfunctional metacognitions, and general psychopathology. So what does the literature reveal about how the factors may result in the development or maintenance of AN?

The recent Cochrane Methodology Review of AN treatment studies for adults and older adolescents found empirical evaluation of AN treatments in this age range is limited, with existing studies finding no difference between specific therapies (e.g., CBT) and control therapies (e.g., “eclectic” therapy, non-specific clinical management; Hay et al., 2009). At the end of treatment, 70% of participants were still not recovered and had BMIs below the normal range (McIntosh et al., 2004; Serfaty, Turkington, Heap, Ledsham, & Jolly, 1999). AN also had a mortality rate of 9.6%, the highest of any mental disorder (Nielson, 2001). The dearth of effective treatments for AN is unsurprising, considering the lack of a sound theoretical basis on which to develop treatment (Fairburn, 2005) and an urgent lack of empirical research from which health professionals and AN sufferers can make informed treatment choices (Kaplan, 2002). Clinically, treatment is guided not by randomised controlled trials but by guidelines developed through pooled clinical experience by bodies such as the APA, the Royal Australian and New Zealand College of Psychiatrists (RANZCP; 2004), and the National Institute for Clinical Excellence (NICE; 2004). Research that provides information regarding antecedents and reinforcers of AN is needed for the development of prevention and treatment interventions and improved care for AN sufferers. This chapter aims to provide an overview of proposed risk and maintenance factors of AN (Section 1). Then, individual factors relevant to the present study and their associations with AN will be reviewed in the following order: perfectionism (Section 2), low self-esteem (Section 3), control-related cognitions (Section 4), and dysfunctional metacognitions (Section 5). Finally, aims and research questions to assess the associations will be described (Section 6).
1. **Brief Overview of Factors Associated with Anorexia Nervosa**

Several researchers have suggested that the development and maintenance of AN result from causal relationships between clusters of “typical” factors grouped into predisposing, precipitating and perpetuating (or maintenance) factors, summarised in Table 4.1. Proposed factors can be classified further into individual variables (Vitousek & Ewald, 1993), sociocultural influences (Crisp, 1980; Garner, Garfinkel, & Olmsted, 1983), family influences (Bruch, 1982), the functional bases for eating disordered behaviour (Vitousek & Ewald, 1993; Vitousek & Orimoto, 1993), neurobiological perspectives (Connan, Campbell, Katzman, Lightman, & Treasure, 2003; Southgate, Tchanturia, & Treasure, 2005), interpersonal theories (Schmidt & Treasure, 2006), cognitive accounts (Garner & Bemis, 1982), thought-action fusion, imagery, and the role of early memories (Cooper, 2005).

Amongst the most prominent of the factors are perfectionism, low self-esteem, and beliefs in the domain of control (Sassaroli, Gallucci, & Ruggiero, 2008). More recently, interest has turned towards the role of metacognition (Cooper et al., 2007; Woolrich et al., 2008; McDermott & Rushford, 2011). It is not the aim of the present study to examine the entire body of factors, but to focus instead on factors that have been theorised to be critical to cognitive theories of AN (elaborated on in Chapter 5), namely, perfectionism, low self-esteem, control-related cognitions, and dysfunctional metacognitions. The following sections will detail the theorised relationships between these factors and AN, and provide a review of existing empirical evidence.
Table 4.1
Factors Hypothesised to Lead to the Development and Maintenance of Anorexia Nervosa

<table>
<thead>
<tr>
<th>Predisposing Factors</th>
<th>Precipitating Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Individual</strong></td>
<td>Life events leading to distress, including: 1, 4, 5, 6, 7, 10</td>
</tr>
<tr>
<td>Biological influences 1, 2</td>
<td>New environmental demands</td>
</tr>
<tr>
<td>e.g., genetics, neuroendocrine factors</td>
<td>e.g., first sexual relationship, shift in</td>
</tr>
<tr>
<td>Perinatal complications 1, 3, 4</td>
<td>attachment from parents to peers/partners</td>
</tr>
<tr>
<td>Female gender 4, 5, 6</td>
<td>Brain development in adolescence</td>
</tr>
<tr>
<td>Body dissatisfaction 2</td>
<td>Biological maturation in puberty</td>
</tr>
<tr>
<td>Negative self-concept 1, 2, 5, 7, 10</td>
<td>Nutritional stress due to illness, dieting, exercise</td>
</tr>
<tr>
<td>Low self-esteem</td>
<td>Separation, loss, bereavement</td>
</tr>
<tr>
<td>Sense of ineffectiveness</td>
<td>Family disruption</td>
</tr>
<tr>
<td>Low sense of autonomy</td>
<td>e.g., parental divorce</td>
</tr>
<tr>
<td>Childhood abuse 2</td>
<td>Interpersonal conflict or difficulties</td>
</tr>
<tr>
<td>Weight disturbances 4</td>
<td>Social hassles and trauma</td>
</tr>
<tr>
<td>Perceptual disturbances 4, 7</td>
<td>Events perceived as threat to or loss of self-esteem</td>
</tr>
<tr>
<td>Personality characteristics 1, 2, 5, 7, 8</td>
<td>e.g., academic failure</td>
</tr>
<tr>
<td>Obsessive-compulsive</td>
<td>Events leading to perceived loss of control</td>
</tr>
<tr>
<td>Compliant</td>
<td>Negative social evaluation</td>
</tr>
<tr>
<td>Dependent</td>
<td></td>
</tr>
<tr>
<td>Perfectionistic</td>
<td></td>
</tr>
<tr>
<td>Rigid</td>
<td></td>
</tr>
<tr>
<td>Shy, socially withdrawn</td>
<td></td>
</tr>
<tr>
<td>Negative affect 2, 9</td>
<td></td>
</tr>
<tr>
<td>e.g., anger, guilt</td>
<td></td>
</tr>
<tr>
<td>Information processing biases 4, 7</td>
<td></td>
</tr>
<tr>
<td>e.g., dichotomous thinking, poor set-shifting</td>
<td></td>
</tr>
<tr>
<td>Cognitive factors 2</td>
<td></td>
</tr>
<tr>
<td>e.g., obsessive thoughts, dissociation</td>
<td></td>
</tr>
<tr>
<td>Weight pathologies 4</td>
<td></td>
</tr>
<tr>
<td>e.g., premorbid obesity</td>
<td></td>
</tr>
<tr>
<td>Other illness 7</td>
<td></td>
</tr>
<tr>
<td><strong>Familial</strong></td>
<td></td>
</tr>
<tr>
<td>Demographic characteristics 2, 4</td>
<td></td>
</tr>
<tr>
<td>Older parental age</td>
<td></td>
</tr>
<tr>
<td>High socio-economic status</td>
<td></td>
</tr>
<tr>
<td>Disturbed family dynamics/interactions 2, 4, 5, 7</td>
<td></td>
</tr>
<tr>
<td>e.g., enmeshed, intrusive, hostile</td>
<td></td>
</tr>
<tr>
<td>Parental expectations 4, 5, 7</td>
<td></td>
</tr>
<tr>
<td>Emphasis on achievement/success</td>
<td></td>
</tr>
<tr>
<td>Unrealistic expectations</td>
<td></td>
</tr>
<tr>
<td>Parental history of affective illness 5</td>
<td></td>
</tr>
<tr>
<td>e.g., depression, substance abuse</td>
<td></td>
</tr>
<tr>
<td>Weight pathologies in family 2</td>
<td></td>
</tr>
<tr>
<td>e.g., mothers with eating disorders</td>
<td></td>
</tr>
<tr>
<td><strong>Socio-cultural</strong></td>
<td></td>
</tr>
<tr>
<td>‘Fashion’; emphasis on thinness 4, 5, 7</td>
<td></td>
</tr>
<tr>
<td>Media 7</td>
<td></td>
</tr>
<tr>
<td>Peer pressure 2</td>
<td></td>
</tr>
<tr>
<td>Shift in societal structure, norms, customs 4, 5</td>
<td></td>
</tr>
<tr>
<td>e.g., increased pressure on women for vocational achievement</td>
<td></td>
</tr>
</tbody>
</table>

1 Treasure, Tchanturia, & Schmidt (2005)
2 Polivy & Herman (2002)
3 Halmi, Goldberg, Eckert, Casper, & Davis (1977)
4 Garfinkel & Garner (1982)
5 Crisp (1980)
6 Southgate, Tchanturia, & Treasure (2005)
2. PERFECTIONISM

2.1 THEORETICAL RELATIONSHIP WITH ANOREXIA NERVOSA

Perfectionism is considered a particularly strong vulnerability factor for AN, involved in both its development (Lilenfeld et al., 2000) and maintenance (Slade, 1982). Early theorists highlighted the link noted clinically between perfectionism and AN, describing the typical profile of individuals with AN as good, successful, compliant, and conscientious, performing well in school and working relentlessly to live up to the expectations of others (Bruch, 1973; Crisp, 1965; Garner & Bemis, 1982). Perfectionism in AN has been observed to be generally maladaptive and pathological, involving an extreme concern about making mistakes, with even minor mistakes perceived as likely to lead to failure (Frost et al., 1990). The conceptual centrality of perfectionism in the individual’s life is also illustrated by its role in a system whereby self-worth is maintained largely on the basis of striving to achieve demanding goals and success at meeting them (Fairburn, Cooper, & Shafran, 2003; Bruch, 1982). In Fairburn et al.’s (2003) maintenance theory of EDs (described in Chapter 5, p. 118-119), the term “clinical” perfectionism was coined, referring to the striving for personally demanding standards despite adverse consequences.

In AN, perfectionist standards are proposed to be applied to aspects of life (e.g., work performance), as well as attempts to control eating, shape and weight. They drive highly restrictive eating (Waller, Cordery et al., 2007) and are applied to dietary restriction in the form of extreme dietary rules (Fairburn, Shafran, et al., 1999). The nature of perfectionism means that no amount of weight loss is ever good enough, with the individual continually striving for increasing dietary restraint and repeatedly shifting goals to lower weights (Schmidt & Treasure, 2006). The accompanying fear of failure (i.e., fear of over-eating or weight gain), frequent and selective attention to performance (e.g., body checking), and striving to meet goals (e.g., weight loss) helps maintain the disorder (Fairburn et al., 2003). Additionally, perfectionism has been thought to contribute to EDs by making normal variation in bodies, perceived as shortcomings or
imperfections, distressing (Hewitt, Flett, & Ediger, 1995). To translate the theoretical propositions to empiricism, the following section will review existing empirical evidence for associations between perfectionism and AN. The mechanisms through which perfectionism are theorised to maintain AN are discussed in detail in Chapter 5.

2.2 Empirical evidence for its association with Anorexia Nervosa

This section will focus on published evidence for the association of perfectionism with AN. Studies linking perfectionism with other psychiatric disorders (for a review, refer to Shafran & Mansell, 2001) and disordered eating behaviours in normal populations (Forbush, Heatherton, & Keel; 2007; McLaren, Gauvin, & White, 2001; Hewitt et al., 1995) will not be included. To present an accurate picture of perfectionism in AN, this review will only focus on studies of clinically diagnosed AN populations. Literature that explores the direct relationship of perfectionism with AN, either as a risk or maintenance factor, will be evaluated. Complex multivariate models combining perfectionism with other vulnerability factors to predict AN symptoms will be discussed in Chapter 5.

Description of studies. Because of the mass of literature, the present review will be organised into findings from existing reviews that included studies up to 2005, then individual publications post-2005. Four existing reviews were identified (Bardone-Cone et al., 2007; Stice, 2002; Cassin & von Ranson, 2005; Franco-Paredes, Mancilla-Díaz, Vázquez-Arévalo, López-Aguilar, & Álvarez-Rayón, 2005), summarised in Table 4.2. Seven individual studies published since 2005 are detailed in Table 4.3, consisting of three cross-sectional group comparison studies (Deas, Power, Collin, Yellowlees, & Grierson, 2011; Bachner-Melman, Zohar, Kremer, & Ebstein, 2007; Castro-Fornieles et al., 2007), three cross-sectional retrospective studies (Wade et al., 2008; Pike et al., 2008; Kim, Heo, Kang, Song, & Treasure, 2010), and one prospective study (Nilsson, Sundbom, & Hägglof, 2008).

Limitations of studies. The four reviews and seven studies had similar methodological limitations. Design weaknesses included an over-reliance on cross-sectional designs, constraining inferences about directionality or the causal role of perfectionism (Bardone-Cone et al., 2007; Stice, 2002), failure to differentiate between state and trait effects, and the impossibility of determining whether perfectionism preceded the onset of AN or was related to transient effects of starvation, such as symptoms of rigidity and obsessionality (Cassin & von Ranson, 2005). Studies using
cross-sectional retrospective designs could have been influenced by inaccurate or selective recall, with recall biased by outcome (Bardone-Cone et al., 2007). For the prospective studies, few assessed AN-specific pathology, focusing instead on BN, BED, or general ED symptomatology (Stice, 2002). Most covered relatively short time intervals (under six months) and rarely had more than two assessment points (Bardone-Cone et al., 2007), resulting in smaller effect sizes (Stice, 2002). Reviewers unanimously recommended that future research should utilise prospective longitudinal designs with multiple time points to clarify the temporal relationship between perfectionism and AN, and to assess whether perfectionism influenced symptom-level change or treatment outcomes. Experimental or treatment studies were also recommended to determine whether changes in perfectionism as a result of treatment was associated with decreases in symptoms or improved treatment outcome.

Additionally, there was an absence of a standard definition for recovery from AN, with an over-reliance of physical indices (e.g., weight and menstruation status), and differences in the length of time that indices of recovery were maintained. No studies assessed recovery in terms of body or eating-related cognitions (Bardone-Cone et al., 2007).

The studies also had limitations related to sampling. A majority of studies used clinical samples from treatment settings, excluding cultural minorities and males, limiting generalisation across all patient groups and non-treatment-seeking individuals (Bardone-Cone et al., 2007), cultural minorities, or males (Cassin & von Ranson, 2005). Sample sizes varied across studies, having implications for power. Comparison groups also varied, with a review finding that about half the studies had healthy controls as a comparison to ED groups, while only 15% of studies included non-eating disordered psychiatric control groups (Bardone-Cone et al., 2007). Reviewers argued that psychiatric control groups were required for specificity tests, to determine if perfectionism was related to psychopathology in general, or only to EDs (Bardone-Cone et al., 2007).

Other limitations included the use of single reporter and self-report measures. Reviewers recommended that these should be interpreted with caution, and further studies should utilise collateral reports, observational data, or interview measures (Stice, 2002; Cassin & von Ranson, 2005; Franco-Paredes et al., 2005).
Despite the above limitations, most findings were consistent, with similar results from a range of designs including cross-sectional, retrospective, case-control, and longitudinal designs, in Western and East Asian samples. The findings will now be discussed.

**Summary of reviews.** The most methodologically rigorous review was a meta-analysis of studies assessing risk and maintenance factors for eating pathology (Stice, 2002). This review included only prospective and experimental studies with well validated scales, to permit the strongest etiological and maintenance inferences. It was concluded that perfectionism had a small but significant effect on increases in eating pathology (average effect size $r = 0.6, p < .025$) and a medium effect on the maintenance of eating pathology ($r = .22, p = .030$). However, the reviewer (Stice, 2002) advised caution in making inferences for AN-specific symptoms, as the conclusions were drawn from six studies focusing on BN pathology and ED symptom composites, with no study focusing solely on AN symptoms. To summarise, perfectionism appeared to be a maintenance factor for general eating pathology including dietary restraint, bingeing, and purging.

Bardone-Cone et al. (2007) conducted a comprehensive review, including 55 studies that focused exclusively on assessing perfectionism in EDs using validated measures. They concluded that individuals with AN consistently had higher levels of perfectionism compared to healthy and psychiatric controls (including individuals with mood disorders, anxiety disorders, and alcohol abuse or dependence). Despite some mixed findings, a majority of studies determined that individuals who had recovered from AN demonstrated no reductions in levels of perfectionism, which remained elevated after recovery, leading reviewers to suggest that perfectionism was not a simple state effect associated with the active phase of illness, but a risk factor or long-term residual effect of AN. Whilst there was a lack of studies considering premorbid perfectionism in predicting AN, reviewers identified one study in which individuals with AN had higher rates of retrospectively-assessed childhood perfectionism compared to healthy and psychiatric controls (Fairburn, Cooper, Doll, & Welch, 1999), and one prospective study which reported that onset of AN in young adults (20 to 24 years old) was predicted by perfectionism at a younger age (12 to 16 years old; Tyrka, Waldron, Graber, & Brooks-Gunn, 2002). Perfectionism was also a negative predictor of outcome for AN, with higher levels of perfectionism associated with treatment dropout (Sutandar-Pinnock, Blake, Carter, Olmsted, & Kaplan, 2003) and with poor prognosis.
five to 10 years post-inpatient admission (Bizeul, Sadowsky, & Rigaud, 2001). To conclude, AN was found to be characterised by elevated perfectionism that endured after recovery, and had predispositional significance for the development of AN. Higher levels of perfectionism also predicted poorer outcome in individuals with AN.

Two further reviews appeared to be less rigorous, as no inclusion or exclusion criteria were reported (Cassin & von Ranson, 2005; Franco-Paredes et al., 2005). Whilst they were more descriptive, with less attention paid to methodological issues, conclusions were similar to the first two reviews. Perfectionism was found to be elevated in individuals with AN compared to normal and psychiatric controls, and remained elevated after recovery from AN or restoration to normal body weight. Perfectionism was also reported to predict the onset of AN (Cassin & von Ranson, 2005) and general ED symptoms (Franco-Paredes et al., 2005). Reviewers suggested that perfectionism may contribute to resistance to treatment and relapse in AN.
### Table 4.2
*Reviews of Studies Assessing Perfectionism in Anorexia Nervosa*

<table>
<thead>
<tr>
<th>Method</th>
<th>Findings</th>
</tr>
</thead>
</table>
| Stice (2002) | **Summary:** Meta-analysis of studies assessing risk and maintenance factors for eating pathology. 6 studies assessing perfectionism were identified, published from 1980 to 2001.  
**Inclusion criteria:** Prospective or experimental study, validated scales used  
- Existing studies focused on BN, BED, or a composite of ED symptoms, with no studies focusing on AN symptoms  
- Perfectionism had a small but significant effect on increases in eating pathology, and a medium effect on the maintenance of eating pathology |
| Bardone-Cone et al. (2007) | **Summary:** Review of 55 studies assessing perfectionism in EDs, published from 1990 to 2005.  
**Inclusion criteria:** n ≥10 per group, samples meeting diagnostic criteria for EDs, validated measures used  
- Individuals with AN were consistently found to have higher levels of perfectionism than normal/psychiatric controls  
- Despite some inconsistent findings, there were generally no reductions in elevated levels of perfectionism in individuals who had recovered from AN  
- Limited evidence that premorbid perfectionism predicted development of AN, and perfectionism was a negative indicator of outcome for AN |
| Franco-Paredes et al. (2005) | **Summary:** Review of 22 studies examining perfectionism and EDs, published from 1995 to 2004.  
No detail on inclusion or exclusion criteria provided.  
- Despite some inconsistent findings, individuals with AN were generally found to have significantly higher scores than normal/psychiatric controls on perfectionism  
- In individuals with AN, elevated perfectionism persisted following restoration to normal body weight  
- Perfectionism was associated with general ED characteristics and was a predictor of general ED symptomatology |
| Cassin & von Ranson (2005) | **Summary:** Review of studies assessing association between personality and EDs. 8 studies assessing perfectionism were identified, published from 1994 to 2004.  
No detail on inclusion or exclusion criteria provided.  
- Studies consistently supported the association between perfectionism and AN  
- Perfectionism prospectively predicted onset of AN symptoms  
- Perfectionism was a correlate of AN in females who were acutely unwell and who had recovered from AN |

*Note.* AN—Anorexia Nervosa; BN—Bulimia Nervosa; BED—Binge Eating Disorder; ED—Eating disorder.

**Individual cross-sectional group comparison studies.** Studies published after the above reviews will now be examined, beginning with three cross-sectional studies that compared perfectionism in AN and control groups. They corroborated findings from the reviews. First, Castro-Fornieles et al. (2007) compared an ED group (n = 108) with psychiatric (n = 86) and normal controls (n = 213) on dimensions of perfectionism, including self-oriented, socially-prescribed, and general perfectionism as measured by the Eating Disorder Inventory—2 (EDI-2; Garner, 1991). The AN and BN participants in the ED group scored significantly higher than either control group on self-oriented (F...
= 10.3, \( p < .001 \)) and general perfectionism (\( F = 17.4, \ p < .001 \)), suggesting that perfectionism was more specific to EDs than to other psychiatric disorders.

Bachner-Melman et al.’s (2007) study compared individuals at different stages of illness: current AN (\( n = 17 \)), partially recovered AN (\( n = 107 \)), and recovered AN (\( n = 71 \)), with normal controls (\( n = 242 \)). All three AN groups scored higher than the control group on perfectionism (\( F = 13.39, \ p < .0001 \)), indicating that perfectionism persisted following recovery from the illness. Researchers suggested that perfectionism was either a premorbid vulnerability trait for AN, or could represent a scarring effect after emerging with the onset of AN (Bachner-Melman et al., 2007).

Deas et al.’s (2011) study assessed perfectionism-related schemas using the Young Schema Questionnaire (YSQ; Young & Brown, 2003) in AN patients (\( n = 40 \)), psychiatric controls (\( n = 44 \)), and healthy controls (\( n = 78 \)). They revealed that the AN group had higher scores for perfectionism-related schemas (including unrelenting standards, defectiveness, and failure) compared to psychiatric controls (effect size \( r = .24-.37 \)) and healthy controls (\( r = .27-.52 \)). Only within the AN group all three perfectionistic schemas correlated positively with general eating pathology. Perfectionism was confirmed as elevated in AN compared to psychiatric and normal controls, and the strength of perfectionism-related beliefs was associated with the severity of ED symptoms in AN.

**Individual retrospective studies.** Three retrospective studies explored the association between perfectionism and AN diagnosis, providing further support for conclusions made in the above reviews. First, Wade et al. (2008) examined data from 1002 female twins, of which 4.3% met criteria for lifetime AN. They found that higher levels of perfectionism, measured by concern over mistakes (\( F = 26.37, \ p < .001 \)) and personal standards (\( F = 11.61, \ p = .001 \)), was significantly associated with AN. Looking at twin pairs, the unaffected twins of individuals with AN reported higher perfectionism, specifically personal standards (OR = 1.75, 95% CI [1.21-2.54], \( p = .003 \)), compared to the unaffected twins of controls. Findings provided evidence that perfectionism acted as a risk factor for disordered eating and predisposed individuals to the development of AN (Wade et al., 2008).

Pike et al. (2008) conducted an age-matched, case-control study of risk factors for AN using retrospective assessment with an AN group, a psychiatric control group, and a normal control group (\( n = 50 \) for each group). The AN group reported more
severe perfectionism ($F(2, 98) = 4.50, p = .013$) and higher parental demands ($F(2, 98) = 6.73, p = .002$) than psychiatric controls. Perfectionism ($\beta = .33$) and high parental demands ($\beta = .29$) were the only two variables to predict AN (versus other psychiatric disorders), accounting for a total of 19.0% of the variance ($p < .001$) and correctly classifying 64% of the AN group and 68% of the psychiatric control group. The study provided evidence that perfectionism, whether prescribed by oneself or others, contributed to the development of AN over other psychiatric disorders.

Kim et al. (2010) assessed childhood risk factors for lifetime AN retrospectively using cross-cultural samples, with an AN ($n = 52$) and a healthy control group ($n = 108$) from Korea, and an AN group from the United Kingdom (UK; $n = 42$). The Korean AN group was more likely to report retrospectively assessed childhood perfectionism than healthy controls (OR = 1.13, 95% CI [1.05-1.22], $p = .001$), and there were no differences between the Korean and UK AN groups in perfectionism scores. The view that childhood perfectionism was an important risk factor for AN, not only in Western but also Asian samples, was supported.

**Individual longitudinal study.** Finally, a longitudinal study by Nilsson et al. (2008) assessed the relationship between perfectionism and outcome in patients with adolescent-onset restricting-subtype AN ($n = 68$), utilising a recovered design with patients allocated into four groups: long-term recovered, almost long-term recovered, newly recovered, and non-recovered. The study’s strength was its long follow-up time after initial assessment, at 8 and 16 years. Perfectionism was assessed at both follow-ups, but not at initial assessment. Findings indicated that on an individual level, levels of perfectionism did not change from the first to second follow-up, suggesting that perfectionism was a longstanding trait that remained after recovery. At 16-year follow-up, compared to non-recovered patients, recovered patients had decreased ED and psychiatric symptoms, however levels of perfectionism did not differ ($p = .174$). Interestingly, the long-term recovered group (recovered at both follow-ups with no relapse) was found to have lower levels of perfectionism than the more recently recovered and non-recovered groups at 8-year ($p < .001$) and 16-year ($p = .001$) follow-up, suggesting that higher levels of perfectionism were related to duration of illness. In summary, whilst ED and psychiatric symptoms improved during recovery, levels of perfectionism remained the same. Also, patients with high levels of perfectionism may be at risk for long illness duration, providing further support for conclusions from earlier reviews. However, as perfectionism was not measured at initial assessment,
results could not distinguish scar effects of traits that developed due to prior ED from true premorbid traits (Nilsson et al., 2008).

Table 4.3
Individual Studies Assessing Perfectionism in Anorexia Nervosa

<table>
<thead>
<tr>
<th>Method and Participants</th>
<th>Measures</th>
<th>Relevant Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cross-sectional group comparison studies</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Castro-Fornieles, et al. (2007)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Aim:</strong> To assess perfectionism in EDs compared to psychiatric and normal controls</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Participants:</strong> From patients treated at hospitals and the general population in Spain,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 108 female ED patients (75 AN, 33 BN)</td>
<td><strong>Self-report measures:</strong></td>
<td>• ED group had higher self-oriented and general perfectionism than psychiatric/normal control groups</td>
</tr>
<tr>
<td>• 86 female psychiatric controls (38 anxiety, 32 depressive, 16 adaptive disorders)</td>
<td>CAPS(^1),</td>
<td>• Self-oriented perfectionism was an independent predictor of EDs</td>
</tr>
<tr>
<td>• 213 female healthy controls</td>
<td>EDI-2(^2),</td>
<td></td>
</tr>
<tr>
<td>Participants aged 10-31 years, diagnosed according to DSM-IV (APA, 1994) criteria in a clinical interview.</td>
<td>EAT(^3)</td>
<td></td>
</tr>
<tr>
<td><strong>Method:</strong> Groups were compared on self-report measures.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Bachner-Melman et al. (2007)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Aim:</strong> To examine the relationship between AN, recovery status, and personality</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Participants:</strong> From an Israeli community, mostly students,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 17 with AN</td>
<td><strong>Self-report measures:</strong></td>
<td>• All three AN groups scored higher than control group on perfectionism</td>
</tr>
<tr>
<td>Mean age = 23.0 years (SD = 2.9), mean BMI = 16.8 kg/m(^2) (SD = 1.0)</td>
<td>BSI(^4), EAT-26(^3), EDI(^2), OEQ(^6), AMS(^7), CAPS(^1), MO-CI(^8), RSES(^9), SATAQ(^10), TPQ(^11)</td>
<td></td>
</tr>
<tr>
<td>• 107 partially recovered from AN</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean age = 23.0 years (SD = 4.4), mean BMI = 20.0 kg/m(^2) (SD = 2.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 71 recovered from AN</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean age = 23.7 years (SD = 3.4), mean BMI = 21.5 kg/m(^2) (SD = 2.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 242 female controls</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean age = 23.9 years (SD = 2.6), mean BMI = 21.9 kg/m(^2) (SD = 2.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCID(^4) used to establish diagnostic status of ED and control groups.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Method:</strong> Groups were compared on self-report measures.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Cross-sectional group comparison studies

**Method and Participants**

| Aim: To investigate perfectionistic schemas and parental bonding on eating attitudes/behaviours |
| Cross-sectional group comparison studies |
| Deas et al. (2011) |
| **Participants:** From patients treated at hospitals and the community in the UK, |
| • 40 female AN patients (28 inpatients, 12 outpatients) |
| Mean age = 29 years \( (SD = 10.3) \), mean BMI = 17.3 kg/m² \( (SD = 3.0) \) |
| • 44 female psychiatric controls (depressed/anxious) |
| Mean age = 37 years \( (SD = 12.1) \), mean BMI = 28.0 kg/m² \( (SD = 8.7) \) |
| • 78 female healthy controls (university students) |
| Mean age = 20 years \( (SD = 5.1) \), mean BMI = 23.3 kg/m² \( (SD = 4.0) \) |
| Participants diagnosed according to DSM-IV (APA, 1994) criteria. |
| **Method:** Groups were compared on self-report measures. |

| Measures |
| Interview: EDE\(^{12}\) |
| Self-report measures: EDE-Q\(^{12}\), PBI\(^{13}\), YSQ\(^{14}\) |

**Relevant Outcomes**

- AN group had higher scores than both control groups in schemas related to perfectionism (unrelenting standards, defectiveness, failure)
- In AN group, all three schemas related to perfectionism correlated positively with EDE scores. For both control groups, only defectiveness and failure were weakly correlated with EDE scores

### Retrospective studies

**Method and Participants**

| Aim: To assess traits associated with AN/share transmitted liabilities with AN, from retrospectively assessed AN diagnoses |
| Retrospective studies |
| Wade et al. (2008) |
| **Participants:** From the Australian Twin Registry, |
| • 1002 female twins |
| Aged 29 to 40 years, mean age = 35 years \( (SD = 2.1) \) |
| Participants diagnosed according to DSM-IV (APA, 1994) criteria. 43 participants (4.3%) met criteria for lifetime AN (amenorrhea not required). |
| **Method:** Within-person and cross-twin associations examined links between AN status and trait measures. |

| Measures |
| Interview: EDE\(^{12}\) |
| Self-report measures: EPQ\(^{15}\), TPQ\(^{11}\), ISM\(^{16}\), BIS\(^{17}\), MPS\(^{18}\) |

**Relevant Outcomes**

- Higher perfectionism (concern over mistakes, personal standards) was significantly associated with AN
- For pairs of twins with Twin 1 with AN vs. Twin 1 without AN, Twin 2 reported higher perfectionism (personal standards) in the former

### Pike et al. (2008)

**Method and Participants**

| Aim: To assess potential risk correlates for AN retrospectively |
| **Participants:** From the community in US, |
| • 50 females with AN |
| Mean age = 26.7 years \( (SD = 6.2) \), mean BMI = 15.0 kg/m² \( (SD = 2.0) \) |
| • 50 female psychiatric controls |
| Mean age = 27.0 years \( (SD = 6.1) \), mean BMI = 25.0 kg/m² \( (SD = 6.3) \) |
| • 50 healthy female controls |
| Mean age = 26.6 years \( (SD = 5.5) \), mean BMI = 23.2 kg/m² \( (SD = 4.6) \) |
| SCID\(^{4}\) and EDE\(^{12}\) used to establish diagnostic status. |
| **Method:** Case-control design used. Group comparisons and prediction of diagnosis assessed. |

**Relevant Outcomes**

- AN group reported more severe perfectionism than psychiatric controls
- High parental demands and perfectionism predicted AN vs. other psychiatric disorders
### Table 4.3 (Continued)

<table>
<thead>
<tr>
<th>Method and Participants</th>
<th>Measures</th>
<th>Relevant Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Retrospective studies</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aim: To assess childhood risk factors for AN retrospectively</td>
<td><strong>Self-report measure:</strong> CRFQ&lt;sup&gt;20&lt;/sup&gt;</td>
<td>• The Korean AN group was more likely to report perfectionism in childhood than the healthy control group&lt;br&gt;• There were no differences between the Korean and UK AN groups in childhood risk factors</td>
</tr>
<tr>
<td>Participants: From hospitals and the community in Korea and the UK,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 52 females with lifetime AN from Korea&lt;br&gt;Mean age = 23.2 years (&lt;i&gt;SD&lt;/i&gt; = 6.3), mean BMI = 16.6 kg/m&lt;sup&gt;2&lt;/sup&gt; (&lt;i&gt;SD&lt;/i&gt; = 2.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 42 females with lifetime AN from the UK&lt;br&gt;Mean age = 24.1 years (&lt;i&gt;SD&lt;/i&gt; = 6.4), mean BMI = 17.8 kg/m&lt;sup&gt;2&lt;/sup&gt; (&lt;i&gt;SD&lt;/i&gt; = 3.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 108 healthy female controls from Korea&lt;br&gt;Mean age = 22.3 years (&lt;i&gt;SD&lt;/i&gt; = 3.6), mean BMI = 20.5 kg/m&lt;sup&gt;2&lt;/sup&gt; (&lt;i&gt;SD&lt;/i&gt; = 2.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDE&lt;sup&gt;12&lt;/sup&gt; and EDE-Q&lt;sup&gt;12&lt;/sup&gt; used to establish diagnostic status of ED and control groups. Groups did not differ on age.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Method: Case-control design used. Groups were compared on a self-report measure.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Longitudinal study</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aim: To assess perfectionism and ED/psychiatric symptoms during recovery from adolescent onset AN</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Participants: From a child and adolescent psychiatric unit in Sweden,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 68 previous AN female patients (restricting subtype)&lt;br&gt;Age range: Initial assessment = 10-17 years, 1&lt;sup&gt;st&lt;/sup&gt; follow-up = 18-25 years, 2&lt;sup&gt;nd&lt;/sup&gt; follow-up = 24-36 years</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial AN assessment was made retrospectively from hospital records.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Method: A recovered design was used, with previously ill patients allocated into four groups: long-term recovered, almost long-term recovered, newly recovered, and non-recovered. Participants were studied at follow-up 8 and 16 years after initial assessment.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interview: Semi-structured interview (Nilsson et al., 2008)</td>
<td></td>
<td>• At 2&lt;sup&gt;nd&lt;/sup&gt; follow-up, recovered patients had decreased ED/psychiatric symptoms compared to non-recovered, but levels of perfectionism remained the same&lt;br&gt;• At 2&lt;sup&gt;nd&lt;/sup&gt; follow-up, the long-term recovered group had lower levels of perfectionism than the other three groups</td>
</tr>
<tr>
<td>Self-report measures: EDI&lt;sup&gt;3&lt;/sup&gt;, SCL-90&lt;sup&gt;21&lt;/sup&gt;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other measures: GAF&lt;sup&gt;22&lt;/sup&gt;</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. AN—Anorexia Nervosa; BN—Bulimia Nervosa; ED—Eating disorder; US—United States; UK—United Kingdom.

---

Conclusion. To summarise, there is compelling evidence for the importance of perfectionism in the development and maintenance of AN. Studies consistently reported that AN was characterised by elevated perfectionism compared to psychiatric or normal controls. Perfectionism predicted AN diagnosis and ED symptomatology, had an effect on the maintenance of ED symptoms, remained elevated after recovery from AN, and was a predictor of poor outcome and longer duration of illness. These findings have clinical implications, providing support that intervention should address hypercritical self-views and the unrelenting tendency to set extremely high goals, both in relation to eating and life in general (Deas et al., 2011; Nilsson et al., 2008).

Whilst the literature review yielded strong support for existing theoretical perspectives of the relationship between perfectionism and EDs, researchers strongly recommended that future studies focus on AN-specific symptomatology or populations, as existing studies focused on BN pathology and ED symptom composites (Stice, 2002). To address the lack of studies in AN-specific symptomatology, the present study intends to compare levels of perfectionism in a larger AN sample with dieting and non-dieting comparison groups. Research questions to achieve this aim are described in Section 6 (p. 86-87).
3. **LOW SELF-ESTEEM**

3.1 *Theoretical relationship with Anorexia Nervosa*

Low self-esteem features prominently in theories of etiology and treatment of EDs (e.g., Fairburn et al., 2003; Schmidt & Treasure, 2006; Garner & Bemis, 1982) and is one of the most commonly reported risk factors for developing EDs (Cervera et al., 2003). A number of theorists have highlighted the deep sense of self-doubt, inadequacy, ineffectiveness, and incompetence in AN sufferers (Bruch, 1973, 1982; Fairburn, Shafran, et al., 1999; Vitousek & Manke, 1994; Garner, Vitousek, & Pike, 1997; Garfinkel & Garner, 1982). Clinicians describe this global negative view of oneself as pervasive and unconditional (Fairburn et al., 2003), observing that individuals with AN spent a lot of time worrying about negative self-evaluations (Vitousek & Hollon, 1990). Low self-esteem or a sense of ineffectiveness has also been linked to the AN patients’ experience of not being in control of their behaviours, needs and impulses, feeling instead like they were under the influence of others (Bruch, 1973).

Low self-esteem has been cited as a predisposing, precipitating, and perpetuating factor in the development and maintenance of EDs (Yellowlees, 1997). Disordered eating is proposed to begin in a society and culture that glorify thinness, where dieting becomes a means of self-improvement. AN sufferers arrive at the conclusion that the attainment of a slim figure would alleviate the distress associated with negative self-evaluation and secure approval and respect from others (Garner & Bemis, 1982; Bruch, 1973; Garner & Garfinkel, 1980; Garner et al., 1983; Rieger et al., 2010). Over time, the preoccupation with dieting and weight control becomes a defence against their doubt about self-worth and value, providing temporary markers for competence, accomplishment, pride, control, attractiveness, and self-discipline (Bruch, 1982; Garner et al., 1997), which in turn reflects self-worth (Vitousek, 1996). As AN progresses, weight and shape is proposed to become the predominant gauge for inferring self-worth (Vitousek, 1996; Fairburn et al., 2003). Low self-esteem then maintains AN by creating hopelessness about patients’ capacity to change, resulting in less compliance with treatment and the persistent pursuit of achievement in valued domains (i.e., eating, shape and weight), making change in these areas more difficult (Fairburn et al., 2003). Whilst these proposed clinical observations regarding the role of self-esteem in AN are noteworthy, it is important to determine if they have been confirmed empirically. The next section will review studies examining the association...
between self-esteem and AN. The mechanisms through which low self-esteem are theorised to maintain AN are further discussed in Chapter 5.

3.2 Empirical Evidence for Its Association with Anorexia Nervosa

This section will focus on evidence linking self-esteem to AN, and will exclude studies linking self-esteem with disordered eating behaviours in BN (Cockerham, Stopa, Bell, & Gregg, 2009; Cooper, Clark, & Fairburn, 1993; Garfinkel et al., 1992; Goldfein, Walsh, & Midlarsky, 2000), in combined ED groups without reporting AN-specific outcomes (Cervera et al., 2003; Sassaroli et al., 2008), and in dieters in normal populations (Mintz & Betz, 1988; Shea & Pritchard, 2007; Chen, Fu, Chen, & Wang, 2007; Hoffmeister, Teige-Mocigemba, Blechert, Klauer, & Tuschen-Caffier, 2010; Meijboom, Jansen, Kampman, & Schouten, 1999). It is necessary to focus on AN-specific populations to present an accurate picture of the role of self-esteem in this disorder. Literature that evaluates the direct relationship of self-esteem on AN, either as a risk or maintenance factor, will be examined in this review. Complex multivariate models involving self-esteem and its interaction with other vulnerability factors in predicting AN symptoms will be discussed in Chapter 5.

Description of studies. Because of the mass of literature, the present review will be organised to present findings from an existing review that included studies up to 2002, then individual publications post-2002. The review (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004) is detailed in Table 4.4. Nine individual studies published since 2002 are detailed in Table 4.5, consisting of five cross-sectional group comparison studies (Wilksch & Wade, 2004; Gila, Castro, Gómez, & Toro, 2005; Brytek-Matera, 2007; Paterson, Power, Yellowlees, Park, & Taylor, 2007, Bachner-Melman et al., 2007), one retrospective study (Halvorsen & Heyerdahl, 2006), and three longitudinal prospective studies (Nicholls & Viner, 2009; Halmi et al., 2005; Karpowicz, Skärsäter, Nevonen, 2009).

Limitations of studies. The review and nine studies had similar methodological limitations. Design weaknesses included the predominant use of cross-sectional designs, limiting inferences about the temporal relation of self-esteem to AN (Jacobi et al., 2004). Studies using retrospective assessment of risk factors to determine precedence could have been biased by retrospective recall or inaccurate memory (Jacobi et al., 2004). The small number of studies using longitudinal prospective designs tended to follow participants only through high school, which may have been insufficient as AN
often developed in late adolescence (Jacobi et al., 2004). Additionally, duration of follow-up was relatively brief in some studies (e.g., three months; Karpowicz et al., 2009). Overall, reviewers concluded that whilst self-esteem was assessed in a large number of studies, the majority of research did not address precedence, thus findings on self-esteem could only be interpreted as correlates (Jacobi et al., 2004). A recent individual study addressed these limitations by assessing risk factors of AN in a birth cohort study, following participants from birth to age 30 years (Nicholls & Viner, 2009) and will be discussed in the present review. Reviewers recommended that future research should use prospective longitudinal designs with longer periods of follow-up to clarify the temporal relationship between low self-esteem and AN-specific syndromes, or randomised controlled trials targeting self-esteem in AN (Jacobi et al., 2004).

Another limitation was that none of the studies in the review controlled for participants’ depressive symptomatology. Depression is a prevalent comorbid condition among women with EDs, with rates of depression found to be three times higher in women with EDs compared to controls (Geller et al., 1998). As low self-esteem and depression are highly correlated, it is unclear if self-esteem deficits reported in AN populations were confounded with possible depressive symptomatology (Jacobi et al., 2004). Two individual studies addressed these limitations by controlling for depression whilst examining the relationship between self-esteem and AN (Wilksch & Wade, 2004; Halvorsen & Heyerdahl, 2006) and will be discussed in the present review.

Existing studies also had sampling limitations. Most studies either collapsed across clinical groups to include AN, BN and EDNOS, or focused on BN and BED syndromes, with few studies of AN-specific syndromes (Jacobi et al., 2004), leading reviewers to raise the issue of the specificity of risk factors for the development of AN, which may differ from those of other EDs. There was also almost a complete absence of psychiatric control groups, which raised the issue of specificity of risk factors for the development of AN over psychopathology in general (Jacobi et al., 2004). Further, the selection of healthy control groups was problematic as most studies utilised university students, confounding findings as this population is known to have a higher prevalence of ED symptoms (Jacobi et al., 2004). The majority of research was also in Caucasian female samples, limiting generalisability to other ethnic groups or males (Jacobi et al., 2004; Gila et al., 2005).

Finally, self-report measures were frequently used to assess self-esteem. As self-beliefs are believed to operate outside of conscious awareness, they may not be
accessible using measures that require conscious reflection on one’s own beliefs. In responding to self-report measures, AN populations also have the propensity to answer defensively, tend to be less objective and aware of anxious feelings, and are impacted by the effects of starvation (Gila et al., 2005). Researchers recommended that future research utilise interview format, which has been found to be superior to questionnaires in measuring some self-esteem related variables (Wilksch & Wade, 2004).

Despite these limitations, most findings were consistent, with similar results from studies using cross-sectional, retrospective, and longitudinal designs, in samples from Australia, UK, US, Israel, Norway, Spain, France, and Sweden. The findings will now be discussed.

**Summary of review.** Jacobi et al. (2004) conducted a methodologically rigorous review of cross-sectional and longitudinal studies assessing low self-esteem in EDs, including studies that conceptualised low self-esteem as negative self-concept or a sense of ineffectiveness. Stringent inclusion criteria were utilised, detailed in Table 4.4. Reviewers identified 24 cross-sectional studies and four longitudinal studies. The majority of cross-sectional studies found AN groups reported lower self-esteem compared to controls. One study retrospectively assessed self-concept, finding that negative self-evaluation was elevated before the onset of AN compared to healthy and psychiatric controls (Fairburn, Cooper, et al., 1999). The four longitudinal studies had mixed findings. Two studies did not find significant relationships between self-esteem and subsequent ED symptomatology (Leon, Fulkerson, Perry, & Early-Zald, 1995; Calam & Waller, 1998). Another study reported that low global self-esteem predicted elevated ED symptomatology four years later, with girls in the lowest self-esteem range having an eightfold increased risk for high ED symptomatology compared to those with high self-esteem (Button, Sonugabarke, Davies, & Thompson, 1996). However, the authors pointed out that it was unclear whether low self-esteem predated the onset of eating disturbances, as these had not been assessed and controlled at baseline. The final longitudinal study reported significantly lower self-esteem at baseline for the incidence group that developed an ED two years later (Ghaderi & Scott, 2001). The review provided strong evidence that low self-esteem, negative self-concept, or a sense of ineffectiveness represented correlates of AN. Despite some mixed findings, longitudinal studies suggested that low self-esteem was present prior to the onset of an ED. The reviewers classified low self-esteem as a risk factor of EDs, with potency ranging from low to high (Jacobi et al., 2004).
Table 4.4
Review of Studies Assessing Low Self-Esteem in Anorexia Nervosa

<table>
<thead>
<tr>
<th>Method</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Summary: Review of cross-sectional and longitudinal risk factor research in EDs. 24 cross-sectional &amp; 4 longitudinal studies assessing low self-esteem were identified, published up to 2002.</td>
<td>• 21 cross-sectional studies reported that AN patients exhibited lower self-esteem, more negative self-concept, and higher levels of ineffectiveness compared to control groups. Only 7 studies included psychiatric control groups, with inconsistent results.</td>
</tr>
<tr>
<td>Inclusion criteria: focusing on risk factors for onset of EDs, control groups included, n ≥10 per group, well validated measures used. For cross-sectional studies, samples meeting full DSM ED criteria. For longitudinal studies, follow-up interval ≥1 year.</td>
<td>• 2 longitudinal studies did not find significant relations between self-esteem and subsequent ED symptomatology. One longitudinal study found that low self-esteem predicted elevated ED symptomatology scores 4 years later. Another longitudinal study reported significantly lower self-esteem at baseline assessment for the group that developed an ED 2 years later.</td>
</tr>
</tbody>
</table>

*Note. AN—Anorexia Nervosa; ED—Eating disorder.*

*Individual cross-sectional group comparison studies.* Five cross-sectional studies conducted comparisons of low self-esteem in AN and control groups. Wilksch and Wade (2004) provided noteworthy novel findings by clarifying the role of depression and its relation to self-esteem. They compared an AN group (n = 19), restrained eaters (n = 20), and unrestrained eaters (n = 21). Participants with AN were significantly more depressed than the other two groups ($F(2, 57) = 29.52, p < .001$). Before controlling for depression, the AN group reported lower global self-esteem than restrained eaters, who in turn had lower global self-esteem compared to unrestrained eaters ($F = 51.19, p < .001$). These findings were consistent with conclusions from the review of earlier studies. However, after controlling for depression, these effects disappeared, with no differences found in global self-esteem between AN and restrained eaters. The change in group differences once depression was controlled for suggested that self-esteem was not an AN-specific correlate or maintenance factor, and was instead highly associated with elevated levels of comorbid depression in the AN group. After controlling for depression, both AN and restrained eaters retained significantly lower global self-esteem than unrestrained eaters ($F(2, 55) = 11.08, p < .05$), suggesting that dietary restriction was associated with self-esteem, regardless of whether it was at a clinical level. However, restrained and unrestrained eaters were not screened to rule out the presence of an ED, thus it is unclear whether some restrained eaters met diagnostic criteria for an ED. Also, about a third of eligible restrained and unrestrained participants did not attend the second stage of the study, raising the possibility of responder biases (Wilksch & Wade, 2004). To conclude, this study provided preliminary evidence that
low self-esteem in AN patients was not independently associated with AN, and was dependent on comorbid depression. However, results need to be replicated in future studies, screening out individuals with ED diagnoses from the restrained eaters sample, before more definite conclusions can be made.

The next three cross-sectional studies reported findings that were consistent with findings from the review. Gila et al. (2005) assessed self-esteem in a female adolescent AN group (n = 115), BN group (n = 55), and a comparison group of schoolgirls (n = 359), finding that the AN group had lower overall self-esteem compared to controls (t = 13.9, p < .001). Paterson et al. (2007) assessed self-esteem in an AN inpatient sample (n = 27) and a control group (n = 62), finding that the AN group scored significantly lower than controls on two dimensions of self-esteem (self-competence, t = -5.24, p < .01; self-liking, t = -6.09, p < .01). Paterson et al. also found that in the AN group, self-esteem was negatively correlated with ED symptoms (self-competence dimension, Pearson’s r = -.499, p < .001), and was the single main predictor of eating pathology, accounting for 25.7% of variance. Brytek-Matera (2007) compared female adolescent AN patients (n = 32) and controls (n = 57) on dimensions of self-esteem, finding that the AN group had lower social, familial, and general self-esteem compared to controls (p < .001). These three studies confirm conclusions from the above review of the association between low self-esteem and AN.

Finally, Bachner-Melman et al. (2007) compared individuals at different stages of illness: current AN (n = 17), partially recovered AN (n = 107), and recovered AN (n = 71), with normal controls (n = 242). Both the currently ill and partially recovered AN groups reported lower self-esteem than the recovered AN group, which in turn reported lower self-esteem than controls (F = 40.62, p < .0001), suggesting that self-esteem increased with recovery (Bachner-Melman et al., 2007).

