School of Psychology and Speech Pathology

A Randomised Controlled Trial of Guided Self-Help Cognitive Behaviour Therapy for Clinical Perfectionism

Kimberley Jo Hoiles

This thesis is presented for Degree of

Doctor of Philosophy

of

Curtin University

October 2016
# Table of Contents

Table of Contents ......................................................................................................................... 2  
Declaration................................................................................................................................. 6  
Acknowledgements .................................................................................................................... 7  
List of Tables .............................................................................................................................. 8  
List of Figures ............................................................................................................................. 10  
List of Acronyms ......................................................................................................................... 10  
Abstract ...................................................................................................................................... 13  

Chapter 1: Introduction to Perfectionism .................................................................................. 16  
1.1. Overview ............................................................................................................................. 16  
1.2. What is Perfectionism? ........................................................................................................ 16  
1.2.1. Construct of Clinical Perfectionism ................................................................................. 19  
1.3. Measurement of Perfectionism ............................................................................................ 22  
1.3.1. Dysfunctional Attitudes Scale – Self Criticism ............................................................... 23  
1.3.2. Burns Perfectionism Scale ............................................................................................. 23  
1.3.3. Eating Disorders Inventory Perfectionism subscale ....................................................... 24  
1.3.4. Multidimensional Measures of Perfectionism ............................................................... 25  
1.3.5. The Perfectionism Cognitions Inventory ....................................................................... 27  
1.3.6. Positive and Negative Perfectionism Scale ................................................................... 28  
1.3.7. The Clinical Perfectionism Questionnaire ................................................................... 28  
1.4. Aetiology of Perfectionism: Nature versus. Nurture ........................................................... 31  
1.4.1. Genetic Predisposition of Perfectionism ...................................................................... 32  
1.4.2. Perfectionism as a Learned Trait .................................................................................. 33  

Chapter 2: Perfectionism and Psychopathology ....................................................................... 38  
2.1. Perfectionism across Disorders .......................................................................................... 38  
2.1.1. Depressive Disorders .................................................................................................... 38  
2.1.2. Anxiety Disorders .......................................................................................................... 40  
2.1.2.1. Social Anxiety Disorder ......................................................................................... 40  
2.1.2.2. Panic with and without Agoraphobia ....................................................................... 41  
2.1.2.3. Generalised Anxiety Disorder ............................................................................... 42  
2.1.3. Post-Traumatic Stress Disorder .................................................................................... 43  
2.1.4. Obsessive Compulsive and Related Disorders ............................................................... 44  
2.1.4.1. Obsessive Compulsive Disorder ............................................................................. 44  
2.1.4.2. Body Dysmorphic Disorder .................................................................................... 45  
2.1.4.3. Hoarding Disorder .................................................................................................... 46  
2.1.4.4. Trichotillomania ....................................................................................................... 47  
2.1.5. Eating Disorders ............................................................................................................ 48  
2.1.6. Perfectionism and Treatment Outcome Across Disorders ......................................... 50
2.2. Perfectionism and Quality of Life .......................................................... 52
2.3. Perfectionism as an Explanation for Co-Morbidity .............................. 54
2.4. Perfectionism as a Transdiagnostic Process ........................................ 55

Chapter 3: Treatment of Perfectionism ...................................................... 58
  3.1 Cognitive Behavioural Treatment for Clinical Perfectionism ................. 58
  3.2. Alternatives to Face-to-Face Treatment .............................................. 62
    3.2.1 Self-help perfectionism treatment in non-clinical samples .............. 65
    3.2.2. Self-help perfectionism treatment in clinical samples ................. 67

Chapter 4: Study 1 - Reliability and Validity of the Clinical Perfectionism
  Questionnaire in a Mixed Clinical Sample .............................................. 69
  4.1. Overview ............................................................................................ 69
  4.2. Rationale and Aims ............................................................................ 70
  4.3. Hypotheses ......................................................................................... 71
  4.4. Method ................................................................................................ 71
    4.4.1. Participants .................................................................................. 71
    4.4.2. Measures ..................................................................................... 73
      4.4.2.1. Mini International Neuropsychiatric Interview-Screen (MINI-
      Screen) ....................................................................................... 73
      4.4.2.2. Mini International Neuropsychiatric Interview (MINI) .......... 73
      4.4.2.3. Anxiety Disorders Interview Schedule-IV (ADIS-IV) .......... 73
      4.4.2.4. Clinical Perfectionism Questionnaire (CPQ) ....................... 74
      4.4.2.5. Frost Multidimensional Perfectionism Scale (FMPS) ............. 74
      4.4.2.6. The Dysfunctional Attitudes Scale (DAS) ............................. 75
      4.4.2.7. The Dichotomous Thinking in Eating Disorders Scale (DTEDS) 75
    4.4.3. Procedure ..................................................................................... 75
  4.5. Results ................................................................................................ 76
    4.5.1. Data Screening ............................................................................ 76
    4.5.1.1. Missing data .......................................................................... 76
    4.5.1.2. Outliers .................................................................................. 77
    4.5.2. Assumption Testing for Correlational Analysis ............................ 77
    4.5.3. Hypothesis Testing ...................................................................... 77
      4.5.3.1. Convergent validity .............................................................. 78
      4.5.3.2. Concurrent validity .............................................................. 78
  4.6. Discussion ........................................................................................... 79

Chapter 5: Study 2. A Randomised Controlled Trial of Cognitive Behavioural
  Guided Self-Help Therapy for Clinical Perfectionism ............................. 82
  5.1. Overview ............................................................................................ 82
  5.2. Rationale and Aims ............................................................................ 83
  5.3. Hypotheses ......................................................................................... 87
    5.3.1. Effects of the Treatment on Perfectionism and Related Constructs 87
    5.3.2. Effects of the Treatment on Psychopathology .............................. 88
    5.3.4. Effect of the Treatment on DSM-IV Diagnoses .......................... 89
5.3.5. Effects of the Treatment on Reliable and Clinically Significant Change in Disorder-Specific Symptoms ......................................................... 89
5.4. Method ........................................................................................................ 89
  5.4.1. Participants ............................................................................................ 89
    5.4.1.1. Recruitment and sampling method .............................................. 91
    5.4.1.2. Inclusion criteria ....................................................................... 92
    5.4.1.3. Exclusion criteria .................................................................. 92
  5.4.2. Measures ............................................................................................. 93
    5.4.2.1. Mini International Neuropsychiatric Interview-Screen (MINI-
              Screen) ...................................................................................... 93
    5.4.2.2. Mini International Neuropsychiatric Interview (MINI) ............. 93
    5.4.2.3. Anxiety Disorders Interview Schedule-IV (ADIS-IV) .............. 93
    5.4.2.4. Clinical Perfectionism Questionnaire (CPQ) ......................... 94
    5.4.2.5. Frost Multidimensional Perfectionism Scale (FMPS) ............. 94
    5.4.2.6. The Dysfunctional Attitudes Scale (DAS) ............................. 94
    5.4.2.7. The Dichotomous Thinking in Eating Disorders Scale (DTEDS) .. 95
    5.4.2.8. Depression Anxiety Stress Scales - 21 (DASS-21) ............... 95
    5.4.2.9. The Quality of Life, Enjoyment and Satisfaction Questionnaire-18
              (QLES-Q) .................................................................................. 95
    5.4.2.10. Penn State Worry Questionnaire (PSWQ) ................................ 96
    5.4.2.11. Fear of Negative Evaluation Scale - Brief Version (FNE-B) .... 96
    5.4.2.12