*Individual retrospective study.* Halvorsen and Heyerdahl (2006) conducted a retrospective study examining the association between self-esteem and outcomes of childhood and adolescent-onset AN. They classified former AN patients according to outcomes at follow-up (8.5 ± 3.4 years after initial treatment) into three groups: no ED or normal attitudes towards eating (n = 21), some strain in relation to eating (n = 15), and with ED (n = 8). At follow-up, former AN patients with no ED had higher self-esteem than the two groups with poorer outcome (p < .001), and similar self-esteem to a large-scale normal population sample. This finding is similar to that of Bachner-Melman et al.’s (2007) study, suggesting that self-esteem improved with recovery from
AN. Halvorsen and Heyerdahl further investigated whether low self-esteem was independently associated with ED symptoms or a non-specific feature of depression. Results indicated that ED outcome had a main effect on self-esteem ($F(2,38) = 4.2, p = .023$), whilst the main effect of depression ($F(1, 38) = 0.2, p = .657$) and the interaction effect between ED outcome and depression on self-esteem was non-significant. These findings suggest a strong association between ED symptomatology and low self-esteem, even after controlling for depression, which are contrary to Wilksch and Wade’s (2004) findings that self-esteem in patients with AN was dependent on comorbid depression. Evidently, the confounding role of depression is unclear, and future research should examine its relationship with self-esteem and AN.

Individual longitudinal studies. Three longitudinal studies were identified. The first utilised data from the 1970 British Cohort Study to examine the associations of childhood risk factors for AN, with self-reported lifetime AN at age 30 years (Nicholls & Viner, 2009). This study had strengths in its large sample size ($N = 11,261$), long follow-up period (30 years), specificity in assessing risk factors for AN instead of EDs in general, and the use of a birth cohort, which was not biased by research hypotheses. At age 30 years, 101 participants (0.9%) reported AN. Results indicated that high self-esteem at childhood (10 years old) was found to be a protective factor from AN (OR = 0.3, 95% CI [0.1-0.9], $p = .04$), providing further compelling support for the association between self-esteem and AN.

The second prospective study assessed factors that led to non-acceptance and non-completion of treatment, through conducting a randomised controlled trial of treatment for AN (Halmi et al., 2005). One hundred and twenty-two AN patients were randomly assigned to one year of CBT treatment, drug treatment, or a combination of CBT and drug treatment. Of the total sample, 37% of participants completed treatment. The only predictor of treatment completion was high self-esteem, with 86% of patients with high self-esteem completing treatment (18 of 21). Surprisingly, type of treatment was not a significant predictor of treatment completion.

The third prospective study assessed self-esteem in female AN patients ($N = 38$) before and after three months of inpatient treatment at a specialist ED ward (Karpowicz et al., 2009). Pre- and post-treatment self-report indicated significant improvements in self-esteem following treatment, measured by the Rosenberg Self-Esteem Scale (RSES; Rosenberg, 1965; $t = -3.8, p < .001$) and EDI ineffectiveness subscale ($t = 5.1, p < .001$). These findings support a significant positive change in patients’ self-esteem with
recovery, determined by significant increases in BMI. Although the conclusions were drawn from a short follow-up period and the study was uncontrolled, findings were consistent with two previously discussed studies that reported improvements in self-esteem with recovery from AN (Bachner-Melman et al., 2007; Halvorsen & Heyerdahl, 2006).

Table 4.5

<table>
<thead>
<tr>
<th>Method and Participants</th>
<th>Measures</th>
<th>Relevant Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cross-sectional group comparison studies</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Aim:</strong> To assess differences in shape/weight concerns and self-esteem between women with AN, restrained, and unrestrained eaters, taking into account depression</td>
<td><strong>Self-report measures:</strong> CES-D&lt;sup&gt;2&lt;/sup&gt;, EDE-Q&lt;sup&gt;3&lt;/sup&gt;, SPPA&lt;sup&gt;4&lt;/sup&gt;</td>
<td>• AN group was significantly more depressed than the other two groups</td>
</tr>
<tr>
<td><strong>Participants:</strong> From clinical and non-clinical (university student) Australian samples,</td>
<td></td>
<td>• Before controlling for depression, AN group had lower global self-esteem than restrained eaters, which in turn had lower global self-esteem than unrestrained eaters</td>
</tr>
<tr>
<td>• 19 female AN patients Mean age = 20.8 years ( (SD = 6.4) ), mean BMI = 16.1 kg/m&lt;sup&gt;2&lt;/sup&gt; ( (SD = 1.9) )</td>
<td></td>
<td>• After controlling for depression, there were no differences in self-esteem in AN and restrained eaters. AN and restrained eaters retained significantly lower global self-esteem than unrestrained eaters</td>
</tr>
<tr>
<td>• 20 female restrained eaters Mean age = 19.7 years ( (SD = 1.7) ), mean BMI = 24.5 kg/m&lt;sup&gt;2&lt;/sup&gt; ( (SD = 4.1) )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 21 female unrestrained eaters Mean age = 19.4 years ( (SD = 1.9) ), mean BMI = 20.5 kg/m&lt;sup&gt;2&lt;/sup&gt; ( (SD = 2.6) )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AN group diagnosed according to DSM-IV (APA, 1994) criteria. Non-clinical groups classified according to responses on the DEBQ-R&lt;sup&gt;1&lt;/sup&gt;.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Method:</strong> Groups were compared on self-report measures before and after controlling for depression.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Table 4.5 (Continued)

<table>
<thead>
<tr>
<th>Method and Participants</th>
<th>Measures</th>
<th>Relevant Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cross-sectional group comparison studies</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Gila et al. (2005)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Aim</strong>: To compare adolescents from the general population with ED patients on self-esteem (social and body self-esteem components)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Participants</strong>: From specialised ED services and the general population in Spain,</td>
<td>Self-report measures: SEED$^5$</td>
<td>• No differences between AN and BN patients in overall self-esteem scores</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Compared to the control group, AN and BN patients had lower overall self-esteem</td>
</tr>
<tr>
<td></td>
<td>170 female adolescent ED patients (115 AN, 55 BN) AN mean age = 15.6 years ($SD = 1.4$), BN mean age = 16.2 years ($SD = 1.4$)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>359 female controls (schoolgirls) Mean age = 14.9 years ($SD = 2.2$)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ED group diagnosed according to DSM-IV (APA, 1994) criteria.</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Method</strong>: Groups were compared on self-report measures.</td>
<td></td>
</tr>
</tbody>
</table>

| **Paterson et al. (2007)** | | |
| **Aim**: To compare AN inpatients with controls on self-esteem (self-competence and self-liking components) and problem-solving style | Self-report measures: EAT-26$^6$, SPSI-R$^7$, SLCS-R$^8$ | • AN group scored significantly lower than controls on both components of self-esteem |
| **Participants**: From specialised ED services and universities in the UK, | | • In the AN group, self-competence was negatively correlated with ED symptoms, and was the single main predictor of eating pathology |
| | 27 AN inpatients Mean age = 26.5 years ($SD = 7.9$), mean BMI = 18.0 kg/m$^2$ ($SD = 3.3$) | |
| | 62 female controls (university students) Mean age = 23.1 years ($SD = 7.8$), mean BMI = 23.5 kg/m$^2$ ($SD = 3.5$) | |
| | AN group diagnosed according to DSM-IV (APA, 1994) criteria, with a lenient weight criteria (15 AN participants had BMI > 17.5 kg/m$^2$). Participants with potential ED were excluded from control group. | |
| | **Method**: Groups were compared on self-report measures. | |

| **Brytek-Matera (2007)** | | |
| **Aim**: To compare adolescent AN patients with controls on dimensions of self-esteem, coping strategies, anger expression, and anger control | Self-report measures: SEI$^9$, Brief COPE$^{10}$, SCS$^{11}$ | • AN group had lower social, familial, and general self-esteem compared to controls |
| **Participants**: From a French hospital and university, | | |
| | 32 female AN patients Mean age = 17.7 years ($SD = 1.3$), mean BMI = 16.7 kg/m$^2$ ($SD = 2.3$) | |
| | 57 female healthy controls (university students) Mean age = 20.8 years ($SD = 1.9$), mean BMI = 21.4 kg/m$^2$ ($SD = 2.8$) | |
| | No detail on procedure for diagnosis or inclusion or exclusion criteria provided. | |
| | **Method**: Groups were compared on self-report measures. | |
Table 4.5 (Continued)

<table>
<thead>
<tr>
<th>Cross-sectional group comparison studies</th>
<th>Participants</th>
<th>Measures</th>
<th>Relevant Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aim:</strong> To examine the relationship between AN, recovery status, and personality</td>
<td>From an Israeli community, mostly students,</td>
<td><strong>Self-report measures:</strong> BSI, EAT-26, EDI, OEQ, AMS, CAPS, MO-CI, RSES, SATAQ, TPQ</td>
<td>No differences between currently ill and partially recovering AN groups. However, both these groups reported lower self-esteem than the recovered AN group, which in turn had lower self-esteem than controls</td>
</tr>
<tr>
<td><strong>Participants:</strong></td>
<td>17 with AN</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean age = 23.0 years (SD = 2.9), mean BMI = 16.8 kg/m² (SD = 1.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>107 partially recovered from AN</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean age = 23.0 years (SD = 4.4), mean BMI = 20.0 kg/m² (SD = 2.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>71 recovered from AN</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean age = 23.7 years (SD = 3.4), mean BMI = 21.5 kg/m² (SD = 2.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>242 female controls</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean age = 23.9 years (SD = 2.6), mean BMI = 21.9 kg/m² (SD = 2.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>SCID-IV used to establish diagnostic status of ED and control groups.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Method:</strong></td>
<td>Groups were compared on self-report measures.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Retrospective studies</th>
<th>Participants: 44 former female AN patients from a Norwegian child psychiatry unit, at follow-up,</th>
<th><strong>Interviews:</strong> EDE, MINI, OCS</th>
<th>At follow-up, former AN patients with no ED (normal eating attitudes) had higher self-esteem than the two groups with poorer outcome, and similar self-esteem to the normal population sample</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aim:</strong> To compare self-esteem, personality, and life satisfaction in former patients with different outcomes of childhood/adolescent-onset AN</td>
<td></td>
<td></td>
<td>ED outcome had a main effect on self-esteem, whilst main effect of depression was non-significant. There was no significant interaction between ED outcome and depression on self-esteem</td>
</tr>
<tr>
<td><strong>Participants:</strong></td>
<td>21 with normal attitudes toward eating</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>15 with some strain in relation to eating</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>8 with ED</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean age = 23.5 years (SD = 3.5), mean BMI = 20.7 kg/m² (SD = 2.6). Former AN diagnosed according to DSM-IV (APA, 1994) criteria. Outcome groups determined according to EDE and body weight.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Method:</strong> Former patients were assessed 8.5 ± 3.4 years after the beginning of treatment for childhood/adolescent-onset AN. Responses on interview and self-report measures were compared to 12,043 women from a large-scale population study.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Table 4.5 (Continued)

<table>
<thead>
<tr>
<th>Method and Participants</th>
<th>Measures</th>
<th>Relevant Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Longitudinal studies</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Nicholls &amp; Viner (2009)</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| **Aim:** To examine how previously identified childhood risk factors predict AN by age 30 years in a prospective birth cohort | **Interview:** Interview to determine lifetime AN (Nicholls & Viner, 2009) | • At age 30 years, 101 participants reported lifetime AN  
• High self-esteem (at 10 years old) was a protective factor |
| **Participants:** From the 1970 British Cohort Study,  
• 11,261 participants | **Self-report measures:** SDS<sup>24</sup>, Rutter Malaise Inventory<sup>25</sup> | |
| Lifetime AN was self-reported. | **Method:** Data collected at birth, 5, 10, and 30 years was examined. | |
| **Karpowicz et al. (2009)** |          |                   |
| **Aim:** To assess factors leading to non-acceptance and non-completion of treatment for AN, through a randomised controlled trial | **Interviews:** OCS<sup>13</sup>, EDE<sup>1</sup>, EDS<sup>26</sup> | • Of the total sample, 37% were treatment completers  
• Type of treatment was not a significant predictor of treatment completion  
• The only predictor of treatment completion was high self-esteem |
| **Participants:** From three US hospitals,  
• 122 AN patients  
Aged 14-50 years, mean BMI = 17.8 kg/m<sup>2</sup>  
SCID-III-R<sup>12</sup> used to establish diagnostic status. | **Self-report measures:** SAS<sup>27</sup>, MPQ<sup>28</sup>, BDI<sup>29</sup>, TFEQ<sup>30</sup>, Side Effects Inventory<sup>31</sup>, RSES<sup>15</sup> | |
| Method: Participants were randomly assigned to one treatment for 1 year: CBT, drug therapy, or CBT + drug therapy. Assessments were administered at baseline and 24, 36, and 52 weeks follow-up. Participants who completed 1 year program were classified as “treatment completers”. | **Self-report measures:** RSES<sup>15</sup>, EDI-2<sup>14</sup> | • Compared to before treatment, self-esteem improved significantly after treatment, measured by both the RSES and EDI-Ineffectiveness subscale |
| **Karpowicz et al. (2009)** |          |                   |
| **Aim:** To assess self-esteem in AN patients before and after treatment at a specialist ED ward | **Self-report measures:** RSES<sup>15</sup>, EDI-2<sup>14</sup> | |
| **Participants:** From a Swedish specialist ED ward,  
• 38 female AN patients  
Aged 17-25 years, mean age = 20.1 years  
AN status determined according to DSM-IV (APA, 1994) criteria. | **Method:** Self-report measures taken before and after three months of inpatient treatment (with cognitive approach) from a multidisciplinary team, with weight gain being the objective of treatment. | |

Note. AN—Anorexia Nervosa; BN—Bulimia Nervosa; ED—Eating disorder; CBT—Cognitive behavioural therapy; US—United States; UK—United Kingdom.

---

2. Centre for Epidemiological Studies Depression Scale (CES-D; Radloff, 1997)
3. Eating Disorder Examination (EDE; Fairburn & Cooper, 1993; EDE-Q; Fairburn & Beglin, 1994)
4. Self-Perception Profile for Adolescents (SPPA; Harter, 1986)
5. Self-Esteem in Eating Disorders Questionnaire (SEED; Gila et al., 2005)
6. Eating Attitudes Test (EAT; Garner & Garfinkel, 1979; EAT-26; Garner et al., 1982)
7. Social Problem Solving Inventory—Revised (SPSI-R; D’Zurilla & Maydeu-Oliveres, 1995)
8. Self-Liking/Competence Scale—Revised (SLCS-R; Tafarodi & Swann, 2001)
<table>
<thead>
<tr>
<th>Test</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self-Esteem Inventory (SEI; Coopersmith, 1984)</td>
<td>9</td>
</tr>
<tr>
<td>Brief COPE (Carver, 1997)</td>
<td>10</td>
</tr>
<tr>
<td>Self-Expression Control Scale (SCS; Van Elderen, Maes, Komproe, &amp; Van Der Kamp, 1997)</td>
<td>11</td>
</tr>
<tr>
<td>Structured Clinical Interview for DSM Disorders (SCID-IV; First et al., 1995; SCID-III-R; Spitzer et al., 1992)</td>
<td>12</td>
</tr>
<tr>
<td>Brief Symptom Inventory (BSI; Derogatis, 1993)</td>
<td>13</td>
</tr>
<tr>
<td>Eating Disorder Inventory (EDI; Garner &amp; Olmsted, 1984; EDI-2; Garner, 1991)</td>
<td>14</td>
</tr>
<tr>
<td>Obligatory Exercise Questionnaire (OEQ; Steffen &amp; Brehm, 1999)</td>
<td>15</td>
</tr>
<tr>
<td>Achievement Motivation Scale (AMS; Nygard &amp; Gjesme, 1973)</td>
<td>16</td>
</tr>
<tr>
<td>Maudsley Obsessive-Compulsive Inventory (MO-CI; Hodgson &amp; Rachman, 1977)</td>
<td>17</td>
</tr>
<tr>
<td>Rosenberg Self-Esteem Scale (RSES; Rosenberg, 1965)</td>
<td>18</td>
</tr>
<tr>
<td>Sociocultural Attitudes Towards Appearance Questionnaire (SATAQ; Heinberg et al., 1995)</td>
<td>19</td>
</tr>
<tr>
<td>Tridimensional Personality Questionnaire (TPQ; Cloninger, 1987)</td>
<td>20</td>
</tr>
<tr>
<td>Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998)</td>
<td>21</td>
</tr>
<tr>
<td>Yale-Brown Obsessive-Compulsive Scale (OCS; Goodman et al., 1989)</td>
<td>22</td>
</tr>
<tr>
<td>Temperament and Character Inventory (TCI; Cloninger et al., 1994)</td>
<td>23</td>
</tr>
<tr>
<td>Social Development Scale (SDS; Nicholls &amp; Viner, 2009)</td>
<td>24</td>
</tr>
<tr>
<td>Rutter Malaise Inventory (Rutter, Tizard, &amp; Whitmore, 1970)</td>
<td>25</td>
</tr>
<tr>
<td>Yale-Brown-Cornell Eating Disorder Scale (EDS; Mazure, Halmi, Sunday, Romano, &amp; Einhorn, 1994)</td>
<td>26</td>
</tr>
<tr>
<td>Social Adjustment Scale (SAS; Weissman &amp; Bothwell, 1976)</td>
<td>27</td>
</tr>
<tr>
<td>Multidimensional Personality Questionnaire (MPQ; Tellegen &amp; Waller, 1994)</td>
<td>28</td>
</tr>
<tr>
<td>Beck Depression Inventory (BDI; Beck et al., 1961)</td>
<td>29</td>
</tr>
<tr>
<td>Three Factor Eating Questionnaire (TFEQ; Stunkard &amp; Messick, 1985)</td>
<td>30</td>
</tr>
<tr>
<td>Side Effects Inventory (Levine &amp; Schooler, 1983)</td>
<td>31</td>
</tr>
</tbody>
</table>

**Conclusion.** To summarise, there is convincing evidence for the role of self-esteem in the development and maintenance of AN. Studies consistently reported that individuals with AN had lower self-esteem compared to controls, and recovery from AN was characterised by improvements in self-esteem. On the other hand, high self-esteem was associated with higher levels of treatment completion and being a protective factor for AN. These studies have clinical implications, highlighting the importance of targeting therapy at improving self-esteem, self-efficacy (Paterson et al., 2007), self-worth related to body issues (Gila et al., 2005), and self-esteem related to family and social domains (Brytek-Matera, 2007). Different treatment protocols are also needed to resolve non-acceptance of treatment and dropout in patients with baseline low self-esteem (Halmi et al., 2005), and attention should be paid to ED prevention interventions targeting self-esteem (Nicholls & Viner, 2009).

Overall, whilst this literature review has yielded substantial support for existing theoretical perspectives of the relationship between self-esteem and AN, due to some inconsistent findings, some issues remain to be clarified through the replication of
existing studies. On the subject of the specificity of the role of self-esteem in AN, only two studies controlled for participants’ depressive symptomatology, with conflicting findings. One study found that low self-esteem was a non-specific feature of comorbid depression in AN, with no differences in self-esteem between the AN and restrained eater groups after controlling for depression (Wilksch & Wade, 2004). However, the restrained eaters in the study were not screened to exclude individuals with EDs, potentially confounding findings. Another study found a strong association between low self-esteem and AN even after controlling for depression (Halvorsen & Heyerdahl, 2006). Future research should seek to clarify the confounding role of depression whilst screening individuals with EDs from the restrained eaters sample. Further, to address the limitations related to sampling in existing studies (e.g., neglect of AN-specific syndromes, collapsing across ED diagnoses to form clinical groups, using only university students for control groups), future research should also seek to clarify the role of self-esteem in AN populations and utilise control groups that are not limited to university students (Jacobi et al., 2004). To address these limitations, the present study intends to compare levels of self-esteem in a larger AN sample with dieting and non-dieting comparison groups from the community. Comparison samples will not be restricted to university students, and will exclude individuals with a current or previous ED. Additionally, to clarify existing inconsistencies in the literature regarding the effects of depression, depression will be controlled for. Research questions to achieve these aims will be determined in Section 6 (p. 86-87).

4. CONTROL-RELATED COGNITIONS

4.1 THEORETICAL RELATIONSHIP WITH ANOREXIA NERVOSA

AN has often been defined as a “disorder of control” (Katzman & Lee, 1997). Instead of being primarily about a problem with eating, theorists have suggested that AN represents a secondary adaptation to more fundamental psychological difficulties, with control-related issues being at the heart of the disorder (Waller, 1998). The disorder has been proposed to arise from a lack of control over one’s life and emotions (Thompson & Sherman, 1989), an internal struggle for self-control (Lawrence, 1979), a battle for control taking place in family contexts between family members (Selvini-Palazzoli, 1974), an attempt to prevent and gain control over physical pubertal maturation (Crisp, 1980), a struggle for control to gain a sense of autonomy and identity in relation to others (Bruch, 1973), an assertion of control in the face of fear and
powerlessness due to socio-political control over women (Orbach, 1978), and more recently, an extreme need for self-control (Fairburn, Shafran, et al., 1999). The issue of control has been considered within different theoretical accounts of AN, although it may have varying meanings between these accounts. For an extended discussion of the theoretical accounts of AN involving psychological control, please refer to Surgenor et al. (2002).

In the papers of original ED theorists, one control-related variable that was frequently considered was the general need for control (Bruch, 1973; Crisp, 1980; Garfinkel & Garner, 1982; Selvini-Palazzoli, 1974; Button, 1985). More recently, the individual’s sense of control being dependent on eating and weight has been implicated in the maintenance of AN (Fairburn, Shafran, et al., 1999). Both these control-related concepts are cognitive and distinct from actual behavioural control, and will now be briefly described.

The need for control, or compulsion and desire to control, has been defined as the obsessive facet of control in EDs. Slade (1982) first highlighted its importance, proposing that it was crucial in the development and maintenance of AN. Slade suggested that in the context of dissatisfaction with life, individuals with AN with perfectionistic tendencies perceived little or no control over their lives. Hence, they responded with a pathological and intrinsic need for control over all life domains. Other theorists proposed that with successful dietary restriction, these individuals then developed a gratifying sense of power by discovering that they could exercise full control over their appetites and body weight compared to other life domains (Bruch, 1973; Selvini-Palazzoli, 1978; Casper & Davis, 1977). Over time, general self-control in AN sufferers then become predicated on the control of eating and weight (Garner & Bemis, 1982). Fairburn and colleagues (1999) elaborated on this concept within a formal cognitive theory of the maintenance of AN (further discussed in Chapter 5, p. 94-96), suggesting that dietary restriction and the resultant weight loss became an important index of self-control in Western societies that valued thinness, with AN individuals’ sense of control becoming dependent on eating/weight. The need for control then became primarily focused on dietary restriction, providing immediate positive reinforcement through feelings of success and an enhanced sense of control over the body and, consequently, life.

Theoretical propositions regarding the general need for control and resultant sense of control becoming dependent on eating and weight have encouraged some
clinicians to recommend control-based treatments for AN (Fairburn, Shafran, et al., 1999; Orsillo & Batten, 2002). However, empirical research underpinning the relationship between control-related factors and AN is scant and inconsistent (Sassaroli et al., 2008; Surgenor, Horn, Plumridge, & Hudson, 2002). At present, we are unclear about the full range and experience of control-related issues in individuals with AN, the meanings ascribed to such control, the relationship between control and specific symptoms, and the types of control that are amenable to treatment (Surgenor et al., 2002). To clarify this, the following section will review the limited existing research, focusing on two aspects of control most relevant to AN: the general need for control and sense of control dependent on eating/weight. The mechanisms through which these control-related factors are theorised to maintain AN are further discussed in Chapter 5.

4.2 Empirical evidence for its association with Anorexia Nervosa

This review will focus on evidence linking the cognitive factors of general need for control and sense of control dependent on eating/weight to AN. Literature that evaluates the direct relationship of these control-related factors with AN, either as a risk or maintenance factor, will be examined. Complex multivariate models involving control-related factors and their interaction with other vulnerability factors in predicting AN symptoms will be discussed in Chapter 5.

Studies excluded from the review. Control-related cognitions are critical in AN-related mechanisms, therefore studies assessing levels of actual behavioural control (e.g., impulse control, delay of gratification) will be excluded (Heilbrun & Bloomfield, 1986; Birgegård, Björck, Norring, Sohlberg, & Clinton, 2009; Mizes, 1992; McLaughlin, Karp, & Herzog, 1985; Strober, 1980). In order to present an accurate picture of AN-specific mechanisms, studies linking control with disordered eating behaviours in non-AN populations will also be excluded (King, 1989; Shearin, Russ, Hull, Clarkin, & Smith, 1993), as will studies collapsing across ED groups without reporting AN-specific results (Sassaroli et al., 2008; Shapiro, Blinder, Hagman, & Pituck, 1993; Waller, 1998; Waller & Hodgson, 1996). Another study was excluded because it was written from a social theory perspective and was largely descriptive, providing no detail on statistical analyses (Dignon, Beardsmore, Spain, & Kuan, 2006). A final study was excluded because of an inadequate number of participants (n = 5) in the AN and control groups (Burn, 1987).
Description of included studies. Three cross-sectional studies were identified and summarised in Table 4.6. Two studies assessed the general need for control (Tiggemann & Raven, 1998; Lee, Chan, Kwok, & Hsu, 2005), and another found evidence for the sense of control dependent on eating/weight (Serpell, Treasure, Teasdale, & Sullivan, 1998).

Limitations of studies. Notably, there was a dearth in studies assessing the association of both control-related factors to AN. Design weaknesses included the overreliance on cross-sectional designs, precluding etiological inferences about cause and effect, making it impossible to determine whether control-related issues preceded the onset of AN or were a product of AN. Prospective longitudinal and experimental research is needed to provide a clearer picture of the general developmental trajectory of control-related cognitions in AN, and of the effect of specific treatments targeting control-related factors.

Existing studies also had sampling limitations, using only clinical samples from treatment settings, hence findings may not generalise to less severely unwell or non-treatment-seeking individuals with AN. Further, in Serpell et al.’s (1999) study, data was collected during therapeutic sessions, influencing issues which patients chose to raise or avoid. Finally, whilst Tiggemann and Raven (1998) compared the AN group with healthy controls, the lack of a psychiatric control group raised the issue of the specificity of the control-related cognitions to only AN, or to psychopathology in general. Despite these limitations, existing studies provided preliminary support for the importance of control-related cognitions in AN. The findings will now be discussed.

Studies relevant to the general need for control. Tiggemann and Raven (1998) assessed issues of control, comparing an ED group (AN: n = 28, BN: n = 24) and female controls (n = 57). The general need for control measure in this study included the need for control over life and others, with no items directly related to food and weight. No differences were found between AN and BN participants on any measure. Surprisingly, the ED group reported a lower general need for control compared to the control group (t(106) = 6.52, p < .001). Further, the need for control was not significantly correlated with ED symptoms (r = .15, p > .01). These results were contrary to the excessive general need for control often described in individuals with AN. Authors suggested that in AN patients, the need for control was possibly only focused on food and weight, with a lack of interest in controlling others or life in
general (Tiggemann & Raven, 1998). There is an urgent need for replication of this study in larger samples.

Lee et al. (2005) utilised a retrospective design to examine the relationship between various domains of control including the general desire for control, and outcome in AN patients. Seventy-four AN patients were assessed an average of 9 years after onset of illness, and classified into good (62.2%), intermediate (32.4%), and poor (5.4%) outcome groups. Patients with good outcome had the least general desire for control ($F(2,71) = 4.36, p = .016$), suggesting that a low need for control could be an indicator of good outcome in AN. However, it should be noted that the DSM-IV (APA, 1994) diagnostic criterion of fear of gaining weight was not required for inclusion in this study. The 18 participants who did not meet this criterion had significantly lower need for control compared to the participants who met full AN criteria ($t(72) = -2.18, p = .033$), confounding interpretations of predictors of “good” outcome, as these patients were less symptomatic initially. This study needs to be replicated in a full-syndrome AN sample before more conclusive interpretations can be made.

**Study relevant to the sense of control being dependent on eating/weight.** Serpell et al. (1999) conducted an exploratory qualitative study to examine attitudes maintaining AN in a sample of AN patients ($N = 18$). Participants were asked to write letters to their AN, and responses were coded into ten key themes using the grounded theory-based approach (Glaser & Strauss, 1967). The theme of “control”, coded when AN appeared to provide a sense of control, willpower, or structure to the lives of patients, was the second most commonly described theme. Researchers suggested that the sense of control gained from AN was viewed positively and highly valued, possibly helping to reduce feelings of ineffectiveness (Serpell et al., 1999). Results from this study suggest that sense of control dependent on eating/weight was an important factor in the maintenance of AN. Due to its exploratory nature, findings need to be confirmed using quantitative methods in larger sample sizes, using a range of data collection methods including self-report questionnaires, in-depth interviews, or focus groups.
Table 4.6

<table>
<thead>
<tr>
<th>Method and Participants</th>
<th>Measures</th>
<th>Relevant Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Studies examining the general need for control</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| **Tiggemann & Raven (1998)** | **Self-report measures:** ICI¹, DCS², Adapted I-EQ³, EDI⁴ | • No differences between AN and BN participants on all measures  
• ED group reported a lower need for control compared to controls  
• In ED group, need for control was not significantly correlated with ED symptoms |
| **Participants:** From an Australian hospital and community.  
• 52 female ED patients (28 AN, 24 BN)  
  Aged 18-57 years, mean age = 28 years  
• 57 female controls  
  Aged 18-45 years, mean age = 30 years  
ED group diagnosed according to DSM-III-R (APA, 1987) criteria. | **Method:** Groups were compared on self-report measures. | |
| **Lee et al. (2005)** | **Self-report measures:** MROAS⁶, SCI⁷, EDI⁴ | • Patients with good outcome had the least desire for control  
• Fat-phobic patients had significantly stronger desire for control compared to non-fat phobic patients |
| **Aim:** To examine the relationship between control and outcome of AN patients  
**Participants:** From a hospital in Hong Kong.  
• 74 AN patients (51 restrictive, 23 binge-purge subtype)  
  Mean age = 27.0 years (SD = 6.9)  
SCID⁵ used to establish diagnostic status. Fear of gaining weight was not required for inclusion (56 fat phobic, 18 non-fat phobic). | **Method:** AN patients were assessed on average 9.0 years after onset of illness, and classified into good (62.2%), intermediate (32.4%), and poor (5.4%) outcome. Outcome groups were compared on self-report measures. | |
| **Studies examining sense of control dependent on eating/weight** | | |
| **Serpell et al. (1999)** | Qualitative responses coded into 10 key themes. | • Control was the second most commonly described theme (i.e., AN allowed patients to feel in control, provided willpower, and gave structure to their lives) |
| **Aim:** To examine AN patients’ attitudes towards AN  
**Participants:** From a specialised ED unit in the UK,  
• 18 AN patients (11 restricting, 7 binge-purge subtype)  
  Median age = 24.1 years.  
Patients diagnosed according to DSM-IV (APA, 1994) criteria. | **Method:** AN patients asked to write letters to their AN. Qualitative responses were coded into themes using the grounded theory-based approach (Glaser & Strauss, 1967). | |

**Note.** AN—Anorexia Nervosa; BN—Bulimia Nervosa; ED—Eating disorder; UK—United Kingdom.

---

¹ Internal Control Index (ICI; Duttweiler, 1984)  
² Desirability of Control Scale (DCS; Burger & Cooper, 1979)  
³ Internal-External Questionnaire—Adapted (Adapted I-EQ; Tiggemann & Raven, 1998)  
⁴ Eating Disorder Inventory (EDI; Garner & Olmsted, 1984)  
⁵ Structured Clinical Interview for DSM-III-R Disorders (SCID; Spitzer et al., 1992)  
⁶ Morgan-Russell Outcome Assessment Scale (MROAS; Morgan & Russell, 1975)  
⁷ Shapiro Control Inventory (SCI; Shapiro, 1994)
Conclusion. Whilst many believe that the role of control in ED pathology has been empirically established, in actuality, little consistent evidence has emerged (Stice, 2002). There is a level of contradiction and tentativeness in the empirical literature on the general need for control in AN, providing incomplete and inconsistent support for this relationship. One study found that contrary to clinical observations and proposed theories, AN patients had a lower general need for control compared to controls (Tiggemann & Raven, 1998). Another study found that a low need for control was an indicator of good outcome in AN (Lee et al., 2005). However, due to the methodological limitations of these studies and an overall lack of studies assessing the general need for control, results need to be replicated before definitive conclusions can be made. For the sense of control dependent on eating/weight, only one qualitative study indicated that this was an important factor in the maintenance of AN (Serpell et al., 1999). The findings need to be confirmed using quantitative methods in larger sample sizes.

Unfortunately, despite the incomplete findings from existing studies, many believe that the role of control-related cognitions in AN are empirically established. Although theories surrounding control-related issues have been discussed, empirical validation is needed to pave the way for development of appropriate psychosocial or cognitive interventions (Horesh, Zalsman, & Apter, 2000). To address the overall dearth of research examining control-related cognitions and AN, the present study will compare two control-related variables—the general need for control and sense of control dependent on eating/weight—in a larger AN sample, with dieting and non-dieting comparison groups. Research questions to achieve this aim will be set up in Section 6 (p. 86-87).

5. Dysfunctional Metacognitions

5.1 Theoretical Relationship with Anorexia Nervosa

An important development in recent years has been the identification of a higher level of cognition: the individual’s thoughts and beliefs about their thoughts, or metacognition (Wells, 2000). Metacognition refers to cognitive processes that appraise, monitor, or cope with thoughts and feelings (Wells & Cartwright-Hatton, 2004). Metacognitive theory was originally developed for generalised anxiety disorder and obsessive-compulsive disorder (Wells & Matthews, 1996), and has since been adapted
for understanding depression and other psychological disorders (Wells, 2009). Whilst studies of risk and maintenance factors of AN have mainly focused on cognitive factors, recent interest has turned towards the role of higher level metacognitions.

Many parallels have been drawn between AN and anxiety disorders, in which metacognitive theory has been established, suggesting the possibility of shared underlying risk factors or vulnerabilities. For example, the extreme nature of perfectionism in individuals with AN has often been likened to obsessive-compulsive personality disorder and obsessive-compulsive disorder (for a review, refer to Cassin & von Ranson, 2005). High levels of anxiety are present in AN, including obsessive-compulsive symptoms (Halmi et al., 2005), and obsessive-compulsive disorder has been found to be more prevalent in AN populations compared to non-clinical populations (Speranza et al., 2001). Another similarity between AN and anxiety disorders, specifically generalised anxiety disorder, are the egosyntonic, positive beliefs held about the value and adaptive function of the disorder, which plays an important role in maintaining the illness (Wells & Butler, 1997). Also, similarly to individuals suffering from anxiety disorders, AN sufferers demonstrate failure of self-regulatory abilities to manage anxiety when confronted with feared stimuli (i.e., weight and body image related; Sassaroli et al., 2008), and have high levels of serotonin and anxious need for control (Frank et al., 2002; Kaye et al., 2000; Strober, 2004). Whilst these similarities between AN and anxiety disorders suggest the possible relevance of metacognitive factors to both, theoretical understanding of the relationship between metacognition and AN is underdeveloped. The relevance of metacognitive theory to AN is further discussed in Chapter 5 (p. 123-128).

Clinically, high levels of metacognition have been observed in AN patients. For instance, patients have described the importance and value of their constant worry about eating, holding beliefs that it helps them maintain dietary restriction and achieve weight-loss goals. These metacognitive beliefs appear to maintain distress and problematic behaviours, including mental checking of what and how much has been eaten throughout the day, and repeated weight or body checking behaviours (Cooper et al., 2007). Because interest in the role of metacognitions in AN has only developed recently, theoretical understanding of the relationship is limited. However, some empirical evidence has provided preliminary support for the association between metacognition and AN, and will be reviewed in the following section. The mechanisms
through which dysfunctional metacognitions are theorised to maintain AN are discussed in Chapter 5.

5.2 **Empirical evidence for its association with Anorexia Nervosa**

This section will focus on evidence for the association between metacognition and AN. To present an accurate picture of AN-specific mechanisms, one study linking metacognitions with disordered eating attitudes in a non-clinical sample was excluded (Konstantellou & Reynolds, 2010). Literature that explores the *direct* relationship of metacognition with AN, either as a risk or maintenance factor, will be evaluated.

*Description of studies.* Three cross-sectional studies (Cooper et al., 2007; Woolrich et al., 2008; McDermott & Rushford, 2011) are detailed in Table 4.7.

*Limitations of studies.* The main limitation was an overreliance on cross-sectional designs, which precluded causal interpretations. However, considering that research in this area is in its infancy, cross-sectional designs are a necessary initial step before more costly longitudinal studies and experimental manipulations of metacognitive variables are conducted to provide evidence of causality.

Sampling limitations included the lack of psychiatric control groups, preventing clear conclusions about the specificity of results to AN, as dysfunctional metacognitions could be due to other difficulties such as comorbid mood disorders. In particular, depression and anxiety have been shown to be associated with elevated levels of dysfunctional metacognitions, and this was not controlled for in existing studies. Researchers recommended that future research should investigate the relationship between metacognitions and AN whilst controlling for depression, anxiety (McDermott & Rushford, 2011), and general cognitive deficits due to low weight (Cooper et al., 2007). Two of the three studies (Cooper et al., 2007, Woolrich et al., 2008) had relatively small sample sizes (*n* = 16 and *n* = 15 in AN groups respectively), limiting statistical comparisons by reducing the potential power to detect differences. One study had a majority of binge-purge subtype in the AN group (10 out of 16, Cooper et al., 2007), which has most often been associated with obsessive-compulsive symptoms (Speranza et al., 2001), thus generalisability of these findings to restricting subtype AN is unclear. Researchers recommended that future studies compare the subtypes of AN, and BN with AN, to determine whether differences in the manifestations of ED syndromes were maintained at this highest level of cognition (McDermott & Rushford, 2011). The studies were mainly conducted in Caucasian female samples, limiting the
generalisability of findings to cultural minorities or males. Diagnostic criteria used for inclusion in the studies also differed, with two of the three studies including patients with AN who did not currently meet the weight criterion due to weight gain during treatment (Cooper et al., 2007; McDermott & Rushford, 2011).

One study included participants who had previously received CBT (Cooper et al., 2007), which possibly increased exposure to the identification and exploration of cognitive constructs. However, as standard CBT for AN does not typically include examination of metacognitions, the experience of CBT may not be particularly significant for metacognitions. Future studies should control for treatment history to ensure that contamination of results resulting from treatment do not occur. Another study applied a considerable number of statistical tests to the data set and did not control statistically for the large number of comparisons (Woolrich et al., 2008). Despite these limitations, most findings from the three studies were consistent, with similar results across clinical samples in the UK and Australia. The findings will now be examined.

Cross-sectional group comparison studies. Cooper et al. (2007) conducted a preliminary investigation of metacognition in AN with the Metacognitions Questionnaire—Brief Version (MCQ-30; Wells & Cartwright-Hatton, 2004), comparing females with AN (n = 16), dieting controls (n = 15), and non-dieting controls (n = 17). Results indicated that the AN group had higher scores than both control groups on four (out of five) dimensions of metacognition: negative beliefs about the uncontrollability/danger of thoughts (F(2,45) = 14.0, p = .001), the need to control thoughts (F(2,45) = 20.9, p = .001), cognitive confidence (F(2,45) = 9.4, p = .001), and cognitive self-consciousness (F(2,45) = 6.6, p = .003). On the final dimension, positive metacognitive beliefs, the AN group did not differ from controls (F(2,45) = 1.2, p = .32). The dieting and non-dieting control groups did not differ on any subscale. The findings indicate that AN patients had significantly higher levels of dysfunctional metacognitions compared to dieters and non-dieters.

Woolrich et al. (2008) explored metacognition in AN patients (n = 15), dieting (n = 17), and non-dieting (n = 18) controls through semi-structured interviews. Qualitative data from interview responses were analysed to identify themes, and trends were identified from group comparisons. The AN group believed that their thoughts were more abnormal and uncontrollable compared to both control groups, which largely reported that their thoughts were normal, momentary, and irrational. When comparing positive beliefs about thoughts about eating/weight/shape, the AN group reported more
advantages of these thoughts compared to both control groups, specifically in helping them achieve humility, survival, and protection. The AN group also used more unhelpful metacognitive coping strategies compared to control groups, such as punishing self mentally, suppressing thoughts, and ruminating, and was less successful at using healthy metacognitive coping strategies compared to control groups, such as thought re-appraisal, and attending to the body and others (all \( p < .05 \)). When asked about the function of metacognitive control strategies, half of the AN group (7 out of 15) reported using the strategies to deliberately make themselves feel worse. The findings correspond with Wells’ (2000) proposal that psychological disorders involve increased metacognitive processes, suggesting that dysfunctional metacognitive beliefs and coping strategies play a role in the maintenance of AN, particularly in reinforcing negative self-evaluations. However, findings of this qualitative study of the AN group having more positive beliefs about thoughts about eating/weight/shape compared to the control group were inconsistent with Cooper et al.’s (2007) finding of no differences between AN and controls on the positive metacognitive beliefs subscale of the MCQ-30. It is possible that whilst individuals with AN did not differ from controls on general positive metacognitive beliefs measured by the MCQ-30, they differed on positive metacognitive beliefs about thoughts specific to eating, weight and shape. Future research should clarify these differences.

McDermott and Rushford (2011) compared metacognition in female AN patients \((n = 74)\) and controls \((n = 93)\) using the MCQ-30. This study had strengths in its larger sample sizes and controlling for BMI, to determine whether group differences were due to effects of starvation, which is known to affect cognition. Before controlling for BMI, the AN group had higher scores on all five domains of dysfunctional metacognitions compared to controls (Wilk’s Lamda = 0.622, \( F(5,154) = 18.73, p < .001, \eta^2 = 0.38 \)). However, after BMI was controlled for, significant group differences only remained in four domains, specifically, negative beliefs about the uncontrollability/danger of thoughts \((F(1,93) = 18.1, p < .001)\), need to control thoughts, \((F(1,93) = 13.4, p < .001)\), cognitive confidence \((F(1,93) = 7.8, p = .006)\), and cognitive self-consciousness \((F(1,93) = 4.3, p = .04)\). There were no longer differences between the AN and control group on positive beliefs about worry \((F(1,93) = 13.2, p = .093)\), which is consistent with Cooper et al.’s (2007) findings. Interestingly, effect sizes indicated that the largest differences between groups were in the control-related metacognitive domains, specifically, negative beliefs about the uncontrollability/danger
of thoughts ($\eta^2 = 0.16$) and the need to control thoughts ($\eta^2 = 0.13$), indicating the disturbing extent to which individuals with AN were affected by metacognitive level control-related factors. These findings suggest that dysfunctional metacognitions, specifically from control-related domains, may play a key role in the maintenance of AN, and was not due to starvation effects in this population.

Table 4.7
**Individual Cross-Sectional Studies Assessing Dysfunctional Metacognitions in Anorexia Nervosa**

<table>
<thead>
<tr>
<th>Method and Participants</th>
<th>Measures</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cooper et al. (2007)</strong></td>
<td><strong>Self-report measure:</strong> MCQ-30&lt;sup&gt;1&lt;/sup&gt;</td>
<td>• AN group had higher scores than both control groups on four dimensions of metacognition: negative beliefs about uncontrollability/danger, need to control thoughts, cognitive confidence, and cognitive self-consciousness.</td>
</tr>
<tr>
<td><strong>Participants:</strong> From specialised ED services and hospital/university staff in UK, 16 female AN patients (6 restricting, 10 binge-purge) Mean age = 29.6 years ($SD = 11.4$), mean BMI = 18.4 kg/m&lt;sup&gt;2&lt;/sup&gt; ($SD = 3.1$) 15 female dieting controls Mean age = 34.0 years ($SD = 12.8$), mean BMI = 24.2 kg/m&lt;sup&gt;2&lt;/sup&gt; ($SD = 4.1$) 17 female non-dieting controls Mean age = 26.2 years ($SD = 9.5$), mean BMI = 21.0 kg/m&lt;sup&gt;2&lt;/sup&gt; ($SD = 1.7$) AN group diagnosed according to DSM-IV (APA, 1994) criteria. 8 (of 16) participants in AN group did not meet weight criterion due to weight gain from treatment. <strong>Method:</strong> Groups were compared on self-report measures.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Woolrich et al. (2008)</strong></td>
<td><strong>Interview:</strong> Semi-structured interview exploring explicit metacognitions/metacognitive control strategies (Woolrich et al., 2008) <strong>Self-report measures:</strong> EAT&lt;sup&gt;4&lt;/sup&gt;. DEBQ-R&lt;sup&gt;5&lt;/sup&gt;</td>
<td>• Compared to control groups, AN group believed their thoughts were more abnormal/uncontrollable, more frequently reported advantages of negative thoughts about eating/weight/shape, used more unhelpful metacognitive coping strategies, and was less successful at using healthy metacognitive coping strategies.</td>
</tr>
<tr>
<td><strong>Participants:</strong> From specialised ED services and the community in UK, 15 female AN patients (11 restricting, 4 binge-purge) Mean age = 31.3 years ($SD = 10.3$), mean BMI = 16.2 kg/m&lt;sup&gt;2&lt;/sup&gt; ($SD = 1.7$) 17 female dieting controls Mean age = 29.3 years ($SD = 5.7$), mean BMI = 25.5 kg/m&lt;sup&gt;2&lt;/sup&gt; ($SD = 4.0$) 18 female non-dieting controls Mean age = 29.8 years ($SD = 8.3$), mean BMI = 20.5 kg/m&lt;sup&gt;2&lt;/sup&gt; ($SD = 1.5$) SCID&lt;sup&gt;2&lt;/sup&gt; used to establish diagnostic status. EDE-Q&lt;sup&gt;3&lt;/sup&gt; used to allocate controls to appropriate group. Women who had a history of ED/psychiatric disorders were excluded from control groups. <strong>Method:</strong> Groups were compared on qualitative responses from a semi-structured interview, which was analysed by a content analysis method (Krippendorff, 1980) to identify themes.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

83
### Method and Participants

**Aim:** To compare metacognition in women with and without AN

**Participants:** From specialised ED services and the community in Australia,
- 74 female AN patients
  Mean age = 24.3 years ($SD = 6.5$)
- 93 female controls
  Mean age = 27.3 years ($SD = 7.1$)

AN group diagnosed according to DSM-IV-TR (APA, 2000) criteria. 12% of patients in AN group no longer met weight criterion due to weight gain from treatment. Women who reported a history of ED or had BMI $< 19$ kg/m$^2$ were excluded from control group.

**Method:** Groups were compared on self-report measures.

### Measures

**Self-report measure:**
- MCQ-30$^1$, VA scales$^6$

### Outcomes

- Before controlling for BMI, AN group had higher scores on all dysfunctional metacognitions compared to controls
- After BMI was controlled for, AN groups scored higher than controls on four domains: negative beliefs about uncontrollability/danger, need to control thoughts, low cognitive confidence, and cognitive self-consciousness
- Largest effect sizes were for negative beliefs about uncontrollability/danger and the need to control thoughts

---

**Note.** AN—Anorexia Nervosa; ED—Eating disorder; UK—United Kingdom; BMI—Body mass index.

---

2. Structured Clinical Interview for DSM-IV Disorders (SCID; First et al., 1995)
3. Eating Disorder Examination—Questionnaire version (EDE-Q; Fairburn & Beglin, 1994)
4. Eating Attitudes Test (EAT; Garner & Garfinkel, 1979)
5. Dutch Eating Behaviour Questionnaire—Restraint subscale (DEBQ-R; Van Strien et al., 1986)
6. Visual Analogue Scales for AN Diagnostic Criteria (VA scales; McDermott & Rushford, 2001)

---

**Conclusion.** To summarise, there is preliminary evidence to suggest that the relationship between dysfunctional metacognitions and AN is clinically relevant. Overall, individuals with AN had higher levels of dysfunctional metacognitions compared to controls on the metacognitive domains of negative beliefs about the uncontrollability/danger of thoughts, the need to control thoughts, low cognitive confidence, and cognitive self-consciousness (Cooper et al., 2007; McDermott & Rushford, 2011; Woolrich et al., 2008). The control-related metacognitions had the largest effect sizes, suggesting a possible metacognitive profile for AN (McDermott & Rushford, 2011). Findings on the domain of positive metacognitive beliefs were mixed, with two studies using the MCQ-30 finding no significant differences between the AN and control groups (Cooper et al., 2007; McDermott & Rushford, 2011), whilst a qualitative study found that positive beliefs about eating/weight-related thoughts were higher in AN compared to controls (Woolrich et al., 2008). Future research needs to
clarify this. In line with proposed metacognitive theories for other psychological disorders, individuals with AN were also found to use more unhelpful metacognitive coping strategies, and use dysfunctional metacognitions to intentionally make themselves feel worse, raising the possibility that dysfunctional metacognitions play a role in the maintenance of AN through reinforcing negative self-evaluations (Woolrich et al., 2008). The role of metacognitions in maintaining AN is further examined in Chapter 5.

The findings also have clinical implications, suggesting that the inefficiency of CBT in treatment of AN is possibly due to the fact that it focuses exclusively on the content of thoughts, which may be modified in treatment, but may not be effective in the long term due to crucial underlying metacognitive processes (McDermott & Rushford, 2011). Metacognitive therapy in the treatment of AN could include—education about the impact of negative metacognitions and metacognitive coping strategies, normalisation of the existence of negative thoughts about eating/weight/shape, and involve strategies focussed specifically on metacognitive processing, such as those outlined by Wells (2009) for anxiety and mood disorders. Taken together, findings from the three studies suggest that dysfunctional metacognitions, particularly control-related metacognitions, may have a role in the maintenance of AN. However, future research should utilise quantitative measures and larger sample sizes to increase statistical power (Woolrich et al., 2008). To address specificity, future research should also seek to clarify the role of depression and anxiety, which have been found to be associated with high levels of dysfunctional metacognitions, and the role of general cognitive deficits due to low weight.

Interestingly, whilst two studies included in the review found no differences in levels of metacognition between dieting and non-dieting controls (Cooper et al., 2007; Woolrich et al., 2008), another study in a non-clinical sample found differences in metacognitions between individuals with problematic and normal eating attitudes (Konstantellou & Reynolds, 2010). These findings raise the possibility that non-clinical individuals with disordered eating attitudes have a slightly different metacognitive profile from diagnosed AN patients and normal controls. Future research should explore this.

To address the above issues related to specificity and sampling, the present study intends to compare levels of dysfunctional metacognitions in a relatively large AN sample with dieting and non-dieting comparison groups. Additionally, to clarify the role
of depression, anxiety, and low weight, the association between metacognitions and ED symptomatology will be assessed whilst controlling for depression, anxiety, and BMI. Research questions to achieve this aim are determined in the following section.

6. AIMS AND RESEARCH QUESTIONS

6.1 AIMS

In his review of risk and maintenance factors for eating pathology, Stice (2002) recommended that to empirically investigate risk and maintenance factors, the first critical step was to establish that an association existed between the factor and eating pathology, so as to not unnecessarily conduct costly longitudinal or experimental studies. He argued that this initial step should precede prospective studies, mediation and moderation analyses, and randomised experimental studies or treatment trials. Additionally, Stice stated that more attention should be directed at identifying new risk factors as most established risk factors have modest effect sizes. Hence, the present study aimed to assess the relationships between AN and perfectionism, self-esteem, control-related cognitions, and dysfunctional metacognitions, which have only recently garnered interest in the field. This aim will be achieved through group comparisons of AN, dieting, and non-dieting comparison groups. Individuals with a current or previous ED were excluded from comparison groups to prevent contamination of results.

To add to the literature, the present study will conduct follow-up analyses to explore the possibility that prominence of the above factors are related to transient effects of starvation and low weight, by assessing differences between the groups whilst controlling for BMI. Low weight has been associated with symptoms of rigidity and obsessionality (relevant to perfectionism and control-related cognitions), depression (relevant to low self-esteem), and cognitive deficits (relevant to dysfunctional metacognitions). Further, to address the limitations and inconsistencies in existing literature about potential confounding effects of depression and anxiety, the present study will explore the possibility that in AN, the prominence of low self-esteem is related to high levels of depression, and the prominence of dysfunctional metacognitions is related to high levels of depression and anxiety. To achieve this aim, group differences on self-esteem and dysfunctional metacognitions will be assessed whilst controlling for depression and anxiety. The aims of this chapter are a precursor
for the further testing of more complex maintenance mechanisms of AN, which will be expanded on in Chapter 5.

6.2 Research Question 2

*Research Question 2(a).* Are there differences between the AN, dieting, and non-dieting comparison groups in levels of perfectionism, self-esteem, the two control-related cognitions, and the five dimensions of dysfunctional metacognitions?

Follow-up analyses:

*Research Question 2(b).* Are any group differences on the variables still significant after controlling for BMI?

*Research Question 2(c).* Are any group differences in self-esteem still significant after controlling for depression?

*Research Question 2(d).* Are any group differences in dysfunctional metacognitions still significant after controlling for depression and anxiety?
Chapter 5: Two Cognitive Maintenance Models of Anorexia Nervosa

Chapter 4 explored a number of cognitive factors associated with AN, namely perfectionism, low self-esteem, control-related cognitions, and dysfunctional metacognitions. However, whilst a large proportion of studies examined isolated factors contributing to AN symptoms, the mechanisms through which these factors develop and maintain AN are unclear. There is relatively little in-depth analysis and knowledge of how these factors develop into a coherent, explicit explanation of the maintenance of the disorder. Hence, to further Chapter 4’s findings of the variables relevant in AN, this chapter will assess the processes through which these cognitive factors maintain the key symptom of AN: dietary restraint or behavioural drive for thinness.

To date, whilst treatment in children and adolescents with AN has been found to be effective, there is a scarcity of treatment research in adults with AN and findings for this population have been disappointing, with no clear leaders in treatment efficacy between CBT, focal psychodynamic therapy, cognitive analytical therapy, interpersonal therapy, or behavioural therapy (for reviews see Wilson et al., 2007; Agras & Robinson, 2008; Bulik et al., 2007; Hay et al., 2009). Research has suggested that a large proportion of AN patients develop chronic disabilities, only about a third are in remission after one year of different specialist treatments (Hay et al., 2003; Treasure & Schmidt, 2005), and standardised mortality rates are significantly high at about 5% (Steinhausen, 2002). As Walsh (2004, p. 6) summarised, “this oldest eating disorder remains impressively resistant to a wide range of interventions”. The dearth of effective treatments for AN is unsurprising, considering the lack of a sound theoretical basis on which to develop treatment (Fairburn, 2005). Presently, the mechanisms of etiology underlying the development and maintenance of AN are poorly understood and have not been adequately investigated (Klein & Walsh, 2005). As Bruch (1982, p. 1537) asserted, “therapy and theory stand in reciprocal interaction”. The lack of treatment success with AN highlights the need for a better understanding of the mechanisms of etiology underlying the disorder and a closer examination of factors that maintain AN and make it so difficult to treat (Wilson et al., 2007). To add to the understanding of mechanisms underlying AN, the present study will focus specifically on theories of AN that are (i) cognitively-based, and (ii) focused on maintenance of the disorder. Reasons for this follow.
Why focus on cognitive theory? Various theoretical models for AN have been proposed from different arms of psychology. These include neurobiological perspectives (Connan et al., 2003; Southgate et al., 2005), psychoanalytic theories (Caparrotta & Ghaffari, 2006; Ripa di Meana, 1999), interpersonal theories (Schmidt & Treasure, 2006; Rieger et al., 2010), emotion regulation-based treatment (Safer & Chen, 2011), family therapy (Dare & Eisler, 1997), and cognitive accounts (Garner & Bemis, 1982; Fairburn, Shafran, et al., 1999). However, most of these theories are poorly understood and have not been adequately investigated (Klein & Walsh, 2005). Interestingly, researchers assert that EDs provide one of the strongest indications for CBT, making this bold claim for two reasons. First, EDs are thought to be essentially cognitive disorders, as they have distinctive core features that are cognitive: the evaluation of self-worth based primarily or exclusively on weight and shape (Cooper & Shafran, 2008) and dysfunctional cognitions and beliefs about food, shape and weight (Garner & Bemis, 1982). These cognitive features are often expressed as a strong drive for thinness combined with an intense fear of weight gain (Murphy, Straebler, Cooper, & Fairburn, 2010). Second, CBT has been demonstrated to be effective in the treatment of BN, for which there is widespread acceptance that CBT is the treatment of choice (NICE, 2004). There is also preliminary evidence that CBT is effective in individuals with EDNOS, the most common ED diagnosis (Fairburn et al., 2009). In line with this reasoning, the present study will focus on cognitive theories of AN.

Why focus on maintenance models? Another important consideration is the differential focus of some theoretical accounts on factors that lead to development of AN, and others on factors that lead to maintenance, although this distinction is not always clear (Cooper, 1997). A basic component of CBT is to assess maladaptive beliefs that maintain the disorder and target them for modification in therapy. Whilst the identification of risk factors is important to prevention program design, the understanding of maintenance processes and factors that sustain the symptoms of AN once it has developed are key in advancing treatment interventions (Shafran & de Silva, 2005; Fairburn, Shafran, et al., 1999; Stice, 2002). This is evidenced in the widely successful treatment of BN based on Fairburn’s (1981) cognitive maintenance model (Cooper, 2005), and in the treatment of other psychiatric disorders, including depression (Beck, 1976) and panic disorder (Clark, 1986). Researchers recommend that more theoretical attention should be focused on a non-causal perspective of how individual maintenance factors work together to promote and perpetuate eating pathology that have already been established, influencing severity, presentation, course of illness, and
response to treatment (Bardone-Cone et al., 2007; Stice, 2002). Hence, the present study will focus on maintenance models of AN, or the vicious cycles that sustain AN symptoms.

This study aims to empirically assess two cognitive maintenance mechanisms of AN to further theoretical understanding of the disorder, providing the first critical step towards future advancement of treatment strategies. To achieve this aim, this chapter will provide a review of existing influential “first generation” cognitive theories of AN (Section 1), from which the first testable maintenance mechanism, Model A, will be based on. Then, newer “second generation” cognitive theories and the relevance of metacognitive theory will be discussed (Section 2), from which the second testable maintenance mechanism, Model B, will be adapted. Finally, Models A and B will be described (Section 3) and research questions to empirically test both models in AN and non-eating disordered comparison samples will be set up (Section 4).

1. **Existing Cognitive Theories for Anorexia Nervosa: Background for Model A**

Cognitive theories of AN emphasise the status of beliefs, the valued nature of thinness, and the automatic influence of biased information processing on perceptions, thoughts and behaviours, leading to psychopathology in individuals with AN. This focus on the critical role of cognitions in maintaining AN can be traced back to the early clinical observations of Bruch (1973), a psychoanalyst renowned for her work in EDs. She highlighted the importance of thinking style, for example, the overwhelming sense of a lack of control, ineffectiveness, and low self-worth in AN sufferers, which permeated their thoughts and behaviours. Bruch (1982) proposed that these thinking styles led to relentless attempts to control weight in a bid to create a sense of competence and effectiveness in at least one life domain. Her therapeutic model focused on exploring and disputing erroneous assumptions and beliefs, and had many parallels with the cognitive-behavioural theory for treatment of depression later proposed by Beck (1976).

Bruch’s (1973) ideas were subsequently extended and elaborated on by Garner and Bemis (1982), whose cognitive theory of AN remained the leading account for many years. The theory of AN has since evolved, with a subsequent cognitive account proposed by Fairburn and colleagues (1999). Both these first generation cognitive
accounts have been critical in shaping current thinking and will now be examined. The basis of Model A, adapted from Fairburn and colleagues’ cognitive theory, will be discussed, followed by an examination of relevant empirical evidence for Model A. Whilst it is the intention of this chapter to include all relevant empirical evidence, it will become evident that although elegant theories for AN have been proposed, most theories have been developed primarily on the basis of clinical observations and anecdotal reports (Cooper, 1997). Empirical data to confirm these theories are incomplete and lacking.

1.1 GARNER AND BEMIS’ (1982) COGNITIVE MODEL

Garner and Bemis’ (1982) cognitive model of AN postulated that the core feature of AN was persistent, dysfunctional beliefs about the importance of shape and weight, such as “thinness is physically attractive” and “thinness is of immeasurable worth”. These extreme concerns about shape, weight, and food were thought to be differentiated from non-eating disordered individuals in terms of the content, intensity, and valence of beliefs, which were proposed to drive eating disordered behaviours, maintaining AN. The model was based on Beck’s (1976) cognitive theory and therapy for depression, and consisted of two parts: the development section and maintenance section.

Development section. In keeping with the focus of this chapter on maintenance models, the development section of the model, summarised in Figure 5.1, will not be described in detail. Put succinctly, Garner and Bemis suggested that the drive for thinness in AN developed in individuals with predisposing personality characteristics (e.g., introversion, sensitivity), precipitated by external stressors or anxiety about physical changes in puberty. Dieting then began as a means of self-improvement under sociocultural pressure to be thin.
Maintenance section. More relevant to the present study is the maintenance section of the model, represented in Figure 5.2. Garner and Bemis suggested that AN symptoms were maintained by patterns of maladaptive thinking, stemming from pervasive low self-esteem and dysfunctional beliefs regarding body shape and weight, resulting in the overvalued influence of shape and weight on self-esteem, or self-worth invested in the body. This led to the behavioural symptom of drive for thinness, which was then maintained by reinforcement contingencies. With successful weight loss or emaciation, positive reinforcement ensued, including from internal factors such as increases in a sense of control, power, and success, and external factors such as positive feedback from others (e.g., compliments about weight loss) and care, concern, or attention elicited due to one’s condition. Negative reinforcement included high levels of anxiety upon eating or when weight loss was not sustained, and the psychological sequelae of starvation (e.g., poor concentration, rigidity in thinking, social withdrawal), which further promoted social isolation, leaving patients increasingly vulnerable to their distorted cognitions. Negative reinforcement also drove eating disordered behaviours through the fear of gaining weight. Garner and Bemis suggested that whilst external reinforcers propelled the eating disordered behaviours initially, they became less significant over time as the behaviours became maintained autonomously through complex internal cognitions and cognitive biases. The disorder eventually became ego-syntonic as a result of the vicious cycle of increasing self-worth invested in the body and the on-going belief that thinness is of immeasurable worth for which suffering and continuous hard work is worthwhile.
Concluding issues. Garner and Bemis’ therapy for AN based on this model had similarities to Beck’s (1976) treatment for depression, with some adaptations to develop initial motivation for therapy. It was the first systematic cognitive-behavioural account of AN and remained the leading theory for more than a decade. The comprehensive treatment program developed from this theory had a pervasive influence on clinical practice, and was the most widely used treatment for AN for many years (Cooper, 1997). However, despite this model having been proposed three decades ago, there has been a lack of empirical studies to support its validity. Treatment based on this theory was also criticised for being excessively complex (Fairburn, Shafran, et al., 1999), requiring an extended period of one to two years of weekly therapy to accommodate for the resistance of AN clients and to accomplish weight restoration (Vitousek, 1996). The theory was also critiqued for being too general and inclusive as it encapsulated both development and maintenance factors and did not provide sufficient detail regarding how these factors operated and interacted (Fairburn, Shafran, et al., 1999). This led researchers to suggest that research had to move beyond cognitive content and specific beliefs, and instead examine the processes through which these beliefs developed, became integrated with the individual’s experience of the world, and became autonomous and highly resistant to change (Vitousek & Orimoto, 1993; Vitousek,
1996). It was also recognised that theories needed to be more specific and detailed, for them to be testable through operationalisation of processes (Shafran & de Silva, 2005), and for adequate conceptualisations to be derived to guide the design of targeted interventions (Cooper, 1997).

1.2 Fairburn and Colleagues’ (1999) Cognitive Model

To elucidate the cognitive mechanisms involved in AN, Fairburn and colleagues’ (1999) proposed a model that had an exclusive focus on the organisational structure of maintenance factors of AN, and a higher level of specification that allowed for testable hypotheses. They furthered Garner and Bemis’ (1982) theory by synthesising it with Slade’s (1982) account that the individual’s need for control was at the heart of maintaining the disorder. In AN sufferers, this need became focused primarily on controlling eating, which was proposed to account for many characteristics of AN including shape and weight concerns, egosyntonicity of the disorder, and resistance to change. This theory aimed to develop a simpler approach for treatment, focusing on the issue of self-control. Fairburn and colleagues’ model is schematically represented in Figure 5.3. It is divided into four mechanisms, the driving mechanism and three feedback mechanisms, for clarity.

Driving mechanism: Prominence of the general need for control. Fairburn and colleagues’ proposed that the general need for control over various aspects of life stemmed from the individual’s perfectionism and pervasive low self-esteem, traits that are well recognised in this population (Bruch, 1973; Vitousek & Manke, 1994). The need for control became focused on restricting eating due to the emphasis on thinness in society, and the disorder was maintained for a number of reasons related to the need for control. For example, restrictive eating provided measurable and instant evidence of self-control, compared to other less predictable life domains such as work, which were often beyond one’s control (Slade, 1982). The general need for control was the key maintenance mechanism that perpetuated drive for thinness, hence this section of Fairburn and colleagues’ model will form the basis of Model A. According to Fairburn and colleagues, AN was maintained and became self-perpetuating through three other feedback mechanisms.

Feedback mechanism 1: Restrictive eating enhanced sense of control. Slade (1982) first pointed out that the act of successful dieting immediately enhanced the sense of being in control, thus reinforcing further restriction. Individuals with AN
described feeling “delighted, inspired, triumphant, proud and powerful” (Vitousek & Ewald, 1993, p. 223) with their success, and over time, in addition to being an index of self-control, restrictive eating also became a measure of one’s self-worth, especially in the context of perceived failure or difficulties in other domains of life (Fairburn, Shafran, et al., 1999). The enhanced sense of self-control and self-worth become fundamentally intertwined with dietary restriction, making this behaviour resistant to change, and contributing to the linking of one’s identity with AN, or “being anorexic”.

Feedback mechanism 2: The starvation syndrome encouraged further dietary restriction. Dietary restriction led to weight loss, which eventually resulted in a starvation syndrome that consisted of a variety of physiological and psychological changes. Some of these changes undermined an individual’s sense of being in control (Fairburn, Shafran, et al., 1999). These included intense hunger, which may be perceived as a threat to control over eating; a heightened sense of fullness, which may be perceived as a loss of self-control; and impaired concentration, which may reduce one’s ability to pay attention to events, which are then perceived as unpredictable and uncontrollable (Fairburn, Shafran, et al., 1999). The perceived loss of control then further fuelled dietary restriction, maintaining the disorder.

Feedback mechanism 3: Extreme concerns about shape and weight promoted dietary restriction. Fairburn and colleagues’ proposed that in Western cultures, there was the propensity for individuals to judge their self-worth largely or entirely by shape and weight. Self-worth invested in the body was a key component of Garner and Bemis’ (1982) theory of AN, and is recognised to be a core symptom of the disorder (APA, 2002). Individuals with AN often closely monitored their weight through hypervigilant body checking (e.g., frequent weighing), which resulted in concern about insignificant weight fluctuations. Small drops in weight were viewed as successes, positively reinforcing dietary restriction, and minute increases in weight were interpreted as evidence of poor control, negatively reinforcing dietary restriction. On the other hand, if body checking became intolerable, individuals may switch to body avoidance (e.g., completely avoiding monitoring) instead. This maintained restrictive eating as there were no means to disconfirm shape and weight concerns. The model postulates that both body checking and avoidance were subject to information processing biases due to AN sufferers’ extreme concerns about shape, leading individuals with AN to think that they are failing to control their shape, thus further promoting and maintaining behavioural drive for thinness (Fairburn, Shafran, et al., 1999).
Concluding issues. Fairburn and colleagues’ control-based model was originally developed for the treatment of BN. It allowed for treatment to be more focused and directed at the issue of self-control and self-worth invested in the body, which was proposed to produce secondary changes in other problem areas (Fairburn, Shafran, et al., 1999). In BN populations, this model has received direct empirical support (Fairburn, Peveler, Jones, Hope, & Doll, 1993), as well as indirect support through a large body of clinical trials with consistent findings that CBT based on this theory had a major and lasting impact on BN patients (NICE, 2004). However, as will become evident in Section 1.4 (p. 97-118), there is little direct empirical evidence to substantiate the control-related mechanisms proposed in AN patients, and treatment based on this model does not appear to be effective for AN. The lack of research to support this theory highlights the need for empirical clarification of AN-specific cognitive mechanisms (Wilson, 2005). The present study aims to contribute to this literature through the testing of Model A.
1.3 The basis of Model A

Slade (1982) first suggested that the general need for control was at the heart of maintaining AN, and his theory was furthered through Fairburn and colleagues’ (1999) cognitive model of AN, for which the general need for control was the central driving mechanism of the model, acting together with perfectionism and low self-esteem to perpetuate drive for thinness (refer to Figure 5.3, p. 96). In Fairburn and colleagues model, this was the critical building block on which the other three feedback mechanisms were based. Hence, empirical clarification of this mechanism is a necessary first step towards understanding how AN is maintained. Model A will be based on the Driving Mechanism section of Fairburn and colleagues’ maintenance model of AN.

Another important control-related mechanism was Feedback Mechanism 1, which was built on Slade’s (1982) premise that dieting resulted in an enhanced sense of control, thus reinforcing further drive for thinness. Feedback Mechanism 1, involving the sense of control dependent on eating/weight, will also be included in Model A. Considering the focus of the present study on psychological factors, Feedback Mechanisms 2 and 3, involving biological starvation-related factors and information processing biases, are beyond the scope of the present study. However, the interested reader may refer to Treasure and Szmukler (1995) for a review of starvation-related factors, and Cooper (1997, 2005) or Vitousek (1996) for a review of studies examining information processing biases (e.g., using the Stroop task) in the domains of food, eating, weight and shape. Model A is further described in Section 3.1 (p. 130-133).

1.4 Empirical evidence for cognitive models of Anorexia Nervosa

There is a wealth of existing studies assessing the individual importance of Garner and Bemis’ (1982) and Fairburn and colleagues’ (1999) proposed maintenance factors. Previously studied factors include low self-esteem, perfectionism, control-related cognitions (refer to Chapter 4 for reviews), dysfunctional beliefs about shape and weight (for reviews, refer to Cooper, 1997, 2005), and information processing biases in the domains of food, eating, weight and shape (Cooper, 1997, 2005; Vitousek, 1996; Polivy & Herman, 2002). However, although the above risk or maintenance factors have been examined individually, more complex studies testing the mechanisms or pathways in maintenance models of AN that include more than one variable is lacking. This section will review empirical studies testing mechanisms of maintenance of AN, focusing specifically on research involving perfectionism, low self-esteem, the
general need for control, and sense of control dependent on eating/weight to inform the development of Model A.

First, direct and indirect evidence for mechanisms involving (i) perfectionism, (ii) self-esteem, and the (iii) two control-related factors will be explored, in that order. Direct evidence refers to studies assessing the validity of proposed mechanisms between maintaining factors (e.g., using moderation or mediation analyses). Indirect evidence for the proposed mechanisms will be inferred from trials of interventions targeting the specific mechanisms in question (e.g., trials targeting perfectionism). Then, indirect evidence for (iv) CBT models in general will be assessed through examining the outcome of treatment trials of CBT in AN.

1.4.1 Evidence for mechanisms involving perfectionism

Description of studies. A systematic review of studies of perfectionism in EDs concluded that whilst there is a wealth of evidence testing the individual importance of perfectionism and its existence in people with EDs, there is an absence of tests of more complex models involving perfectionism in the ED literature (Bardone-Cone et al., 2007). For example, there is very limited work testing moderator or mediator models involving perfectionism in interaction with other vulnerabilities in the prediction of ED symptoms or diagnosis. A search of existing literature confirmed this, with only three studies identified and summarised in Table 5.1. The first two provided direct evidence for mechanisms related to perfectionism in EDs (Sassaroli et al., 2008; Davis, 1997). The third study provided preliminary indirect evidence through the effectiveness of CBT targeting perfectionism, in treating individuals with BN (Steele & Wade, 2008).

Limitations of studies. The two studies assessing mechanisms related to perfectionism (Sassaroli et al., 2008; Davis, 1997) were cross-sectional, constraining inferences about the directionality of mechanisms being tested. The studies also had sampling limitations, with Sassaroli et al. (2008) combining across ED categories of AN, BN and controls for regression analyses. The mixed sample of ED and control participants raise concerns about interpretation, as significant interactive effects could have been artefacts of the qualitative differences between both groups, causing spurious correlations. Davis (1997) combined the ED categories of AN, BN, and EDNOS, impeding the interpretation and understanding of AN-specific mechanisms, and did not have a control group, making it unclear whether or not the mechanisms being tested were specific to EDs or generally existed in non-disordered individuals or individuals with other psychiatric disorders.
The third study, a randomised trial, had small group sample sizes ($n = 15$ to $17$ in each condition), resulting in groups that were heterogeneous in demographic and clinical features despite randomisation procedures (Steele & Wade, 2008). The small sample size also limited ability to identify significant differences between groups if they were not of a moderate effect size or larger (Steele & Wade, 2008). As the participants in this trial had BN, findings need to be replicated in AN samples to determine if targeting perfectionism in therapy will result in similar outcomes for AN symptomatology.

Despite these shortcomings, results from the three studies provide some, albeit limited, support for the role of perfectionism in the maintenance of AN, across Italian, Canadian, and Australian samples, using cross-sectional and experimental designs.

**Direct evidence.** Sassaroli et al.’s (2008) study assessed the main and moderation effects of perfectionism, global self-esteem, and perception of low control (over emotional reactions and external threats) on three ED symptoms: drive for thinness, body dissatisfaction, and bulimia. Their study included a sample of $55$ ED patients ($32$ AN, $23$ BN) and $38$ non-eating disordered controls. Only perfectionism had a significant main effect on drive for thinness ($t = 2.152, p = .037$). Whilst perception of low control and self-esteem had no main effects on drive for thinness, they moderated the effect of perfectionism on drive for thinness ($t = -2.098, p = .041$). These findings clarify that the constructs of perfectionism, self-esteem, and control-related factors are tightly interwoven in their relationship to ED symptoms. Interestingly, although the sample included BN patients, there were no significant main or interaction effects of the three variables in predicting the ED symptom of bulimia, suggesting that these variables were specifically relevant to the restrictive subtype of AN. Whilst it is not appropriate to infer causal relationships from correlations and interactions, this study provides preliminary empirical support that drive for thinness may be an attempt to retrieve a personal sense of control and increased self-esteem, via the pursuit of perfection in the narrow domain of eating, weight, and shape (Sassaroli et al., 2008).

Davis (1997) assessed the relationship between perfectionism and a dimension of self-esteem: body esteem, or body image perceptions, in a sample of $123$ female ED patients. In this study, perfectionism was conceptualised into two separate components: normal perfectionism and neurotic perfectionism, of which the latter, setting impossibly high standards whilst experiencing an intense need to avoid failure, is maladaptive and more relevant to the maintenance of AN. Findings suggested that both components of
perfectionism (normal perfectionism, $t = 2.83, p = .0056$; neurotic perfectionism, $t = 2.59, p = .019$), as well as the interaction between them ($t = -3.55, p = .0006$), were statistically significant predictors of body esteem ($R^2 = 0.44$). In analysing the regression plots to understand the interactive influence of both components of perfectionism on body esteem, Davis concluded that at high levels of neurotic perfectionism, normal perfectionism became associated with low body esteem. From these results, Davis proposed that the rigid adherence to self-imposed, typically unrealistic body size aspirations, in conjunction with anxiety about the perceived consequences of failure, resulted in poor body esteem. This provides some support for the complex interactive relationship between perfectionism and self-esteem, specifically in the domain of body esteem.

**Indirect evidence.** Steele and Wade (2008) conducted a randomised trial of CBT for perfectionism in individuals with BN. The participants were randomly allocated to three groups, CBT for perfectionism ($n = 17$), “traditional” CBT for BN ($n = 15$), and placebo (based on mindfulness techniques; $n = 16$). Post-intervention, there were no group differences for rate of treatment completers (Fisher’s probability exact test, $p = .24$), or significant main effects of group or interaction effects on any of the outcome variables, suggesting the three conditions had similar effects. There was a significant main effect of time, with participants significantly improved on 10 of 12 outcome variables post-treatment, including objective and subjective binge episodes, purging (specifically vomiting), excessive exercise, shape/weight concerns, global ED measures, two dimensions of perfectionism (concern over mistakes and personal standards), self-esteem, and depression (all $p < .05$), with effect sizes ranging from medium to large (0.64 to 3.19). Most gains were sustained at 6-month follow-up. Although participants still experienced some clinical symptoms post-intervention and at follow-up, findings suggest a substantial reduction in bingeing and purging episodes and a reduction of ED attitudes in all three treatment conditions. Whilst all treatment conditions were equally effective, suggesting that non-specific effects of therapy could be crucial in BN treatment, this study provides preliminary support that BN symptoms may be alleviated by targeting perfectionism in treatment, highlighting the role of perfectionism in maintaining the disorder. However, further research is required to ascertain the importance of non-specific effects of therapy, and findings need to be replicated in AN samples to determine if the same gains are obtained for AN symptomatology.
Conclusion. The above studies provide preliminary support for mechanisms related to perfectionism in AN. First, the findings suggest that perfectionism has a complex interactive relationship with low self-esteem (Sassaroli et al., 2008; Davis, 1997) and control-related factors (in this case, low perception of control; Sassaroli et al., 2008) in ED patients, but particularly in the restrictive subtype of AN. Second, findings suggest that perfectionism has a main effect on drive for thinness, and this effect is moderated by low self-esteem and control-related factors (Sassaroli et al., 2008). Third, there is evidence that targeting perfectionism in a CBT intervention led to improvement in BN (Steele & Wade, 2008), however this is yet to be confirmed in an AN sample. As Bardone-Cone and colleagues (2007) concluded in their review, largely missing from the literature on perfectionism is the specification of any explanatory mechanism leading from perfectionism to EDs. Whilst these studies provide preliminary support for the role of perfectionism in maintaining ED symptoms, much work remains to address whether perfectionism interacts with other variables to explain AN-specific symptoms (i.e., moderator models), and what mechanisms explain the link between perfectionism and AN symptoms (i.e., mediator models).

Table 5.1
Studies Assessing Mechanisms of Perfectionism

<table>
<thead>
<tr>
<th>Method and Participants</th>
<th>Measures</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Direct evidence (studies testing proposed mechanisms)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Aim:</strong> To assess main and interactive effects of perfectionism (concern over mistakes), self-esteem, and perception of control on ED symptoms of drive for thinness, body dissatisfaction, and bulimia</td>
<td><strong>Self-report measures:</strong> EDI-3², MPS¹, RSES⁴, ACQ³</td>
<td>• Perfectionism had a significant main effect on ED symptoms of drive for thinness and body dissatisfaction, but not bulimia</td>
</tr>
<tr>
<td><strong>Participants:</strong> From Italian specialised ED services &amp; the community.</td>
<td></td>
<td>• Self-esteem had a significant main effect on ED symptom of body dissatisfaction, but not the other two</td>
</tr>
<tr>
<td>• 55 ED patients (32 AN, 23 BN)</td>
<td></td>
<td>• Perception of control had no significant main effect on all three ED symptoms</td>
</tr>
<tr>
<td>Mean age = 32.7 ± 7.8 years. 53 females, 2 males</td>
<td></td>
<td>• There was an interaction between independent variable of perfectionism, and moderators of perception of control and self-esteem, in predicting ED symptoms of drive for thinness and body dissatisfaction, but not bulimia</td>
</tr>
<tr>
<td>• 38 controls</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean age = 30.0 ± 9.3 years. 37 females, 1 male</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCID-IV¹ used to establish diagnostic status of ED and control groups.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Method:</strong> Groups were compared on self-report measures.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Method and Participants</td>
<td>Measures</td>
<td>Outcomes</td>
</tr>
<tr>
<td>-------------------------</td>
<td>----------</td>
<td>----------</td>
</tr>
<tr>
<td><strong>Direct evidence (studies testing proposed mechanisms)</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| **Aim:** To assess the relationship between perfectionism (adaptive and maladaptive dimensions) and self-esteem (specifically body esteem) | **Self-report measures:** | • Neurotic perfectionism, normal perfectionism, and the interaction between the two (neurotic x normal perfectionism) were statistically significant predictors of body esteem  
• At low levels of neurotic perfectionism, normal perfectionism was positively related to body esteem. However, at high levels of neurotic perfectionism, normal perfectionism became inversely related to body esteem |
| **Participants:** From a Canadian hospital, | BES\(^4\), MPS\(^3\), EPQ\(^7\), NPQ\(^8\) | |
| • 123 female ED patients (42 AN, 59 BN, 22 EDNOS)  
Mean age = 27.8 years (SD = 7.6)  
Participants diagnosed according to DSM-III-R (APA, 1987) criteria. | | |
| **Method:** Participants completed self-report measures as part of a larger-scale study. | | |
| **Steele & Wade (2008)** | **Interview:** EDE\(^9\) | **Pre-intervention:**  
• No differences from baseline to pre-treatment (after six-week waitlist period) in all groups  
• No significant main effect of group or interaction effects; three conditions had similar effects  
• Significant main effect of time for 10 (of 12) outcome variables, maintained at 6-month follow-up |
| **Aim:** To assess the effectiveness of CBT for perfectionism in treating BN | **Self-report measures:** RSES\(^4\), DASS\(^10\), FMPS\(^11\) |  
Post-intervention: |
| **Participants:** 48 BN participants (47 female, 1 male) from Australia, | | |
| • 17 CBT for perfectionism group  
Mean age = 24.7 years (SD = 5.5), mean BMI = 21.4 kg/m\(^2\) (SD = 2.4)  
• 15 “traditional” CBT for BN group  
Mean age = 25.7 years (SD = 5.6), mean BMI = 22.5 kg/m\(^2\) (SD = 3.7)  
• 16 placebo group (adapted MBCT treatment)  
Mean age = 27.8 years (SD = 6.4), mean BMI = 25.4 kg/m\(^2\) (SD = 6.1) | | |
| Participants diagnosed according to DSM-IV (APA, 1994) criteria, with lenient criteria for binge/purge frequency and amount of food during binge. | **Methods:** Participants allocated to groups using computer-generated randomisation. Six-week waitlist period before treatment. Treatment consisted of eight individual sessions of manual-based guided self-help over six-week period. Measures taken at baseline (before waitlist period), pre- and post-intervention, and at 6-month follow-up. | |

**Note.** AN—Anorexia Nervosa; BN—Bulimia Nervosa; ED—Eating disorder; EDNOS—Eating Disorder Not Otherwise Specified; CBT—Cognitive behavioural therapy; MCBT—Mindfulness-based cognitive therapy; UK—United Kingdom.

1. Structured Clinical Interview for DSM Disorders (SCID-IV; First et al., 1995; SCID-IV-TR; First, Spitzer, Gibbon, & Williams, 2002)
2. Eating Disorder Inventory (EDI-3; Garner, 2004)
4. Rosenberg Self-Esteem Scale (RSES; Rosenberg, 1965)
1.4.2 Evidence for mechanisms involving self-esteem

Description of studies. Six studies are summarised in Table 5.2. Four provided direct evidence for mechanisms related to self-esteem in AN (Blechert, Ansorge, Beckmann, & Tuschen-Caffier, 2011; Surgenor, Maguire, Russell, & Touyz, 2007; Woolrich, Cooper, & Turner, 2006; Cooper, Todd, & Wells, 1998), and two provided indirect evidence from trials of interventions targeting self-esteem, one of which was in an ED sample (Newns, Bell, & Thomas, 2003) and another in a non-clinical sample of adolescents (O’Dea & Abraham, 2000). A seventh study assessing the relationship between self-esteem and ED behaviours in college women (Hesse-Biber, Marino, & Watts-Roy, 1999) from a sociological perspective was excluded from the literature review as it was largely descriptive and did not provide details or results of statistical analyses.

Limitations of studies. For the four studies providing direct evidence, three used cross-sectional designs (Blechert et al., 2011, Woolrich et al., 2006; Cooper et al., 1998), limiting conclusions about the directionality of mechanisms as it was unclear whether they predated the development of AN or were a consequence of AN. Whilst the remaining study used a prospective design (Surgenor et al., 2007), it only allowed a relatively brief time during which changes in self-esteem and eating disturbance were prospectively observed (mean treatment duration = 50 days, $SD = 38.1$). Coupled with the relatively long duration of illness in that sample (mean age of participants = 22.5 years, $SD = 8.5$; mean age of AN onset = 16.7 years, $SD = 4.9$), cause and effect of the mechanisms involved were also difficult to establish. Sampling limitations included two studies with small sample sizes for the AN and other comparison groups due to the qualitative nature of the studies ($n = 15$, Woolrich et al., 2006; $n = 12$, Cooper et al., 1998), limiting generalisability of findings. Also, none of the studies used a psychiatric control group, making it unclear whether the mechanisms tested were AN-specific or generally existed in individuals with psychiatric disorders.

5 Anxiety Control Questionnaire (ACQ; Rapee, Craske, Brown, & Barlow, 1996)
6 Body Esteem Scale (BES; Franzoi & Shields, 1984)
7 Eysenck Personality Questionnaire-Revised (EPQ; Eysenck & Eysenck, 1991)
8 Neurotic Perfectionism Scale (NPQ; Mitzman, Slade, & Dewey, 1994)
9 Eating Disorder Examination (EDE; Fairburn & Cooper, 1993)
10 Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995)
11 Frost Multidimensional Perfectionism Scale (FMPS; Frost, Marten, Lahart, & Rosenblate, 1990)
For the two studies providing indirect evidence, one used an ED sample (Newns et al., 2003) which included patients with AN, BN, EDNOS, and BED, however did not specify the diagnostic criteria used for inclusion in the study. It should be noted that all participants had BMI >17.5 kg/m$^2$, suggesting that lenient criteria for AN was used. Also, AN patients formed only 13.3% of the heterogeneous group. Further, this study did not provide follow-up after conclusion of the intervention, therefore it is unclear whether effects of the intervention were maintained. The other study providing indirect evidence was conducted in a non-clinical sample of secondary school students (O’Dea & Abraham, 1999), thus needs to be replicated in AN samples to determine if these mechanisms exist, possibly to a stronger degree, in AN populations.

A final methodological limitation of all the studies was the dependence on self-reported self-esteem, with the exception of Blechert et al. (2011) who used an experimental method to assess implicit self-esteem. As self-beliefs may be less accessible or amenable to self-report than other types of cognitions (Woolrich et al., 2006), it is unclear whether there were other important beliefs that were unreported.

Despite these shortcomings, results from all six studies provided support for proposed cognitive maintenance mechanisms related to self-esteem in AN, with similar findings across German, Australian, and UK samples, using interview and self-report measures, cross-sectional and longitudinal designs, and experimental, quantitative and qualitative methods.

**Direct evidence.** Looking first at direct evidence, four studies assessed mechanisms involving self-esteem and AN symptomatology. Blechert et al. (2011) used an experimental design to explore the relationship between self-esteem (in a non-appearance related domain), and shape, weight, and eating concerns with an affective priming procedure (detailed in Table 5.2). They included three groups of participants, 20 AN, 20 BN, and 28 healthy controls. Following the procedure, all measures showed differential affective priming effects for the different groups, consistent with the general idea that self-esteem and shape, weight and eating concerns were linked in EDs but not in controls. Comparing reaction times, affective priming effects were found to be significant in the AN group ($t(19) = 2.65, p = .016, d = 0.19$), but not BN ($t(19) = 1.87, p = .077, d = 0.10$) or healthy controls ($t < 1.0$), suggesting that individuals with AN associated shape, weight and eating concerns with non-appearance related self-esteem to a stronger degree than individuals with BN and healthy individuals. These findings provide support for the excessive influence of shape, weight, and eating behaviours on
self-evaluation in individuals with AN, even in non-appearance related domains of self-esteem. Researchers suggested that in AN, efforts to increase general self-esteem were directed towards shape and weight control, and failure to achieve desired weight or shape could negatively affect general self-esteem in a vicious cycle (Blechert et al., 2011).

Surgenor et al.'s (2007) prospective study of 77 inpatients with AN investigated the relationship between self-esteem and common ED characteristics measured by the EDI-2 (Garner, 1991), at admission and discharge from hospital treatment. At admission, the severity of ED disturbance was significantly associated with self-esteem (self-competence component, $r = -0.73$, $p < .001$; self-liking component, $r = -0.47$, $p < .001$). Interestingly, the study also assessed subscales of the EDI-2 in predicting self-esteem, and found that higher levels of perfectionism ($t = -2.6$, $p = .01$), together with ineffectiveness and interpersonal distrust, were independent predictors of lowered self-esteem (self-competence component, $R^2 = 0.66$, $p < .001$). At discharge, a decreased drive for thinness was the only EDI-2 subscale associated with an increase in self-esteem (self-competence component, $t = -2.24$, $p = .03$; self-liking component, $t = -2.39$, $p = .02$). These findings provide support for the link between self-esteem, perfectionism, and drive for thinness. Specifically, they suggest that high perfectionism predicted low self-esteem, and increased self-esteem was associated with decreased drive for thinness.

Woolrich et al. (2006) assessed the mechanisms related to self-esteem by comparing an AN group ($n = 15$) with two control groups, dieting ($n = 17$) and non-dieting ($n = 18$), using qualitative results from an interview. They reported that failure and defectiveness were two of the three most strongly endorsed negative self-beliefs in the AN group, with at least two-thirds of the AN group endorsing these beliefs. These negative self-beliefs resulted in higher distress ratings for the AN group compared to both control groups ($p < .001$). To cope, participants with AN most frequently reported using behavioural dietary restraint as a strategy (86% of AN group) to reduce the cognitive and emotional distress associated with negative self-beliefs, reporting that this strategy was effective in the short term (Woolrich et al., 2006). The findings suggest a functional link between drive for thinness and low self-esteem, as measured by negative self-beliefs.

Cooper et al. (1998) conducted a similar study, utilising a qualitative interview to elicit the assumptions linking negative self-beliefs to disturbed eating behaviours in
an AN group, BN group, and control group \((n = 12 \text{ for all groups})\). They found that only AN and BN patients had negative self-beliefs and assumptions about dietary restraint. Specifically, their beliefs linked dietary restraint to increased “social- and self-desirability” or self-esteem, as well as to self-control (Cooper et al., 1998). These qualitative results highlight the use of dietary restraint as a strategy to cope with the negative emotions experienced as a result of low self-esteem in AN, providing evidence for the link between drive for thinness, an increased sense of control, and increased self-esteem.

**Indirect evidence.** Moving on to indirect evidence, Newns et al. (2003) conducted a trial to examine the effectiveness of an eight-session self-esteem group that was based broadly on CBT techniques for ED patients. The intervention was aimed at breaking the link between low self-esteem and shape and weight concerns, and to encourage the use of avenues other than body image to evaluate self-esteem. It included little direct focus on body image or eating attitudes. 33 female ED patients completed treatment (out of 41 that began treatment), with significant improvements in self-esteem \((t = -3.993, p = .001)\), depression \((t = 5.673, p < .001)\) and, importantly, eating attitudes \((t = 3.262, p = .004)\) post-intervention. Although patients still had some eating disordered attitudes at the end of treatment, results suggest that addressing self-esteem rather than the ED directly may potentially improve eating attitudes. The results confirm the role of low self-esteem in maintaining EDs, possibly contributing to the vicious cycle whereby low self-esteem drives a desire for a thin ideal, which in turn reduces self-esteem further.

Finally, O’Dea and Abraham’s (2000) study provided indirect evidence for mechanisms related to self-esteem, albeit in a non-clinical sample. They conducted a school-based self-esteem education program focusing on body image, eating attitudes, and behaviours. A total of 470 male and female students aged 11-14 years participated, including 116 students with low self-esteem and higher anxiety who were considered at high risk of developing EDs. Post-intervention, body image in the intervention group significantly improved \((I \rightarrow II; F(1, 463) = 8.6, p < .01)\). Whilst weight-losing behaviours in the control group increased significantly by 8% (McNemar \(\chi^2 = 3.85, df = 1, p < .05\)), there were no significant changes in weight-losing behaviours in the intervention group (McNemar \(\chi^2 = 0.38, df = 1, p > .05\)). The students considered at high risk had significantly greater body satisfaction \((F(1, 111) = 4.8, p < .05)\), decreased drive for thinness \((F(1, 111) = 4.0, p < .05)\), and decreased importance of physical
appearance on self-esteem ($F(1, 108) = 4.30, p < .05$) after the intervention. Many of these changes were still significant at 12-month follow-up. The study provides preliminary evidence that it is possible to increase body satisfaction, reduce dieting and weight loss, and decrease self-worth invested in the body, through focusing directly on cognitive changes aimed at strengthening self-esteem without directly targeting weight or ED related themes. It supports the hypothesis that self-esteem is a maintaining factor of EDs, with specific links to drive for thinness, body image, eating attitudes, and self-worth invested in the body.

**Conclusion.** The above studies provide preliminary evidence for mechanisms related to low self-esteem in AN. First, the findings suggest that weight, shape and eating behaviours have an excessive influence on self-esteem (Blechert et al., 2011). Second, further evidence was provided for the complex relationship between perfectionism and low self-esteem, with high levels of perfectionism found to predict low self-esteem (Surgenor et al., 2007). Third, empirical support was provided for the association between low self-esteem, control-related factors, and AN symptomatology, in particular drive for thinness (Surgenor et al., 2007; Woolrich et al., 2006; Cooper et al., 1998). Specifically, drive for thinness was reported to be a coping strategy for low self-esteem, and resulted in an increased sense of control and increased self-esteem in individuals with AN (Woolrich et al., 2006; Cooper et al., 1998). Fourth, indirect evidence from treatment trials targeting self-esteem support the role of self-esteem in maintaining ED behaviours (Newns et al., 2003; O’Dea & Abraham, 1999), showing that targeting self-esteem in treatment will disrupt maintenance mechanisms of AN, resulting in improved eating attitudes and body satisfaction, and decreased drive for thinness and self-worth invested in the body. Collectively, these studies provide consistent support for the role of self-esteem in maintaining AN. However, the studies do not assess specific pathways though which self-esteem interacts with other AN maintenance variables. More work is required to address more complex moderator or mediator models of self-esteem to determine the mechanisms that lead to maintenance of AN symptomatology.
### Table 5.2
**Studies Assessing Mechanisms of Self-Esteem**

<table>
<thead>
<tr>
<th>Method and Participants</th>
<th>Measures</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Direct evidence (studies testing proposed mechanisms)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Blechert et al. (2011)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Aim:</strong> To assess associations between self-esteem (interpersonal relationships &amp; achievement/ performance domains) and shape/weight/eating concerns</td>
<td>Self-report measures: EDE-Q, BDI, RSES, MSES</td>
<td>• There was a significant group x congruence interaction, indicating that the group modulated the congruence effect for event related potentials and reaction times (i.e., self-esteem was linked with weight/shape/eating concerns in ED groups but not controls)</td>
</tr>
<tr>
<td><strong>Participants:</strong> From a German community,</td>
<td></td>
<td>• To follow-up on the interaction effect, reaction times were pooled across self-esteem domains. Post-hoc t-tests comparing congruent and incongruent conditions found that the AN group showed significant affective priming effects, but not BN or controls</td>
</tr>
</tbody>
</table>
| • 20 AN females  
  Mean age = 23.1 years ($SD = 4.6$), mean BMI = 16.8 kg/m$^2$ ($SD = 0.8$) | Event related potentials, reaction times | |
| • 20 BN females  
  Mean age = 26.5 years ($SD = 7.8$), mean BMI = 22.5 kg/m$^2$ ($SD = 3.4$) | | |
| • 28 healthy controls  
  Mean age = 25.4 years ($SD = 4.8$), mean BMI = 20.8 kg/m$^2$ ($SD = 2.5$) | | |
| EDE$^1$ and SCID-IV$^2$ used to establish diagnostic status. | | |
| **Method:** Used affective priming procedure with shape, weight or eating related prime sentences, complemented by affectively congruent and incongruent target words from self-esteem domains. | | |
| **Surgenor et al. (2007)** | | |
| **Aim:** To assess associations between self-esteem (self-competence and self-liking components) and AN symptoms in a prospective study | Self-report measures: EDI-2, RSES | • At admission, overall severity of ED disturbance was significantly associated with both components of self-esteem |
| **Participants:** Data from two Australian specialist treatment centres, | | • EDI subscales of perfectionism and ineffectiveness made independent contributions to self-competence |
| • 77 AN inpatients  
  Mean age = 22.5 years ($SD = 8.5$), mean BMI = 16.4 kg/m$^2$ ($SD = 2.2$) | | • At discharge, a decrease in drive for thinness was significantly associated with an increase in both components of self-esteem |
| Participants diagnosed according to DSM-IV (APA, 1994) criteria. | | |
| **Method:** Measures were recorded at admission and discharge from inpatient treatment. | | |
Table 5.2 (Continued)

<table>
<thead>
<tr>
<th>Method and Participants</th>
<th>Measures</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Direct evidence (studies testing proposed mechanisms)</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Woolrich et al. (2006)**

**Aim:** To assess links between negative self-beliefs and drive for thinness

**Participants:** From UK,
- 15 AN females
  - Mean age = 31.3 years ($SD = 10.3$), mean BMI = 16.2 kg/m$^2$ ($SD = 1.7$)
- 17 dieting female controls
  - Mean age = 29.3 years ($SD = 5.7$), mean BMI = 25.5 kg/m$^2$ ($SD = 4.0$)
- 18 non-dieting female controls
  - Mean age = 29.8 years ($SD = 8.3$), mean BMI = 20.5 kg/m$^2$ ($SD = 1.5$)

SCID-IV$^2$ used to establish diagnostic status. EDE$^1$ used to screen for dieting behaviours in control groups.

**Method:** Groups were compared on results of a semi-structured interview. Themes were identified from qualitative data using content analysis (Krippendorff, 1980).

**Interview:** Semi-structured interview (Cooper, Todd, & Wells, 1998)

**Self-report measures:** DEBQ-R$^7$, EAT$^8$, BDI-II$^3$, RSES$^4$

- In AN participants, failure and defectiveness were in the top three most reported negative self-beliefs
- AN participants reported significantly more distress associated with negative self-beliefs than both control groups
- To cope with negative self-beliefs, AN participants most frequently endorsed restricting food intake, which was reported to decrease negative self-beliefs in the short term. Few control participants used this coping behaviour

---

**Cooper et al. (1998)**

**Aim:** To assess self-beliefs and beliefs linking eating behaviour with weight/shape

**Participants:** From UK,
- 12 female AN patients
  - Mean age = 26.0 years ($SD = 7.9$), mean BMI = 15.5 kg/m$^2$ ($SD = 1.3$)
- 12 female BN patients
  - Mean age = 25.7 years ($SD = 6.3$), mean BMI = 21.6 kg/m$^2$ ($SD = 2.8$)
- 12 female controls
  - Mean age = 25.7 years ($SD = 5.3$), mean BMI = 23.2 kg/m$^2$, $SD = 3.4$

SCID-III-R$^2$ used to establish diagnostic status.

**Method:** All participants completed self-report measures and were interviewed individually.

**Interview:** Semi-structured interview to elicit automatic thoughts and underlying assumptions (Cooper et al., 1998)

**Self-report measures:** EAT$^8$, BDI$^3$, RSES$^4$

- Two types of qualitatively distinct beliefs were elicited in patients with AN and BN, but not controls. They were:
  a) Negative unconditional beliefs about the self (being worthless, useless, inferior, a failure, abandoned, and alone)
  b) Beliefs about weight, shape, and eating (linking thoughts about weight/shape and negative self-beliefs)
- All patients believed that dietary restraint helped with their negative self-beliefs (e.g., to feel more successful, less of a failure, and more in control)
<table>
<thead>
<tr>
<th>Indirect evidence (studies assessing effectiveness of treatment targeting self-esteem)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Method and Participants</strong></td>
</tr>
<tr>
<td><strong>Aim:</strong> To assess the effectiveness of a self-esteem group intervention on patients with EDs from a mental health service in UK,</td>
</tr>
<tr>
<td><strong>Participants:</strong> From a mental health service in UK,</td>
</tr>
<tr>
<td>• 41 female ED patients (11 AN, 32 BN, 15 BED, 12 EDNOS) Aged 17–56 years, mean age = 29.03 years, BMI &gt; 17.5 kg/m²</td>
</tr>
<tr>
<td><strong>Method:</strong> The intervention was skills-teaching in nature, based broadly on CBT techniques, and consisted of 8 sessions of 1.5 hours. Measures were taken pre- and post-intervention.</td>
</tr>
<tr>
<td><strong>Newns et al. (2003)</strong></td>
</tr>
<tr>
<td><strong>Self-report measures:</strong></td>
</tr>
<tr>
<td>• EAT-26², BDI¹, GHQ⁹,SES¹⁰</td>
</tr>
<tr>
<td><strong>Outcomes:</strong></td>
</tr>
<tr>
<td>• 33 completed treatment</td>
</tr>
<tr>
<td>• Treatment completers showed improvement in eating attitudes, depressive features, and self-esteem</td>
</tr>
<tr>
<td><strong>O'Dea &amp; Abraham (1999)</strong></td>
</tr>
<tr>
<td><strong>Aim:</strong> To assess the effectiveness of a school-based, self-esteem educational intervention on body image and eating attitudes</td>
</tr>
<tr>
<td><strong>Participants:</strong> 470 secondary students at Australian schools (37% males, 63% females), Aged 11–14 years. Sub-sample of 116 students classified as high risk (due to low self-esteem/high anxiety)</td>
</tr>
<tr>
<td><strong>Method:</strong> Students were randomly allocated to control or intervention groups. The intervention was an educational program aimed at building general self-esteem (O’Dea, 1995), consisting of nine weekly 50-80 minute lessons, administered by teachers. Measures were taken pre- and post-intervention and at 12-month follow-up.</td>
</tr>
<tr>
<td><strong>Self-report measures:</strong></td>
</tr>
<tr>
<td>• EDI⁶, Self-Perception Profile for Adolescents¹¹, BDI³, STAI¹², Physical Appearance Ratings¹³</td>
</tr>
<tr>
<td><strong>Outcomes:</strong></td>
</tr>
<tr>
<td>• Post intervention, there was increased body satisfaction and no increase in weight-losing behaviours</td>
</tr>
<tr>
<td>• In students considered at high risk, there was increased body satisfaction and decreased drive for thinness and importance of physical appearance on self-esteem</td>
</tr>
</tbody>
</table>

**Note.** AN—Anorexia Nervosa; BN—Bulimia Nervosa; ED—Eating disorder; BED—Binge Eating Disorder; EDNOS—Eating Disorder Not Otherwise Specified; CBT—Cognitive behavioural therapy; UK—United Kingdom.

---

¹ Eating Disorder Examination (EDE; Fairburn & Cooper, 1993; EDE-Q; Fairburn & Beglin, 1994)
² Structured Clinical Interview for DSM Disorders (SCID-III-R; Spitzer et al., 1992; SCID-IV; First et al., 1995)
³ Beck Depression Inventory (BDI; Beck et al., 1961; BDI-II; Beck et al., 1996)
⁴ Rosenberg Self-Esteem Scale (RSES; Rosenberg, 1965)
⁵ Multidimensional Self-Esteem Scale (MSES; Fleming & Courtney, 1984)
⁶ Eating Disorder Inventory (EDI; Garner & Olmsted, 1984; EDI-2; Garner, 1991)
⁷ Dutch Eating Behaviour Questionnaire—Restraint subscale (DEBQ-R; Van Strien et al., 1986)
⁸ Eating Attitudes Test (EAT; Garner & Garfinkel, 1979; EAT-26; Garner et al., 1982)
⁹ General Health Questionnaire (GHQ; Goldberg & Hillier, 1979)
¹⁰ Robson Self-Esteem Self Report Inventory (SESR; Robson, 1989)
¹¹ Self-Perception Profile for Adolescents (Harter, 1982)
¹² State-Trait Anxiety Inventory (STAI; Spielberger et al., 1970)
¹³ Physical Appearance Ratings (O’Dea, Abraham, & Heard, 1996)
1.4.3 Evidence for mechanisms involving control-related cognitions

Description of studies. The two control-related cognitions relevant to the present study are the general need for control and sense of control dependent on eating/weight. Surprisingly, considering the widely accepted notion that the general need for control is critical in the maintenance of AN, no studies assessing mechanisms related to the need for control were uncovered in the literature. Only one study providing direct evidence for having a sense of control dependent on dietary restraint in AN was identified (Shafran, Fairburn, Nelson, & Robinson, 2003) and summarised in Table 5.3. Two studies assessing another control-related factor (e.g., perceived levels of control) were also identified, however were not included in the present review as these variables are conceptually different from the general need for control and sense of control dependent on eating/weight (Sassaroli et al., 2008). For the reader’s interest, the studies provided direct evidence that behavioural drive for thinness was used as a coping strategy for perceived loss of control, resulting in an increased sense of control in individuals with AN (Woolrich et al., 2006) and restrained eaters (Rezek & Leary, 1998). The findings from these studies will not be further discussed as they are beyond the scope of the present study.

Limitations of the study. Shafran and colleagues (2003) used a cross-sectional design, constraining inferences about the directionality of mechanisms being tested. Their study also lacked a psychiatric control group, making it unclear whether the mechanisms in question existed generally in psychiatric disorders or are ED-specific. Methodological flaws included combining across AN, BN, and EDNOS categories to form the clinical comparison group.

Direct evidence. Shafran et al. (2003) tested whether individuals with EDs interpreted symptoms of dietary restraint in terms of control over eating, weight, shape, or over oneself in general, comparing 44 ED patients (10 AN, 12 BN, 21 EDNOS) to 80 controls. They found that significantly more of the ED group interpreted symptoms of dietary restraint or starvation in terms of control, including symptoms of hunger \( \chi^2 = 28.2, df = 1, p < .001 \), poor concentration \( \chi^2 = 29.0, df = 1, p < .001 \), heightened satiety \( \chi^2 = 11.7, df = 1, p = .01 \), dizziness \( \chi^2 = 9.7, df = 1, p = .02 \), and reduction in rate of weight loss \( \chi^2 = 34.5, df = 1, p < .001 \). Also, about one-quarter of the ED group positively valued the symptoms of hunger (25%), heightened satiety (22.7%), and dizziness (27.3%).
Conclusion. The above study provided support for the hypothesis that certain symptoms of dietary restraint were interpreted in terms of control over eating, weight, shape, or oneself, also suggesting that individuals with EDs have the tendency to use control over eating as an index of self-control in general. The findings are consistent with Fairburn and colleagues’ (1999) Feedback Mechanism 1, which proposes that in individuals with AN, one’s sense of control becomes dependent on eating and weight. However, there is a dismal lack of evidence for mechanisms related to the general need for control. In a discussion paper of psychological control and AN, Surgenor et al. (2002, p. 95) asserted, “Fairburn et al.’s (1999) appeal for an examination of control-based treatments for AN must be accompanied by more complex empirical data regarding the full range and experience of control issues and processes reported by people with AN, the meanings ascribed to such control, and the relationship between control issues and specific symptoms”. Studies testing models of control-related factors, especially the general need for control, in interaction with other vulnerabilities in predicting AN symptoms, is urgently needed.

<table>
<thead>
<tr>
<th>Method and Participants</th>
<th>Measures</th>
<th>Outcomes</th>
</tr>
</thead>
</table>
| **Direct evidence (studies testing proposed mechanisms)** | **Self-report measures:** EDE\(^1\), BDI\(^2\), Ambiguous Sentence Completion Task\(^3\) | • Significantly more of ED group interpreted symptoms of starvation/dietary restraint in terms of control, including hunger, poor concentration, heightened satiety, dizziness, and reduction in rate of weight loss  
• About ¼ of ED patients positively valued symptoms of hunger, heightened satiety, and dizziness |
| **Shafran et al. (2003)** | **Aim:** To determine if individuals with EDs interpret symptoms of dietary restraint in terms of control  
**Participants:** From specialised ED services & the community in UK,  
• 44 female ED patients (10 AN, 12 BN, 21 EDNOS)  
  Mean age = 28.4 years (SD = 9.8)  
• 80 community controls  
  Mean age = 31.0 years (SD = 13.3)  
Participants diagnosed according to DSM-IV (APA, 1994) criteria.  
**Method:** Groups were compared on self-report measures. | |

Note. AN—Anorexia Nervosa; BN—Bulimia Nervosa; ED—Eating disorder; EDNOS—Eating Disorder Not Otherwise Specified; UK—United Kingdom.

---

1 Eating Disorder Examination (EDE; Fairburn & Cooper, 1993)  
2 Beck Depression Inventory (BDI; Beck et al., 1961)  
3 Ambiguous Sentence Completion Task, Adapted (Butler & Mathews, 1983)
1.4.4 Indirect evidence from treatment trials of cognitive therapy

Findings from reviews. Numerous reviews examining the effectiveness of therapy for AN have been published. Findings from the “gold standard” of reviews, the Cochrane Methodology Reviews, will now be examined. Most recently updated in 2009 (Hay et al., 2009), no changes in the status of treatment efficacy of outpatient treatment for AN were reported, compared to when the initial review was first published in 2003 (Hay et al., 2003). Utilising a rigorous, systematic approach, reviewers only identified seven small randomised controlled outpatient trials with acceptable methodologies, of which three assessed CBT. The three studies, which compared CBT to interpersonal therapy (IPT) and nonspecific clinician management (McIntosh et al., 2005), to dietary advice (Serfaty et al., 1999), and to behavioural therapy and “eclectic” therapy (Channon, de Silva, Hemsley, & Perkins, 1989), are summarised in Table 5.4.

First, considering participants’ compliance to different outpatient psychological therapies, no significant differences were found in the number of participants completing therapy for CBT compared to IPT, non-specific therapy (McIntosh et al., 2005), behavioural therapy, or “eclectic” therapy (Channon et al., 1989). However, participants favoured non-specific therapy over CBT and IPT (McIntosh et al., 2005). Compared to dietary advice, CBT was more acceptable to participants, with one study reporting that all participants in the dietary advice group dropped out of treatment compared to two in the CBT group (Serfaty et al., 1999).

Next, considering the outcomes of the outpatient trials, findings were mixed. One study reported improvement in the CBT group compared to IPT group in global clinician ratings (RR = 0.76, 95% CI [0.54 to 1.06]) and dietary restraint (SMD = -0.74, 95% [CI -1.38 to -0.09]; McIntosh et al., 2005). Another study reported higher BMI in the CBT group at the end of treatment compared to the dietary advice group, however this difference failed to meet significance (SMD = 0.71, 95% CI [-0.05 to 1.46]; Serfaty et al., 1999). On the other hand, no differences in outcome were found when comparing CBT to behavioural therapy, “eclectic” therapy (Channon et al., 1989), or non-specific therapy (McIntosh et al., 2005). At the end of treatment, one study reported that a large proportion of participants (70%) were still not recovered (McIntosh et al., 2005), and another study reported that BMI was still not within the normal range (Serfaty et al., 1999).

Authors of the Cochrane Methodology Review were unable to conduct a meta-analysis due to the small number of outpatient trials (Hay et al., 2009). The trials had
small sample sizes, thus power was a major problem. Also, there was a possibility of bias in the trials due to the lack of blind outcome assessments. These issues and the insufficient replication of findings made conclusions severely limited. There appeared to be few consistent differences between specific psychotherapies in terms of treatment compliance and outcomes. As such, the authors concluded that no specific approach could be recommended from the review, and an urgent need for large, well-designed trials in AN was required (Hay et al., 2009).

Whilst the Cochrane Methodology Review did not include trials with inpatient samples, other reviews of psychotherapy for adults with AN included trials with inpatient and outpatient samples, or a combination of both, coming to similar conclusions. These reviews cited that limitations including the small numbers of trials, small sample sizes, large drop-out rates, failure to report randomisation or blinding procedures, absence of power analyses, lack of controls, absence of an intention-to-treat approach, and failure to report funding sources, have prevented definitive conclusions to be drawn (Agras & Robinson, 2008; Bulik et al., 2007; Wilson et al., 2007). Although earlier isolated studies have found CBT to be effective in improving AN outcomes and preventing relapse in certain conditions (e.g., post-hospitalisation; Pike, Walsh, Vitousek, Wilson, & Bauer, 2003), these findings have not been replicated, and more recent randomised controlled trials (e.g., Carter et al., 2011) continued to find no significant differences in the outcome of patients participating in CBT compared to other psychotherapies. Overall, evidence for any AN treatment is weak, with no clear differences found between CBT and other comparison conditions.

**Conclusion.** Evidently, although much has been written about theoretical models for CBT, empirical evaluation of treatments is limited (Treasure & Schmidt, 2002). No clear or consistent differences have been found between CBT and other comparison conditions including behavioural therapy, IPT, or non-specific clinical management. Most AN patients remain unwell at the end of treatment.
<table>
<thead>
<tr>
<th>Method and Participants</th>
<th>Measures</th>
<th>Outcomes</th>
</tr>
</thead>
</table>
| McIntosh et al. (2005)                  | **Self-report measures:** EDI\(^1\), EDE\(^2\), HRSD\(^3\) **Other measures:** Clinician global rating, GAF (DSM-IV; APA, 1994), height, weight | **Compliance:**  
- No significant differences in number of participants not completing therapy for all three groups  
- Non-specific therapy was favoured over CBT and IPT  
**Outcome:**  
- CBT vs. IPT: CBT group more participants rated as significantly improved on global scale, more improvement in dietary restraint. No differences for weight, global assessment of function, or depression.  
- CBT vs. non-specific management: No differences on any measures  
- At end of treatment, 70% of participants still not recovered |
| Serfaty et al. (1999)                   | **Self-report measures:** EDI\(^1\), DAS\(^4\), LCB\(^5\), BDI\(^6\) **Other measures:** BMI | **Compliance:**  
- CBT appeared more acceptable. All patients in dietary advice group dropped out vs. 2 in CBT group  
**Outcome:**  
- CBT vs. dietary advice: CBT group higher mean BMI at the end of treatment (although non-significant), but still not in normal range (<18 kg/m\(^2\)) |
| Channon et al. (1989)                   | **Interview:** Morgan & Russell Interview\(^7\) **Self-report measures:** EDI\(^1\), BDI\(^6\), MOCI\(^8\) **Other measures:** BMI, preferred weight | **Compliance:**  
- CBT vs. BT & CBT vs. “eclectic” therapy: No differences in number of participants not completing treatment  
- CBT group subjects missed significantly fewer sessions over control treatment  
**Outcome:**  
- Participants in all treatment groups improved |

**Note.** AN—Anorexia Nervosa; CBT—Cognitive behavioural therapy; IPT—Interpersonal therapy; BT—Behavioural therapy.

\(^1\) Eating Disorder Inventory (EDI; Garner & Olmsted, 1984; EDI-2; Garner, 1991)  
\(^2\) Eating Disorder Examination (EDE; Fairburn & Cooper, 1993)
1.4.5 Implications for the present study

Whilst a number of studies providing support for the role of low self-esteem in maintaining AN were identified, support for the role of perfectionism, the general need for control, and sense of control dependent on eating/weight in maintaining AN was weak or non-existent. Some interesting findings relevant to Model A were derived from the literature review. First, there is a complex interactive relationship between perfectionism and self-esteem (Sassaroli et al., 2008; Davis, 1997; Surgenor et al., 2007), and both perfectionism and self-esteem with control-related factors (Sassaroli et al., 2008). Second, perfectionism, self-esteem, and control-related variables have main and moderating effects on drive for thinness (Sassaroli et al., 2008; Surgenor et al., 2007; Woolrich et al., 2006; Cooper et al., 1998). Third, indirect evidence from treatment trials targeting perfectionism (Steele & Wade, 2008) and self-esteem (Newns et al., 2003; O’Dea & Abraham, 2000) show that changing these factors will disrupt maintenance mechanisms of EDs, providing preliminary support for their role in maintaining AN. Finally, in individuals with EDs, dietary restraint is interpreted in terms of control over eating, weight, and shape (Shafran et al., 2003). Although these studies yielded interesting findings, replication of the findings is limited. Also, existing studies have methodological flaws in sampling, combining across ED categories or even combining ED and control samples (e.g., Sassaroli et al., 2008; Davis, 1997; Shafran et al., 2003). Additionally, some studies used relatively small sample sizes due to the qualitative nature of the studies (e.g., Woolrich et al., 2006; Cooper et al., 1998). Replicating these findings using quantitative methods and a larger AN sample will make results more robust.

It is evident that empirical evidence for maintenance mechanisms proposed in AN cognitive models is lacking. Cognitive theory of AN remains largely undeveloped, and we still have a relatively poor understanding of the specific cognitions and cognitive processes that are involved in maintenance of the disorder, especially those

---

3 Hamilton Rating Scale for Depression (HRSD; Hamilton, 1986)
4 Dysfunctional Attitudes Scale (DAS; Weissman and Beck, 1978)
5 Locus of Control of Behaviour (LCB; Craig, Franklin, & Andrews, 1984)
6 Beck Depression Inventory (BDI; Beck et al., 1961)
7 Morgan and Russell Interview (Morgan & Russell, 1975)
8 Maudsley Obsessional-Compulsive Inventory (MOCI; Hodgson & Rachman, 1977)
that influence the most distinctive symptom of AN, drive for thinness (Woolrich et al., 2006). More studies assessing complex moderator and mediation models of the variables of interest in predicting AN symptoms is urgently required (Bardone-Cone et al., 2007). In a review of risk and maintenance factors of EDs, Stice (2002) asserted that more AN research should be directed at elucidating how maintenance factors worked together to promote and perpetuate eating pathology in this disorder. To address the shortcomings of the existing literature, Model A in the present study will test the direct and mediating effects of perfectionism, low self-esteem, general need for control, and sense of control dependent on eating/weight on drive for thinness, in a larger AN sample. The above findings will guide the development of Model A, which is further described in Section 3.1 (p. 130-133).

Despite the wide recommendation for the use of CBT in the treatment of AN, the literature review also brought to light the limited empirical evidence for treatment of adults with AN. As discussed, several reviews of treatment efficacy, including a “gold standard” Cochrane Methodology Review, found no clear or consistent differences between CBT and other comparison treatments, including behavioural therapy, IPT, or non-specific clinical management (Hay et al., 2009; Agras & Robinson, 2008; Bulik et al., 2007; Wilson et al., 2007). These reviews assessed outpatient, inpatient, or a combination of outpatient and inpatient treatments, concluding that most AN patients remained unwell at the end of treatment. Although earlier isolated studies have found CBT to be effective in improving outcomes (e.g., Pike et al., 2003), these findings have not been replicated, and more recent randomised controlled trials (e.g., Carter et al., 2011) continued to find no significant differences in the outcome of patients participating in CBT compared to other psychotherapies. The small numbers of existing trials with various methodological limitations (i.e., small sample sizes, large drop-out rates, failure to report randomisation or blinding procedures, absence of power analyses, lack of controls, absence of an intention-to-treat approach, and failure to report funding sources) have prevented definitive conclusions from being drawn, highlighting an urgent need for large, well-designed trials in AN (Hay et al., 2009; Agras & Robinson, 2008; Bulik et al., 2007; Wilson et al., 2007). Whilst there have been many treatment follow-up studies supporting the effectiveness of CBT in BN (for reviews, refer to Wilson et al., 2007; Cooper, 1997), CBT does not appear to have consistent effects on AN symptomatology, highlighting the need for empirical clarification of AN-specific cognitive mechanisms (Wilson, 2005). Cognitive theory of AN could benefit from the consideration of new variables, with researchers recommending that more attention
should be directed at identifying new factors or vulnerabilities in AN (Stice, 2002). The following section of this chapter will discuss newer enhanced cognitive models of AN, and examine a topic that has been of recent interest: the role of metacognitive theory in AN.

2. ADVANCEMENTS IN COGNITIVE THEORY FOR ANOREXIA NERVOSA: BACKGROUND FOR MODEL B

New second generation ED theories that appear considerably different from existing cognitive theories have been developed (Cooper, 2005), with the inclusion of novel components and an attempt to tease apart detailed cognitions, precise content, and links to emotion and behaviour. Three new theories, the transdiagnostic model of EDs (Fairburn et al., 2003), schema-focused CBT for EDs (Waller, Kennerley, & Ohanian, 2007), and the cognitive-interpersonal theory for AN (Schmidt & Treasure, 2006), will be briefly discussed with the objective of providing a more complete picture to the evolution of cognitive theory in AN. Issues and weaknesses that have led to the exclusion of these theories in the present study’s model development will be discussed. Then, recent interest in the relevance of metacognition to AN and metacognitive theory will be explored, on which Model B will be based. Whilst it is the intention of this chapter to provide a literature review of studies assessing mechanisms involved in Model B, unsurprisingly, no relevant studies were discovered in literature searches. This is likely because of relatively recent interest in the role of metacognition in AN and the novelty of ideas proposed in Model B.

2.1 ENHANCED COGNITIVE THEORIES

Transdiagnostic model of EDs. Fairburn and colleagues (2003) proposed a “transdiagnostic” theory that did not differentiate between the ED diagnostic categories, attempting to provide a single framework within which to conceptualise all EDs. Their maintenance model was developed on the premise that all EDs shared the same distinctive underlying mechanisms that were expressed in different diagnostic states, accounting for the frequent migration of patients between the diagnostic categories over time, the similar clinical features of EDs, and common ED behaviours. The transdiagnostic model suggested that in addition to the mechanisms proposed in the cognitive theory of AN (Fairburn, Shafran, et al., 1999) discussed earlier, four new maintaining factors prevented change: clinical perfectionism, core low self-esteem,
mood intolerance, and interpersonal difficulties. Whilst the transdiagnostic model broadened existing theory, it did not elucidate cognitions and mechanisms specific to AN. For example, mood intolerance (or the difficulty coping with intense mood states) is more pertinent in individuals with BN, impacting on binge eating and self-induced vomiting as mood modulatory behaviours (Fairburn et al., 2003). Whilst clinical perfectionism and core low self-esteem are recognised to be critical in the maintenance of AN, the transdiagnostic theory does not add much to Fairburn et al.’s (1999) initial AN model, which already encompassed these factors. One study examined the validity of the transdiagnostic model in a community sample of females, finding support for the model (Hoiles, Egan, & Kane, 2012). However, to the knowledge of the researcher, no studies have assessed the validity of the model in AN or ED samples.

Three studies have tested the model therapeutically in ED outpatients. In the first study, 149 BN or EDNOS patients received 20 50-minute sessions of therapy, with results indicating that 66.4% of those who completed treatment had a good outcome (Fairburn et al., 2009). However, this study excluded individuals with AN who had BMI < 17.5 kg/m². In the second study, 125 ED outpatients (34 AN, 40 BN, 51 EDNOS) received 20 to 40 50-minute sessions of therapy, with patients with BMI ≥ 18.5 kg/m² receiving about 20 sessions, whilst underweight patients received about 40 sessions to allow for increasing motivation and weight restoration (Byrne et al., 2011). This study included patients with BMI > 14 kg/m². Whilst full or partial remission was achieved by two thirds of treatment completers in the total sample, the authors acknowledged that outcomes were poorer for AN compared to BN and EDNOS, due to the high drop-out rate of AN patients (50%, compared to 35% and 37.3% respectively). Of the treatment completers, full or partial remission was also achieved by fewer AN patients compared to BN and EDNOS (50%, compared to 66.7% and 73.3% respectively). These findings suggest that therapy based on the transdiagnostic theory of EDs may be less effective for AN than for other EDs (Byrne et al., 2011). In the third recent study, 99 adult AN patients from two sites (in the UK and Italy) received 40 50-minute individual sessions of therapy, with results indicating that two-thirds of patients completed treatment (Fairburn et al., 2013). Of the treatment completers, 62% achieved a BMI of > 18.5 kg/m² and 88% had minimal residual ED psychopathology at the end of treatment, with changes largely maintained at 60-week follow-up (Fairburn et al., 2013). While findings from this new study show promise for the utility of AN therapy based on the transdiagnostic model, the findings are substantially more optimistic compared to earlier findings from Byrne et al.’s (2011) study, hence need to be
replicated before more definitive conclusions can be made. Further, for the UK site of the study, only AN patients with BMI between 15.0 and 17.5 kg/m² were recruited, hence findings cannot be generalised to patients with BMI < 15.0 kg/m² (Fairburn et al., 2013).

It should also be noted that none of the above studies have assessed the effectiveness of therapy based on the transdiagnostic model in ED inpatients. This therapy was designed for ED patients where outpatient management was appropriate and who are not underweight (BMI > 17.5 kg/m²). As AN patients tend to be more severely unwell with complex medical risk, with rates of hospitalisation twice those of BN patients (Newman et al., 1996), this therapy is possibly less suitable for AN compared to other EDs. To summarise preliminary findings, existing studies have inconsistent conclusions, with one study indicating that therapy based on the transdiagnostic model shows promise for AN treatment (Fairburn et al., 2013), and another study determining that it is less effective for AN compared to other EDs (Byrne et al., 2011). Further research is required to clarify these findings. Considering the lack of robust empirical support for the utility of the transdiagnostic model in individuals with AN, and taking into account the rationale for maintaining EDs as separate categorical diagnoses as discussed earlier (p. 3-4), the present study is targeted towards the understanding of AN-specific mechanisms. The relevant variables of interest from this model—perfectionism and low self-esteem—have been examined in Chapter 4 and are already included in Models A and B.

Schema-focused CBT for EDs. Schema-focused CBT for EDs (Waller, Kennerley, et al., 2007) was developed based on Young’s (1990, 1999) schema-focused cognitive therapy for personality disorders. Like the transdiagnostic theory of EDs, this theory did not differentiate between different diagnostic categories. It took into account deeper level cognitions to address the role of schema content and process, accommodating both restrictive and bulimic features of EDs (Waller, Kennerley, et al., 2007). The model proposed that maladaptive schemas or beliefs about the self, others, and the world influenced an individual’s interpretation of events, further activated existing schemas, which were then maintained through information processing biases and behaviours that were consistent with schema compensation or avoidance (Young, 1990). Put briefly, AN occurred through a process of schema compensation, characterised by primary avoidance that is demonstrated through not eating. Dietary restraint was proposed to be a way of preventing distressing cognitions and emotions
(generated from core beliefs) from being experienced in the first place. Whilst core beliefs or negative self-beliefs are recognised to be critical in the maintenance of AN, these overlap conceptually with low self-esteem, hence, schema-focused CBT may not add much to existing knowledge of AN; specifically, that drive for thinness is being used as a coping strategy for low self-esteem (Woolrich et al., 2006; Cooper et al., 1998), as discussed earlier (p. 105-106).

Whilst limited single-case studies have indicated positive outcomes of schema-focused CBT for BN and EDs (Waller, Kennerley, et al., 2007), no controlled trials of therapy were identified in the literature. Also, no studies about schema-driven processes were found in AN samples. Further, researchers have warned that schema-focused CBT should be used with caution in EDs (James, 2001), and that conventional CBT should be the default treatment option as it has a more established empirical basis and is less complex and costly than schema-focused CBT (Waller, Kennerley, et al., 2007). Due to its focus on the development of AN rather than maintenance, the lack of empirical support in AN populations, and the complexity of schema-focused CBT, this theory will not be further discussed. The relevant variable of interest from this model—negative self-beliefs or low self-esteem—has been examined in Chapter 4 and is already included in Models A and B.

Cognitive-interpersonal theory for AN. The cognitive-interpersonal maintenance model of AN (Schmidt & Treasure, 2006) focuses particularly on the restricting subtype of AN, which is conceptualised to be distinct from other EDs, and the most puzzling and difficult to treat of all EDs (Schmidt & Treasure, 2006). The theory combined four intra- and interpersonal factors, namely perfectionism/cognitive rigidity, experiential avoidance, pro-anorectic beliefs, and response of close others. It differed from other previous models of AN in that it did not emphasise the role of weight and shape-related factors in the maintenance of the disorder, adding a new consideration that in avoidant individuals with AN, pro-anorectic beliefs emerged about the value of AN in helping to manage their emotions, thus maintaining the disorder (Schmidt & Treasure, 2006). This “numbing” of emotions occurred because the individual with AN became so focused on eating and food that their emotions became less salient, and was highly valued as it was perceived to help manage emotions (Schmidt & Treasure, 2006). Aside from this component, the other intra-personal factors in the cognitive-interpersonal theory for AN have parallels with Fairburn and colleagues’ (1999) theory of AN, and the interpersonal perspective of this model is similar to that of Garner & Bemis’ (1982) model.
Whilst this model highlights some novel ideas and emphasises the interpersonal factors involved in AN which are undoubtedly important, interpersonal theories are beyond the scope of the current study. Focusing primarily on cognitive factors, the cognitive-interpersonal maintenance model does not add much to Garner & Bemis’ (1982) and Fairburn and colleagues’ (1999) cognitive models of AN. Hence, this theory will not be further discussed. The variable of interest in this model—perfectionism—has been examined in Chapter 4 and is already included in Models A and B.

Concluding issues. To summarise, there have been advancements in the cognitive theory of AN. Two of the three new theories have highlighted the overlap and similarities between diagnostic groups and proposed a transdiagnostic approach to classification and treatment (Fairburn et al., 2003; Waller, Kennerley, et al., 2007). However, as previously discussed in Chapter 2 (p. 3-4), there is robust evidence that AN should be considered a distinct and separate condition from the other EDs (Birmingham et al., 2009; Clinton et al., 2004; Keel et al., 2004; for a review, see Collier & Treasure, 2004). Further, Fairburn et al.’s theory does not add new knowledge of AN-specific mechanisms to first generation theories of AN, and Waller et al.’s theory is focused on the development of AN instead of maintenance. The third theory, the cognitive-interpersonal theory (Schmidt & Treasure, 2006), has strengths in its specific focus on the restrictive subtype of AN. However, of its two components, the cognitive component does not add much new knowledge to existing theories of AN, and the interpersonal component is beyond the scope of the present study. Finally, there is no evidence for effectiveness of therapies based on these enhanced theories in AN samples.

Whilst these new theories have highlighted a number of previously neglected factors that are indisputably important, there is relatively little in depth analysis and knowledge of how these issues and concerns might be developed or translated into a coherent, explicitly cognitive explanation of the maintenance of AN. Cooper (2005) suggested that while the replication of existing knowledge and theories are important, there is limited innovation and originality in many existing AN studies. It is necessary for researchers of AN to remain open to the study of new cognitive constructs derived from research and theory development in other psychological disorders. The next section addresses this by considering an important area of burgeoning research—the role of metacognition in AN.
2.2 The relevance of metacognitive theory

Whilst CBT is widely used to treat AN, it is inadequately evaluated in existing studies, with no clear or consistent advantages of CBT found over comparison therapies or non-specific clinical management, as discussed earlier (p. 113-115). Unfortunately, most AN patients remain unwell at the end of treatment (Wilson et al., 2007). Traditional CBT models focus on dysfunctional beliefs about the self, others, and the world that cause psychological disorders, which have been criticised for being predominantly focused on the content of cognitions at the expense of underlying cognitive mechanisms that control and regulate thinking itself (Woolrich et al., 2008).

In recent years, the relevance of metacognitive theory, which focuses instead on the higher level processes that regulate cognition, has received increasing attention in the field of EDs.

Metacognitive theory for psychological disorders. Metacognitive theory was initially developed for anxiety disorders (Wells, 1995) and later adapted to depression (Wells, 2000) and alcohol abuse (Spada & Wells, 2009). It introduced a clear distinction between cognition and metacognition, taking the focus of intervention up from the content-based cognition level to higher level metacognitive beliefs and processes (Wells, 2009). A schematic representation of the metacognitive model for psychological disorders, also called the self-regulatory executive function model (S-REF; Wells & Matthews, 1996), is illustrated in Figure 5.4. It includes cognitive level and metacognitive level factors involved in the top-down maintenance of psychological disorders. Metacognitive theory is built on the premise that psychological disorders develop due to a toxic, inflexible, and recurrent style of thinking called the cognitive attentional syndrome, which consists of excessive amounts of on-going worry, rumination, threat monitoring, and resultant unhelpful coping behaviours that prevent adaptive learning (Wells, 2009). In healthy individuals, negative thoughts and emotions (e.g., anxiety, anger, sadness) are normally temporary or fleeting because healthy coping strategies are applied to reduce threat or control cognitions. However, due to maladaptive coping strategies, individuals with psychological disorders are trapped in the cognitive attentional syndrome, resulting in intensified and prolonged negative emotional experiences (Wells, 2009). As illustrated in Figure 5.4, the cognitive attentional syndrome is monitored and controlled by higher level metacognitive beliefs, and unhelpful coping behaviours (e.g., avoidance) further reinforce these dysfunctional metacognitive beliefs. These processes use up valuable attentional resources and may
Impair clear and controlled thinking (Wells, 2009). Ultimately, these processes lead to helplessness and a sense of loss of control over cognition and emotion, strengthening negative metacognitive beliefs in a vicious cycle.

Research on metacognitive theory for various psychological disorders has gained momentum in recent years, with disorder-specific metacognitive models garnering extensive support from path analyses and structural equation modelling data in patients with generalised anxiety disorder (Wells, 2005), obsessive-compulsive disorder (Myers, Fisher, & Wells, 2007), post-traumatic stress disorder (Roussis & Wells, 2006), depression (Papageorgiou & Wells, 2009; Roelofs et al., 2007), and alcohol abuse (Spada & Wells, 2009).

**Figure 5.4.** Schematic representation of the metacognitive model of psychological disorder or the Self-Regulatory Executive Function model, adapted from Wells and Matthews’ (1996) diagram.

**Links between AN and anxiety disorders.** Metacognitive theory was originally developed for anxiety disorders and interestingly, various researchers have drawn the link between AN and some anxiety disorders. For instance, Crisp (1970) suggested that AN represented a variant of phobic disorders, specifically a weight phobia, distinguishable from other phobic disorders only in the choice of feared object.
Conceptualised within the framework of phobic disorders which often involve avoidance behaviours, drive for thinness and weight loss in AN was proposed to serve the avoidance of full normal adult body weight and shape during puberty. Crisp proposed that individuals with AN strived for weight loss and maintenance of body weight at a sub-pubertal level as they feared the loss of control over their body weight/shape, as well as social and psychological changes that occurred during puberty.

Several researchers have also drawn the link between AN and obsessive-compulsive spectrum disorders (Jacobi et al., 2004), as they share many features such as rigidity and concern over mistakes. About 50% of individuals with EDs suffer from obsessive-compulsive spectrum disorders including obsessive-compulsive disorder (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004) and obsessive-compulsive personality disorder (Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003), with those who do not fulfil criteria for formal diagnoses also showing elevated levels of obsessive-compulsive or anxiety traits from childhood (Kaye et al., 2004). Even after recovering from AN, recovered women had elevations on certain obsessive-compulsive traits, particularly excessive concern with symmetry and exactness (Srinivasagam et al., 1995). Looking more closely at the links between AN and obsessive-compulsive spectrum disorders, researchers have conceptualised restrictive eating as a compulsive “safety behaviour” (Waller, Kennerley, et al., 2007) which serves to temporarily reduce anxiety, creating a self-perpetuating cycle of anxiety (Treasure et al., 2005). Further, starvation and weight loss appear to increase compulsive behaviours. Examples of this are studies finding that compulsive behaviours (e.g., hoarding) developed during experimental starvation (Keys, Brozek, & Henschel, 1950), the onset of AN led to worsening of rigidity and perfectionism (Anderluh et al., 2003), and rituals and obsessional behaviours which form the clinical picture of AN decreased with weight gain (Channon & De Silva, 1985). The interested reader may wish to refer to reviews (e.g., Cassin & von Ranson, 2005) examining the link between AN and obsessive-compulsive spectrum disorders, or to empirical studies (Diaz-Marsa, Carrasco, & Siaz, 2000; Gillberg, Rastam & Gillberg, 1995; Milos, Spindler, Ruggiero, Klaghofer, & Schnyder, 2002; Anderluh et al., 2003).

Whilst similarities have been drawn between AN and certain anxiety disorders, these links are anecdotal, with no evidence provided for the actual underlying shared mechanisms between AN and anxiety disorders. The key point is whether parallels exist between the mechanisms or pathways proposed to maintain AN in existing cognitive
theories of AN, and those proposed to maintain anxiety disorders in metacognitive theory.

**Parallels between cognitive theories of AN and metacognitive theory.** Whilst this topic has not been explored or empirically tested in EDs, parallels can be drawn between cognitive mechanisms proposed to maintain AN in existing cognitive theories and those proposed to maintain other psychological disorders in metacognitive theory, specifically surrounding the issue of control. Wells (2009) asserted that in psychological disorders, the individual’s key subjective experience is one of being “out of control” (p. 7) and helpless against internal cognitions and emotions, with maladaptive coping strategies often consisting of attempts to control the nature of thinking. These maladaptive coping strategies (e.g., thought suppression, obsessive prediction of the future to avoid problems) tend to be counterproductive in the long term, further maintaining the sense of threat to wellbeing, perceived lack of control, and negative sense of self through information processing biases (Wells, 2009). Similarly, the theme of control has been widely described to be important in AN, as previously discussed in Chapter 4 (p. 72-74). The need for control has been theorised to result in severe drive for thinness, resulting in the individual’s sense of control being dependent on eating, and perpetuating the individual’s perceived loss of control through information processing biases (Fairburn, Shafran, et al., 1999). The similarities between metacognitive theory of psychological disorders and cognitive theory of AN in the domain of control raises the question of whether control-related factors that are theorised to drive AN symptomatology in existing cognitive models (e.g., the general need for control or perceived loss of control; Fairburn, Shafran, et al., 1999) extend beyond the individual’s life events and behaviours, to the individual’s internal thoughts and processes, or metacognition.

Whilst existing research on metacognitions in EDs is scant, Chapter 4 (p. 80-86) examined three studies of ED or AN populations assessing the relevance of individual metacognitive factors in AN. Two of the three studies utilised the MCQ-30 (Wells & Cartwright-Hatton, 2004), which operationalises dysfunctional metacognitions and metacognitive coping strategies comprising the cognitive attentional syndrome into five domains. As previously discussed (p. 81-83), the studies found that the two control-related metacognitive domains, the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts, were elevated in individuals with AN compared to controls (Cooper et al., 2007; McDermott & Rushford, 2011). Compared to
the other metacognitive domains, effect sizes indicated that the largest differences between AN and control groups were in both the control-related metacognitive domains, indicating the disturbing extent to which individuals with AN were affected by metacognitive level control-related factors (McDermott and Rushford, 2011). The differences between groups remained significant after controlling for BMI, providing support that the association between dysfunctional metacognitions and AN was not due to starvation effects. These findings correspond with Wells’ (2000) proposal that psychological disorders involve increased metacognitive processes, and suggest that control-related metacognitive factors may play an important role in the maintenance of AN. Intriguingly, both these control-related metacognitive factors parallel the control-related cognitive factors proposed to be of importance in Fairburn and colleagues’ (1999) cognitive model of AN, specifically, the general need for control and perceived loss of control. Based on these empirical findings, it appears that the pathological need for control and perceived loss of control experienced by individuals with AN extends to the metacognitive domain.

Unfortunately, as research of metacognitive factors in AN is in its early stages, little existing empirical evidence is available to guide the understanding of maintenance mechanisms of AN that involve metacognitive factors. Woolrich and colleagues’ (2008) qualitative study provided some insight into metacognitive processes that occur in individuals with AN. They found that compared to control groups, the AN group used more unhelpful metacognitive coping strategies compared to control groups (e.g., thought suppression, rumination) and was less successful at using healthy metacognitive coping strategies (e.g., thought re-appraisal). This is consistent with the metacognitive model of psychological disorders (Wells, 2009). Woolrich and colleagues also proposed ways that dysfunctional metacognitions and metacognitive coping strategies could be relevant to maintaining AN. For example, they suggested that patients with AN may hold positive metacognitive beliefs that excessive worrying about their eating is helpful in ensuring that they do not eat too much. Also, as part of the cognitive attentional syndrome in AN, individuals may engage in dysfunctional coping strategies such as strenuous efforts to suppress thoughts about weight or shape, which serve to maintain their disorder (Woolrich et al., 2008). Further, Woolrich and colleagues’ proposed that individuals with dysfunctional metacognitions or metacognitive coping strategies are more likely to turn to behavioural strategies (e.g., dietary restraint) to cope with distressing thoughts, maintaining AN. This is consistent with Fairburn and colleagues’ (1999) cognitive-level theory of AN, which posits that drive for thinness results from
the pathological need for control and perceived loss of control. Additionally, Woolrich and colleagues found that individuals with AN frequently reported using dysfunctional metacognitive strategies to deliberately make themselves feel worse, suggesting that dysfunctional metacognitions may maintain AN through the perpetuation of negative self-evaluation. This also has similarities to Fairburn et al.’s cognitive-level theory, which postulated that low self-esteem was involved in the maintenance of drive for thinness. Whilst these proposed mechanisms are speculative at this stage, they are consistent with existing cognitive theory of AN (Fairburn, Shafran, et al., 1999), and would benefit from empirical testing in an AN-specific metacognitive model.

**Concluding issues.** Due to the lack of evidence for the effectiveness of AN treatment based on CBT, researchers have recommended that more attention should be directed towards the consideration of new variables in developing both theory and treatment interventions for AN (Stice, 2002). Given the demonstrated effectiveness of metacognitive therapy in several psychological disorders, metacognitive theory may hold therapeutic potential for AN (McDermott & Rushford, 2011). Whilst metacognitive dysfunction is a broad vulnerability factor for psychological disorders, different psychological disorders have distinct metacognitive formulations and disorder-specific metacognitive profiles (Wells, 2009). To the knowledge of the researcher, no study has explored the role of metacognition within a cognitive framework of AN due to the infancy of interest in this area. However, the parallels between characteristics of AN and anxiety disorders, and between cognitive and metacognitive control-related factors prominent in AN, bring to light the possibility of adapting an existing cognitive model to a higher level metacognitive model, by shifting the focus from control-related cognitions to higher level control-related metacognitions. This study will develop a novel metacognitive maintenance mechanism of AN, Model B, which will be built on this premise.

### 2.3 The Basis of Model B

Considering the parallels between control-related metacognitive factors prominent in AN and Fairburn and colleagues’ (1999) control-based cognitive model of AN, their cognitive model, namely the Driving Mechanism section, would be an ideal basis from which to develop a metacognitive model of AN. Model B, focusing on control-related dysfunctional metacognitions, will attempt to shift the focus from control-related cognitions to higher level metacognitions. Model B will be adapted from Model A (which is based on Fairburn and colleagues’ proposed Driving Mechanism of
AN), incorporating existing empirical evidence relevant to metacognitions in AN, and maintenance mechanisms proposed by other researchers (e.g., Woolrich et al., 2008). Model B is further described in Section 3.2 (p. 133-134).

3. MODEL TESTING

Whist theories of AN have progressed over time, they remain largely untested in individuals with AN. Cooper (2005) stated that more empirical research is required to examine the proposed causal relationships, mechanisms, or vicious cycles between cognition and eating disordered behaviours that maintain the disorder. She suggested that one way to achieve this was through cross-sectional designs, whilst controlling for the relevant variables statistically. In line with this recommendation, the present study aims to further the understanding of maintenance theories of AN through testing two mechanisms or models, derived from existing theories and empirical evidence. The following points were taken into consideration whilst developing the models. First, a good model should have a level of specification that allows it to be empirically testable. It should also have predictive power (i.e., agree with known facts and future observations) and be consistent with related theories that have a high probability of being true (Cooper, 2005). Finally, it should explain the phenomena in the simplest possible way, containing as few principles as possible (Cooper, 2005). The models to be tested in the present study were developed with these considerations in mind, to provide a clear link between beliefs, cognition, and AN symptomatology. The models will also include direct and indirect effects through mediating variables, which may provide an important point at which therapeutic interventions may be targeted (Baron & Kenny, 1986).

As there is presently a dearth of empirical evidence for existing models of AN, it is not feasible to include all possible factors that are impinging on AN at this initial stage of model testing. Instead, the testing of core elements of proposed models is a more realistic goal. The Driving Mechanism section of Fairburn and colleagues’ (1999) cognitive theory of AN (illustrated in Figure 5.5), centred on the general need for control, is widely accepted to maintain the disorder. However, no existing evidence supports this notion. Hence, Model A will be primarily based on Fairburn and colleagues’ Driving Mechanism, incorporated with Feedback Mechanism 1 (illustrated in Figure 5.6), to test the importance of a control-related maintenance mechanism of AN at the cognitive level. Model B will test Model A at a higher level of cognition, the
metacognitive level, synthesising it with metacognitive theory (Wells, 2000) and existing knowledge about metacognition in EDs. Both models include indirect (or mediation) effects between variables, whereby a third control-related variable (or mediator) explains the process or mechanism by which the independent variable predicts the dependent variable. The main outcome variable for both Models A and B will be drive for thinness, the operational definition of the main symptom of AN: the refusal to maintain minimally normal weight. Both models will now be described.

3.1 MODEL A

As described, Model A was adapted from Fairburn et al.’s (1999) Driving Mechanism (Figure 5.5, described in text on p. 94), involving the interactive relationship between perfectionism and low self-esteem, and their indirect effect on drive for thinness through the mediating variable of the general need for control. Additionally, Fairburn et al.’s Feedback Mechanism 1 (Figure 5.6, described in text on p. 94-95) will be incorporated into Model A. It involves the direct effect of drive for thinness on the individual’s sense of control being dependent on eating/weight, which in turn further perpetuates drive for thinness in a vicious cycle. Whilst this relationship is proposed to be a two-way effect in Fairburn et al.’s model, limitations of the statistical analyses used in the present study, namely path analyses, preclude the testing of feedback loops or bi-directional relationships (Stage, Carter, & Nora, 2004; Blunch 2008). Hence, a causal progression from drive for thinness to the sense of control dependent on eating/weight will be tested in Model A, as it is the initial stage in the bi-directional effect proposed by Fairburn and colleagues.

Figure 5.5. Schematic representation of the Driving Mechanism section from Fairburn et al.’s (1999) cognitive maintenance model of anorexia nervosa.
Taking into account Fairburn and colleagues’ proposed AN maintenance mechanisms and empirical findings from the literature review, Model A was developed. It is schematically outlined in Figure 5.7. The following findings and theory correspond to specific pathways in Figure 5.7:

(i) **Perfectionism and self-esteem covary.** Although there is some evidence that high levels of perfectionism predict low self-esteem (Surgenor et al., 2007) and self-esteem moderates the effect of perfectionism on drive for thinness (Sassaroli et al., 2008), the directionality of this association is speculative. There is a complex interactive relationship between both variables (Davis, 1997). Whilst some researchers argue that perfectionism emerges as a defence against low self-esteem (Bruch, 1982; Slade, 1982), some propose that the inability to achieve perfectionistic standards may instead lead to lowered self-esteem (Surgenor et al., 2007), and others suggest that perfectionism and self-esteem mediate the relationship between other risk factors (e.g., personality) and ED symptomatology (Mas et al., 2011). Due to the lack of clarity on this issue, both will be allowed to covary in Model A, to account for the self-defeating behavioural trap of perfectionism and self-esteem in AN without imposing directional constraints.

(ii) **Perfectionism has a direct effect on drive for thinness.** There is evidence for the direct effect of perfectionism on drive for thinness (Sassaroli et al., 2008) and indirect evidence that targeting perfectionism in interventions leads to improvement in some EDs (Steele & Wade, 2008). Perfectionist or unrelenting standards have been proposed to be applied to aspects of life, including attempts to control eating, shape and weight (Waller, Cordery, et al., 2007), and are possibly applied to dietary restriction in the form of extreme dietary rules (Fairburn, Shafran, et al., 1999).
(iii) **Self-esteem has a direct effect on drive for thinness.** There is evidence that drive for thinness is used by individuals with AN as a coping strategy for low self-esteem (Woolrich et al., 2006; Cooper et al., 1998), that increased self-esteem is associated with decreased drive for thinness (Surgenor et al., 2007), and indirect evidence that targeting self-esteem in treatment trials leads to improvement in ED behaviours including drive for thinness (Newns et al., 2003; O’Dea & Abraham, 1999). Many theorists have proposed that individuals with AN believe that the attainment of a slim figure would alleviate the distress associated with negative self-evaluation, thus maintaining drive for thinness (Garner & Bemis, 1982; Bruch, 1973; Garner & Garfinkel, 1980; Garner et al., 1983; Rieger et al., 2010).

(iv) **Perfectionism and self-esteem have an indirect effect on drive for thinness through the general need for control.** This was the key mechanism theorised in Fairburn and colleagues’ (1999) Driving Mechanism. There is evidence that the effect of perfectionism on drive for thinness is moderated by control-related factors (Sassaroli et al., 2008), and evidence for the complex interactive relationship between the three factors in predicting drive for thinness (Surgenor et al., 2007; Woolrich et al., 2006 Cooper et al., 1998). Low self-esteem has also been linked to AN patients’ experience of not being in control of their behaviours, needs and impulses, feeling instead like they were under the influence of others (Bruch, 1973).

(v) **Drive for thinness has a direct effect on the sense of control being dependent on eating/weight.** This was the key mechanism theorised in Fairburn and colleagues’ Feedback Mechanism 1. There is evidence that drive for thinness is interpreted in terms of control over eating, weight or shape in individuals with EDs (Shafran et al., 2003).
3.2 **MODEL B**

As discussed, two control-related metacognitive domains have been found to be relevant to EDs, the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts (Cooper et al., 2007; McDermott & Rushford, 2011). Both these metacognitive factors will be included in Model B. Taking into account Fairburn and colleagues’ (1999) Driving Mechanism (Figure 5.5, p. 130), Model A (Figure 5.7, on current page), and possible AN metacognitive mechanisms proposed by researchers, Model B was developed and is schematically outlined in Figure 5.8. Put simply, the control-related cognitive factor in Model A (i.e., the general need for control) was replaced by higher level control-related metacognitive factors (i.e., the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts). The following theoretical hypotheses correspond to specific pathways in Figure 5.8:

(i) **Perfectionism and self-esteem covary; perfectionism and self-esteem have a direct effect on drive for thinness.** The theory and evidence for these three pathways were included in the description of Model A (p. 131-132).

(ii) **Perfectionism and self-esteem have an indirect effect on drive for thinness through the need to control thoughts.** These pathways take the original pathways from Model A (i.e., perfectionism and self-esteem predict the general need for control) up to the metacognitive level. This is a realistic adaption as the need for control over thoughts is a component of the general need for control. The need to control thoughts has been found to be of relevance to AN, with studies finding individuals with AN reporting a higher
need to control thoughts compared to controls (Cooper et al., 2007; McDermott and Rushford, 2011). Researchers have also highlighted the complex relationship between dysfunctional metacognitive strategies and the perpetuation of negative self-evaluation (Woolrich et al., 2008) or low self-esteem. Further, Woolrich et al. (2008) proposed that individuals with dysfunctional metacognitions and metacognitive coping strategies are more likely to turn to behavioural coping strategies such as dietary restraint to cope with distressing thoughts, maintaining AN.

(iii) *The need to control thoughts has an indirect effect on drive for thinness through negative beliefs about the uncontrollability/danger of thoughts.* This mediation pathway is a novel concept proposed by the present study. Taking into consideration that both these control-related metacognitive factors are of relevance to AN, the researcher was interested in exploring the mechanisms through which they impact on drive for thinness. Theoretically, it is likely that the strong pathological need to control cognitions results in intrusive thoughts being negatively perceived as uncontrollable or dangerous, which in turn results in dietary restraint. This is consistent with Fairburn and colleagues’ (1999) proposed Feedback Mechanisms 2 and 3, which suggest that a perceived loss of control (in this case, a higher level metacognitive loss of control) maintains drive for thinness. This is also consistent with the theory that due to dysfunctional metacognitions and lack of success with metacognitive coping strategies, individuals with AN are more likely to turn to drive for thinness as a behavioural coping strategy, maintaining AN (Woolrich et al., 2008).

*Figure 5.8. Schematic representation of Model B. Pathways and variables in red indicate additions or alterations to Fairburn et al.’s (1999) proposed theory of anorexia nervosa.*
4. **AIMS AND RESEARCH QUESTIONS**

4.1 **AIMS**

The present study aimed to investigate particular predispositions, cognitions, and metacognitions in AN, to clarify the extent to which they explained the main symptom of AN, the refusal to maintain minimally normal weight, operationalised as drive for thinness. The mediation mechanisms proposed in Model A (Figure 5.7, p. 133) and Model B (Figure 5.8, p. 134) were empirically tested in an AN sample. To elucidate the mechanisms specific to individuals with AN that served to maintain the disorder, Models A and B were also tested in a comparison group of non-eating disordered females, to highlight differences in cognitive and metacognitive mechanisms between the groups. Ultimately, the clinical aim of the present study was to contribute to the theoretical understanding of maintenance mechanisms of AN, from which future optimal therapeutic interventions could be derived.

4.2 **RESEARCH QUESTION 3**

**Research question 3(a).** In (i) the AN group and (ii) the comparison group: Are the proposed individual pathways in Model A (Table 5.5) significant? Do the data provide a good overall fit to Model A?

**Research question 3(b).** In (i) the AN group and (ii) the comparison group: Are the proposed individual pathways in Model B (Table 5.5) significant? Do the data provide a good overall fit to Model B?

<table>
<thead>
<tr>
<th>Table 5.5</th>
<th>Proposed Individual Pathways in Models A and B</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model A</strong></td>
<td><strong>Model B</strong></td>
</tr>
<tr>
<td>• Perfectionism and self-esteem have direct effects on the drive for thinness;</td>
<td>• Perfectionism and self-esteem have direct effects on the drive for thinness;</td>
</tr>
<tr>
<td>• Perfectionism and self-esteem have indirect effects on the drive for thinness through the mediating variable, the general need for control;</td>
<td>• Perfectionism and self-esteem have indirect effects on the drive for thinness through the mediating variable, the need to control thoughts;</td>
</tr>
<tr>
<td>• Drive for thinness has a direct effect on the sense of control dependent on eating/weight.</td>
<td>• The need to control thoughts has an indirect effect on the drive for thinness through the mediating variable, negative beliefs about the uncontrollability/danger of thoughts.</td>
</tr>
</tbody>
</table>
CHAPTER 6: METHOD

This chapter will detail the participants (Section 1), procedure (Section 2), measures used (Section 3) and statistical analyses involved (Section 4).

1. PARTICIPANTS

   AN patient sample. The clinical sample consisted of 110 female patients (66 inpatients, 27 day patients, and 17 outpatients) who met all cognitive/psychological DSM-IV-TR criteria for AN (APA, 2002) and had weight loss sufficient to cause health problems. Fulfilment of the physical AN criteria (weight and amenorrhea) was not required for inclusion in the AN group, as subgroup analyses on the variables of interest in the present study indicated no significant differences between patients who strictly met full AN diagnostic criteria, and patients who met all AN criteria except weight and/or amenorrhea (p. 159). Participants were from two specialist eating disorder services in Melbourne, the Royal Melbourne Hospital and The Melbourne Clinic. Description of the inpatient, day patient, and outpatient treatment modalities are provided in Appendix A. For the AN sample, age ranged from 18 to 45 years, with a mean of 24.4 years ($SD = 6.7$). BMI ranged from 11.3 kg/m$^2$ to 26.8 kg/m$^2$, with a mean of 17.8 kg/m$^2$ ($SD = 3.1$). Of the patient sample, 41 patients (37.3%) were amenorrheic, 37 (33.6%) were menstruating regularly or infrequently, and menstruation status could not be determined for the remaining patients (due to use of the OCP [$n = 28$, 25.5%] or missing data [$n = 4$, 3.6%]). Fifty-two patients (47.3%) had BMI < 17.5 kg/m$^2$ and 58 (52.7%) had BMI $\geq$ 17.5 kg/m$^2$. Patients who were unable to give informed consent because they were mentally or medically compromised, as determined by their treating clinicians, were not approached for participation in the study. Patients on a stable dose of psychotropic medication without change in AN symptoms were included. Patients with comorbid psychiatric disorders were included if their AN was severe enough to stand out as the main diagnosis, as AN populations typically have other serious comorbid psychopathology including depression, anxiety disorders, personality disorders, and substance-use disorders (Agras et al., 2004).

   Non eating-disordered comparison group. For the non-eating disordered comparison group, individuals who reported a current or previous eating disorder ($n = 2$) and who were underweight (BMI < 18.5 kg/m$^2$, $n = 4$) or obese (BMI $\geq$ 30.0 kg/m$^2$, $n = 4$) according to World Health Organisation (2000) guidelines were excluded, to rule
out contamination of the comparison group data from participants with potential eating disorders. Please note that bingeing, purging, or excessive exercise in this group was not evaluated. Hence, it is possible that despite not reporting a current or previous eating disorder, individuals from the comparison group could be engaging in behaviours related to disordered eating. The final comparison sample consisted of 132 non-eating disordered females from the community. Whilst the comparison group was not a true control group (i.e., it was not the same size as the AN group; participants were not pair-matched with participants from the AN group), participants in the comparison group did not differ significantly from the AN group on age, with age ranging from 18 to 45 years with a mean of 25.8 years ($SD = 7.3$). BMI ranged from 18.6 kg/m$^2$ to 29.8 kg/m$^2$, with a mean of 22.3 kg/m$^2$ ($SD = 2.5$). Of the total non-eating disordered sample, 66 (50%) were dieters and 66 (50%) were non-dieters. Dieters and non-dieters were categorised based on their responses to self-report measures relating to demographic and weight-related information, further described in Section 3.1 (p. 139). Specifically, responses to the item, “Do you try to restrict food intake or to diet?”, for which they were given the option of responding “Yes” or “No”, was utilised to differentiate participants into dieting and non-dieting subgroups. With regards to menstrual status, no participants were not menstruating, 70 (53.0%) were menstruating regularly, 8 (6.1%) were menstruating infrequently, and 53 (40.2%) were using the OCP. Menstruation status could not be determined for one participant due to missing data.

**Exclusion criteria.** Individuals below the age of 18 years and above 45 years and males were excluded from the study as their potential representation in the patient sample was too small for meaningful statistical analyses. Participants who were unable to understand, read, or write in English were also excluded.

2. **Procedur**

Ethics approval for the study was obtained from three participating organisations: The Royal Melbourne Hospital, The Melbourne Clinic and the University of Melbourne. All participants were assured of confidentiality through the de-identification of collected data. Recruitment of participants is detailed as follows.

**For the AN group.** Patients were recruited from Eating Disorders Units at the two hospitals. Following assessment and consultation with the ward psychologist and psychiatric registrar, competent patients were invited to participate in the study then
privately interviewed for consent. During the interview, patients were given the Participant Information and Consent Form (Appendix B) and provided with a brief verbal explanation of the study including issues regarding privacy, confidentiality, and disclosure of information. Participation was emphasised as voluntary, confidential, and having no effect on treatment. Consent to participate was given by signing the consent form. Patients who had agreed to participate were provided with a questionnaire pack, which they completed in their own time and returned to the researcher. All patient questionnaires were de-identified and had unique ID numbers. Each pack included an ID for the patient’s record which enabled them to remove their data from the study if they changed their minds about participating.

For the non-eating disordered comparison group. The comparison group was recruited from the community at large, through the distribution of questionnaire packs to first and second-degree acquaintances of the researchers using a snowballing technique. This was done to preserve anonymity due to the sensitivity of the topic. The questionnaire packs included prepaid envelopes, and consent to participate was implicitly given by the posted return of completed questionnaires. The questionnaire packs also included a checklist entitled ‘Is your weight too low for good health?’ (Appendix C) with relevant contact details should participants require help with an ED. All participant questionnaires were anonymous and labelled with ID numbers to allow for participants to remove their data from the study, as above, if they changed their minds about participating.

3. Measures

All information was collected through self-report measures to help protect participant confidentiality. The exception was current weight for patients, which was obtained from medical records, as patients were not informed of their weight during admission for therapeutic reasons. The measures used in the present study constitute a component of a battery of instruments completed by participants as part of a larger study. Instruments not included in this study were the Readiness to Recover visual analogue scales (Rushford, 2006), the Modified Distressing Thoughts Questionnaire (Clark & de Silva, 1985; Clark, Feldman, & Channon, 1989), the Revised Beliefs about Voices Questionnaire (Chadwick, Lees and Birchwood, 2000), and the Young Schema Questionnaire (YSQ; Young & Brown, 2003). Only the instruments relevant to this
study are described. Some instruments were altered or introduced after commencement of data collection, resulting in sample size variation for each instrument.

3.1 DEMOGRAPHIC AND WEIGHT-RELATED INFORMATION

The questionnaire is provided in Appendix D, and included information collected on age, education, employment status, partner status, menstruation status, dieting habits (e.g., “Do you try to restrict food intake or diet?”, “How old were you when you started restricting?”), exercise habits (e.g., “Do you exercise?”, “What is the average time per week you would typically exercise?”), bingeing behaviours (e.g., “Do you have episodes of bingeing? If so, how often?”), purging behaviours (e.g., “Do you have episodes of purging? If so, how often?”, “Have you taken laxatives, purgatives, or diuretics to help you lose or control weight?”), and current or previous eating disorder diagnoses. For the community sample, self-reported height and weight was also collected. Despite the clinically observed tendency towards reporting lower weights, there is evidence for the reliability of self-reported height and weight, with high correlations between actual and self-reported weights ($r = .97$; Attie & Brooks-Gunn, 1989).

3.2 DIAGNOSTIC CRITERIA FOR ANOREXIA NERVOSA

The diagnostic criteria assessed in the present study were the fear of gaining weight, self-worth invested in the body, body image disturbance, and drive for thinness.

Fear of gaining weight and self-worth invested in the body. Fear of gaining weight and self-worth invested in the body, two cognitive diagnostic criteria of AN (APA, 2002), were measured using visual analogue scales corresponding to the items “How afraid are you of gaining weight?” (Rushford, 2006) and “How important are your weight/body shape for how you feel about yourself?” respectively. Each scale was a 100 mm horizontal line with poles labelled “Not at all” and “Completely” corresponding to each item, as illustrated in Figure 6.1. The participant was requested to place a vertical line intersecting each scale at the point that best indicated how she felt about each item. Scales were scored by measuring the distance (to the nearest mm) from the minimal end point to the marked intersection line, ranging from 0 to 100. Higher scores indicated stronger symptom severity. Visual analogue scales have been established as reliable and valid in a range of clinical and research applications (McCormack, Horne, & Sheather, 1988). The fear of gaining weight scale has been shown to be valid and reliable in AN samples, with established concurrent validity ($r =$}
.836) with the Goldfarb Fear of Fat Scale (Goldfarb, Dykens, & Gerrad, 1985), construct validity with the EDI (Garner & Olmsted, 1984), predictive validity with drive for thinness ($r = 0.771$), and test-retest reliability ($r = 0.636$; Rushford, 2006).

![Visual analogue scales](image)

*Figure 6.1.* Visual analogue scales measuring fear of gaining weight and self-worth invested in the body.

**Body image disturbance.** Body image disturbance or distortions, another cognitive diagnostic criteria of AN (APA, 2002), was also measured by a visual analogue scale corresponding to the item “How fat I feel I am” (Rushford & Ostermeyer, 1997). The scale was a horizontal 130 mm line with poles labelled “Skinny” and “Fat” with a central zero, as illustrated in Figure 6.2. Participants were requested to place a vertical line intersecting the scale at the point that best indicated how they felt, and the scale was scored by measuring the distance (to the nearest mm) from the central zero, using BMI as a covariate, and taking the two poles as representing a BMI of 13 kg/m$^2$ and 30 kg/m$^2$. Scores were standardised to range from -1 (*perceived emaciation when obese*) through 0 (*accurate perception of body size*) to 1 (*perceived obesity when emaciated*), through the use of a normative sample. Rushford and Ostermeyer (1997) reported that controls had a mean score of 0.00 ($SD = 0.14$) whilst individuals with AN over-estimated their size, with a mean positively-skewed score of 0.38 ($SD = 0.29$), indicating that individuals with AN had a significantly inflated sense of feeling fatter than other young women, $F(1,17) = 30.6$, $p < .001$, despite being almost six BMI units lighter in the study. Using a visual analogue scale to measure body image disturbance reduced the reliance on external prompts in eliciting responses (e.g., figures or questionnaires), decreasing the probability of experimental bias (Rushford & Ostermeyer, 1997).
Drive for thinness. Behavioural drive for thinness or dietary restraint, the main symptom of AN, was measured using the drive for thinness subscale of the EDI-3 (Garner, 2004; Appendix E), which measures psychological traits or constructs identified to be clinically relevant in individuals with EDs. The drive for thinness scale consists of seven items (e.g., “I think about dieting”, “I feel extremely guilty after overeating”), which participants were required to rate on a 6-point scale, scored as 4 (Always), 3 (Usually), 2 (Often), 1 (Sometimes), and 0 (Rarely and Never), with one item reverse scored. The seven subscale items were totalled to obtain a subscale score ranging from 0 to 28, with higher scores indicating a stronger drive for thinness. Studies have provided empirical support for the psychometric properties of the EDI-3 in eating disordered and control populations, including its factor structure (Clausen, Rosenvinge, Friborg, & Rokkedal, 2011), internal consistency (high .80s to low .90s for the subscales; Garner, 2004), test-retest reliability (.93 to .98 for the subscales; Garner, 2004), convergent and discriminant validity (Clausen et al., 2011; Cumella, 2006), and sensitivity and specificity (Clausen et al., 2011). In the present study, Cronbach’s alpha was .94 for drive for thinness, indicating high internal consistency.

3.3 COMMON EATING DISORDER CHARACTERISTICS

The common ED characteristics assessed in the present study were perfectionism and low self-esteem.

Perfectionism. As the majority of studies assessing perfectionism in EDs have utilised the EDI perfectionism subscale (69%; Bardone-Cone et al., 2007), the present study used the perfectionism subscale of the EDI-3 (Garner, 2004; Appendix E). The subscale consists of six items (e.g., “I hate being less than best at things”, “My parents have expected excellence of me”), which participants were required to rate on a 6-point
scale. Ratings were recoded into 4 (Always), 3 (Usually), 2 (Often), 1 (Sometimes), and 0 (Rarely and Never). The six items were totalled to obtain subscale scores ranging from 0 to 24, with higher scores indicating a higher level of perfectionism. Studies have provided empirical support for the psychometric properties of the EDI-3, as described earlier (p. 141). In the present study, Cronbach’s alpha was .77 for the perfectionism subscale, indicating acceptable internal consistency.

Self-esteem. In the AN literature, self-esteem is commonly measured through self-report with the Rosenberg Self-Esteem Scale (RSES; Rosenberg, 1965; Appendix F). The present study used the RSES, a ten-item questionnaire requiring participants to rate how each item applied to them on a four-point scale (e.g., “I feel that I have a number of good qualities”, “On the whole I am satisfied with myself”). On each item, the following scores were given, 3 (Strongly agree), 2 (Agree), 1 (Disagree), and 0 (Strongly Disagree), with some items reverse scored. Total scores ranged between 0 and 30, with higher scores indicative of higher self-esteem. Many studies have provided evidence of the validity and reliability of the RSES in normative samples (e.g., Rosenberg, 1965; McCarthy & Hoge, 1982), with reliability estimates found to range from .72 to .88 (Bleiler, Boo, & Vispoel, 2001). In ED samples, the RSES has been found to have sounder construct and convergent validity compared to other measures of self-esteem (Griffiths et al., 1999). In the present study, Cronbach’s alpha was .94, indicating a high level of internal consistency.

3.4 CONTROL-RELATED COGNITIONS

Interestingly, although control-related factors have been theorised to play a key role in cognitive models of AN, they have not been incorporated into commonly used measures of ED themes such as the EDI (Garner & Olmsted, 1984) or EDE (Fairburn & Cooper, 1993). Whilst the EDI-3 included an “overcontrol” composite, this was made up of the primary subscales of perfectionism and asceticism. Although somewhat overlapping with control, the EDI-3 perfectionism subscale related to the setting and striving of excessively high personal standards, whilst the asceticism subscale referred to the avoidance of pleasure and human desires. These two subscales forming the EDI-3 overcontrol composite were conceptually different from the two control-related cognitions relevant to this study: the general need for control and sense of control dependent on eating/weight.
The general need for control. There was no intent to assess the general need for control at the beginning of this study, thus no validated measures of this variable were included. However, due to the surprising dearth of empirical research involving this variable, there was an imperative need for investigation of this construct in AN. Hence, a variable for the general need for control was created from relevant existing data, including two EDI-3 items, one MCQ-30 item, and three YSQ items. The YSQ (Young & Brown, 2003) assesses unconditional, schema-level cognitions about oneself and the world, consisting of 75 items on which participants were required to rate on a six-point scale, ranging from 1 (Completely untrue of me) to 6 (Describes me perfectly), with a higher score reflecting a more maladaptive, unhealthy self-belief.

The items forming the general need for control variable are listed in Table 6.1, including items relevant to the need for control over emotions, body, behaviour, interpersonal situations, and cognitions. All the items were rated on a 6-point scale in their original measures except for the MCQ-30 item, which was rated on a 4-point scale. Hence, the MCQ-30 item was rescaled such that original ratings of 1, 2, 3, and 4 were rescored into 1, 2.67, 4.33, and 6 respectively, to scale it in line with the other items. All six items were summed to form the general need for control score, with some items reverse scored. Scores ranged from 6 to 36, with higher scores indicating a greater need for control. For the present study, Cronbach’s alpha was .73, indicating acceptable internal consistency. Results indicated that Cronbach’s alpha was reduced with the removal of any single item, indicating that none of the items was required to be removed from the scale. Evidence for the construct validity of this scale from confirmatory factor analysis is reported in the Results chapter (p. 151-153).

Table 6.1

<table>
<thead>
<tr>
<th>Original measure</th>
<th>Item</th>
<th>Original rating scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDI-3</td>
<td>44: “I worry that my feelings will get out of control”</td>
<td>6-point</td>
</tr>
<tr>
<td>EDI-3</td>
<td>68: “I would like to be in total control of my bodily urges”</td>
<td>6-point</td>
</tr>
<tr>
<td>YSQ-S2</td>
<td>59: “I control myself so much that people say I’m unemotional”</td>
<td>6-point</td>
</tr>
<tr>
<td>YSQ-S2</td>
<td>63: “I must meet all my responsibilities”</td>
<td>6-point</td>
</tr>
<tr>
<td>YSQ-S2</td>
<td>66: “I have a lot of trouble accepting ‘no’ for an answer when I want something from other people.”</td>
<td>6-point</td>
</tr>
<tr>
<td>MCQ-30</td>
<td>27: “I should be in control of my thoughts all the time”</td>
<td>4-point</td>
</tr>
</tbody>
</table>

Note. EDI-3—Eating Disorder Inventory, 3rd Edition (Garner, 2004); YSQ-S2—Young Schema Questionnaire, Short Form (Young & Brown, 2003); MCQ-30—Metacognitions Questionnaire, Brief version (Wells & Cartwright-Hatton, 2004).
Sense of control dependent on eating/weight. To the knowledge of the researcher, this aspect of control did not appear to have been empirically examined previously. The present study utilised a visual analogue scale for the item “To what extent does your sense of control depend on your control of eating/weight?” to measure this construct. The scale was a 100 mm horizontal line, illustrated in Figure 6.3, and was scored by measuring the distance (to the nearest mm) from the minimal end point to the marked intersection line, ranging from 0 to 100. Higher scores indicated stronger symptom severity. Visual analogue scales have been established as reliable and valid in a range of clinical and research applications (McCormack et al., 1988).

![Visual analogue scale measuring sense of control dependent on eating/weight.](image)

Figure 6.3. Visual analogue scale measuring sense of control dependent on eating/weight.

3.5 Dysfunctional metacognitions

The Metacognitions Questionnaire (Cartwright-Hatton & Wells, 1997) and its short-form version, the MCQ-30 (Wells & Cartwright-Hatton, 2004; Appendix G), have been developed to test the metacognitive theory of psychological disorders. The MCQ-30 is more frequently used in research as it is shorter and retains the factor structure of the original Metacognitions Questionnaire (Wells, 2009). It was used to assess the level of dysfunctional metacognitions in the present study, on five separate subscales, positive beliefs about worry, negative beliefs about uncontrollability/danger of thoughts, the need to control thoughts, cognitive confidence, and cognitive self-consciousness. The instrument is made up of 30 items, with six items corresponding to each subscale. Examples of these items are provided in Table 6.2. Participants were required to rate whether each item applied to them on a 4-point scale ranging from 1 (Do not agree) to 4 (Agree very much), and items in each subscale were summed to form subscale scores. Higher scores indicated an increased presence of the corresponding metacognitive belief or strategy. The MCQ-30 has demonstrated construct validity (Spada, Mohiyyeddini, & Wells, 2008). Cronbach’s alphas for the individual subscales were reported to range from .72 to .93 (Wells, 2009) and test-retest reliabilities from .59 to .87 (Wells & Cartwright-Hatton, 2004). McDermott and Rushford (2011) found strong internal
consistency for the MCQ-30 in an AN sample ($\lambda = .98$). For the present study, Cronbach’s alpha ranged from .90 to .95 for the five subscales, indicating a high level of internal consistency.

Table 6.2
Subscales of the MCQ-30 and Examples of Relevant Items

<table>
<thead>
<tr>
<th>Domain</th>
<th>Example of item</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive beliefs about worry</td>
<td>“Worrying helps me cope”</td>
</tr>
<tr>
<td>Negative beliefs about uncontrollability/danger of thoughts</td>
<td>“I could make myself sick with worrying”</td>
</tr>
<tr>
<td>Need to control thoughts</td>
<td>“I should be in control of my thoughts all of the time”</td>
</tr>
<tr>
<td>Cognitive confidence</td>
<td>“I do not trust my memory”</td>
</tr>
<tr>
<td>Cognitive self-consciousness</td>
<td>“I constantly examine my thoughts”</td>
</tr>
</tbody>
</table>


3.6 GENERAL PSYCHOPATHOLOGY

The short-form of the Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995; Appendix H) assessed symptoms of general psychopathology with a 21-item questionnaire measuring three subscales, depression (e.g., “I couldn’t seem to experience any positive feeling at all”), anxiety (e.g., “I experienced breathing difficulty”) and stress (e.g., “I found it difficult to relax”). Participants were required to rate how each item applied to them in the past week on a four-point scale. On each item, the following scores were given, 0 (Did not apply to me at all), 1 (Applied to me to some degree), 2 (Applied to me to a considerable degree), 3 (Applied to me very much). The seven items corresponding to each subscale were summed then doubled to be equivalent to the original 42-item DASS. Higher subscale scores indicated a greater degree of psychopathology. Reliability and validity of the short-form of the DASS has been previously established with normative samples and populations with depressive and anxiety disorders, with reliability in normative samples measured by Cronbach’s alpha reported to be 0.91 for depression, 0.84 for anxiety, and 0.90 for stress (Lovibond & Lovibond, 1995). For the present study, Cronbach’s alpha was .98 for depression, .95 for anxiety, and .97 for stress, indicating high internal consistency.
4. **Statistical Analyses**

The present study employed a cross-sectional design. Analyses were performed using SPSS (Version 20), AMOS (Version 20), and G*Power (Version 3.1).

4.1 **Research Question 1**

*Research Question 1(a).* This research question involved group comparisons of the full syndrome and subthreshold AN groups. Multivariate analysis of variance (MANOVA) required the number of cases in each cell to be larger than the number of dependent variables (DV; Pallant, 2007; Tabachnick & Fidell, 2007). This requirement was not met in the present study ($n = 10$ to 16 in each cell with 16 DVs), resulting in the cells becoming singular, hence Box’s Test of Equality of Covariance Matrices could not be computed. Due to the failed assumption test for homogeneity of group variance-covariance matrices, MANOVA was not used. For Research Question 1(a), separate one-way between-subjects analyses of variance (ANOVA) were used for each variable. When assumptions of normality were violated, the Kruskal-Wallis test, an alternative non-parametric analysis, was carried out. When large numbers of comparisons during statistical analysis are performed, it is usual to set alpha at a more stringent level than $p < .05$ to control for familywise error (e.g., McIntosh et al., 2004, Watson & Andersen, 2003). Although there were many comparisons in the present study, an alpha level of $p < .05$ was used, because it was more important to find a significant difference under the hypothesis than to avoid Type 1 errors, since the present study aimed to determine the salience of the weight and amenorrhea criteria in differentiating the types of EDs. Eta-squared ($\eta^2$) was computed to provide a measure of effect size, with the effect sizes of significant group differences interpreted using .01, .06, and .138 to indicate small, medium and large effects respectively (Cohen, 1988). Post-hoc power analysis was conducted using G*Power.

*Research Question 1(b).* This research question involved group comparisons of the OCP-using and non-OCP-using AN subgroups. MANOVA required the number of cases in each cell to be larger than the number of DVs (Pallant, 2007; Tabachnick & Fidell, 2007). This requirement was not met in the present study ($n = 14$ in one cell with 16 DVs), resulting in the cells becoming singular, hence Box’s Test could not be computed. Due to the failed assumption test for homogeneity of group variance-covariance matrices, MANOVA was not used. Separate independent-samples $t$-tests were used to compare the two groups for Research Question 1(b). When assumptions of normality were violated, the Mann-Whitney U test, an alternative non-parametric
analysis, was carried out. Whilst alpha is usually set at a more stringent level when large
numbers of comparisons during statistical analysis are performed, due to the exploratory
nature of this research question, an alpha level of \( p < .05 \) was used. In accordance
with Cabin and Mitchell’s (2000) recommendations for dealing with multiple tests of
statistical significance, no statistical control was applied to the data set as this can
reduce the purpose and meaning of exploratory studies. \( \eta^2 \) was computed to provide a
measure of effect size, with the effect sizes of significant group differences interpreted
using .01, .06, and .138 to indicate small, medium and large effects respectively (Cohen,
1988).

4.2 Research Question 2

This research question involved group comparisons of the AN, dieting, and non-
dieting comparison groups, whilst controlling for the effects of low weight, depression,
and anxiety. For the group comparisons, a MANOVA was used for Research Question
2(a), multivariate analyses of covariance (MANCOVAs) were used for Research
Questions 2(b) and (d), and an analysis of covariance (ANCOVA) was used for
Research Question 2(c). Due to the large number of comparisons on Research Questions
2(a) and (b), separate univariate \( F \)-tests were evaluated at Bonferroni-adjusted alpha
levels of \( p < .006 \) and \( p < .008 \) respectively. For Research Questions 2(c) and (d), an
alpha level of \( p < .05 \) was used. Partial eta-squared (\( \eta_{p}^2 \)) was reported for all analyses,
to provide a measure of effect size, with the effect sizes of significant group differences
interpreted using .01, .06, and .138 to indicate small, medium and large effects
respectively (Cohen, 1988). For the MANOVA in Research Question 2(a), Tukey post-
hoc comparisons were conducted to determine which specific group mean differences
were statistically significant.

4.3 Research Question 3

Research Questions 3(a) and (b) involved the testing of Models A and B in AN
and non-eating disordered comparison groups. Prior to testing of the models, Pearson
correlations were undertaken to explore relationships between the variables in Models A
and B, and were determined to be significant at \( p < .01 \). The size of the correlations
were interpreted according to Cohen’s (1988) guidelines, with .10, .30, and .50
indicating small, medium and large correlations respectively.

Path analysis, an extension of multiple regression models and a variant of
structural equation modelling, was used to test the models. Path analysis takes a
confirmatory (i.e., hypothesis testing) approach to the multivariate analysis of an *a priori* proposed theory (Byrne, 2010). Through this method, the causal associations under investigation are represented by a series of structural (i.e., regression) equations, and these structural equations can be modeled pictorially to allow for a clear conceptualisation of the theory under study. In Model A (Figure 5.7, p. 133) and Model B (Figure 5.8, p. 134), the observed variables are represented by boxes, with perfectionism and self-esteem as exogenous variables (i.e., influenced by factors external to the model), and the other variables, including the main outcome variable of drive for thinness, endogenous variables (i.e., dependent variables, influenced either directly or indirectly by variables in the model).

In path analysis or structural equation modelling, there is no consensus among researchers as to what constitutes an appropriate sample size. Although some recommend a rule of thumb of $N > 200$ (e.g., Hair, Anderson, Tatham, & Black, 1999; Boomsma & Hoogland, 2001), researchers have argued that this is conservative and simplistic, finding that structural equation models can perform well even with small samples (i.e., $N = 50$ to $100$; Iacobucci, 2010). The required sample size depends largely on the complexity of the path model, with more complex models (i.e., those with more parameters) requiring larger sample sizes than do parsimonious models, for which smaller samples suffice (Kline, 2005; Bollen, 1990). Although there is no absolute standard in the literature, it is recommended that a realistic target is for the ratio of number of participants to number of model parameters to be 10:1, with the statistical ability of results doubtful when the ratio is less than 5:1 (Kline, 2005; Bentler & Chou, 1987). Although structural equation modelling is a more powerful method than path analysis as it controls for measurement error, structural equation models of Models A and B would increase the complexity and number of model parameters, hence required sample size. Conventional wisdom implies that researchers should limit the number of parameters to be considered when sample size is small (Hau & Marsh, 2004). Due to the relatively small sample size of the present study (AN group, $n = 110$; non-eating disordered comparison group, $n = 132$), path analysis was performed in the present study instead of structural equation modelling.

Path analysis can be conducted by two approaches, step-by-step model fitting using separate multiple regression equations for each endogenous variable, or one-step estimation with a model-fitting program. The latter is preferred as it solves for all coefficients simultaneously through an iterative procedure, provides overall fit indices,
provides indirect and total effects of the predictor variables (Meyers, Gamst, & Guarino, 2006; Kline, 2005), and allows for all predicted relationships to be statistically controlled for and partialled out, producing more consistent estimates (Iacobucci, 2008). Hence, path analyses using maximum likelihood estimation with AMOS were carried out to test Models A and B. Individual path coefficients were assessed for statistical significance at \( p < .05 \). Whilst AMOS automatically provides the statistical significance of direct effects, this is not done for combined indirect (or mediation) effects. Bootstrapping was implemented in AMOS to derive the statistical significance of the indirect effects in the models. Bootstrapping is a resampling technique that takes a large number of samples from the data, sampling with replacement, and deriving confidence intervals for the mediated effects based on the bootstrapped sampling distribution (Byrne, 2010). Whilst other methods can be used for assessing indirect (or mediation) effects (e.g., Baron and Kenny’s [1986] causal steps approach or the Sobel [1982] test), these methods have been criticised for having low statistical power and Type I error rates (MacKinnon, Lockwood, Hoffman, & West, 2002), not being able to address suppression effects (Shrout & Bolger, 2002), not providing tests of statistical significance of the mediation effect, and requiring large sample sizes and normality of the sampling distribution (Preacher & Hayes, 2004). Bootstrapping was found to be more accurate than these traditional mediation analyses (Preacher & Hayes, 2004; Bollen & Stine, 1990), and can be applied to small samples with confidence (i.e., \( N = 20 \) to \( 80 \); Efron & Tibshirani, 1993). To assess the significance of indirect effects, AMOS was requested to perform a bootstrap on 2000 samples using the maximum likelihood estimator, and to provide bias-corrected 95% confidence intervals (CIs) and \( p \)-values (two-tailed) for each of the parameter bootstrap estimates.

The overall fit of the hypothesised models was evaluated using the following indices reported by AMOS: (i) the chi-square test, (ii) the comparative fit index, and (iii) the root mean square error of approximation. Chi-square \( (\chi^2) \) tests the null hypothesis of perfect fit, indicating the amount of difference between the expected and observed covariance matrices. A small \( \chi^2 \) value that is non-significant \( (p > .05) \) indicates a close fit between the hypothesised model and perfect fit (Byrne, 2010). The comparative fit index (CFI) assesses the fit of the proposed model relative to the independence model, which assumes that there are no relationships in the data. It ranges from 0 to 1, with acceptable model fit indicated by a value of .95 or greater (Hu & Bentler, 1999). The root mean square error of approximation (RMSEA) is a fit index
that takes into account the error of approximation in the population (Byrne, 2010). It ranges from 0 to 1, with values less than .05 indicating good fit, values less than .08 indicating reasonable errors of approximation in the population, values of .08 to .10 indicating mediocre fit, and values above .10 indicating unacceptable fit (Browne & Cudeck, 1993).
CHAPTER 7: RESULTS

This chapter presents results from preliminary analyses (Section 1), followed by results for Research Questions 1 to 3 (Sections 2 to 4), with findings from the relevant tests of assumptions for univariate and multivariate analyses.

1. PRELIMINARY ANALYSES

Prior to analyses, variables were examined for accuracy of data entry and missing values (Section 1.1). Validation of the general need for control variable was tested with confirmatory factor analysis (Section 1.2). Before determining the final patient and comparison groups, differences between subgroups within the patient and non-eating disordered comparison groups were assessed for BMI and AN diagnostic criteria of fear of gaining weight, self-worth invested in the body, body image disturbance, and drive for thinness (Sections 1.3 and 1.4). Descriptive statistics for the final patient and comparison groups were presented (Section 1.5).

1.1 MISSING DATA

There was a low proportion of missing data (4.79%) on the variables and missing values analysis indicated that data were “missing completely at random” as described by Little and Rubin (1987), indicating that most methods of imputing missing data could be used accurately (Tabachnick & Fidell, 2007; Roth, 1994). For replacing missing data in Likert scales, person mean substitution (i.e., estimating missing items from each individual’s responses to other items in the scale) was reported to be the most efficient and accurate method if < 20% of items were missing (Hawthorne & Elliot, 2005; Downey & King, 1998). Person mean substitution was conducted to increase statistical power in the present study. Due to sample size variation between the data collected for each instrument, sample sizes differed between analyses and are reported in each section.

1.2 VALIDATION OF NEWLY CREATED “GENERAL NEED FOR CONTROL” VARIABLE

To test the factor structure of the newly developed general need for control variable measured by six items (refer to Table 6.1, p. 143), confirmatory factor analysis was conducted with AMOS. This method was preferred as it is a theory-based approach and provides goodness-of-fit statistics, compared to other more data-driven methods such as principal components analysis or exploratory factor analysis (Meyers et al.,
Researchers have cautioned that imputation strategies for missing data prior to factor analyses will artificially increase the clarity of factor structure, recommending instead that listwise or pairwise deletion of missing data should be used in factor analyses (Roth, 1994). In line with these recommendations, due to sample size variation between the data collected for each instrument in the study, only participants with complete data for the six items prior to imputation of missing data were included in the analysis (N = 148). Prior to analysis, the variables were tested for possible violations of univariate and multivariate normality. No violations of assumptions were noted, indicated by Mardia’s test of multivariate kurtosis, with an acceptable range of multivariate kurtosis indicated by a critical ratio (c.r.) within the range of ±1.96 (Mardia’s coefficient = 1.20, c.r. = 0.74). No outliers were identified (highest Mahalanobis $d^2$ value = 19.89, $p^2 = .35$). Requirements for model identification were met and the model was overidentified.

The hypothesised model in Figure 7.1 with six indicator variables (or questionnaire items) loading on one factor (the general need for control) was specified in AMOS and assessed by maximum likelihood estimation. All three fit indices indicated excellent fit of the proposed model to the data, (i) $\chi^2 = 9.98$, $df = 9$, $p = .354$, (ii) CFI = .99, (iii) RMSEA = .027. Standardised regression weights and variance explained for each indicator are reported in Figure 7.1. Regression weights for all six indicator variables were statistically significant ($p < .001$), indicating the importance of all six variables to the model. These results provide evidence for the construct validity of the general need for control variable in the present study.
1.3 **COMBINING PATIENT GROUPS: INPATIENTS, DAY PATIENTS, AND OUTPATIENTS**

Mean BMI and scores on AN diagnostic criteria for the AN inpatients (n = 66), day patients (n = 27), and outpatients (n = 17) are detailed in Table 7.1. Preliminary assumption testing for univariate and multivariate analyses was conducted, with no serious violations noted. A one-way ANOVA with BMI as dependent variable revealed statistically significant differences between the patient groups, $F(2,107) = 11.39, p < .001$. The effect size ($\eta^2 = .18$) was large (Cohen, 1988). Post-hoc comparisons using Tukey HSD indicated that the mean BMI for inpatients ($M = 16.8$ kg/m$^2$, $SD = 2.5$) was significantly lower than day patients ($M = 19.6$ kg/m$^2$, $SD = 3.1$) and outpatients ($M = 18.9$ kg/m$^2$, $SD = 3.5$), which did not differ significantly from each other. This is expected as inpatients tend to be more severely underweight and physically compromised, thus requiring hospitalisation. In a MANCOVA with BMI as covariate, group comparisons of the combined diagnostic criteria indicated no statistically significant differences between the three groups ($F(8, 198) = 0.71, p = .681$, Pillai’s Trace = 0.06, $\eta_p^2 = .03$). Inpatients, day patients, and outpatients were therefore combined into a single AN patient group in the present study.
Table 7.1
Mean BMI and Scores on AN Diagnostic Criteria for Inpatients, Day Patients, and Outpatients

<table>
<thead>
<tr>
<th></th>
<th>Inpatients</th>
<th>Day Patients</th>
<th>Outpatients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 66)</td>
<td>(n = 27)</td>
<td>(n = 17)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>16.8 (2.5)</td>
<td>19.6 (3.1)</td>
<td>18.9 (3.5)</td>
</tr>
<tr>
<td>Diagnostic criteria</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fear of gaining weight</td>
<td>77.9 (23.9)</td>
<td>80.5 (25.2)</td>
<td>82.6 (19.1)</td>
</tr>
<tr>
<td>Self-worth invested in the body</td>
<td>75.8 (17.9)</td>
<td>83.7 (15.8)</td>
<td>74.4 (25.4)</td>
</tr>
<tr>
<td>Body image disturbance</td>
<td>0.44 (0.25)</td>
<td>0.34 (0.27)</td>
<td>0.35 (0.25)</td>
</tr>
<tr>
<td>Drive for thinness</td>
<td>19.5 (7.6)</td>
<td>20.4 (6.6)</td>
<td>20.5 (6.2)</td>
</tr>
</tbody>
</table>

Note. AN—Anorexia Nervosa; BMI—Body mass index.

1.4 Differentiating Comparison Groups: Dieters and Non-Dieters

Mean scores for the dieters (n = 66) and non-dieters (n = 66) in the comparison group on BMI and AN diagnostic criteria are detailed in Table 7.2. As there were violations to the assumption of homogeneity of variance-covariance matrices for MANOVA indicated by Box’s Test, separate univariate ANOVAs were conducted to compare both groups. Levene’s test indicated that the assumption of homogeneity of variances was violated for BMI and drive for thinness. However, alternative robust tests of equality of means (Welch and Brown-Forsythe) produced similar significant results, confirming the robustness of the effects. ANOVA results are shown in Table 7.2, indicating that there were statistically significant differences between the dieters and non-dieters for BMI (F(1,130) = 7.22, p = .008), fear of gaining weight (F(1,130) = 13.61, p < .001), and drive for thinness (F(1,127) = 38.74, p < .001), all of which were higher in the dieters compared to non-dieters. Although the effect size for BMI was small (η² = .05), the effect sizes for fear of gaining weight (η² = .09) and drive for thinness (η² = .23) were medium and large respectively (Cohen, 1988). Because the dieters and non-dieters were different on these clinically significant AN diagnostic criteria, they were distinguished and treated as separate comparison groups in the present study.
## Table 7.2
### Mean Scores and ANOVA Results for BMI and AN Diagnostic Criteria for Dieting and Non-Dieting Comparison Groups

<table>
<thead>
<tr>
<th></th>
<th>Dieters (n = 66)</th>
<th>Non-Dieters (n = 66)</th>
<th>df</th>
<th>F</th>
<th>p</th>
<th>η²</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BMI (kg/m²)</strong></td>
<td>22.9 (2.7)</td>
<td>21.8 (2.1)</td>
<td>1, 130</td>
<td>7.22</td>
<td>.008*</td>
<td>.05</td>
</tr>
<tr>
<td><strong>Diagnostic criteria</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fear of gaining weight</td>
<td>59.1 (25.4)</td>
<td>42.1 (27.5)</td>
<td>1, 130</td>
<td>13.61</td>
<td>&lt;.001*</td>
<td>.09</td>
</tr>
<tr>
<td>Self-worth invested in the body</td>
<td>68.8 (18.8)</td>
<td>63.7 (16.8)</td>
<td>1, 130</td>
<td>2.74</td>
<td>.100</td>
<td>.02</td>
</tr>
<tr>
<td>Body image disturbance</td>
<td>-0.02 (0.18)</td>
<td>-0.02 (0.19)</td>
<td>1, 130</td>
<td>0.00</td>
<td>.992</td>
<td>.00</td>
</tr>
<tr>
<td>Drive for thinness</td>
<td>10.6 (7.6)</td>
<td>3.8 (4.4)</td>
<td>1, 127</td>
<td>38.74</td>
<td>&lt;.001*</td>
<td>.23</td>
</tr>
</tbody>
</table>

**Note.** *p < .01. ANOVA—Analysis of variance; BMI—Body mass index; AN—Anorexia Nervosa.

### 1.5 Descriptive Statistics

Table 7.3 presents descriptive statistics for the demographics, AN diagnostic criteria, and general psychopathology of the final comparison groups, AN (n = 110), dieters (n = 66), and non-dieters (n = 66). As the data violated the assumption of homogeneity of variance-covariance matrices for MANOVA indicated by Box’s Test, separate univariate ANOVAs were conducted to compare the three groups. Levene’s test indicated that the assumption of homogeneity of variances was violated for BMI, body image disturbance, drive for thinness, depression, anxiety, and stress. However, alternative robust tests of equality of means (Welch and Brown-Forsythe) produced similar significant results, confirming the robustness of the effects. ANOVA results are reported in Table 7.3, indicating that with the exception of age, there were statistically significant differences between the three groups on all clinical characteristics (*p < .001*). According to Cohen’s guidelines (1988), effect sizes were large on all variables (η² > .138) except self-worth invested in the body, which had a medium effect size (η² = .10). Tukey post-hoc comparisons indicated that group differences were in the expected direction, with the AN group having significantly lower BMI and higher fear of gaining weight, self-worth invested in the body, body image disturbance, drive for thinness, and general psychopathology (depression, anxiety, and stress) compared to dieting and non-dieting comparison groups (*p < .05*). Additionally, the dieting comparison group also had significantly higher BMI, fear of gaining weight, and drive for thinness compared to non-dieting comparison group (*p < .05*). Dieting and non-dieting comparison groups did not differ on self-worth invested in the body, body image disturbance, or general psychopathology.
Table 7.3
Mean Scores and Univariate ANOVA Results for Demographic and Clinical Characteristics of AN, Dieting, and Non-Dieting Comparison Groups

<table>
<thead>
<tr>
<th></th>
<th>AN (n = 110)</th>
<th>Dieters (n = 66)</th>
<th>Non-dieters (n = 66)</th>
<th>df</th>
<th>F</th>
<th>p</th>
<th>( \eta^2 )</th>
<th>Post-hoc tests (Tukey HSD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>24.4 (6.7)</td>
<td>25.1 (7.2)</td>
<td>26.4 (7.4)</td>
<td>2, 239</td>
<td>1.71</td>
<td>&lt;.001*</td>
<td>.01</td>
<td>-</td>
</tr>
<tr>
<td>BMI (kg/m(^2))</td>
<td>17.8 (3.1)</td>
<td>22.9 (2.7)</td>
<td>21.8 (2.1)</td>
<td>2, 239</td>
<td>84.19</td>
<td>&lt;.001*</td>
<td>.41</td>
<td>AN &lt; ND &lt; D</td>
</tr>
<tr>
<td>Diagnostic criteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fear of gaining weight</td>
<td>79.3 (23.4)</td>
<td>59.1 (25.4)</td>
<td>42.1 (27.5)</td>
<td>2, 239</td>
<td>46.43</td>
<td>&lt;.001*</td>
<td>.28</td>
<td>AN &gt; D &gt; ND</td>
</tr>
<tr>
<td>Self-worth invested in the body</td>
<td>77.5 (18.9)</td>
<td>68.8 (18.8)</td>
<td>63.7 (16.8)</td>
<td>2, 239</td>
<td>12.63</td>
<td>&lt;.001*</td>
<td>.10</td>
<td>AN &gt; D, ND</td>
</tr>
<tr>
<td>Body image disturbance</td>
<td>0.40 (0.26)</td>
<td>-0.02 (0.18)</td>
<td>-0.02 (0.19)</td>
<td>2, 239</td>
<td>108.01</td>
<td>&lt;.001*</td>
<td>.47</td>
<td>AN &gt; D, ND</td>
</tr>
<tr>
<td>Drive for thinness</td>
<td>19.9 (7.1)</td>
<td>10.6 (7.6)</td>
<td>3.8 (4.4)</td>
<td>2, 231</td>
<td>124.27</td>
<td>&lt;.001*</td>
<td>.52</td>
<td>AN &gt; D &gt; ND</td>
</tr>
<tr>
<td>General psychopathology</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>25.8 (12.0)</td>
<td>8.1 (7.6)</td>
<td>5.3 (8.4)</td>
<td>2, 239</td>
<td>109.69</td>
<td>&lt;.001*</td>
<td>.48</td>
<td>AN &gt; D, ND</td>
</tr>
<tr>
<td>Anxiety</td>
<td>20.1 (11.0)</td>
<td>5.8 (6.3)</td>
<td>3.9 (5.1)</td>
<td>2, 239</td>
<td>95.17</td>
<td>&lt;.001*</td>
<td>.44</td>
<td>AN &gt; D, ND</td>
</tr>
<tr>
<td>Stress</td>
<td>28.0 (10.3)</td>
<td>13.0 (9.9)</td>
<td>9.5 (8.3)</td>
<td>2, 239</td>
<td>91.43</td>
<td>&lt;.001*</td>
<td>.44</td>
<td>AN &gt; D, ND</td>
</tr>
</tbody>
</table>

Note. * p < .001. AN—Anorexia Nervosa; BMI—Body mass index; D—Dieters, ND—Non-dieters.
2. **Research Question 1**

This research question involved group comparisons of full syndrome and subthreshold AN subgroups, and OCP-taking and non-OCP-taking AN subgroups.

2.1 **Analysis Specific Participants**

In the AN patient group, 106 participants had complete data regarding weight, amenorrhea, and use of OCP. Five subgroups were identified and summarised in Table 7.4. The subgroups were meeting full AN criteria (A: \(n = 28\)); subthreshold, not meeting amenorrhea criterion (B: \(n = 11\)); subthreshold, not meeting weight criterion (C: \(n = 13\)); subthreshold, not meeting both weight and amenorrhea criteria (D: \(n = 26\)); and using the OCP (E: \(n = 28\)).

<table>
<thead>
<tr>
<th>Meet amenorrhea criterion?</th>
<th>Meet weight criterion?</th>
<th>A: Full criteria</th>
<th>B: Subthreshold, not meeting amenorrhea</th>
<th>C: Subthreshold, not meeting weight</th>
<th>D: Subthreshold, not meeting both</th>
<th>E: Using OCP</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td></td>
<td>28</td>
<td></td>
<td></td>
<td></td>
<td>12</td>
<td>51</td>
</tr>
<tr>
<td>No</td>
<td></td>
<td>13</td>
<td></td>
<td></td>
<td></td>
<td>16</td>
<td>55</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>41</td>
<td>37</td>
<td>28</td>
<td></td>
<td>106</td>
<td></td>
</tr>
</tbody>
</table>

*Note. AN—Anorexia Nervosa; OCP—Oral contraceptive pill.*

**Research Question 1(a).** Participants who reported using the OCP (\(n = 28\)) were excluded from the analyses for Research Question 1(a), resulting in a final sample size of 78. Due to the relatively small sample sizes of subgroups B and C, these groups were combined to increase power, forming a new group, *subthreshold, not meeting one criterion*. The three final groups of AN patients used for analyses for Research Question 1(a) are outlined in Table 7.5. They were meeting full criteria (\(n = 28\)); subthreshold, not meeting one criterion (\(n = 24\)); and subthreshold, not meeting both criteria (\(n = 26\)). To extract the maximum information from the data, all variables were tested within the full data set of \(N = 78\), with cases with missing values excluded by analysis for each variable according to the completeness of data.
Table 7.5
Final Three Groups Used in Analyses for Research Question 1(a)

<table>
<thead>
<tr>
<th>AN Criteria Met</th>
<th>Full criteria</th>
<th>Subthreshold, Missing One</th>
<th>Subthreshold, Missing Both</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Amenorrhea</td>
<td>Weight</td>
<td></td>
</tr>
<tr>
<td>n =</td>
<td>28</td>
<td>24</td>
<td>26</td>
</tr>
</tbody>
</table>

Note. AN—Anorexia Nervosa.

Research Question 1(b). The two groups of AN patients in analyses for Research Question 1(b) were participants who reported using the OCP (here on referred to as the OCP group; n = 28) and participants who reported not using the OCP (here on referred to as the non-OCP group; n = 78). To extract the maximum information from the data, all variables were tested within the full data set of N = 106, with cases with missing values excluded by analysis for each variable according to the completeness of data.

2.2 ASSUMPTION TESTING

Research Question 1(a). Variables were tested for possible violation of assumptions for using parametric tests in the sample. Upon inspection of histograms, normal probability plots, and tests of normality, distributions of fear of gaining weight, self-worth invested in the body, positive beliefs about worry, and depression appeared to violate the assumption of normality, thus the Kruskal-Wallis test was carried out for these variables. However, as the Kruskal-Wallis test resulted in similar conclusions for these variables (i.e., no evidence to reject the null hypotheses), statistical findings from ANOVA were reported to allow for further information (e.g., effect sizes) to be computed. Visual inspection of boxplots indicated that outliers were randomly distributed across groups and no action was taken as they were not found to influence overall results. Levene’s test of homogeneity of variances indicated equal variances across groups for all variables (p > .05).

Research Question 1(b). Variables were tested for possible violation of assumptions for using parametric tests in the sample. Upon inspection of histograms, normal probability plots, and tests of normality, distributions of fear of gaining weight, self-worth invested in the body, and positive beliefs about worry appeared to violate the assumption of normality, thus the Mann-Whitney U test was carried out for these variables. However, as the Mann-Whitney U test resulted in similar conclusions for
these variables (i.e., no evidence to reject the null hypotheses), statistical findings from ANOVA were reported as these are more informative. Visual inspection of boxplots indicated that outliers were randomly distributed across groups and no action was taken as these were not found to influence overall results. Levene’s test for equality of variances indicated equal variances across groups for all variables \((p > .05)\), hence the “equal variances assumed” output from \(t\)-tests were reported.

2.3  **Research Question I(a): Univariate ANOVA & post-hoc power analysis results**

*Univariate ANOVA results.* One-way between-groups ANOVA was conducted to compare the AN groups meeting full criteria, subthreshold (not meeting one criterion), and subthreshold (not meeting both criteria) on the variables of clinical interest. Group means and ANOVA results are reported in Table 7.6. Interestingly, comparing the means, the subthreshold, not meeting one criterion group had more highly pathological scores than the other two groups on a majority of variables. However, none of the group differences reached statistical significance \((p < .05)\), with ANOVA results indicating no significant differences between the three groups on any of the clinical characteristics and variables of interest. This included measures of diagnostic criteria, perfectionism, low self-esteem, control-related cognitions, dysfunctional metacognitions, and general psychopathology.

*Post-hoc power analyses.* Computed between-groups comparison effect sizes (Cohen’s \(f\)) ranged from .05 to .31. A post-hoc power analysis using G*Power revealed that power for one-way ANOVAs in the present study (Cohen’s \(f = .05\) to \(.31\), \(\alpha = .05\), \(N = 78\), \(k = 3\)) ranged from .06 to .67. To detect a medium-sized effect (Cohen’s \(f = .25\)) at the recommended statistical power level of .80 (Cohen, 1988), the required sample size would have to be \(N = 159\) in the present study. Hence, the current study was underpowered to detect medium- or small-sized effects.
Table 7.6
Mean Scores and Univariate ANOVA Results for Full Syndrome and Subthreshold AN Groups on Variables of Interest

<table>
<thead>
<tr>
<th></th>
<th>Meeting full criteria (n = 28)</th>
<th>Subthreshold, missing one (n = 24)</th>
<th>Subthreshold, missing two (n = 26)</th>
<th>df</th>
<th>F</th>
<th>p</th>
<th>η²</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diagnostic criteria</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fear of gaining weight</td>
<td>73.9 (26.1)</td>
<td>86.3 (19.2)</td>
<td>80.0 (24.3)</td>
<td>2.75</td>
<td>1.78</td>
<td>.18</td>
<td>.05</td>
</tr>
<tr>
<td>Self-worth invested in the body</td>
<td>76.0 (18.0)</td>
<td>79.5 (17.6)</td>
<td>79.2 (17.0)</td>
<td>2.75</td>
<td>.32</td>
<td>.72</td>
<td>.01</td>
</tr>
<tr>
<td>Body image disturbance</td>
<td>0.49 (0.27)</td>
<td>0.44 (0.26)</td>
<td>0.34 (0.22)</td>
<td>2.75</td>
<td>2.50</td>
<td>.09</td>
<td>.06</td>
</tr>
<tr>
<td>Drive for thinness</td>
<td>18.1 (8.4)</td>
<td>21.7 (5.3)</td>
<td>19.4 (6.9)</td>
<td>2.71</td>
<td>1.60</td>
<td>.21</td>
<td>.04</td>
</tr>
<tr>
<td><strong>Common ED characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perfectionism</td>
<td>13.2 (6.3)</td>
<td>10.9 (4.0)</td>
<td>12.9 (5.1)</td>
<td>2.45</td>
<td>0.68</td>
<td>.51</td>
<td>.03</td>
</tr>
<tr>
<td>Self-esteem</td>
<td>11.7 (5.6)</td>
<td>7.7 (5.4)</td>
<td>10.8 (3.8)</td>
<td>2.47</td>
<td>2.36</td>
<td>.11</td>
<td>.09</td>
</tr>
<tr>
<td><strong>Control-related cognitions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>General need for control</td>
<td>22.2 (6.8)</td>
<td>25.2 (4.3)</td>
<td>23.5 (5.7)</td>
<td>2.41</td>
<td>0.83</td>
<td>.44</td>
<td>.04</td>
</tr>
<tr>
<td>Sense of control dependent on eating/weight</td>
<td>75.3 (22.4)</td>
<td>71.8 (27.5)</td>
<td>62.4 (26.0)</td>
<td>2.48</td>
<td>1.30</td>
<td>.28</td>
<td>.05</td>
</tr>
<tr>
<td><strong>Dysfunctional metacognitions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive beliefs about worry</td>
<td>10.6 (5.0)</td>
<td>11.9 (5.7)</td>
<td>10.1 (4.7)</td>
<td>2.75</td>
<td>0.87</td>
<td>.42</td>
<td>.02</td>
</tr>
<tr>
<td>Negative beliefs about thoughts</td>
<td>17.1 (5.2)</td>
<td>19.3 (3.9)</td>
<td>17.1 (5.3)</td>
<td>2.75</td>
<td>1.73</td>
<td>.19</td>
<td>.04</td>
</tr>
<tr>
<td>Need to control thoughts</td>
<td>15.6 (5.0)</td>
<td>16.6 (5.2)</td>
<td>15.5 (6.3)</td>
<td>2.75</td>
<td>0.33</td>
<td>.72</td>
<td>.01</td>
</tr>
<tr>
<td>Cognitive confidence</td>
<td>12.0 (4.9)</td>
<td>14.3 (5.0)</td>
<td>13.5 (4.6)</td>
<td>2.75</td>
<td>1.61</td>
<td>.21</td>
<td>.04</td>
</tr>
<tr>
<td>Cognitive self-consciousness</td>
<td>17.3 (4.2)</td>
<td>19.0 (4.4)</td>
<td>16.9 (4.5)</td>
<td>2.75</td>
<td>1.54</td>
<td>.22</td>
<td>.04</td>
</tr>
<tr>
<td><strong>General psychopathology</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>25.3 (12.7)</td>
<td>29.0 (11.7)</td>
<td>27.3 (10.1)</td>
<td>2.75</td>
<td>0.65</td>
<td>.53</td>
<td>.02</td>
</tr>
<tr>
<td>Anxiety</td>
<td>18.1 (11.0)</td>
<td>22.7 (10.2)</td>
<td>20.7 (11.3)</td>
<td>2.75</td>
<td>1.19</td>
<td>.31</td>
<td>.03</td>
</tr>
<tr>
<td>Stress</td>
<td>28.0 (11.5)</td>
<td>29.1 (10.3)</td>
<td>28.1 (9.1)</td>
<td>2.75</td>
<td>0.10</td>
<td>.91</td>
<td>.00</td>
</tr>
</tbody>
</table>

*Note.* No comparisons were significant at $p < .05$. AN—Anorexia Nervosa; ANOVA—Analysis of variance.
2.4 RESEARCH QUESTION 1(B): INDEPENDENT SAMPLES T-TEST RESULTS

Independent samples t-tests were conducted to compare the two AN subgroups, OCP and non-OCP, on the variables of clinical interest. Group means and t-test results are reported in Table 7.7. Group differences reached statistical significance for only two variables, cognitive confidence ($t(104) = -2.43, p = .017$) and depression ($t(104) = -2.22, p = .029$), for which the non-OCP group had significantly greater pathological scores. The magnitude of differences between the means for cognitive confidence (mean difference = -5.59, 95% CI [-4.70, -0.48]) and depression (mean difference = -5.73, 95% CI [-10.86, -0.61]) were small ($\eta^2 = .054$ and .045 respectively). On all other measures of diagnostic criteria, common ED characteristics, control-related cognitions, dysfunctional metacognitions, or general psychopathology, none of the group differences reached statistical significance for $p < .05$.

<table>
<thead>
<tr>
<th>Variable</th>
<th>OCP (n = 28)</th>
<th>Non-OCP (n = 78)</th>
<th>df</th>
<th>t</th>
<th>p</th>
<th>$\eta^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnostic criteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fear of gaining weight</td>
<td>77.4 (23.9)</td>
<td>79.8 (23.8)</td>
<td>104</td>
<td>-0.46</td>
<td>.65</td>
<td>.00</td>
</tr>
<tr>
<td>Self-worth invested in the body</td>
<td>73.7 (23.0)</td>
<td>78.1 (17.4)</td>
<td>104</td>
<td>-1.05</td>
<td>.30</td>
<td>.01</td>
</tr>
<tr>
<td>Body image disturbance</td>
<td>0.37 (0.24)</td>
<td>0.42 (0.26)</td>
<td>104</td>
<td>-0.90</td>
<td>.37</td>
<td>.01</td>
</tr>
<tr>
<td>Drive for thinness</td>
<td>20.3 (7.2)</td>
<td>19.6 (7.2)</td>
<td>99</td>
<td>0.40</td>
<td>.69</td>
<td>.00</td>
</tr>
<tr>
<td>Common ED characteristics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perfectionism</td>
<td>10.9 (5.5)</td>
<td>12.6 (5.4)</td>
<td>62</td>
<td>-1.08</td>
<td>.29</td>
<td>.02</td>
</tr>
<tr>
<td>Self-esteem</td>
<td>11.4 (4.7)</td>
<td>10.5 (5.1)</td>
<td>64</td>
<td>0.65</td>
<td>.52</td>
<td>.01</td>
</tr>
<tr>
<td>Control-related cognitions</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>General need for control</td>
<td>20.0 (6.3)</td>
<td>23.4 (5.9)</td>
<td>57</td>
<td>-1.87</td>
<td>.07</td>
<td>.06</td>
</tr>
<tr>
<td>Sense of control dependent on eating/</td>
<td>72.9 (23.3)</td>
<td>69.9 (25.2)</td>
<td>65</td>
<td>0.43</td>
<td>.67</td>
<td>.00</td>
</tr>
<tr>
<td>weight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dysfunctional metacognitions</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive beliefs about worry</td>
<td>11.1 (5.4)</td>
<td>10.8 (5.1)</td>
<td>104</td>
<td>0.27</td>
<td>.79</td>
<td>.00</td>
</tr>
<tr>
<td>Negative beliefs about thoughts</td>
<td>16.9 (5.3)</td>
<td>17.8 (4.9)</td>
<td>104</td>
<td>-0.84</td>
<td>.40</td>
<td>.01</td>
</tr>
<tr>
<td>Need to control thoughts</td>
<td>14.6 (4.4)</td>
<td>15.9 (5.5)</td>
<td>104</td>
<td>-1.05</td>
<td>.30</td>
<td>.01</td>
</tr>
<tr>
<td>Cognitive confidence</td>
<td>10.6 (4.7)</td>
<td>13.2 (4.9)</td>
<td>104</td>
<td>-2.43</td>
<td>.02</td>
<td>.05</td>
</tr>
<tr>
<td>Cognitive self-consciousness</td>
<td>16.1 (4.3)</td>
<td>17.7 (4.4)</td>
<td>104</td>
<td>-1.69</td>
<td>.10</td>
<td>.03</td>
</tr>
<tr>
<td>General psychopathology</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>21.4 (12.2)</td>
<td>27.1 (11.5)</td>
<td>104</td>
<td>-2.22</td>
<td>.03</td>
<td>.05</td>
</tr>
<tr>
<td>Anxiety</td>
<td>18.6 (11.5)</td>
<td>20.4 (10.9)</td>
<td>104</td>
<td>-0.71</td>
<td>.48</td>
<td>.01</td>
</tr>
<tr>
<td>Stress</td>
<td>26.6 (11.1)</td>
<td>28.3 (10.3)</td>
<td>104</td>
<td>-0.76</td>
<td>.45</td>
<td>.01</td>
</tr>
</tbody>
</table>

Note. * $p < .05$. OCP—Group using oral contraceptive pills; Non-OCP—Group not using oral contraceptive pills; AN—Anorexia Nervosa.
3. **Research Question 2**

This research question involved group comparisons of the AN, dieting, and non-dieting comparison groups, whilst controlling for the effects of low weight, depression, and anxiety.

3.1 **Analysis Specific Participants**

The full AN (n = 110), dieting (n = 66) and non-dieting (n = 66) comparison samples were used in Research Questions 2(a) to (d), with cases with missing values excluded listwise as required by the respective analyses. Due to sample size variation between the data collected for each instrument, final sample sizes for the AN, dieting, and non-dieting comparison groups differed for each analysis, and are reported in the respective sections below.

3.2 **Assumption Testing**

For the univariate analysis, Research Question 2(c), Levene’s test of homogeneity of variances indicated equal variances across groups (p > .05). For the multivariate analyses, Research Questions 2(a), (b), and (d), as the sample sizes in each cell were more than 20, the MANOVAs and MANCOVAs were reasonably robust to modest violations of normality and equality of variance (Tabachnick & Fidell, 2007). No violations of assumptions of the homogeneity of variance-covariance matrices were noted for 2(a) and (b), indicated by Box’s Test (p > .05). For 2(d), Box’s Test indicated violations to the assumption of homogeneity of variance-covariance matrices (p < .05), however Box’s M can tend to be too strict when sample sizes are large (Tabachnick & Fidell, 2007), which was the case in this analysis. Pillai’s Trace, which is more robust to violations of assumptions (Tabachnick & Fidell, 2007), was reported for all the multivariate tests. For the analyses involving covariates, Research Questions 2(b), (c), and (d), no violations to the assumption of homogeneity of regression slopes were noted in F-tests on the interaction of the independent variables with the covariates, and internal consistency of the covariate scales were high (Cronbach’s alpha = .98 and .95 for depression and anxiety respectively).

3.3 **Research Question 2(a): MANOVA Results**

In a MANOVA with all the variables of interest included as dependent variables, multivariate tests of significance indicated statistically significant differences between the three groups ($F(18, 220) = 6.66, p < .001$, Pillai’s Trace = 0.71, $\eta^2_p = .35$). Mean scores and univariate F-tests for each variable are reported in Table 7.8. Mean scores
indicated that the AN group reported more highly pathological scores on all the variables. When results for each variable were considered separately, with the exception of three metacognitive domains (positive beliefs about worry, cognitive confidence, and cognitive self-consciousness), there were statistically significant differences between the three groups on all other variables (Bonferroni-adjusted $p < .006$). Tukey post-hoc comparisons indicated that group differences were in the expected direction, with the AN group having significantly lower self-esteem and higher perfectionism, general need for control, sense of control dependent on eating/weight, negative beliefs about the uncontrollability/danger of thoughts, and need to control thoughts, compared to both comparison groups (Bonferroni-adjusted $p < .006$). Effect sizes were large for all significant variables ($\eta_p^2 > .138$) with the exception of perfectionism, which had a medium effect size ($\eta_p^2 = .10$). To determine if the medium-sized group difference for perfectionism was clinically significant, error bars for the 95% CI for group means were compared and are presented in Figure 7.2. Error bars indicate that the CIs for perfectionism for the AN group have little overlap with the dieting comparison group and no overlap with the non-dieting comparison group, suggesting a clinically significant difference.

![Figure 7.2](image_url)

*Figure 7.2.* Error bars representing 95% confidence intervals for mean perfectionism scores for AN, dieting, and non-dieting comparison groups.
Comparing the dieting and non-dieting comparison groups, no significant differences were observed between them, with the exception of the sense of control dependent on eating/weight, for which dieters had a significantly higher mean than non-dieters (mean difference = 15.45, 95% CI [0.59, 20.32]).

The follow-up analyses, Research Questions 2(b) to (d), were conducted only for the six variables for which statistically significant differences were found between the AN and comparison groups.
Table 7.8
Mean Scores and Separate Univariate F-tests from MANOVA for AN, Dieting, and Non-Dieting Comparison Groups on Variables of Interest

<table>
<thead>
<tr>
<th>Mean (SD)</th>
<th>AN</th>
<th>Dieters</th>
<th>Non-Dieters</th>
<th>F</th>
<th>p</th>
<th>η²</th>
<th>Post-hoc tests (Tukey HSD)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 57)</td>
<td>(n = 38)</td>
<td>(n = 25)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Common ED characteristics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perfectionism</td>
<td>12.4 (5.5)</td>
<td>9.6 (5.6)</td>
<td>8.4 (4.3)</td>
<td>6.14</td>
<td>.003*</td>
<td>.10</td>
<td>AN &gt; D, ND</td>
</tr>
<tr>
<td>Self-esteem</td>
<td>10.4 (4.8)</td>
<td>20.5 (5.0)</td>
<td>21.6 (5.4)</td>
<td>67.58</td>
<td>&lt;.001*</td>
<td>.54</td>
<td>AN &lt; D, ND</td>
</tr>
<tr>
<td>Control-related cognitions</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>General need for control</td>
<td>22.7 (6.1)</td>
<td>16.5 (4.7)</td>
<td>15.3 (4.5)</td>
<td>23.47</td>
<td>&lt;.001*</td>
<td>.29</td>
<td>AN &gt; D, ND</td>
</tr>
<tr>
<td>Sense of control dependent on eating/weight</td>
<td>71.1 (23.0)</td>
<td>46.1 (26.3)</td>
<td>30.6 (24.0)</td>
<td>27.63</td>
<td>&lt;.001*</td>
<td>.32</td>
<td>AN &gt; D &gt; ND</td>
</tr>
<tr>
<td>Dysfunctional metacognitions</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive beliefs about worry</td>
<td>10.3 (5.2)</td>
<td>9.7 (4.2)</td>
<td>9.5 (3.3)</td>
<td>0.34</td>
<td>.712</td>
<td>.01</td>
<td>-</td>
</tr>
<tr>
<td>Negative beliefs about thoughts</td>
<td>17.0 (5.8)</td>
<td>10.2 (4.7)</td>
<td>10.5 (4.8)</td>
<td>24.15</td>
<td>&lt;.001*</td>
<td>.29</td>
<td>AN &gt; D, ND</td>
</tr>
<tr>
<td>Need to control thoughts</td>
<td>15.2 (5.8)</td>
<td>9.3 (3.2)</td>
<td>8.8 (3.0)</td>
<td>27.07</td>
<td>&lt;.001*</td>
<td>.32</td>
<td>AN &gt; D, ND</td>
</tr>
<tr>
<td>Cognitive confidence</td>
<td>11.7 (5.0)</td>
<td>10.2 (4.0)</td>
<td>9.3 (3.8)</td>
<td>3.06</td>
<td>.051</td>
<td>.05</td>
<td>-</td>
</tr>
<tr>
<td>Cognitive self-consciousness</td>
<td>17.0 (4.7)</td>
<td>15.0 (5.3)</td>
<td>13.3 (5.1)</td>
<td>4.98</td>
<td>.008</td>
<td>.08</td>
<td>-</td>
</tr>
</tbody>
</table>

* Bonferroni-adjusted p < .006. AN—Anorexia Nervosa; MANOVA—Multivariate analysis of variance; D—Dieting comparison; ND—Non-dieting comparison.
3.4 Research Question 2(b): MANCOVA Results

In this analysis, only the six dependent variables that were found to have statistically significant differences between the groups in Research Question 2(a) were included in the MANCOVA with BMI as covariate. Multivariate tests of significance indicated statistically significant differences between the three groups ($F(12, 224) = 7.06, p < .001$, Pillai’s Trace = 0.55, $\eta_p^2 = .27$). Mean scores for each variable were the same as reported in Table 7.8. Estimated marginal means (i.e., adjusted means with the effect of BMI statistically removed) and univariate $F$-tests for each variable are reported in Table 7.9. Estimated marginal means indicated that the AN group still had more highly pathological scores on the six variables. After adjusting for BMI, the differences between the three groups on all six variables continued to be statistically significant (Bonferroni-adjusted $p < .008$). Effect sizes continued to be large for all significant variables ($\eta_p^2 > .138$) with the exception of perfectionism, which continued to have medium effect size ($\eta_p^2 = .09$).

<table>
<thead>
<tr>
<th>Common ED characteristics</th>
<th>Estimated Marginal Mean (SE)</th>
<th>AN $(n = 57)$</th>
<th>Dieters $(n = 38)$</th>
<th>Non-Dieters $(n = 25)$</th>
<th>$F$ (2, 116)</th>
<th>$p$</th>
<th>$\eta_p^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perfectionism</td>
<td>12.7 (0.8)</td>
<td>9.3 (1.0)</td>
<td>8.2 (1.1)</td>
<td>5.54</td>
<td>.005*</td>
<td>.09</td>
<td></td>
</tr>
<tr>
<td>Self-esteem</td>
<td>10.1 (0.8)</td>
<td>20.7 (0.9)</td>
<td>21.8 (1.0)</td>
<td>46.86</td>
<td>&lt;.001*</td>
<td>.45</td>
<td></td>
</tr>
<tr>
<td>Control-related cognitions</td>
<td>General need for control</td>
<td>22.8 (0.8)</td>
<td>16.5 (1.0)</td>
<td>15.3 (1.1)</td>
<td>15.58</td>
<td>&lt;.001*</td>
<td>.21</td>
</tr>
<tr>
<td></td>
<td>Sense of control dependent on eating/weight</td>
<td>71.2 (3.7)</td>
<td>46.0 (4.4)</td>
<td>30.6 (5.0)</td>
<td>19.96</td>
<td>&lt;.001*</td>
<td>.26</td>
</tr>
<tr>
<td>Dysfunctional metacognitions</td>
<td>Negative beliefs about thoughts</td>
<td>17.2 (0.8)</td>
<td>9.9 (1.0)</td>
<td>10.3 (1.1)</td>
<td>17.57</td>
<td>&lt;.001*</td>
<td>.23</td>
</tr>
<tr>
<td></td>
<td>Need to control thoughts</td>
<td>15.3 (0.7)</td>
<td>9.2 (0.8)</td>
<td>8.7 (0.9)</td>
<td>18.36</td>
<td>&lt;.001*</td>
<td>.24</td>
</tr>
</tbody>
</table>

Note. * Bonferroni-adjusted $p < .008$. AN—Anorexia Nervosa; BMI—Body mass index; MANCOVA—Multivariate analysis of covariance.

3.5 Research Question 2(c): ANCOVA Results

In an ANCOVA with self-esteem as the dependent variable and depression as a covariate, the differences between the three groups on self-esteem continued to be statistically significant ($F(2, 153) = 21.58, p < .001$), with a large effect size ($\eta_p^2 = .22$).
The means and estimated marginal means (i.e., adjusted means with the effect of depression statistically removed) are reported in Table 7.10, indicating that the AN group had lower self-esteem compared to the comparison groups.

Table 7.10  
Means and Estimated Marginal Means (Controlling for Depression) for AN, Dieting, and Non-Dieting Comparison Groups on Self-Esteem

<table>
<thead>
<tr>
<th></th>
<th>AN (n = 68)</th>
<th>Dieters (n = 53)</th>
<th>Non-Dieters (n = 36)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (SD)</td>
<td>10.5 (5.2)</td>
<td>20.3 (5.2)</td>
<td>21.3 (5.0)</td>
</tr>
<tr>
<td>Estimated Marginal Mean (SE)</td>
<td>13.0 (0.6)</td>
<td>18.6 (0.6)</td>
<td>19.0 (0.8)</td>
</tr>
</tbody>
</table>

Note. AN—Anorexia Nervosa.

3.6 Research Question 2(d): MANCOVA results

Out of the five metacognitive variables, only the two control-related metacognitive variables were found to be associated with the AN group in Research Question 2(a) and included in this analysis. In a MANCOVA with the control-related metacognitions as dependent variables and depression and anxiety as covariates, multivariate tests of significance indicated that the differences between the three groups continued to be statistically significant ($F(4, 472) = 5.03, p = .001$, Pillai’s Trace = 0.08, $\eta_p^2 = .04$). The means and estimated marginal means (i.e., adjusted means with the effect of depression and anxiety statistically removed) are reported in Table 7.11, indicating that the AN group had higher levels of dysfunctional metacognitions compared to the comparison groups, although the differences between the AN and comparison groups became smaller after the effect of depression and anxiety were removed. Univariate $F$-tests indicated that after adjusting for depression and anxiety, the differences between the three groups continued to be significant for both negative beliefs about the uncontrollability/danger of thoughts ($F(2, 236) = 5.75, p = .004$) and the need to control thoughts ($F(2, 236) = 8.49, p < .001$), albeit with small effect sizes ($\eta_p^2 = .05$ and .07 respectively).
Table 7.11
Means and Estimated Marginal Means (Controlling for Depression and Anxiety) for AN, Dieting, and Non-Dieting Comparison Groups on Control-Related Metacognitive Variables

<table>
<thead>
<tr>
<th></th>
<th>Negative Beliefs about the Uncontrollability/Danger of Thoughts</th>
<th>Need to Control Thoughts</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AN (n = 110)</td>
<td>Dieters (n = 65)</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>17.8 (5.0)</td>
<td>10.0 (5.0)</td>
</tr>
<tr>
<td>Estimated Marginal Mean (SE)</td>
<td>14.9 (0.5)</td>
<td>12.1 (0.6)</td>
</tr>
</tbody>
</table>

Note. AN—Anorexia Nervosa.

4. RESEARCH QUESTION 3

This research question involved the testing of Model A and Model B in AN and non-eating disordered comparison groups.

4.1 ANALYSIS SPECIFIC PARTICIPANTS

The full AN patient group (n = 110) and non-eating disordered comparison group (n = 132) were used in path analyses for Research Questions 3(a) and (b). The AN patient group was combined across all subgroups (meeting full AN syndrome, subthreshold AN, and on the OCP) as earlier analyses indicated that there were no differences across these subgroups for the variables included in Models A and B. The comparison group was combined across subgroups (dieters and non-dieters) to allow for an adequate sample size for path analyses. Due to sample size variation between the data collected for each variable, missing values were estimated using a full information maximum likelihood (FIML) approach, which is the default option in AMOS.

4.2 ASSUMPTION TESTING

Variables were tested for possible violation of assumptions of univariate and multivariate normality in the sample. Univariate normality was assessed through the inspection of histograms, normal probability plots and boxplots. Distributions for the AN group appeared normal for all variables except drive for thinness and sense of control dependent on eating/weight, which were slightly negatively skewed, as would be expected in this sample. Distributions for the comparison group for drive for thinness and both metacognitive variables (need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts) were positively skewed. No action was taken as no extreme outliers were present in either group. No violations of assumptions of multivariate normality were noted, indicated by Mardia’s test of multivariate kurtosis.
with an acceptable range of multivariate kurtosis indicated by a c.r. within the range of ±1.96. For the AN sample, Mardia’s coefficient was .52 (c.r. = .18). For the comparison sample, Mardia’s coefficient was 5.28 (c.r. = 1.87). No multivariate outliers were identified in the AN sample (highest Mahalanobis $d^2$ value = 18.39, $p^2 = .45$) or the comparison sample (highest Mahalanobis $d^2$ value = 18.22, $p^2 = .50$). Requirements for model identification were met, with both models being recursive and overidentified.

### 4.3 Correlations between Variables in Models A & B

Pearson correlations between the variables in Models A and B are reported in Table 7.12 for the AN group, and in Table 7.13 for the comparison group.

**AN group.** Inspection of the correlation coefficients for the AN group in Table 7.12 showed that all variables had significant medium to strong positive correlations with the main outcome variable, drive for thinness, with the exception of self-esteem, which had a significant strong negative correlation with drive for thinness. Focusing on the relationships between the other variables proposed in Models A and B, the complex relationship between perfectionism and self-esteem in individuals with AN was demonstrated by the significant negative correlation between both variables. As had been hypothesised by the mediating mechanisms proposed in Model A (centered on the general need for control) and Model B (centered on the need to control thoughts), perfectionism and self-esteem had strong significant correlations with both variables.

As detailed in Chapter 5 (p. 133), Model B was developed on the central premise that in an individual with AN, the general need for control, central to Model A, extended to the higher level metacognitive domain. This was proposed to occur through two dysfunctional metacognitive variables: the need to control thoughts, which then resulted in negative beliefs about thoughts being perceived as uncontrollable/dangerous in nature. The correlations observed in the AN group supported the correspondence between both levels of control-related cognitions proposed in Models A and B, with strong positive correlations observed between the general need for control and both higher order metacognitive factors, the need to control thoughts ($r = .77$, $p < .001$) and negative beliefs about the danger/uncontrollability of thoughts ($r = .65$, $p < .001$).

**Non-eating disordered comparison group.** Inspection of the correlation coefficients for the comparison group in Table 7.13 showed that all variables had significant small to medium correlations with the main outcome variable, drive for thinness, with the exception of sense of control dependent on eating/weight, which did
not have a significant correlation with drive for thinness \( (r = .21, p = .10) \). Focusing on the relationships between the other variables proposed in Models A and B, the relationship between perfectionism and self-esteem that was observed in individuals with AN was not observed in the comparison sample, for which there was no significant correlation between both variables \( (r = .00, p = .99) \). The correlations between perfectionism and self-esteem with the central mechanisms proposed in Model A (the general need for control) and Model B (the need to control thoughts) were non-significant, with the exception of the significant correlation between perfectionism and the general need for control \( (r = .37, p < .001) \).

Interestingly, the corresponding relationship between the general need for control and higher level metacognitive control-related variables was also observed in the comparison sample, although to a lesser degree than the AN group, with the general need for control observed to be significantly correlated with the need to control thoughts \( (r = .61, p < .001) \) and negative beliefs about the danger/uncontrollability of thoughts \( (r = .34, p = .001) \).
### Table 7.12
Means, Standard Deviations, and Correlations Between Variables in Models A and B for the AN Group (n = 110)

<table>
<thead>
<tr>
<th>Correlation (r)</th>
<th>Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P</td>
</tr>
<tr>
<td>Perfectionism (P)</td>
<td>--</td>
</tr>
<tr>
<td>Self-esteem (SE)</td>
<td>-.44**</td>
</tr>
<tr>
<td>General need for control (GNFC)</td>
<td>.64**</td>
</tr>
<tr>
<td>Sense of control dependent on eating/weight (SoC)</td>
<td>.26</td>
</tr>
<tr>
<td>Need to control thoughts (NCont)</td>
<td>.48**</td>
</tr>
<tr>
<td>Negative beliefs about uncontrollability/danger of thoughts (Neg Bel)</td>
<td>.39*</td>
</tr>
<tr>
<td>Drive for thinness (DT)</td>
<td>.36*</td>
</tr>
</tbody>
</table>

*Note. *p < .01, **p < .001 (2-tailed). AN—Anorexia Nervosa.*

### Table 7.13
Means, Standard Deviations, and Correlations Between Variables in Models A and B for the Non-Eating Disordered Comparison Group (n = 132)

<table>
<thead>
<tr>
<th>Correlation (r)</th>
<th>Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P</td>
</tr>
<tr>
<td>Perfectionism (P)</td>
<td>--</td>
</tr>
<tr>
<td>Self-esteem (SE)</td>
<td>.00</td>
</tr>
<tr>
<td>General need for control (GNFC)</td>
<td>.37**</td>
</tr>
<tr>
<td>Sense of control dependent on eating/weight (SoC)</td>
<td>.28</td>
</tr>
<tr>
<td>Need to control thoughts (NCont)</td>
<td>.22</td>
</tr>
<tr>
<td>Negative beliefs about uncontrollability/danger of thoughts (NegBel)</td>
<td>-.03</td>
</tr>
<tr>
<td>Drive for thinness (DT)</td>
<td>.29*</td>
</tr>
</tbody>
</table>

*Note. *p < .01, **p < .001 (2-tailed).
4.4 RESEARCH QUESTION 3(A): MODEL A PATH ANALYSIS RESULTS

In the following, results of the standardised coefficients (β) were reported.

4.4.1 Anorexia Nervosa group

Proposed Model A. Model A was evaluated via AMOS in the AN data, with the standardised regression coefficients and variance explained for each endogenous variable ($R^2$) illustrated in Figure 7.3. Looking at the standardised path coefficients, all pathways predicted in Model A were significant ($p < .05$) with the exception of the direct effect of perfectionism on drive for thinness ($β = .00, p = .99$). Overall fit of the proposed model was excellent, as indicated by the following fit indices (i) $\chi^2 = 2.15, df = 3, p = .542$, (ii) CFI = 1.000, and (iii) RMSEA = .000.

![Figure 7.3](image)

*Figure 7.3*. Proposed Model A assessed in AN sample, with standardised regression weights and variance explained for each endogenous variable (italicised). Note. * $p < .05$.

Revised Model A: Individual pathways. To obtain a more parsimonious model, the non-significant pathway from perfectionism to drive for thinness was trimmed, and revised Model A was evaluated in the AN data, with standardised regression coefficients and variance explained for each endogenous variable illustrated in Figure 7.4. Looking at the standardised path coefficients, all the pathways predicted in revised Model A were significant ($p < .05$). Perfectionism and self-esteem covaried significantly ($r = .43, p = .001$). The general need for control demonstrated a strong amount of variance explained by perfectionism and self-esteem ($R^2 = .47$), and the outcome variable of drive for thinness demonstrated a strong amount of variance explained by the endogenous variables ($R^2 = .37$). Looking at direct effects, self-esteem
had a significant negative direct effect on drive for thinness ($\beta = -.41, p < .001$), which in turn, had a strong direct effect on the sense of control dependent on eating/weight ($\beta = .42, p < .001$). Looking at the bias-corrected bootstrapped estimates for indirect effects, perfectionism had a significant indirect effect on drive for thinness through the general need for control ($\beta = .18, p = .002, 95\%$ CI [.054, .363]), and self-esteem had a significant negative indirect effect on drive for thinness through the general need for control ($\beta = -.08, p = .019, 95\%$ CI [-.218, -.011]).

*Revised Model A: Overall fit.* Overall fit of the revised model continued to be excellent, as indicated by the following fit indices, (i) $\chi^2 = 2.15$, $df = 4$, $p = .709$, (ii) CFI = 1.000, and (iii) RMSEA = .000.

4.4.2 *Non-eating disordered comparison group*

*Proposed Model A: Individual pathways.* Model A was evaluated via AMOS in the comparison group data, with the standardised regression coefficients and variance explained for each endogenous variable illustrated in Figure 7.5. Interestingly, in contrast with the AN sample, perfectionism and self-esteem did not covary significantly ($r = .01, p = .89$). Also in contrast with the AN sample, the general need for control demonstrated a relatively small amount of variance explained by perfectionism and self-esteem ($R^2 = .18$), and the outcome variable of drive for thinness demonstrated a relatively small amount of variance explained by the endogenous variables ($R^2 = .23$). Finally, the key differentiating point between the AN and comparison groups was that in the comparison group, standardised path coefficients between drive for thinness and
the important control-related variables were non-significant (general need for control on drive for thinness, $\beta = .19, p = .06$; drive for thinness on the sense of control dependent on eating/weight, $\beta = .20, p = .11$). Instead, both perfectionism and self-esteem had significant direct effects on drive for thinness. The present study was focused on assessing the importance of control-related mechanisms in maintaining AN, and these were found to be non-significant in the comparison sample. Hence, examination of indirect effects and revisions to Model A were not further pursued for the comparison sample.

**Proposed Model A: Overall fit.** Whilst overall fit of the proposed model was acceptable as indicated by chi-square, $\chi^2 = 7.73$, $df = 3$, $p = .052$, this was approaching significance ($p < .05$). The other fit indices indicated that the fit of Model A to the comparison group data was poor and unacceptable, CFI = .879, RMSEA = .110.

![Figure 7.5. Proposed Model A assessed in non-eating disordered comparison sample, with standardised regression weights and variance explained for each endogenous variable (italicised). Note. * $p < .05$.](image)

### 4.4.3 Conclusion

In the AN data, all hypothesised pathways in Model A were significant with the exception of the direct effect of perfectionism on drive for thinness, despite the significant correlation between perfectionism and the drive for thinness. Of particular importance were the significant indirect effects of perfectionism and self-esteem on drive for thinness through the mediating variable, the general need for control. Findings also supported the direct effect of drive for thinness on the AN individual’s sense of
control becoming dependent on eating/weight. Overall fit of proposed and revised Model A to the AN data was excellent.

In the comparison group data, hypothesised pathways of the direct effects of perfectionism and self-esteem on drive for thinness were significant. However, in contrast to the AN group, the key control-related mechanisms did not exist in the comparison group, with predicted pathways between drive for thinness and both control-related variables found to be non-significant. Overall fit of proposed Model A to the comparison group was unacceptable.

4.5 **RESEARCH QUESTION 3(b): MODEL B PATH ANALYSIS RESULTS**

In the following, results of the standardised coefficients (β) were reported.

4.5.1 **Anorexia Nervosa group**

*Proposed Model B.* Model B was evaluated via AMOS for the AN data, with standardised regression coefficients and variance explained for each endogenous variable illustrated in Figure 7.6. Looking at the standardised path coefficients, most pathways predicted in Model B were significant (p < .05) with the exception of the direct effect of perfectionism on drive for thinness (β = .06, p = .60), the direct effect of need to control thoughts on drive for thinness (β = .10, p = .43), and the effect of negative beliefs about the uncontrollability/danger of thoughts on drive for thinness (β = .21, p = .06), although this was approaching significance. The following fit indices indicated good overall fit of the model to the AN data, (i) $\chi^2 = 3.65$, df = 2, $p = .161$, (ii) CFI = .988. However, the final fit index, (iii) RMSEA = .087, suggested mediocre fit.

![Figure 7.6](image-url)

*Figure 7.6.* Proposed Model B assessed in AN sample, with standardised regression weights and variance explained for each endogenous variable (italicised). Note. *p < .05.*
**Revised Model B: Individual pathways.** To obtain a more parsimonious model, the two clearly non-significant pathways, from perfectionism and the need to control thoughts to drive for thinness, were trimmed. Revised Model B was evaluated in the AN data, with standardised regression coefficients and variance explained for each endogenous variable illustrated in Figure 7.7. Looking at the standardised path coefficients, all the pathways predicted in revised Model B were significant ($p < .05$). The need to control thoughts demonstrated a strong amount of variance explained by perfectionism and self-esteem ($R^2 = .33$), and the outcome variable of drive for thinness demonstrated a strong amount of variance explained by the endogenous variables ($R^2 = .36$). Looking at direct effects, self-esteem had a significant negative direct effect on drive for thinness ($\beta = -.45$, $p < .001$). Looking at the bias-corrected bootstrapped estimates for indirect effects, perfectionism had a significant indirect effect on drive for thinness through the metacognitive control variables ($\beta = .06$, $p = .035$, 95% CI [.003, .194]), self-esteem had a significant negative indirect effect on drive for thinness through the metacognitive control variables ($\beta = -.07$, $p = .027$, 95% CI [-.186, -.007]), and the need to control thoughts had a significant indirect effect on drive for thinness through negative beliefs about the uncontrollability/danger of thoughts ($\beta = .20$, $p = .047$, 95% CI [.002, .422]).

**Revised Model B: Overall fit.** Overall fit of the revised model was excellent, as indicated by all three fit indices, (i) $\chi^2 = 5.09$, $df = 4$, $p = .279$, (ii) CFI = .992, and (iii) RMSEA = .050.

*Figure 7.7. Revised Model B assessed in AN sample, with standardised regression weights and variance explained for each endogenous variable (italicised). Note. * $p < .05$.*
4.5.2 Non-eating disordered comparison group

Proposed Model B: Individual pathways. Model B was evaluated via AMOS in the comparison group data, with standardised regression coefficients and variance explained for each endogenous variable illustrated in Figure 7.8. Looking at the standardised path coefficients, all pathways predicted in Model B were significant ($p < .05$) with the exception of the direct effect of need to control thoughts on drive for thinness ($\beta = .10, p = .27$). However, in contrast with the AN sample, the need to control thoughts demonstrated a relatively small amount of variance explained by perfectionism and self-esteem ($R^2 = .11$), and the outcome variable of drive for thinness demonstrated a relatively small amount of variance explained by the endogenous variables ($R^2 = .26$). Whilst perfectionism and self-esteem had significant direct effects on drive for thinness, the present study was more interested in maintenance mechanisms involving the metacognitive control-related variables. Looking at the bias-corrected bootstrapped estimates for indirect effects, in contrast to the AN group, the indirect effect of perfectionism on drive for thinness through the metacognitive control variables was non-significant ($\beta = .03, p = .120, 95\% \text{ CI } [-.007, .113]$). The indirect effect of self-esteem on drive for thinness through the metacognitive control variables was also non-significant ($\beta = -.04, p = .147, 95\% \text{ CI } [-.140, .012]$). The only significant indirect effect was of the need to control thoughts on drive for thinness, through negative beliefs about the uncontrollability/danger of thoughts ($\beta = .09, p = .043, 95\% \text{ CI } [.002, .272]$).

Proposed Model B: Overall fit. Whilst there were some significant direct effects and one significant indirect effect involving control-related metacognitive mechanisms in the comparison group data, overall fit of the proposed model was poor and unacceptable, as indicated by all three fit indices, (i) $\chi^2 = 20.32, df = 2, p < .001$, (ii) CFI = .775, (iii) RMSEA = .264. Even when a more parsimonious revised Model B was tested (with removal of the non-significant pathway between need to control thoughts and drive for thinness), it had little impact on the individual path coefficients and made negligible improvement to overall fit indices, with all three still indicating bad fit, (i) $\chi^2 = 21.59, df = 3, p < .001$, (ii) CFI = .772, (iii) RMSEA = .217.
4.5.3 Conclusion

In the AN data, most hypothesised pathways in Model B were significant, with the exceptions of the direct effects of perfectionism and the need to control thoughts on drive for thinness, despite the significant correlations between both these variables and the drive for thinness. Of particular importance were the significant indirect effects of perfectionism and self-esteem on drive for thinness through the mediating metacognitive control variables, as well as the significant indirect effect of the need to control thoughts on drive for thinness through negative beliefs about the uncontrollability/danger of thoughts. Overall fit of proposed and revised Model B to the AN data was excellent.

In the comparison group data, hypothesised pathways of the direct effects of perfectionism and self-esteem on drive for thinness were significant. However, in contrast to the AN group, of the three key metacognitive control-related mechanisms proposed, only one was significant, the indirect effect of the need to control thoughts on drive for thinness through negative beliefs about the uncontrollability/danger of thoughts. Overall fit of proposed Model B to the comparison group was unacceptable.
CHAPTER 8: DISCUSSION

This chapter will examine and discuss findings for the research project. For each research question, interpretation of results will be provided, the application of findings in the context of existing literature and implications will be discussed, and strengths, limitations, and future directions will be considered (Sections 1 to 4). Then, general limitations of the research project and suggestions for future research will be examined (Section 5). Lastly, final conclusions for the research project will be considered (Section 6).

1. RESEARCH QUESTION 1(a)

|Research Question 1(a). Are there differences between the groups with full syndrome AN and subthreshold AN (not meeting diagnostic criteria for weight \(\text{BMI} < 17.5 \text{ kg/m}^2\) and/or amenorrhea) on the variables of interest (Table 3.3, p. 44)?|

1.1 INTERPRETATION OF FINDINGS

The aim of Research Question 1(a) was to establish whether there were differences between the full syndrome and subthreshold AN (not meeting weight and/or amenorrhea criteria) groups on diagnostic criteria, variables proposed to be involved in the maintenance of AN, or general psychopathology. No statistically significant group differences were found for any cognitive, psychological or behavioural diagnostic criteria of AN (fear of gaining weight, self-worth invested in the body, body image disturbance, drive for thinness), variables relevant to the maintenance of AN (perfectionism, self-esteem, control-related cognitions, dysfunctional metacognitions), or general psychopathology (depression, anxiety, stress).

Whilst post-hoc power analyses suggested that with an increased sample size of \(N = 159\) (instead of \(N = 78\) in the present study), the power to detect significant medium-sized effects would have been higher at the recommended level of .80 (Cohen, 1988), it is also important that any differences be of clinical significance. On visual inspection of the group means, the only variables for which mean differences could have clinical significance are fear of gaining weight, self-esteem, and sense of control dependent on eating/weight. Each of these measures would be in the clinically severe spectrum, so what that might mean for treatment is limited. The remaining results...
suggest that there are no clinically important differences signified by the weight or amenorrhea criteria in diagnosing AN.

1.2 APPLICATION OF FINDINGS IN CONTEXT OF PREVIOUS LITERATURE

The clinical differences signified by the weight and amenorrhea criteria were of interest in the present study due to (i) the substantial proportion of individuals with subthreshold AN who were diagnosed with EDNOS and did not meet these physical criteria, (ii) inconsistencies and difficulties with assessing them, and (iii) limitations of existing empirical studies examining their clinical utility. The findings of the present study will now be discussed in the context of these issues.

Substantial proportion of individuals with subthreshold AN not meeting physical criteria. As reviewed earlier (p. 11-15), EDNOS was the most prevalent ED in clinical and non-clinical samples in Western countries, and individuals diagnosed with EDNOS more commonly had subthreshold AN than subthreshold BN (Bunnell et al., 1990; Dancyger & Garfinkel, 1995). Of individuals with subthreshold AN, a substantial one third failed to meet full diagnosis because they did not meet the weight criterion (Turner & Bryant-Waugh, 2004), and another third because they did not have amenorrhea (Gendall et al., 2006). These findings raised the issue of the clinical utility of both criteria, as the EDNOS diagnosis has major implications for individuals in clinical and research settings.

Inconsistencies and difficulties in assessing physical criteria. Various difficulties with assessing the physical criteria came to light (p. 19-20, 27). The weight criterion has not been applied consistently, with clinicians and researchers using a variety of approaches to determine underweight status (e.g., different BMI cut-points, a range of published norms and tables; Thomas et al., 2009). Assessment of the amenorrhea criterion was also problematic as it depended largely on patients’ self-report of menstrual status, often with no independent confirmation by clinicians. Furthermore, it cannot be applied to males, and females who are pre-menarchal, post-menopausal, or on hormone replacement therapy such as OCPs. These inconsistencies further prompted the question regarding the clinical utility of both criteria, as their inconsistent application resulted in substantial differences between the patients eligible for diagnosis of AN.

Limitations of existing studies. Since the purpose of diagnostic classification is to aid clinicians in making appropriate treatment decisions, the above issues highlighted
the importance of examining the clinical differences signified by the weight and amenorrhea criteria. A literature review of four empirical studies assessing the clinical utility of the weight criterion (p. 20-25), and seven studies assessing that of the amenorrhea criterion (p. 30-39), found few statistically significant differences signified by the absence or presence of both physical criteria in community, inpatient, and outpatient samples. Specifically, existing studies found no differences between underweight and non-underweight, and amenorrheic and non-amenorrheic AN patients, in behavioural symptoms of AN (drive for thinness through dieting, excessive exercise, purging or using laxatives/diuretics), physical measures, ED history, common ED characteristics measured by the EDI (e.g., ineffectiveness, perfectionism, maturity fears, interpersonal distrust, interoceptive awareness, social insecurity, impulse regulation, and asceticism), psychiatric comorbidities, distress, secondary impairment of functioning, service utilisation, and treatment outcome (McIntosh et al., 2004; Watson & Anderson, 2003; Williamson et al., 1992; Bunnell et al., 1990; Cachelin & Maher, 1998; Dalle Grave et al., 2008; Roberto et al., 2008; Dellava et al., 2011; Gendall et al., 2006; Garfinkel et al., 1996). Whilst the findings were confirmed in meta-analyses (Thomas et al., 2009), existing studies had common limitations of small sample size and failure to assess cognitive or psychological diagnostic criteria of AN, clinically important as embodiments of severity, thus affecting treatment planning and outcome.

Present findings in the context of existing literature. The present study contributes to the debate around the physical criteria of AN through addressing some limitations of previous findings by using a larger sample size. Results for Research Question 1(a) confirm and extend previous studies, finding no evidence for the clinical utility of the weight and amenorrhea criteria. Novel findings were contributed through the assessment of cognitive or psychological diagnostic criteria of AN, and the examination of other cognitive and metacognitive factors known to be pertinent in maintaining the disorder, all having implications for treatment. The results provide compelling evidence that there are no clinically important differences signified by the physical AN criteria, and have implications for DSM-V, as well as clinical and research practice. They are discussed in the following section.

1.3 IMPLICATIONS: DSM-V, CLINICAL AND RESEARCH

Implications for DSM-V. Notably, the present findings signify that the existing physical diagnostic criteria for AN, which were established in a non-empirical manner and have changed with different versions of the DSM over the years, are too restrictive.
Results provide support for proposed changes to the weight and amenorrhea criteria in the DSM-V (APA, 2012).

Focusing first on the weight criterion, results contribute to the argument against an absolute weight cut-off of 85% of ideal weight or BMI ≤ 17.5 kg/m². Methods of calculating underweight status are not consistent or comparable, and they do not take into account an individual’s pre-morbid weight range. Interestingly, in light of increases in obesity and average body weights at the population level (Hedley et al., 2004), which may render diagnostically low weights increasingly difficult to achieve (Thomas et al., 2009), some clinicians have suggested that percentage weight loss would be a more meaningful criterion (McIntosh et al., 2004), for example suggesting that a weight loss of more than 20% should be required for diagnosis (Andersen et al., 2001). Proponents of this view argue that self-induced starvation to a point of impairment does not necessarily require a final lowest weight of < 85% of ideal weight, as individuals who are pre-morbidly at a higher weight and lose weight with significant medical consequences may still be above the 85% criterion (Watson & Andersen, 2003). From clinical observation, pre-morbidly overweight AN patients who have experienced a large or rapid weight loss, although remaining in the normal weight range, are commonly medically compromised and present with disordered eating patterns, cognitions, and psychopathology similar to patients with full syndrome AN. As about half of patients who meet all criteria for AN except the weight criterion were more than 10% overweight before their illness onset (Watson & Andersen, 2003), changing the absolute weight cut-off criterion to a weight loss criterion could possibly reduce the large proportion of EDNOS diagnoses. However, strict application of this criterion could be difficult as it depends on having a reliable pre-morbid weight, and more empirical evidence is required before the criterion can be applied reliably. With regards to the DSM-V criteria of AN, the Eating Disorders Work Group has recommended that it is not desirable to set a specific numerical standard for weight, with the judgement of excessively low weight best made by the clinician in the context of individual factors including age, sex, developmental trajectory, and physical health (APA, 2012). This is consistent with the present findings.

Considering the amenorrhea criterion, as previously discussed (p. 29-30), diagnostic specificity is not increased as amenorrhea does not discriminate between ED diagnoses, presence or absence of EDs, full syndrome or subthreshold EDs, and does not develop consistently across individuals. Amenorrhea is not a phenomenon exclusive
to AN, and there is evidence that it occurs secondary to weight loss, nutritional status, BMI, or relative energy deficits (Gendall et al., 2006; Dalle Grave et al., 2008; Copeland et al., 1995; Cachelin & Maher, 1998; Pinheiro et al., 2007; Abraham et al., 2005), rather than being associated with psychiatric status. Importantly, most of the biological effects associated with amenorrhea (e.g., lower bone mineral density) are reversible following nutritional rehabilitation, weight gain, and the resuming of normal menstrual function (Copeland et al., 1995). The present findings provide evidence that amenorrhea is not associated with clinically meaningful AN psychopathology variables, thus there is no clinical utility in differentiating individuals with AN who do and do not menstruate. Results support the Eating Disorders Work Group’s recommendation for deletion of the amenorrhea criterion for DSM-V (APA, 2012).

Clinical implications. The present findings suggest that prior to publication of the DSM-V, flexibility should be used when applying the weight and amenorrhea criteria in clinical situations. Evidently, the criteria have no discernible effect on clinically meaningful psychopathological variables. In the case of the rigid weight criterion, adhering strictly to it detracts from the essence of Criterion A for diagnosis of AN: the relentless, intentional weight loss that is the crux of the disorder and the main source of devastating physical health consequences. It can occur at any weight and patients often range across being overweight, underweight, or at normal weight throughout the course of the illness, regardless of their weight status at the time of the assessment (Beumont et al., 1994). Underweight and non-underweight individuals fulfilling the other AN criteria were found to have similar highest and lowest premorbid weights on admission to hospital, suggesting that the higher weights of the subthreshold AN group were likely to be an artefact of the time of assessment (Watson & Andersen, 2003). Adhering strictly to the physical criteria may exclude patients with clinically significant illnesses.

Clinically, relaxing the physical criteria for diagnosis of AN will allow for the early detection of individuals with clinically significant illnesses, increase their chances of receiving appropriate treatment or reimbursement for treatment in some countries, and facilitate efficient treatment administration. This is crucial as the subthreshold diagnosis may be a transition period for individuals in the process of developing a full disorder (Herzog et al., 1993), as a majority of those with subthreshold EDs continue to have ED symptomatology and progress to full syndrome EDs at follow-up (Herzog et al., 1993; Striegel-Moore et al., 1989; Yager et al., 1987).
Research implications. The findings suggest that prior to publication of the DSM-V, flexibility should be used when applying the weight and amenorrhea criteria in research. Relaxing the criteria will allow individuals with subthreshold AN and clinically significant illnesses, who form the majority of the treatment-seeking population, to be included in AN research, providing a better understanding of the course of illness, predictors of treatment response, outcome, and differential treatment needs in this heterogeneous group (Agras et al., 2004). In light of the findings, fulfilment of the weight and amenorrhea criteria was not required for inclusion in the AN sample for Research Questions 2 and 3.

1.4 Strengths, Limitations, and Future Directions

Strengths. The present study addressed gaps in existing literature by assessing the previously neglected core cognitive diagnostic criteria of AN, namely, body image disturbance, fear of gaining weight and self-worth invested in the body. Further, it added to the literature by including novel clinically relevant factors proposed to be critical to the maintenance of AN, specifically, low self-esteem, control-related cognitions, and dysfunctional metacognitions. The present study had increased statistical power to find differences when compared with the small sample sizes in previous studies (e.g., Bunnell et al., 1990; Williamson et al., 1992; Cachelin & Maher, 1998; Dalle Grave et al., 2008; Gendall et al., 2006). It also increased our understanding of amenorrhea by documenting OCP use, eliminating the possibility that the non-amenorrheic group was menstruating because of the use of these medications (e.g., Dellava et al., 2011).

Limitations and future directions. General limitations of the research project as a whole are discussed later (p. 208-210). Specific to this study, the more relaxed selection criteria for subthreshold AN groups was a limitation. The three comparison groups included the full-syndrome (meeting all criteria) AN group, the subthreshold (not meeting both physical criteria) AN group, and the subthreshold (not meeting one criterion) AN group, which included patients who did not meet the weight \( n = 13 \) or amenorrhea criterion \( n = 11 \). This makes it difficult to draw conclusions about differences based purely on weight or amenorrhea. Future research should aim to clearly delineate the selection criteria for subthreshold AN groups to allow for comparisons based purely on presence and absence of each of the physical criteria.
Another limitation of the present study was assessment of amenorrhea status through patient self-report, with no independent confirmation by researchers or clinicians. The present study also did not explore differences signified by the weight and amenorrhea criteria in demographics, early experiences, and personality. Previous individual studies reported that the full syndrome AN group had higher full-scale intelligence, less physical or sexual abuse (Watson & Andersen, 2003), less family control, more recreational family experiences, higher levels of family expressiveness (Cachelin & Maher, 1998), higher persistence, and lower novelty-seeking (Gendall et al., 2006), compared to the non-underweight or non-amenorrheic subthreshold AN groups. Future research should aim to replicate these findings so that more conclusive statements about demographics, early experiences, or personality can be made.

2. RESEARCH QUESTION 1(B)

Research Question 1(b). Are there differences between AN patients on the OCP and those who are not, on the variables of interest (Table 3.3, p. 44)?

2.1 INTERPRETATION OF FINDINGS

Research Question 1(b) aimed to explore possible differences between AN patients on the OCP and those who were not, on AN diagnostic criteria, variables proposed to be involved in the maintenance of AN, and general psychopathology. Results indicated no differences between the OCP and non-OCP groups on most variables including AN diagnostic criteria (fear of gaining weight, self-worth invested in the body, body image disturbance, drive for thinness), variables relevant to the maintenance of AN (perfectionism, self-esteem, control-related cognitions, dysfunctional metacognitions), or general psychopathology.

The exceptions were low cognitive confidence and depression, which were more severe in the non-OCP group. A statistical possibility for the difference could have been chance, due to an increased probability of Type 1 errors. Despite the large number of tests (16) applied to the data set, no statistical control was applied to correct for familywise error rate due to the exploratory nature of this study, to accord with Cabin and Mitchell’s (2000) recommendations. The conventional alpha level of $p < .05$ was used to define significance, resulting in an increased risk of Type 1 errors, or wrongly rejecting the null hypothesis in favour of the alternative (Pallant, 2007; Tabachnick &
Fidell, 2007). Even if the results were not due to Type 1 error, the effect sizes of differences between groups were small, suggesting that the differences in depression and cognitive confidence may have little clinical significance.

2.2 APPLICATION OF FINDINGS IN CONTEXT OF PREVIOUS LITERATURE

To the knowledge of the researcher, there have been no previous studies examining differences between AN patients on the OCP and those who were not. Typically, AN patients on the OCP have been excluded from studies assessing the clinical utility of the amenorrhea criterion (e.g., Dalle Grave et al., 2008; Gendall et al., 2006; Roberto et al., 2008). Because a substantial proportion of patients used the OCP in the present study, Research Question 1(b) provided novel findings through addressing the lack of knowledge about potential differences between the OCP and non-OCP AN subgroups on clinically important psychological factors. Implications of the findings for DSM-V, as well as for clinical and research practice, are discussed in the following section.

2.3 IMPLICATIONS: DSM-V, CLINICAL AND RESEARCH

Implications for DSM-V. In the present study, OCP use was distributed evenly between the underweight and non-underweight AN groups, with a quarter of the AN sample (25.5%) and 40.2% of the non-eating disordered comparison sample using the OCP. Of the seven studies previously examined in the literature review assessing the clinical utility of the amenorrhea criterion (p. 30-39), three excluded individuals using the OCP and of those, only one US study reported the proportion of their patient sample using the OCP (7.1%; Roberto et al., 2008). The proportion of OCP users in the Australian treatment-seeking sample in the present study was substantially larger, possibly due to the wide availability and low-cost of OCPs which are subsidised under the Pharmaceutical Benefits Scheme by Medicare, by the Australian Government. The findings further diminish the utility of the amenorrhea criterion, when a substantial proportion of the treatment-seeking population in Australia uses the OCP. Hence, for Australian samples at the very least, results support the exclusion of the amenorrhea criterion in DSM-V proposed by the Eating Disorders Work Group (APA, 2012).

Clinical and research implications. As this is the first study examining OCP use in AN samples, the finding that there is little difference between the OCP and non-OCP groups tentatively suggests that, in clinical and research practice, these subgroups of patients need not be differentiated. Importantly, no differences were found between the
OCP and non-OCP groups on cognitive and physical AN diagnostic criteria, indicating that they had comparable illness severity.

The only differences found between the OCP and non-OCP AN groups, for cognitive confidence and depression, had small effect sizes, suggesting that they have little clinical or practical significance for treatment. These statistical findings, possibly due to increased Type 1 error, could not be planned for or predicted a priori. However, in an attempt to explain the differences, a literature search was conducted on pharmacological findings for psychological effects of the OCP. To the knowledge of the researcher, no studies have been published on the OCP and cognitive confidence, or on dysfunctional metacognitions in general. Some studies reported no short-term effects of OCP use on general cognitive function (e.g., Grinspoon et al., 2003; Silber, Almkvist, Larsson, Stock, & Uvnäs-Moberg, 1987). The effects of OCP use on mood or depression were inconsistent or contradictory, with some studies reporting improvements in depression (e.g., Freeman et al., 2001), worsening of depression (e.g., Lewis & Hoghughi, 1969), or no effect on mood (e.g., Grinspoon et al., 2003) resulting from OCP use. Findings were also dependent on several potential confounding factors such as pre-existing mood disorders, type and strength of OCP dose, length of use of OCPs, and psychosocial factors, hence, there appears to be individual and subgroup variation in response to OCPs (Bancroft & Sartorius, 1990). Despite the statistically significant results in the present study, in light of the small effect sizes, possibility of Type 1 error, and inconsistent pharmacological findings in existing literature, it would be unacceptable to suggest that OCP use resulted in higher cognitive confidence and lower depression in AN patients. Until more empirical evidence is available, no definite conclusions can be made.

In summary, there were mostly no differences found between the groups for diagnostic criteria of AN, factors proposed to maintain the disorder, and general psychopathology, suggesting that there is no utility in differentiating individuals with AN on the OCP and those who are not, in clinical and research settings. Hence, the AN patient sample in Research Questions 2 and 3 were not differentiated based on OCP use.

2.4 STRENGTHS, LIMITATIONS, AND FUTURE DIRECTIONS

A strength of this study was its originality in exploring a previously neglected area in the field of AN. General limitations of the research project as a whole are discussed later (p. 208-210). A limitation specific to the study was that due to its
psychological focus and exploratory nature, necessary understanding and interpretation of the biological mechanisms underpinning the effects of OCP use were beyond the scope of discussion. Future research should apply statistical control for familywise error rates, and take into account potential confounding factors including reasons why OCPs are sought or prescribed (e.g., for bone health, contraceptive purposes, to treat severe premenstrual cramps) and the types of health professionals consulted (e.g. general practitioner only, general practitioner and psychologist/psychiatrist).

3. **RESEARCH QUESTIONS 2(A) TO (D)**

<table>
<thead>
<tr>
<th>Research Question 2(a).</th>
<th>Are there differences between the AN, dieting, and non-dieting comparison groups in levels of perfectionism, self-esteem, the two control-related cognitions, and the five dimensions of dysfunctional metacognitions?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Follow-up analyses:</td>
<td></td>
</tr>
<tr>
<td>Research Question 2(b).</td>
<td>Are any group differences on the variables still significant after controlling for BMI?</td>
</tr>
<tr>
<td>Research Question 2(c).</td>
<td>Are any group differences in self-esteem still significant after controlling for depression?</td>
</tr>
<tr>
<td>Research Question 2(d).</td>
<td>Are any group differences in dysfunctional metacognitions still significant after controlling for depression and anxiety?</td>
</tr>
</tbody>
</table>

3.1 **INTERPRETATION OF FINDINGS**

The aim of Research Question 2(a) was to test for differences between the AN, dieting, and non-dieting comparison groups on the variables of interest. Statistically significant group differences were found between the groups in the expected direction, with the AN group reporting more highly pathological scores on perfectionism, self-esteem, and the control-related cognitions of general need for control and sense of control dependent on eating/weight, compared to both comparison groups. Of the five dysfunctional metacognition domains, statistically significant group differences were found only for the two control-related domains, negative beliefs about the uncontrollability/danger of thoughts and the need to control thoughts, with the AN group reporting a higher level of metacognitive dysfunction compared to both comparison groups. Effect sizes were large on all variables with significant differences with the exception of perfectionism, which had a medium effect size. To determine that
statistical significance was translated to clinical significance even for the medium-sized effect for perfectionism, an inspection of error bars representing 95% CIs for means of each group (Figure 7.2, p. 163) was conducted and indicated that the medium-sized difference was clinically significant, with the CIs for the AN group outside those of both comparison groups. Hence, the significant differences found between the AN and comparison groups for perfectionism, self-esteem, two control-related cognitive factors, and two control-related dysfunctional metacognitions were clinically significant, with practical relevance for treatment. The results confirm previous findings reviewed in Chapter 4, that perfectionism, self-esteem, and control-related issues are pertinent to AN, and that control-related issues in AN extend beyond the cognitive domain into higher level metacognitive domains. Comparing the dieting and non-dieting comparison groups, the only statistically significant difference was a stronger sense of control dependent on eating/weight in the dieting group. Contrasting the comparison groups with AN highlights the salience of perfectionism, self-esteem, control-related cognitions, and control-related dysfunctional metacognitions for AN.

Research Questions 2(b) to (d) were follow-up analyses aimed to determine whether the significant group differences between the AN, dieting, and non-dieting groups found for the six variables in Research Question 2(a) still existed after controlling for the potentially confounding factors of low weight, depression, and anxiety. Results for Research Question 2(b) indicated that after adjusting for low weight, the differences on all six variables continued to be statistically significant, with effect sizes still large for all variables except perfectionism (which continued to have a medium effect size), suggesting that the more highly pathological scores reported by AN patients were not just a result of cognitive deficits due to low weight. Results for Research Question 2(c) indicated that after adjusting for depression, the AN group still had significantly lower self-esteem than the comparison groups, with a large effect size, indicating that it was not a consequence of comorbid depression. Results for Research Question 2(d) indicated that after controlling for depression and anxiety, the AN group still had significantly higher control-related dysfunctional metacognitions (negative beliefs about the uncontrollability/danger of thoughts and need to control thoughts) than the comparison groups. While effect sizes were reduced, suggesting that the relationships between dysfunctional metacognitions, depression and anxiety were of importance, they did not fully explain the more dysfunctional level of control-related metacognitions in AN patients. To conclude, results for Research Questions 2(b) to (d) provide novel findings for the confounding effects of low weight, depression and
anxiety on the variables of interest in AN. Findings suggest that the pathological levels of the variables that characterised individuals with AN were attributable to more than cognitive deficits resulting from low weight, comorbid depression, or anxiety, and were instead features specific to AN.

3.2 Application of Findings in Context of Previous Literature

At present, no treatment for adults with AN have been found to be consistently effective (McIntosh et al., 2004; Serfaty et al., 1999) and mortality rates for the disorder are high (Nielson, 2001), postulated to be due to the lack of an empirically supported theoretical basis on which to develop treatment (Fairburn, 2005). Whilst a number of predisposing, precipitating and perpetuating factors have been proposed for AN (p. 46-47), the present study focused on factors theorised to be critical in cognitive theories of AN: perfectionism, low self-esteem, control-related cognitions, and dysfunctional metacognitions. Research Questions 2(a) to (d) were developed to assess the relationship between these factors and AN, a requisite first step before more complex maintenance models can be tested. Results of the present study will now be compared with the previously discussed literature reviews for each factor.

Perfectionism. Literature on the association between perfectionism and AN is broad, with four existing reviews and seven recent individual studies included in the literature review (p. 49-59). Results from the cross-sectional and longitudinal studies were generally consistent, finding that perfectionism had an effect on increases in and the maintenance of general eating pathology, AN was characterised by elevated perfectionism that endured after recovery, perfectionism had predispositional significance for the development of AN, and higher levels of perfectionism predicted poorer outcome in individuals with AN (Stice, 2002; Franco-Paredes et al., 2005; Bardone-Cone et al., 2007; Cassin & von Ranson, 2005; Castro-Fornieles et al., 2007; Bachner-Melman et al., 2007; Deas et al., 2011; Wade et al., 2008; Pike et al., 2008; Kim et al., 2010; Nilsson et al., 2008). However, a major sampling limitation of existing studies was the focus on ED symptom composites or BN pathology, with a lack of studies focusing on AN-specific symptomatology or populations (Stice, 2002). Research Question 2(a) addressed these limitations by focusing on an AN patient population that met all cognitive diagnostic criteria, confirming that individuals with AN possessed elevated levels of perfectionism compared to dieting and non-dieting comparison groups, with group differences of a medium effect size and as previously examined (Figure 7.2, p. 163), clinically significant. Because the dieting and non-
dieting comparison groups did not differ significantly, elevated levels of perfectionism were specific to individuals with clinical levels of EDs.

Further, to the knowledge of the researcher, no existing study controlled for the transient effects of starvation and low weight, which are associated with symptoms of rigidity and obsessionality, possibly linked to elevated levels of perfectionism. Research Question 2(b) addressed this gap in the literature, contributing novel findings that the medium effect size was retained after controlling for BMI.

*Low self-esteem.* A mass of literature was found to examine the association between low self-esteem and AN, with one existing review and nine recent individual studies included in the literature review (p. 61-72). Findings from the cross-sectional and longitudinal studies were generally consistent, reporting that low self-esteem had an effect on increases in and the maintenance of general eating pathology, AN was characterised by lowered self-esteem which improved with recovery, low self-esteem was a risk factor for the development of AN, and low self-esteem predicted poorer outcome in individuals with EDs (Jacobi et al., 2004; Wilksch & Wade, 2004; Gila et al., 2005; Brytek-Matera, 2007; Paterson et al., 2007, Bachner-Melman et al., 2007; Halvorsen & Heyerdahl, 2006; Nicholls & Viner, 2009; Halmi et al., 2005; Karpowicz et al., 2009). However, a major sampling limitation was most existing studies either collapsed across clinical groups to include AN, BN and EDNOS, or focused on BN and BED syndromes, with few studies of AN-specific syndromes, raising the issue of the specificity of risk factors for the development of AN (Jacobi et al., 2004). Further, the selection of healthy control groups was problematic as most studies utilised university students, confounding findings, as this population is known to have a higher prevalence of ED symptoms (Jacobi et al., 2004). Also, some studies did not screen the healthy control groups of restrained and unrestrained eaters to rule out the presence of an ED (e.g., Wilksch & Wade, 2004), making it unclear whether some restrained eaters met diagnostic criteria for an ED. Research Question 2(a) addressed these limitations by utilising an AN sample that met all cognitive diagnostic criteria, and comparison groups that were not limited to university students and excluded those who reported a current or previous ED, confirming that individuals with AN possessed lower self-esteem compared to the dieting and non-dieting comparison groups, with group differences having a large effect size, consistent with clinical significance. Because the dieting and non-dieting comparison groups did not differ significantly, low self-esteem was specific to individuals with clinical levels of EDs.
To date, only two studies have controlled for the confounding effects of depressive symptomatology whilst assessing the relationship between self-esteem and AN. Depression is prevalent in individuals with EDs and highly correlated with low self-esteem (Geller et al., 1998; Jacobi et al., 2004). The two studies had contradictory results, with one finding that group differences in self-esteem between AN and dieters disappeared after controlling for depression (Wilksch & Wade, 2004), and another finding a strong association between ED symptomatology and low self-esteem even after controlling for depression (Halvorsen & Heyerdahl, 2006). Further, to the knowledge of the researcher, no studies took into account possible confounding effects of starvation and low weight, which are associated with depression, possibly further influencing self-esteem. To address these disparities in the literature, Research Questions 2(b) and (c) clarified the confounding role of low weight and depression respectively, finding that individuals with AN retained significantly lower self-esteem with a large effect size compared to comparison groups, even after controlling for BMI and depression.

Control-related cognitions. Although the role of control has been widely cited in theoretical accounts and clinical observations of AN (e.g., Bruch, 1973; Slade, 1982; Waller, 1998), there is surprisingly limited and inconsistent empirical research underpinning the relationship between control-related factors and AN (Sassaroli et al., 2008; Surgenor et al., 2002; Stice, 2002). The two control-related cognitions pertinent to the present study were the general need for control and the sense of control being dependent on eating and weight. Only three cross-sectional studies were identified in a literature search, providing somewhat contradicting and tentative evidence for the importance of the control-related factors in AN (p. 74-78).

Considering the general need for control, one study found that AN patients had a lower general need for control compared to controls and the need for control was not significantly correlated with ED symptoms (Tiggemann & Raven, 1998), whilst another study found that a low need for control was an indicator of good outcome in AN (Lee et al., 2005). However, both these studies had methodological limitations, including data collection during therapeutic sessions, which influenced issues patients chose to disclose or avoid (Tiggemann & Raven, 1998), and the inclusion of less symptomatic ED patients who did not meet the AN diagnostic criterion of fear of gaining weight, confounding interpretation (Lee et al., 2005). For the sense of control being dependent on eating/weight, a qualitative exploratory study found that AN patients commonly
described their AN as providing a sense of control to their lives (Serpell et al., 1999). However, due to the exploratory nature of this single study in a small sample, the findings need to be replicated. To address the overall dearth of studies examining both the control-related cognitions and AN, and limitations of existing studies, Research Question 2(a) used quantitative measures and a larger sample size. For the AN patient sample, the present study required the fear of gaining weight criterion and all other AN cognitive diagnostic criteria to be met, and used de-identified data collected solely for research, with participation being emphasised to patients as confidential and having no effect on treatment. Findings disputed Tiggemann and Raven’s (1998) study, indicating, instead, an elevated general need for control and sense of control dependent on eating/weight in the AN patients compared to the comparison groups, supporting theories centered on control-related issues and Serpell et al.’s (1999) results. The group differences had large effect sizes, suggesting clinical significance. Perhaps unsurprisingly, the dieting comparison group also reported a significantly higher sense of control dependent on eating/weight compared to the non-dieting comparison group.

No existing study controlled for the transient effects of starvation and low weight, which are associated with symptoms of rigidity and obsessivity, possibly linked to elevated levels of need for control. To take the findings one step further, Research Question 2(b) confirmed that the group differences in each control-related variable remained even after controlling for BMI, with effect sizes continuing to be large.

Dysfunctional metacognitions. There has been recent interest in the role of metacognitions in AN, with the literature review identifying three recent cross-sectional studies providing preliminary support for this association (p. 80-86). As discussed, individuals with AN used more unhelpful metacognitive strategies which reinforced negative self-evaluations (Woolrich et al., 2008), and had higher levels of dysfunctional metacognitions compared to controls on four (out of five) dimensions of the MCQ-30, the need to control thoughts, negative beliefs about the uncontrollability/danger of thoughts, cognitive confidence, and cognitive self-consciousness (Cooper et al., 2007; McDermott & Rushford, 2011). These differences were retained even after controlling for low weight, with both control-related metacognitive domains having the largest effect sizes (McDermott & Rushford, 2011). However, limitations of existing studies included small sample sizes and failure to control statistically for large numbers of comparisons. To address the lack of empirical evidence in this new area, Research
Question 2(a) used a larger sample size and applied statistical control for the number of comparisons. Results were somewhat in contrast to previous findings. Whilst the AN group reported more dysfunctional metacognitions compared to both comparison groups, this was only specifically in the two control-related domains (negative beliefs about the uncontrollability/danger of thoughts and the need to control thoughts). Effect sizes were large, indicating a clinically significant difference.

Another point of interest was that two studies in the review found no differences in levels of metacognition between dieting and non-dieting controls (Cooper et al., 2007; Woolrich et al., 2008) while, in contrast, Konstantellou and Reynolds’ (2010) study of a non-clinical sample found that individuals with problematic eating attitudes had significantly greater levels of dysfunctional metacognitions compared to those with normal eating attitudes. Findings from Research Question 2(a) confirmed Cooper et al.’s (2007) and Woolrich et al.’s (2008) findings that there were no differences in levels of dysfunctional metacognitions in dieting and non-dieting comparison groups.

To date, only one study assessing dysfunctional metacognitions in AN controlled for general cognitive deficits due to low weight (McDermott & Rushford, 2011). To the knowledge of the researcher, no studies have controlled for depression and anxiety, which have been shown to be associated with elevated levels of dysfunctional metacognitions. To address specificity, Research Questions 2(b) and (d) assessed the association between dysfunctional metacognitions and AN whilst controlling for BMI, depression, and anxiety. Findings confirmed McDermott and Rushford’s (2011) results that elevated levels of control-related dysfunctional metacognitions in individuals with AN were not due to starvation effects in this population, with group differences still significant with a large effect size after controlling for BMI. Group differences on control-related dysfunctional metacognitions also continued to be significant after controlling for depression and anxiety, despite a reduction in effect size, indicating the disturbing extent to which individuals with AN were affected by metacognitive level control-related factors.

**Conclusion.** Results confirmed existing knowledge regarding the association of perfectionism and low self-esteem with AN, and clarified the association between control-related cognitive factors (the general need for control and sense of control dependent on eating/weight) and dysfunctional metacognitions with AN, for which existing evidence is scant and contradictory. Findings suggest that the above variables may be involved in maintenance of the disorder. Novel findings were also provided
through follow-up analyses, which provided clarification that the above variables were independently associated with or features of AN, and were not due to the potential confounding role of transient effects of starvation and low weight, or the effects of comorbid depression or anxiety. The findings have various clinical and research implications, discussed in the following section.

3.3 CLINICAL AND RESEARCH IMPLICATIONS

Clinical implications. The findings pave the way for development of appropriate psychosocial or cognitive interventions. Specifically, findings highlight the importance of targeting therapy at addressing hypercritical self-views and the unrelenting tendency to set extremely high goals, improving self-esteem and self-efficacy, and modifying the strong need for control shown in AN. Findings also raise the possibility that the poor efficacy of CBT for treatment of AN is due to its focus on the content of thoughts, which may be modified in treatment, but may not be effective in the long term due to crucial underlying control-related metacognitive processes. Metacognitive therapy could be utilised in the treatment of AN. However, before resources are directed towards targeting these factors, the testing of complex maintenance models to confirm the role of each factor in the maintenance of AN is required, as discussed in the following research implications.

Research implications. The present study clarifies the association between AN and perfectionism, self-esteem, control-related cognitive variables, and control-related dysfunctional metacognitions, a critical first step in examining maintenance factors of the disorder (Stice, 2002). Findings set the groundwork for further testing of more complex maintenance mechanisms of AN, such as moderation or mediation models involving more than one variable. The testing of more complex models is required to determine the amount of variance in AN symptoms possibly explained by the above factors, to ensure that clinical resources are not prematurely directed towards minor factors. Building on the findings, two such models were developed and tested in Research Question 3.

3.4 STRENGTHS, LIMITATIONS, AND FUTURE DIRECTIONS

Strengths. The present study addressed various methodological limitations in previous studies. This included using an AN-specific sample that met all cognitive diagnostic criteria. Although the physical diagnostic criteria were not required for inclusion in the AN sample, findings for Research Question 1 indicated that
subthreshold AN patients who did not meet the weight and/or amenorrhea criteria did not differ from full syndrome AN patients on the variables of interest (p. 159), suggesting that the subthreshold group should not be excluded in research practice, as previously discussed (p. 184). Another strength of the present study was not limiting comparison groups to university students, excluding individuals with ED diagnoses from comparison groups, utilising a larger sample size and quantitative measures, and using de-identified data collected solely for research. Importantly, the study also addressed specificity and contributed unique findings by conducting follow-up analyses to control for the potentially confounding effects of low weight, depression, and anxiety. Whilst a large body of empirical evidence exists for the association between perfectionism and low self-esteem with AN, studies examining control-related cognitions and dysfunctional metacognitions were scant and contradictory. The present study clarified these relationships. In accordance with Stice’s (2002) recommendation that more attention should be directed at identifying novel risk or maintenance factors, the present study also provided evidence that the specific control-related domain of dysfunctional metacognitions is pertinent to AN, suggesting a possible metacognitive profile for the disorder.

Limitations and future directions. General limitations of the research project as a whole are discussed later (p. 208-210). To further our understanding of the issue of control in AN, future research should examine other control-related factors such as perceived loss of control (Fairburn, Shafran, et al., 1999) and locus of control (Waller, 1998), which were beyond the scope of the present study. Additionally, future studies should seek to compare subtypes of AN on dysfunctional metacognitions, as the binge-purge subtype has most often been associated with obsessive-compulsive symptoms (Speranza et al., 2001), hence possibly control-related dysfunctional metacognitions.
4. Research Questions 3(a) and (b)

Research question 3(a). In (i) the AN group and (ii) the comparison group: Are the proposed individual pathways in Model A (Table 5.5, p. 135) significant? Do the data provide a good overall fit to Model A?

Research question 3(b). In (i) the AN group and (ii) the comparison group: Are the proposed individual pathways in Model B (Table 5.5, p. 135) significant? Do the data provide a good overall fit to Model B?

4.1 Interpretation of Findings

The aim of Research Question 3(a) was to assess the fit (of individual pathways and overall) of Model A to data from the AN and non-eating disordered comparison group, centred on the mechanism involving the general need for control, at the cognitive level. In the AN group, the variables in Model A (perfectionism, self-esteem, the general need for control, and sense of control dependent on eating/weight) were shown to have significant medium to strong correlations with the main outcome variable of drive for thinness. In the comparison group, correlations were small to medium, with no correlation found between the sense of control dependent on eating/weight and drive for thinness.

The aim of Research Question 3(b) was to assess the fit (of individual pathways and overall) of Model B to data from the AN and non-eating disordered comparison group. Model B was adapted from Model A through taking the control-related mechanism in Model A (centred on the general need for control) up to the metacognitive level (centred on control-related dysfunctional metacognitions, namely the need to control thoughts and negative beliefs about the danger/uncontrollability of thoughts). In the AN group, the variables in Model B were shown to have significant medium to strong correlations with the main outcome variable of drive for thinness. Importantly, both of the higher order control-related metacognitions had strong correlations with the general need for control, confirming the validity of adapting Model B (metacognitive level control-related factors) from Model A (cognitive level control-related factors). In the comparison group, the variables had small to medium correlations with drive for thinness, and whilst the corresponding relationship between metacognitive and cognitive level control-related variables was also observed, the correlations were smaller in magnitude.
AN sample. Path analyses confirmed that in the AN sample, all direct and indirect pathways predicted in Model A and Model B were significant (p. 172-173, 176), with the exception of the direct effects of perfectionism and the need to control thoughts, on drive for thinness. Taking into account the significant positive correlation between both variables with drive for thinness, as well as the significant indirect effect of both variables on drive for thinness, this finding suggests that the effect of perfectionism and the need to control thoughts on drive for thinness was fully mediated by other variables in the model. Importantly, the cognitive control-related mechanisms in Model A (direct and indirect pathways involving the general need for control and sense of control dependent on eating/weight) and metacognitive control-related mechanisms in Model B (direct and indirect pathways involving the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts) were confirmed. The variables of both models explained a considerable proportion of the variance in the outcome variable, drive for thinness. Fit indices indicated that overall fit of Model A and Model B to the AN data was excellent, providing evidence that the proposed cognitive and metacognitive control-related mechanisms maintain drive for thinness in AN.

Comparison group. In the comparison group, whilst some individual pathways were significant (e.g., the direct effects of perfectionism and self-esteem on drive for thinness), the key control-related mechanisms in Model A (direct and indirect pathways involving the general need for control and sense of control dependent on eating/weight) and Model B (direct and indirect pathways involving the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts) were mostly non-significant (p. 173-174, 177). A relatively small amount of variance in the outcome variable, drive for thinness, was explained by the variables of both models. Fit indices indicated that overall fit of Models A and B were unacceptable, suggesting that in the non-eating disordered comparison group, other factors not included in the models may be more important in predicting drive for thinness. It must be kept in mind that the size of drive for thinness in the comparison group was smaller compared with that of AN, possibly resulting in a diminished range of drive for thinness scores and limiting correlations. This is further discussed later in the chapter (p. 207).
4.2 APPLICATION OF FINDINGS IN CONTEXT OF PREVIOUS LITERATURE

Background to Model A. To date, empirical evaluation of AN treatment is limited and findings have been disappointing. Overall, evidence for any AN treatment is weak, with no clear leaders in treatment efficacy when comparing CBT with other conditions (p. 113-115). Most AN patients remained unwell and developed chronic disabilities even after receiving specialist treatment (McIntosh et al., 2005; Serfaty et al., 1999; Hay et al., 2003; Treasure & Schmidt, 2005), highlighting the need for a better understanding of maintenance mechanisms that sustain the symptoms of the disorder and make it so resistant to treatment (Wilson et al., 2007).

Whilst various cognitive maintenance theories have been proposed, they have not been empirically investigated (Klein & Walsh, 2005). Many studies have examined isolated factors associated with AN; however the mechanisms or processes through which the factors maintain AN are unclear and poorly understood. Research Question 3(a) aimed to address the gap in the literature and further theoretical understanding of the disorder by empirically testing Model A, a maintenance mechanism adapted from Fairburn and colleagues’ (1999) cognitive theory of AN focused on control-related cognitions, in particular the pathological need for control (described in Chapter 5, p. 94-96).

Background to Model B. Due to the lack of evidence for the effectiveness of AN treatment based on CBT, researchers recommended that attention should be directed towards the consideration of new variables in developing both theory and treatment interventions for AN (Stice, 2002). The present study focused on the recent interest in higher level metacognitions and the relevance of metacognitive theory. Similarities were noted in the focus on issues of control between Fairburn and colleagues’ (1999) cognitive theory for AN and metacognitive theory (p. 126-128). Interestingly, a literature review (p. 81-83) revealed that the two control-related metacognitive domains, the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts, were elevated in individuals with AN compared to controls (Cooper et al., 2007; Woolrich et al., 2008), with the largest effect sizes compared to other metacognitive domains (McDermott & Rushford, 2011). This was confirmed by Research Question 2, which found that this group difference retained significance even after controlling for confounding effects of low weight, depression, and anxiety. Overall, evidence indicated the disturbing extent to which individuals with AN were...
affected by metacognitive level control-related factors, suggesting that the control-related issues experienced by individuals with AN extends to the metacognitive domain.

Whilst links have been drawn between dysfunctional metacognitions and AN, no theory has been developed to explain the specific mechanisms or pathways through which metacognitive variables could maintain symptoms of AN. Research Question 3(b) aimed to examine this novel concept by empirically testing Model B, a maintenance mechanism that was adapted from Model A, through shifting the focus from control-related cognitions to higher level control-related metacognitions.

*Development of Models A and B.* Model A and Model B were built on existing findings in the literature, clinical observations (including Fairburn, Shafran, et al.’s [1999] proposed model), and results from Research Question 2, which indicated that the following individual variables were associated with AN even after controlling for potentially confounding factors of low weight, depression, and anxiety: perfectionism, self-esteem, the general need for control, sense of control dependent on eating/weight, the need to control thoughts, and negative beliefs about the uncontrollability/danger of thoughts. The present study was interested in the specific pathways (direct and mediating effects) in which these variables worked together to maintain the key symptom of AN, dietary restraint or drive for thinness.

The rationale behind specific pathways in Model A was previously discussed (p. 130-133), and direct and mediation pathways tested were presented schematically in Figure 5.7 (p. 133) and summarised in Table 5.5 (p. 135). The rationale behind specific pathways in Model B was previously discussed (p. 133-134), and direct and mediation pathways tested were presented schematically in Figure 5.8 (p. 134) and summarised in Table 5.5 (p. 135). Put simply, the control-related cognitive factor in Model A (i.e., the general need for control) was replaced by higher level control-related metacognitive factors in Model B (i.e., the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts). Findings from the present study justified this adaptation, finding that both of the control-related metacognitive factors were significantly correlated with the general need for control in the AN and comparison samples.

Models A and B were tested using a cross-sectional design, to contribute to the theoretical understanding of AN from which future therapeutic interventions could be derived. To elucidate cognitive mechanisms specific to AN, both models were tested in
an AN and non-eating disordered comparison group. Findings for mechanisms involving perfectionism and low self-esteem will now be discussed in the context of literature reviews. Whilst it was the intention of the researcher to discuss findings for mechanisms involving control-related cognitive and metacognitive variables in the context of existing evidence, few or no studies were found, hence results are discussed in the context of clinical observations or theoretical propositions.

Specific mechanisms involving perfectionism. The literature review revealed an absence of studies of more complex models involving perfectionism, with only three studies identified (p. 98-102). The studies provided limited support for an interactive relationship between perfectionism, self-esteem, and control-related cognitive factors, which have main and moderating effects on drive for thinness (Sassaroli et al., 2008; Davis, 1997). There was also indirect evidence that targeting perfectionism in therapy resulted in improvement in BN, although it is unclear if the role of perfectionism in maintaining BN extends to AN (Steele & Wade, 2008). Evidently, empirical evidence of any explanatory mechanism leading from perfectionism to ED symptoms is lacking. Limitations of existing studies included small group sample sizes (e.g., Steele & Wade, 2008). Also, studies combined ED categories of AN, BN, and EDNOS (e.g., Davis, 1997) or even ED and control samples (e.g. Sassaroli et al., 2008), raising concerns about interpretation due to qualitative differences between the groups, thus impeding the understanding of AN-specific mechanisms. To address the lack of evidence for the role of perfectionism in maintaining AN as well as limitations of existing studies, Models A and B were tested in a larger AN-specific sample that met all cognitive diagnostic criteria of the disorder, and a non-eating disordered comparison group.

Results clarified and extended the role of perfectionism in the maintenance of AN, showing in the AN group only, (i) there was a strong association between perfectionism and self-esteem, (ii) perfectionism had a direct effect on the general need for control and the need to control thoughts, (iii) perfectionism had an indirect effect on drive for thinness through the mediating control-related cognitive mechanism (involving the general need for control) and metacognitive mechanism (involving the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts). At the cognitive level, results are consistent with Fairburn and colleagues’ (1999) assertion that perfectionism has a complex interactive relationship with low self-esteem, and combined with the unrelenting high standards of individuals with AN, the general need for control develops, also encompassing the metacognitive need to control thoughts.
With eating, food, and weight being more within one’s control than other life domains in an unpredictable life, the cognitive and metacognitive need for control becomes focused on drive for thinness.

Specific mechanisms involving self-esteem. Six studies assessing maintenance mechanisms involving self-esteem were included in the literature review (p. 103-110). The studies provided consistent evidence that, in individuals with AN, there was an interactive relationship between perfectionism and low self-esteem, both of which predicted drive for thinness (Surgenor et al., 2007), drive for thinness was used to cope with distress from negative self-beliefs, resulting in an increased sense of control and self-esteem (Woolrich et al., 2006; Cooper et al., 1998), and dysfunctional metacognitive strategies were used to deliberately diminish self-esteem by making one feel worse about themselves (Woolrich et al., 2008). Further, indirect evidence from treatment trials targeting self-esteem in ED patients (Newns et al., 2003) and non-clinical samples (O’Dea & Abraham, 2000) reported that changing this factor was effective in disrupting ED maintenance mechanisms, resulting in improved eating attitudes and body satisfaction, and decreased drive for thinness and self-worth invested in the body. The findings provided support for the role of self-esteem in maintaining AN. Limitations of existing studies included small sample sizes due to the qualitative nature of some studies, and using ED samples that combined patients with AN, BN, EDNOS and BED (e.g., Newns et al., 2003). To address these limitations, Models A and B were tested using quantitative methods, in a larger AN-specific sample that met all cognitive diagnostic criteria of the disorder, and a non-eating disordered comparison group.

Results confirmed and extended the role of low self-esteem in the maintenance of AN, showing in the AN group only, (i) there was a strong association between self-esteem and perfectionism, as above, (ii) self-esteem had a direct effect on the general need for control and the need to control thoughts, (iii) self-esteem had a direct effect on drive for thinness, and (iv) self-esteem had an indirect effect on drive for thinness through the mediating control-related cognitive mechanism (involving the general need for control) and metacognitive mechanism (involving the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts). The findings confirm existing knowledge of the role of low self-esteem in maintaining AN. Low self-esteem directly feeds into drive for thinness, in a possible attempt to increase self-esteem through achieving a thinner, more attractive body. Low self-esteem also interacts with
perfectionism, leading to a strong general need for control that encompasses the metacognitive need to control thoughts, perpetuating drive for thinness. Findings confirm Woolrich et al.’s (2008) proposition that negative self-evaluation interacts with dysfunctional metacognitions to perpetuate AN. In individuals with AN, dietary restraint is possibly a strategy to cope with negative self-evaluation by controlling and performing well in at least one area of various uncontrollable life domains.

*Specific mechanisms involving control-related cognitive factors.* The factors relevant to Model A were the general need for control and sense of control being dependent on eating/weight. Despite the widely accepted notion that the general need for control is critical to the maintenance of AN, to the knowledge of the researcher, no studies testing the notion have been published. One study has examined mechanisms involving the sense of control dependent on eating/weight and was included in the literature review (p. 111-112). It reported that, in individuals with EDs, symptoms of dietary restraint or starvation were positively valued and interpreted in terms of control (Shafran et al., 2003). The major limitation of the study was combining AN, BN, and EDNOS categories to form the clinical comparison group. Research Question 3(a) addressed the dismal lack of empirical data for AN maintenance mechanisms involving control-related cognitive factors through testing Model A in an AN-specific sample that met all cognitive diagnostic criteria of the disorder, and a non-eating disordered comparison group.

Results revealed that in the AN group only, (i) a large proportion of variance in the general need for control was explained by the predictors, perfectionism and low self-esteem, (ii) the general need for control had a significant direct effect on drive for thinness, and (iii) drive for thinness had a significant direct effect on the sense of control being dependent on eating/weight. Results provide the first empirical confirmation for the strength of relationships between AN and cognitive aspects of control, in particular the general need for control and sense of control dependent on eating/weight, indicating that they are critical to maintaining AN. Findings support Fairburn and colleagues’ (1999) proposed cognitive maintenance mechanism of AN, specifically, that the general need for control develops from elevated perfectionism and pervasive low self-esteem. The need for control becomes fixated on control over eating/weight-related phenomena, encapsulated in drive for thinness, which is then maintained as eating/weight becomes an index of self-control to the individual with AN.
Specific mechanisms involving control-related metacognitive factors. Due to the infancy of interest in the area and the novelty of ideas proposed in Model B, no studies examining mechanisms involving metacognitive factors in AN were found. Woolrich and colleagues (2008) proposed that individuals with dysfunctional metacognitive coping strategies were more likely to turn to behavioural strategies (such as dietary restraint) to cope with distressing thoughts, maintaining AN. This is consistent with Fairburn and colleagues’ (1999) cognitive-level theory of AN, which posits that drive for thinness resulted from the pathological need for control and was maintained by perceived loss of control. Research Question 3(b) confirmed these clinical observations and extended them by testing Model B in an AN group that met all cognitive diagnostic criteria of the disorder, and a non-eating disordered comparison group.

Results revealed that, in the AN group only, (i) perfectionism and self-esteem had significant indirect effects on drive for thinness through the mediating metacognitive variables (the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts), and (ii) the need to control thoughts had a significant indirect effect on drive for thinness through the mediating variable of negative beliefs about the uncontrollability/danger of thoughts. The findings provide the first empirical confirmation for the strength of relationships between AN and aspects of metacognitive control, confirming Woolrich and colleagues’ (2008) clinical observations and providing information about the specific dysfunctional metacognitions that are pertinent in AN, and the pathways through which they maintain the disorder. Specifically, the strong need for control that develops from the interactive relationship between perfectionism and low self-esteem extends to the metacognitive domain in the form of the need to control thoughts. When thoughts are perceived as uncontrollable or dangerous in the context of this strong metacognitive need for control, individuals with AN resort to behavioural control strategies instead, specifically, dietary restraint or the drive for thinness.

Overall fit of the theoretical model. Although elegant theories for AN have been proposed, the literature review highlighted the observation that most theories have been developed primarily on the basis of clinical observations (Cooper, 1997). Research Question 3(a) was aimed at addressing the urgent need for generation of more complex empirical data to test existing proposed maintenance theories of AN, whilst Research Question 3(b) tested a novel metacognitive maintenance mechanism of AN. In the AN group only, variables in Models A and B explained a considerable proportion of the
variance in the outcome measure, drive for thinness, and the overall fit of both models was excellent. The fit was poor in the comparison group. Evidently, the vicious cycle involving perfectionism, self-esteem, and control-related cognitive and metacognitive variables, played out in drive for thinness, is fulfilled in AN populations and serves to maintain the disorder.

**Conclusion.** Results confirmed and extended existing knowledge regarding the role of low self-esteem in the maintenance of AN. Novel findings were contributed for the role of perfectionism, control-related cognitive variables (the general need for control and sense of control dependent on eating/weight), and control-related metacognitive variables (the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts). In Models A and B, the fit of control-related cognitive and metacognitive individual pathways, as well as overall fit of the models, were strong only in the AN sample, confirming part of Fairburn and colleagues’ (1999) proposed cognitive model of AN and the importance of metacognitive processes in maintaining AN. The findings have clinical and research implications, discussed in the following section.

### 4.3 Clinical and Research Implications

**Clinical implications.** The testing of mediation pathways in Models A and B pave the way for development of appropriate treatment for AN, providing new points at which therapy can be targeted, specifically, the mediating control-related cognitive and metacognitive variables. It would obviate the need to directly tackle other longstanding, pervasive characteristics (e.g., perfectionism).

At the cognitive level, findings suggest that a focus in therapy on methods to change or redirect control in AN would disrupt a central maintenance mechanism of the disorder, with the potential to achieve secondary changes in drive for thinness. Suggested therapeutic techniques include addressing the strong need for control through cognitive restructuring, shifting the focus of control and self-worth away from dietary restriction or weight/shape by helping patients derive a sense of achievement and satisfaction form other activities, and tackling behaviours (e.g., hypervigilant body checking) that result in a perceived threat to or loss of control (Fairburn, Shafran, et al., 1999).

At the metacognitive level, findings suggest that changing metacognitive-level control issues in therapy for AN would disrupt the central maintenance mechanism of
the disorder, potentially resulting in secondary changes in drive for thinness. Given the demonstrated effectiveness of metacognitive therapy in several psychological disorders (Wells, 2009), it could hold therapeutic potential for AN. Metacognitive therapy could include psycho-education about the impact of dysfunctional metacognitions and metacognitive coping strategies, normalisation of the existence of negative thoughts about eating/weight/shape, and strategies focussed specifically on metacognitive processing, such as those outlined by Wells (2009) for anxiety and mood disorders.

Research implications. The present study confirms the importance of control-related issues in maintaining AN, setting the groundwork for clinical trials of AN treatment targeting control-related issues at the cognitive and metacognitive level. To the knowledge of the researcher, no published trials have specifically assessed CBT focused on control-related factors or metacognitive therapy in AN samples, potentially promising areas for further study. However, existing empirical evidence does not provide a clear picture of the full range of control-related issues in individuals with AN, meanings ascribed to such control, and the types of control that are amenable to treatment (Surgenor et al., 2002). The refinement of the definition and operationalisation of control in its possibly many manifestations in AN is an important area that needs further development through research. Future research should test mechanisms involving other control-related factors proposed to be important in maintaining AN, such as perceived loss of control (Fairburn, Shafran, et al., 1999) and locus of control (Waller, 1998).

4.4 Strengths, Limitations, and Future Directions

Strengths. The present study addressed various methodological limitations in previous studies, through using a larger AN-specific sample that met all cognitive diagnostic criteria and quantitative measures, leading to increased power. Although the physical diagnostic criteria were not required for inclusion in the AN sample, findings for Research Question 1 indicated that subthreshold AN patients who did not meet the weight and/or amenorrhea criteria did not differ from full syndrome AN patients on the variables of interest (p. 159), suggesting that the subthreshold group should not be excluded in research practice, as previously discussed (p. 184). To address specificity, the models were also tested in a non-eating disordered comparison group, to elucidate cognitive mechanisms unique to AN.

In line with recommendations that cognitive theory of AN should move towards the consideration of new factors (Stice, 2002), the present study also contributed novel
findings, specifically, testing maintenance mechanisms involving control-related cognitive and metacognitive factors, for which there is a dearth of existing empirical evidence.

Limitations and future directions. General limitations of the research project as a whole are discussed later (p. 208-210). Statistical and design limitations include the use of path analyses to test Models A and B instead of the more powerful structural equation modelling, necessary because of the statistical need to reduce the complexity and number of model parameters so that the sample sizes of the present study would suffice (AN group, n = 110; comparison group, n = 132). To enable the use of structural equation modelling, which controls for measurement error, future research should aim to test the models in larger samples (N > 200; Hair et al., 1999; Boomsma & Hoogland, 2001).

Path analyses also precluded the testing of feedback loops or bi-directional relationships (Stage et al., 2004; Blunch, 2008), restricting analyses in the present study to the testing of a priori determined relationships. Future research should utilise prospective or longitudinal designs to test the bi-directional pathways proposed in Fairburn et al.’s (1999) cognitive maintenance model of AN (e.g., the drive for thinness predicts the sense of control being dependent on eating/weight, which in turn further perpetuates drive for thinness in a vicious cycle).

Further, it should also be noted that drive for thinness was smaller in the comparison group, resulting in a diminished range of drive for thinness scores that could limit correlations and consequently, analyses of predicted pathways. It was also not possible to run separate analyses for the dieting and non-dieting comparison subgroups, as they were combined to ensure a sufficient sample size for path analysis. However, since the dieters had a significantly greater drive for thinness than the non-dieters in the comparison group (p. 154), further research with larger numbers (N > 200; Hair et al., 1999; Boomsma & Hoogland, 2001) would help clarify potential significance and differences between the dieting and non-dieting subgroups.

Findings suggest that both Models A and B provide an excellent fit to the AN data. It would be interesting to combine both models to test a more complex control-related maintenance model of AN that included cognitive and metacognitive level factors. This was beyond the scope of the present study since the samples were of insufficient size to test a model of increased complexity. Future research with larger
sample sizes ($N > 200$; Hair et al., 1999; Boomsma & Hoogland, 2001) should aim to further these findings.

While the present study was one of the first to test more complex maintenance models of AN, the models confined themselves to a handful of maintenance factors and did not attempt to assess all potential factors in AN. It should be highlighted that a substantial amount of variance in drive for thinness remains unexplained. There are many other factors that have been proposed to maintain AN (listed in Table 4.1, p. 47), and other models from different clinical or theoretical perspectives wait to be developed and tested. Future research should investigate the relationships of other factors with those currently identified, in order to construct an integrated biopsychosocial model of maintenance of AN.

5. General Limitations and Recommendations for Future Research

The main limitation of the research project was the use of a cross-sectional design, which may be a necessary initial step in the research of risk and maintenance factors, but precludes causal interpretations. For Research Questions 1(a) and (b), which tested for differences between full syndrome, subthreshold, and OCP-using AN subgroups, the use of a cross-sectional design that differentiated patients based on presenting symptoms made it impossible to determine the course of illness or potential shifts across subgroups (e.g., Dalle Grave et al., 2008). For Research Questions 2(a) to (d), which tested for differences in variables of interest between AN, dieting, and non-dieting comparison groups, the use of a cross-sectional design constrained temporal inferences about whether the variables of interest preceded the onset of AN (i.e., trait effects), were related to transient effects of the disorder (i.e., state effects), or were a product of AN (e.g., Stice, 2002; Jacobi et al., 2004). For Research Questions 3(a) and (b), which tested two proposed maintenance models of AN in an AN and non-eating disordered comparison group, the cross-sectional design constrained inferences about the directionality of the mechanisms being tested (Byrne, 2010; Blunch, 2008). Although path analyses are intended to test a priori determined unidirectional relationships, the cross-sectional design precludes causal conclusions, thus findings of relationships between variables can only be interpreted confidently as correlates. To address these limitations, future research should utilise prospective longitudinal designs with multiple time points and long periods of follow-up to clarify the temporal
relationship between the variables of interest and AN, and to assess whether the variables of interest influenced symptom-level change or treatment outcomes. Experimental or treatment studies (e.g., randomised controlled trials targeting specific variables of interest) should also be conducted to determine whether changes in the variables of interest as a result of treatment is associated with decreases in symptoms or improved treatment outcome, to allow for more meaningful conclusions to be drawn about causation and for bidirectional hypotheses of the development and maintenance of AN to be tested.

Another limitation of the research project was the exclusion of males, an increasingly affected but under researched population (Ziora et al., 2006). The majority of the sample was Caucasian Australian females, limiting generalisability of results to males and other ethnic groups. The AN group also utilised a patient sample, limiting findings to individuals of sufficient severity to warrant prolonged treatment. Individuals with AN who were less severely unwell or not actively seeking treatment were not represented. Findings should be replicated in other countries and cultures, in samples that include males and non-treatment seeking AN populations, to increase generalisability.

For the non-eating disordered comparison group, participants were excluded if they reported a current or previous eating disorder. However, bingeing, purging, and excessive exercise was not evaluated, presenting the possibility that the comparison group data could have been contaminated by participants with subthreshold or undiagnosed eating disorders. The non-eating disordered comparison group was further categorised into dieting and non-dieting subgroups, based on endorsement of a single item regarding dieting/restriction from self-report measures. Aside from the general limitations of self-report measures (discussed below, p. 210), the use of a single item with binary responses (“Yes” or “No”) does not capture varying degrees of dieting/restriction (e.g., time spent restricting, calories/kilojoules per day, food groups eliminated). Future research should assess potential eating disorder symptoms and dieting/restriction behaviours in more detail using stringent assessment procedures (e.g., EDE interview; Fairburn & Cooper, 1993), to prevent contamination of comparison group data.

The research project also did not include BN, EDNOS, or non-eating disordered psychiatric control groups, raising the issue of specificity of risk factors for the development of AN over other eating disorders and psychopathology in general.
(Bardone-Cone et al., 2007). Future research should include control groups with other ED or psychiatric diagnoses to clarify AN-specific mechanisms.

Finally, the use of single reporter and self-report measures was a limitation. Some of the variables of interest assessed in the research project (e.g., self-esteem) may operate outside of conscious awareness, thus may not be accessible using measures that require conscious reflection on one’s own beliefs. In responding to self-report measures, AN populations also have the propensity to answer defensively, tend to be less objective and aware of anxious feelings, and are impacted by the effects of starvation (Gila et al., 2005). However, given the subjective nature of the data for all variables of a psychological nature, existing subjective processes at work in participants influenced all the data in a coherent manner, as correlations were found between variables for which relationships were proposed to exist. Future studies should utilise a range of data collection methods including collateral reports, observational data, implicit measures, focus groups, or interview measures (Stice, 2002; Cassin & von Ranson, 2005), which have been found to be superior to questionnaires in measuring some variables of interest (e.g., self-esteem; Wilksch & Wade, 2004).

6. CONCLUSION

In view of the controversy regarding the existing diagnostic criteria for AN and the lack of effective treatments for the disorder, the present study examined the utility of physical diagnostic criteria for AN, existing and novel maintenance factors for AN, and two control-related mechanisms, at the cognitive and metacognitive level, through which these factors maintained the disorder. Some findings extend current knowledge whilst most findings are novel, discussed below.

6.1 FINDINGS THAT CLARIFY AND EXTEND CURRENT KNOWLEDGE

Clinical utility of the weight/amenorrhea criteria for AN. Present findings confirm previous knowledge that there is no evidence for the clinical utility of the weight and amenorrhea criteria when considering variables including drive for thinness, body image disturbance, perfectionism, and general psychopathology. Findings extend existing knowledge through the assessment of novel variables: cognitive or psychological diagnostic criteria of AN (fear of gaining weight, self-worth invested in the body) and other factors known to be pertinent in maintaining the disorder (self-esteem, control-related cognitions, and dysfunctional metacognitions), providing
compelling evidence that there are no clinically important differences in the variables of interest to the present study signified by the physical AN criteria.

Association of perfectionism and low self-esteem with AN. Present findings confirm existing knowledge that perfectionism and low self-esteem are more severe in individuals with EDs than dieting and non-dieting controls, adding to current knowledge by confirming that this difference exists at a clinically significant level in an AN-specific sample. The research project also addresses the paucity of and contradictory evidence regarding the potentially confounding effects of low weight and depression, contributing novel findings that the clinically significant associations between perfectionism and low self-esteem with AN are retained even after controlling for BMI and depression, suggesting that both are independently associated with or features of AN.

6.2 Novel Findings

Clinical significance of OCP use in individuals with AN. There has been a lack of knowledge about potential differences between AN patients signified by OCP use, with previous studies typically excluding individuals on the OCP from AN groups when examining clinical utility of the amenorrhea criteria. The present research project contributes new findings for the proportion of and differences signified by OCP use in AN patients. Despite individuals with AN being commonly infertile due to low weight, a quarter of the Australian AN sample in the present study were using OCPs. No clinically significant differences were found for AN patients using the OCP and those who were not, for most clinically important psychological factors, including AN diagnostic criteria, variables pertinent to the maintenance of AN, and general psychopathology.

Association of the general need for control with AN. The present research project examines the general need for control in an AN-specific group for the first time. In contrast to a single previous study (Tiggeman & Raven, 1998) which reported that individuals with EDs had a lower general need for control compared to controls, the AN group had an elevated general need for control compared to dieting and non-dieting comparison groups, with differences of large, clinically significant effect sizes. Findings provide the first support for theories centered on control-related issues (e.g., Fairburn, Shafran, et. al., 1999; Slade, 1982) in an AN sample. The research project also
addresses a novel area, the potential confounding effect of low weight, contributing novel findings that the clinically significant association between general need for control with AN are retained even after controlling for BMI, suggesting that it is a feature of the disorder.

**Association of sense of control being dependent on eating/weight with AN.** The present study is the first quantitative study examining the sense of control dependent on eating/weight in AN, extending findings from a single previous qualitative study (Serpell et al., 1999), confirming that the sense of control dependent on eating/weight is elevated in the AN patients compared to dieting and non-dieting comparison groups, with differences of large, clinically significant effect sizes. Findings address the lack of empirical evidence for AN theories centered on control-related issues (e.g., Fairburn, Shafran, et al., 1999). The research project also addresses a novel area, the potentially confounding effect of low weight, contributing new findings that the clinically significant association between sense of control dependent on eating/weight with AN is retained even after controlling for BMI, suggesting that it is a feature of the disorder.

**Association of control-related dysfunctional metacognitions with AN.** The research project found that in contrast to two existing studies, for which individuals with AN were elevated on four (out of five) dimensions of dysfunctional metacognitions compared to controls (Cooper et al., 2007; McDermott & Rushford, 2011), the AN group was elevated on only two dimensions of dysfunctional metacognitions, both of which were control-related, compared to dieting and non-dieting comparison groups. The control-related domains for which large and clinically significant differences were found were the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts. Further, the research project explores another novel area, the potential confounding effects of low weight, depression and anxiety, contributing new findings that the significant association between the two control-related dysfunctional metacognitions with AN are retained even after controlling for BMI, depression, and anxiety, despite a reduction in effect size. Findings provide novel evidence that control-related issues in AN extend beyond the cognitive domain into higher level metacognitive domains, providing an AN-specific metacognitive profile.

**Evidence of maintenance of AN through a control-related cognitive mechanism.** The research project is one of the early few to test a complex maintenance mechanism of AN, and the first to test a maintenance mechanism involving control-related
cognitive factors. Findings provide support for a cognitive-level model which proposes that perfectionism and low self-esteem predict drive for thinness, the operational definition of the main symptom of AN, refusal to maintain minimally normal weight, through the mediating variable of the general need for control. The model was an excellent fit to AN data but not to the non-eating disordered comparison group data, providing the first empirical confirmation for the strength of relationships between AN and cognitive aspects of control, in particular the general need for control and sense of control dependent on eating/weight. Findings indicate that both control-related cognitive factors are critical in maintaining AN, confirming part of Fairburn and colleagues’ (1999) proposed cognitive theory of AN.

Evidence of maintenance of AN through a control-related metacognitive mechanism. The research project is the first to propose a novel, metacognitive-level maintenance mechanism of AN and test it empirically, as interest in this area in the AN field is in its infancy. The model proposes that perfectionism and low self-esteem predict drive for thinness, through the mediating mechanism centred around control-related dysfunctional metacognitions (the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts). The model was an excellent fit to AN data but not to the non-eating disordered comparison group data, providing the first empirical confirmation for the strength of relationships between AN and metacognitive aspects of control, in particular the need to control thoughts and negative beliefs about the uncontrollability/danger of thoughts. The results indicate that they are critical to maintaining AN, and confirm that the pathological general need for control in AN extends to the metacognitive domain in the form of the need to control thoughts. Hence, when thoughts are perceived as uncontrollable or dangerous, individuals with AN resort to behavioural control strategies instead, specifically, drive for thinness.

6.3 Summary

The research project has a number of important implications. On the basis of findings, current DSM criteria should be applied flexibly in clinical and research practice, and results support changes to the weight criterion and deletion of the amenorrhea criterion proposed for DSM-V. Evidently, crucial underlying control-related cognitive and metacognitive mechanisms maintain AN, providing new points at which therapy can be targeted in clinical practice, obviating the need to directly tackle other longstanding, pervasive characteristics associated with AN. New AN therapies targeting control-related issues at the cognitive and metacognitive level can be further
tested in prospective longitudinal or experimental studies. Finally, present findings pave the way for future research to test AN maintenance models of increased complexity involving both cognitive and metacognitive factors, other control-related variables, bi-directional pathways, and factors (e.g., sociocultural, familial, interpersonal, neurobiological) proposed in other theories of AN, to develop a more comprehensive understanding of maintenance of the disorder.
REFERENCES


Clinton, D. N., & Glant, R. (1992). The eating disorders spectrum of DSM-III-R: Clinical features and psychosocial concomitants of 86 consecutive cases from a Swedish urban catchment area. *Journal of Nervous and Mental Disease, 180*, 244-250.


239


APPENDICES

APPENDIX A: DESCRIPTION OF TREATMENT MODALITIES

**Inpatient treatment.** Treatment involves intensive 24-hour acute care and supervision, with the primary aim to achieve weight restoration. Treatment may be voluntary or involuntary, and is normally for a duration of four to five weeks. Some goals unrelated to weight gain are also a focus of treatment, with dietary, medical, psychological, social and family therapeutic assistance provided if required. During admission, treatment plans are made twice weekly, with the multi-disciplinary treating team and patient participating in decision-making. The inpatient group program includes shopping with staff, cooking and preparing meals, guided relaxation, music therapy, psychological therapy, and art therapy.

**Day patient treatment.** This is an intensive form of outpatient treatment that involves daily weekday involvement over eight-week blocks. Day patient treatment sometimes follows an inpatient admission. The program focuses on motivational enhancement, developing practical life skills, and coping with eating in individual and social situations. During admission, treatment plans are made twice weekly, with the multi-disciplinary treating team and patient participating in decision-making. The day patient group program includes planning and cooking meals, food challenges (e.g., eating in public at restaurants, ordering takeaway), and intensive psychological therapy focusing on higher-level skills (e.g., cognitive restructuring, assertiveness training).

**Outpatient treatment.** Treatment sometimes follows an inpatient admission or day patient treatment. It involves regular individual appointments with a consultant psychiatrist and dietician, and focuses on weight maintenance.
PARTICIPANT INFORMATION AND CONSENT FORM (PICF)

Full Project Title: Weight-related cognitions, their causes and effects, in women with and without anorexia nervosa

Principal Researcher: Dr Nola Rushford

Associate Researchers: Dr Harry Derham, Siew Soon, Susan Bullock, Dana Ben-Israel, Julia Nicholls, Cressida McDermott

1. Introduction

You are invited to take part in this research project which aims to help us understand similarities and differences in the experiences of women with anorexia nervosa compared with women across a range of body weights and who do not have an eating disorder. We can then gain a better understanding of concerns of women about their bodies, regardless of whether they have an eating disorder or not. We then hope to be able to offer help to those whose concerns about their bodies are adversely affecting their lives. For some of you, you are being approached by us because you are receiving treatment for an eating disorder at the Royal Melbourne Hospital or The Melbourne Clinic and your normal treatment will not be affected by your decision to participate or not. For others, you are being invited to participate through a system where the student researchers give information about the study and questionnaire packs to other women in the community to distribute to women they are acquainted with. That means that we will not be able to identify you.

This Participant Information and Consent Form tells you about the research project. It explains what is involved to help you decide if you want to take part.

Please read this information carefully. Ask questions about anything that you don’t understand or want to know more about. Before deciding whether or not to take part, you might want to talk about it with a relative, friend or your local health worker.

Participation in this research is voluntary. If you don’t wish to take part, you don’t have to. If you are a patient, participation or not will not affect treatment in any way.

If you decide you want to take part in the research project, you may be asked to sign the consent section. By signing it you are telling us that you:

- understand what you have read;
- consent to take part in the research project;
- consent to be involved in the procedures described;
- consent to the use of your personal and health information as described.

You will be given a copy of this Participant Information and Consent Form to keep.

If you are someone who does not have an eating disorder your consent will be implied by mailing us back the questionnaire pack in the stamped envelope provided.

2. What is the purpose of this research project?

- The purpose of this project is to understand the experiences of young women with or without anorexia nervosa related to thoughts and feelings that affect attitudes to body weight and shape, so that we can use the findings to better
educate people about anorexia nervosa and improve care available to sufferers. The intrusiveness of weight/shape-related thoughts and the experience of them as voices will be a particular focus. By comparing the two groups, relevant similarities and differences will be clarified. By recording the measures of the study on more than one occasion we can determine prediction of change over time;

- We would like to find out how to reduce the distress of the thoughts by studying their qualities, such as their strength and frequency, and the degree of distress they cause before designing treatment. We would also look at their relationships to the way people think about their thoughts (metacognitions), factors related to anorexia nervosa (eg. fear of gaining weight, body image disturbance, the drive to be thin, their weight), perfectionism and other characteristics we all possess, and depression, anxiety and stress measures. Some of these connections have been studied by us previously but this project will add to and unite previous work;

- A total of 284 people will participate in this project;

- We hope to recruit 60 inpatients, 50 day patients and 30 outpatients with anorexia nervosa from the Eating Disorders Units at the Royal Melbourne Hospital and the Melbourne Clinic and 144 participants from the Melbourne community who don't have an eating disorder. The community participants will let us see what the thoughts are like normally and the outpatient group will show us more clearly what the anxiety related to being admitted to the Eating Disorders Unit might do to the thoughts in the other groups. The participants will be female and between the ages of 18 and 45 years;

- In the participants with anorexia nervosa we would also like to collect the information about six weeks later and then at six-monthly intervals so that we can build up over time a picture of these thoughts and their relationships to the strength of anorexia and readiness to recover;

- This study is an extension of a study conducted in 2006, so that we can better understand what happens to the thoughts in relation to the body over a longer period of time. It will also help us work out how to better help;

- Dr Nola Rushford and Dr Harry Derham have affiliations with the Royal Melbourne Hospital and University of Melbourne and Dr Derham also has an affiliation with The Melbourne Clinic, and student researchers attend the University of Melbourne;

- The results of this research will be used by the student researchers to obtain degrees as follows: Honours theses for Julia Nicholls and Cressida McDermott, Masters degrees for Susan Bullock and Dana Ben-Israel, and a PhD for Siew Soon.

3. What does participation in this research project involve?

Participation in this project will involve:

- Deciding whether to give consent to participate and then if you would like to participate, taking the questionnaire pack that will be given to you by the student and completing it in your own time.

- If you are an inpatient, day patient or an outpatient you would sign a consent form after being satisfied with the information on the project and give back the completed questionnaires to the student researcher.

- If you are a community participant you will post back the questionnaires in a sealed pre-paid envelope.

- The questionnaires would take about 45 minutes to complete. They are related to anorexia, the thoughts about anorexia, your thoughts about the thoughts, to depression, anxiety and stress and to personality
characteristics. Some demographic information will be included. There are no right or wrong answers and if you don’t want to answer a question or you don’t want to continue, you can do so.

- **Reimbursement:** You will not be paid for your participation in this research but the costs of any postage for the project will be covered.

4. **What are the possible benefits?**

We cannot guarantee that you will receive any benefits from this project. However, the opportunity to reflect upon your thoughts might be beneficial to your understanding of them. We will also provide a summary of our findings for the groups so that you can see what conclusions we drew from the study. Possible future benefits include designing therapy to help reduce the distress caused by the thoughts in individuals with anorexia nervosa and training of health professionals about our findings.

5. **What are the possible risks?**

Possible risks, side effects and discomforts include being upset by thinking about your thoughts. If this should happen - and according to previous research this is unlikely - then we have included contacts for you to talk it over with.

If you feel uncomfortable and would like to talk to some-one other than the researchers, you can contact the Eating Disorders Foundation of Victoria (9885 0318) for advice. If you are a patient, a member of the nursing staff would be some-one to approach initially. You can cease your participation at any time if you decide you no longer wish to continue.

The risk of any participant being identified is minimal. Questionnaires will not include your name and if you sign a consent form it will be kept in a different locked filing cabinet to the questionnaires. The record of your responses to the questionnaires will have an Identification number (ID) only. The ID will belong to you for the life of the project. The ID will allow us to match subsequent sets of questionnaires (if any) to the first set. It will also enable you to withdraw your information at any time before we combine it with the information from the other participants. The ID also enables patients to give separate consent for a summary of results to be given to a clinician attached to the Eating Disorders Unit they attend, if they would like to.

There is very little risk of identification of community participants by any-one. Once they are returned it is not possible to discover who sent them back because the answers to the questionnaires are anonymous. The researchers do not get to see any of the returned questionnaires they themselves distributed, so that they can't even guess who completed them. For the patients, the risk may be slightly higher but because the patient group is large and from two Eating Disorders services, if some-one did see a person’s questionnaire the probability of identifying her is remote. It is possible, however, that a questionnaire set for a patient might be subject to the legal requirement for subpoena of information under some circumstances.

Sometimes a person might become upset by thinking about associations to the items in a questionnaire. If you become upset or distressed as a result of your participation in the research, we are able to arrange for counselling or other appropriate support if you contact one of us on 9326 4774 or 9342 4041. Any counselling or support will be provided by staff who are not members of the research team. You could also contact the Eating Disorders Foundation of Victoria (9885 0318) or LifeLine (13 1114), for example.
6. **Do I have to take part in this research project?**

Participation in any research project is voluntary. If you do not wish to take part, you do not have to. If you decide to take part and later change your mind, you are free to withdraw from the project at a later stage. If you do consent to participate, you may only withdraw prior to generation of the group results after your information has been combined with that of all the other participants.

If you decide to withdraw, please notify a member of the research team if you are a patient and quote your ID. This will allow that person or the research supervisor to withdraw your information if group results haven’t been generated. If you are a community participant you can phone on 0326 4774 or 9342 4041 and quote your ID. You will not need to give your name.

If you decide to leave the project at a later stage, the researchers would like to keep the personal and/or health information about you that has been collected. This is to help them make sure that the results of the research can be measured properly. If you do not want them to do this, you must tell them before you withdraw from the research project.

If you became too ill to participate in the project at any stage, this would be discussed with you and participation at that stage would be withdrawn.

Your decision whether to take part or not, or to take part and then withdraw, will not affect your relationship with the researchers or if you are a patient your access to treatment. Participation will not affect your treatment in any way.

7. **How will I be informed of the final results of this research project?**

A summary of group results will be available through the Eating Disorders Unit you attend, or the websites for the Centre of Excellence for Eating Disorders and the Eating Disorders Foundation of Victoria. The summary should be available in mid-2010.

8. **What will happen to information about me?**

Any information obtained in connection with this research project that can identify you will remain confidential and will only be used for the purpose of this research project. Because we intend to use the information to develop therapy that can help individuals with anorexia nervosa, so we would like to compare the information we collect in this phase of the project with information collected when we test the new therapy.

The information provided by you will be stored in a locked filing cabinet in the Chief Investigator’s room. A copy of your consent form and ID will be kept in another locked cabinet. The student researchers will also have access to the questionnaires so they can enter the information from them into a computer file for group analysis. Your information will only be disclosed with your permission, except as required by law.

Information from the community sample is anonymous while information from the participants with anorexia nervosa is potentially re-identifiable by linking it to your ID. In any publication and/or presentation, information will be provided in such a way that you cannot be identified, except with your permission.

Once the project has been written up by the researchers, data will be kept for another seven years then disposed of by a secure disposal system.

9. **Can I access research information kept about me?**

In accordance with relevant Australian and/or Victorian privacy and other relevant laws, you have the right to access the information collected and stored by the
researchers about you. Please contact one of the researchers named at the end of this document if you would like to access your information. Please quote the ID given in Section 11.

In addition, in accordance with regulatory guidelines, the information collected in this research project will be kept for at least seven years.

10. **Is this research project approved?**

The ethical aspects of this research project have been approved by the Human Research Ethics Committee of Melbourne Health.

This project will be carried out according to the *National Statement on Ethical Conduct in Human Research (2007)* produced by the National Health and Medical Research Council of Australia. This statement has been developed to protect the interests of people who agree to participate in human research studies.
11. Consent

If you are a community participant, you will give your implicit consent by posting back your questionnaires, so are not required to sign this consent form. Please keep this Participant Information Sheet so you can refer to it at a later time. It also has your ID on it in case you wish to withdraw your information from the study.

I have read, or have had this document read to me in a language that I understand, and I understand the purposes, procedures and risks of this research project as described within it.

I have had an opportunity to ask questions and I am satisfied with the answers I have received.

I freely agree to participate in this research project, as described.

I understand that I will be given a signed copy of this document to keep.

Participant’s name (printed) ……………………………………………………
Signature
Date

Declaration by researcher*: I have given a verbal explanation of the research project, its procedures and risks and I believe that the participant has understood that explanation.

Researcher’s name (printed) ……………………………………………………
Signature
Date

Note: All parties signing the consent section must date their own signature.

This is your ID to quote if you wish to withdraw your information. Please do not give your name.

If you are a patient of either the Eating Disorders Unit at the Royal Melbourne Hospital or The Melbourne Clinic and you would like a summary of your information to go to a clinician at the Eating Disorders Unit, please write his or her name here.
12. Who can I contact?
The person you may need to contact will depend on the nature of your query. Therefore, please note the following:

**For further information or appointments:**
If you want any further information concerning this project or if you have any problems which may be related to your involvement in the project (for example, feelings of distress), you can contact the principal researcher,

Name: Nola Rushford  
Position: Chief Investigator  
Telephone: 9326 4774 or 9342 4041  
or any of the following people:  
Dr Harry Derham on 9819 3224, or  
Siew Soon, Susan Bullock, Dana Ben-Israel, Julia Nicholls or Cressida McDermott on 9326 4774 or 9342 404.  
You may also send an email to nolar@unimelb.edu.au.

**For complaints:**
If you have any complaints about any aspect of the project, the way it is being conducted or any questions about being a research participant in general, then you may contact:

Name: Ms Michelle Clemson  
Position: Manager, Mental Health Research Ethics Committee, Melbourne Health  
Telephone: 03 9342 7215
IS YOUR WEIGHT TOO LOW FOR GOOD HEALTH?*

In our society, the fashion, fitness and diet industries promote a ‘thin ideal’ weight or shape for women that is well below what is required to maintain health. As a consequence, many women feel uncomfortable about their own body weight or shape because they think they can only be attractive and worthwhile if they are like the ‘thin ideal’. So they diet. Sometimes, if they do lose weight and feel a bit better about themselves, it is very easy to fall into the trap of putting even more energy into weight loss strategies. A person who remains for some time at a low weight is at risk of long-term adverse consequences to all aspects of her life, such as health, education, work, and social and personal relationships.

Here are some pointers to help you decide whether you should be concerned about your weight. Just place a tick or cross on the box beside each question.

Is your Body Mass Index (BMI) below 18.5 kg/m²?  
The normal range for BMI is 20 – 25 kg/m². However, if you have had a recent, rapid weight loss even if your BMI is above 18.5 kg/m², you may need assistance to prevent further weight loss.

You can calculate your BMI from your weight and height.  
BMI = weight in kilograms / (height in metres x height in metres).  
For example, if you were 60 kg and were 1.62 m (ie 162cm) tall,  
your BMI would be 60 / (1.62 x 1.62) = 60 / 2.6244 =  22.9 kg/m².

Do you refuse to consider putting on more weight even though people have seemed to be worried about you?  
It is sometimes difficult to trust others around us when we have lost weight; we think that they are not being truthful or that we need to lose more weight anyway.

Have you ceased menstruating or are having irregular periods?  
When thinking about this, remember that if you are taking the oral contraceptive pill it is difficult to tell whether you really are menstruating normally and regularly.

Do you often feel that you are fat even when other people have expressed concern that you appear to be losing weight?  
One of the reasons that a person finds it hard to put on weight when she is underweight is that she feels that she is larger than she really is. This is known as disturbed or distorted body image.

How important are your weight/shape for how you feel about yourself?  
Think of the numbers between 0 and 10. If 0 is no importance at all and 10 is complete importance, where would you rate yourself? If you rate above 7 then it is a bit high.
Do you find it hard to believe that low weight has serious consequences for a person’s health?
Low weight adversely affects all aspects of our physical health, including causing the development of osteoporosis. It can also affect our concentration, memory and how easily we can think. The longer a person stays at low weight the more severe the effects.

Do you spent a lot of time in activities designed to lose weight?
Some activities are running, walking or bicycling long distances; running up and down stairs carrying something heavy; doing many sit-ups or star jumps. The person may even be heartily sick of doing it but is too concerned about gaining weight to stop.

Do you spend a lot of time each day working out what you can or can’t eat or how to avoid some meals altogether?
People who have lost weight can become pre-occupied with what not to eat. They might even decide that once they’ve resisted eating a particular food they cannot eat it again.

Do you vomit after eating or take laxatives or some other substance because you believe it will help you lose weight?
These are activities that are dangerous to a person’s health in their own right so add to the health risks associated with low weight.

If your BMI indicates that you are underweight and you have also ticked any of the other boxes it is possible that you require assistance to regain a normal weight. If you are concerned please contact the

Eating Disorders Foundation (Inc)
Address 1513 High St Glen Iris 3146
General Enquiries (03) 9885 6563
Helpline 1300 550 236
help@eatingdisorders.org.au
www.eatingdisorders.org.au

The Eating Disorders Foundation of Victoria offers a range of services to provide advice and support for individuals who may suffer from an eating disorder and also to their family members, and friends who are close to them. Your call will remain confidential.

*Prepared by Dr Nola Rushford, Chief Researcher for Project MHREC 2008.605: Weight-related cognitions, their causes and effects in women with or without anorexia nervosa.
Telephone: (03) 9326 4774
Would you please complete the following questions? If you don't know the answer to an item please just leave it blank.

Age: __________  Partner status: Single / Partner / Married / Separated or divorced

Are you studying?  NO / DEFERRED / YES (please describe) ________________

Are you employed?  NO / DEFERRED / YES (please describe) ________________

Education level attained:  Year 10 or less / Year 11 / Higher Schools Certificate (VCE) / Undergraduate tertiary / Postgraduate tertiary / Other (please specify): ________________

Have you been trained for a particular type of employment?  NO / YES (please describe): ________________

Height: ______ cm  Weight now: _____ kg

or ______ ft, inches  or _____ stone, lb

Since puberty, what has been your lowest weight: ____________ kg (lb)

In the last three months, * * * * lowest weight: ____________ kg (lb)

Since puberty, what has been your highest weight: ____________ kg (lb)

What weight would you ideally like to be? ____________ kg (lb)

What weight would you be afraid to go above? ____________ kg (lb)

Have you ever been teased or bullied?  NO / YES About your weight? NO / YES

Do you try to restrict food intake or to diet?  NO / YES

If so, how many calories kilojoules would you try to restrict to? _______ cal or Kj / Don't Know

Or do you just cut out foods?  NO / YES Such as? ________________

And, how old were you when you started restricting? _______ years

In the last year, how long would you have spent trying to diet or restrict? All year / > 6 months / > 3 months / month or more / Not at all

Do you exercise?  NO / YES

If you exercise, what would you typically do? ________________

Average time/week you would typically exercise _______ hours

If you exercise, is it for any of the following reasons (please circle all that apply)?  Sport / recreation / weight loss / fitness / sense of obligation or compulsion / relieve guilt / allow you to eat / other - 

_________________________

_________________________

Do you have episodes of bingeing?  NO / YES Or vomiting?  NO / YES

If so, how often? Daily/ weekly / monthly / less

If so, how often? Daily/ weekly / monthly / less
This is a scale which measures a variety of attitudes, feelings and behaviours. Some of the items relate to food and eating. Others ask you about your feelings about yourself. **THERE ARE NO RIGHT OR WRONG ANSWERS SO TRY VERY HARD TO BE COMPLETELY HONEST IN YOUR ANSWERS. RESULTS ARE COMPLETELY CONFIDENTIAL.** Read each question and fill in the circle under the column which applies best to you. Please answer each question very carefully. Thank you.

**Appendix E: Eating Disorder Inventory, 3rd Edition (EDI-3; Garner, 2004)**

<table>
<thead>
<tr>
<th></th>
<th>U</th>
<th>S</th>
<th>O</th>
<th>M</th>
<th>E</th>
<th>R</th>
</tr>
</thead>
<tbody>
<tr>
<td>S</td>
<td>A</td>
<td>S</td>
<td>E</td>
<td>R</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L</td>
<td>U</td>
<td>O</td>
<td>T</td>
<td>A</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>W</td>
<td>A</td>
<td>F</td>
<td>I</td>
<td>R</td>
<td>E</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>L</td>
<td>T</td>
<td>M</td>
<td>E</td>
<td>V</td>
<td></td>
</tr>
<tr>
<td>Y</td>
<td>L</td>
<td>E</td>
<td>E</td>
<td>L</td>
<td>E</td>
<td></td>
</tr>
<tr>
<td>S</td>
<td>Y</td>
<td>N</td>
<td>S</td>
<td>Y</td>
<td>R</td>
<td></td>
</tr>
</tbody>
</table>

1. I eat sweets and carbohydrates without feeling nervous…….. | o | o | o | o | o | o
2. I think that my stomach is too big………………………….. | o | o | o | o | o | o
3. I wish that I could return to the security of childhood…….. | o | o | o | o | o | o
4. I eat when I am upset………………………………………… | o | o | o | o | o | o
5. I stuff myself with food………………………………………… | o | o | o | o | o | o
6. I wish that I could be younger………………………………….. | o | o | o | o | o | o
7. I think about dieting……………………………………………. | o | o | o | o | o | o
8. I get frightened when my feelings are too strong…………….. | o | o | o | o | o | o
9. I think that my thighs are too large……………………………. | o | o | o | o | o | o
10. I feel ineffective as a person……………………………………. | o | o | o | o | o | o
11. I feel extremely guilty after overeating………………………… | o | o | o | o | o | o
12. I think that my stomach is just the right size…………………… | o | o | o | o | o | o
13. Only outstanding performance is good enough in my family.. | o | o | o | o | o | o
14. The happiest time in life is when you are a child…………….. | o | o | o | o | o | o
15. I am open about my feelings……………………………………. | o | o | o | o | o | o
16. I am terrified of gaining weight…………………………………. | o | o | o | o | o | o
17. I trust others…………………………………………………….. | o | o | o | o | o | o
18. I feel alone in the world…………………………………………. | o | o | o | o | o | o
19. I feel satisfied with the shape of my body………………………. | o | o | o | o | o | o
20. I feel generally in control of things in my life…………………. | o | o | o | o | o | o
21. I get confused about what emotion I am feeling…………………. | o | o | o | o | o | o
22. I would rather be an adult than a child…………………………. | o | o | o | o | o | o
23. I can communicate with others easily……………………………. | o | o | o | o | o | o
24. I wish I were someone else………………………………………. | o | o | o | o | o | o
25. I exaggerate or magnify the importance of weight……………. | o | o | o | o | o | o
26. I can clearly identify what emotion I am feeling……………o o o o o o
27. I feel inadequate………………………………………………o o o o o o
28. I have gone on eating binges where I have felt that I could not stop………………………………………………………o o o o o o
29. As a child, I tried very hard to avoid disappointing my parents and teachers………………………………………………………o o o o o o
30. I have close relationships…………………………………………………………o o o o o o
31. I like the shape of my buttocks…………………………………………………o o o o o o
32. I am preoccupied by the desire to be thinner…………………………………o o o o o o
33. I don’t know what’s going on inside me………………………………………o o o o o o
34. I have trouble expressing my emotions to others……………………………o o o o o o
35. The demands of adulthood are too great………………………………………o o o o o o
36. I hate being less than best at things……………………………………………o o o o o o
37. I feel secure about myself………………………………………………………..o o o o o o
38. I think about bingeing (overeating)……………………………………………o o o o o o
39. I feel happy that I am not a child any more………………………………………o o o o o o
40. I get confused as to whether or not I am hungry………………………………o o o o o o
41. I have a low opinion of myself……………………………………………………o o o o o o
42. I feel that I can achieve my standards……………………………………………o o o o o o
43. My parents have expected excellence of me……………………………………o o o o o o
44. I worry that my feelings will get out of control…………………………………o o o o o o
45. I think that my hips are too big……………………………………………………o o o o o o
46. I eat moderately in front of others and stuff myself when they’re gone…………………………………………………………o o o o o o
47. I feel bloated after eating a normal meal………………………………………o o o o o o
48. I feel that people are happiest when they are children……………………o o o o o o
49. If I gain a pound, I worry that I will keep gaining……………………………o o o o o o
50. I feel that I am a worthwhile person……………………………………………o o o o o o
51. When I am upset, I don’t know if I am sad, frightened, or angry…………………………………………………………o o o o o o
52. I feel that I must do things perfectly or not do them at all……………………o o o o o o
53. I have the thought of trying to vomit in order to lose weight……………………o o o o o o
54. I need to keep people at a certain distance (feel uncomfortable if someone tries to get too close)…………………o o o o o o
55. I think that my thighs are just the right size
56. I feel empty inside (emotionally)
57. I can talk about personal thoughts or feelings
58. The best years of your life are when you become an adult
59. I think my buttocks are too large
60. I have feelings that I can’t quite identify
61. I eat or drink in secrecy
62. I think that my hips are just the right size
63. I have extremely high goals
64. When I am upset, I worry that I will start eating
65. People I really like end up disappointing me
66. I am ashamed of my human weakness
67. Other people would say that I am emotionally unstable
68. I would like to be in total control of my bodily urges
69. I feel relaxed in most group situations
70. I say things impulsively that I regret having said
71. I go out of my way to experience pleasure
72. I have to be careful of my tendency to abuse drugs
73. I am outgoing with most people
74. I feel trapped in relationships
75. Self-denial makes me feel stronger spiritually
76. People understand my real problems
77. I can’t get strange thoughts out of my head
78. Eating for pleasure is a sign of moral weakness
79. I am prone to outbursts of anger or rage
80. I feel that people give me the credit I deserve
81. I have to be careful of my tendency to abuse alcohol
82. I believe that relaxing is simply a waste of time
83. Others would say that I get irritated easily
84. I feel like I am losing out everywhere
85. I experience marked mood shifts
86. I am embarrassed by my bodily urges
87. I would rather spend time by myself than with others.
88. Suffering makes you a better person
89. I know that people love me
90. I feel like I must hurt myself or others
91. I feel that I really know who I am
**APPENDIX F: ROSENBERG SELF-ESTEEM SCALE**
(RSES; Rosenberg, 1965)

Please read each question and fill in the circle under the column which applies best to you for each statement.

<table>
<thead>
<tr>
<th>Statement</th>
<th>Strongly Agree</th>
<th>Agree</th>
<th>Disagree</th>
<th>Strongly Disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. On the whole I am satisfied with myself</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>2. At times I feel no good at all</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>3. I feel that I have a number of good qualities</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>4. I am able to things as well as most other people</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>5. I feel I do not have much to be proud of</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>6. I certainly feel useless at times</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>7. I feel I am a person of worth, at least on an equal level with others</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>8. I wish I could have more respect for myself</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>9. All in all I am inclined to think I'm a failure</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>10. I take a positive attitude towards myself</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
</tbody>
</table>
APPENDIX G: METACOGNITIONS QUESTIONNAIRE, BRIEF VERSION
(MCQ-30; Wells & Cartwright-Hatton)

The questionnaire is to help us to understand more about how you think about your own thoughts. There are no right or wrong responses. Please read each statement and tick the box that is most accurate for you.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Do not agree</th>
<th>Agree slightly</th>
<th>Agree moderately</th>
<th>Agree very much</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>I do not trust my memory</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>I have a poor memory</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>I have little confidence in my memory for actions</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>I have little confidence in my memory for places</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>I have little confidence in my memory for words and names</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>My memory can mislead me at times</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7</td>
<td>Worrying helps me get things sorted out in my mind</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8</td>
<td>Worrying helps me cope</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9</td>
<td>I need to worry in order to work well</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10</td>
<td>Worrying helps me solve problems</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11</td>
<td>I need to worry in order to remain organized</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>12</td>
<td>Worrying helps me to avoid problems in the future</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>13</td>
<td>I am constantly aware of my thinking</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>14</td>
<td>I pay close attention to the way my mind works</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>15</td>
<td>I think a lot about my thoughts</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>16</td>
<td>I constantly examine my thoughts</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>17</td>
<td>I monitor my thoughts</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>18</td>
<td>I am aware of the way my mind works when I am thinking through a problem</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>19</td>
<td>My worrying thoughts persist, no matter how I try to stop them</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>20</td>
<td>When I start worrying I cannot stop</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>21</td>
<td>I could make myself sick with worrying</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>22</td>
<td>I cannot ignore my worrying thoughts</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>23</td>
<td>My worrying could make me go mad</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>24</td>
<td>My worrying is dangerous for me</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Question</td>
<td>Score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>-------------------------------------------------------------------------</td>
<td>---------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>If I cannot control my thoughts I would not be able to function</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>Not being able to control my thoughts is a sign of weakness</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>27</td>
<td>I should be in control of my thoughts all of the time</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>It is bad to think certain thoughts</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>29</td>
<td>If I did not control a worrying thought and then it happened it would be my fault</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>I will be punished for not controlling certain thoughts</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### DASS

**Date:**

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

*The rating scale is as follows:*

<table>
<thead>
<tr>
<th>Number</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Did not apply to me at all</td>
</tr>
<tr>
<td>1</td>
<td>Applied to me to some degree, or some of the time</td>
</tr>
<tr>
<td>2</td>
<td>Applied to me to a considerable degree, or a good part of time</td>
</tr>
<tr>
<td>3</td>
<td>Applied to me very much, or most of the time</td>
</tr>
</tbody>
</table>

1. I found myself getting upset by quite trivial things 0 1 2 3
2. I was aware of dryness of my mouth 0 1 2 3
3. I couldn't seem to experience any positive feeling at all 0 1 2 3
4. I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion) 0 1 2 3
5. I just couldn't seem to get going 0 1 2 3
6. I tended to over-react to situations 0 1 2 3
7. I had a feeling of shakiness (eg, legs going to give way) 0 1 2 3
8. I found it difficult to relax 0 1 2 3
9. I found myself in situations that made me so anxious I was most relieved when they ended 0 1 2 3
10. I felt that I had nothing to look forward to 0 1 2 3
11. I found myself getting upset rather easily 0 1 2 3
12. I felt that I was using a lot of nervous energy 0 1 2 3
13. I felt sad and depressed 0 1 2 3
14. I found myself getting impatient when I was delayed in any way (eg, lifts, traffic lights, being kept waiting) 0 1 2 3
15. I had a feeling of faintness 0 1 2 3
16. I felt that I had lost interest in just about everything 0 1 2 3
17. I felt I wasn't worth much as a person 0 1 2 3
18. I felt that I was rather touchy 0 1 2 3
19. I perspired noticeably (eg, hands sweaty) in the absence of high temperatures or physical exertion 0 1 2 3
20. I felt scared without any good reason 0 1 2 3
21. I felt that life wasn't worthwhile 0 1 2 3
Author/s: 
SOON, SIEW PENG

Title: 
Anorexia nervosa: controversy regarding diagnostic criteria and evidence for control-related cognitive and metacognitive mechanisms maintaining the disorder

Date: 
2012

Citation: 

Persistent Link: 
http://hdl.handle.net/11343/38124

File Description: 
Anorexia nervosa: controversy regarding diagnostic criteria and evidence for control-related cognitive and metacognitive mechanisms maintaining the disorder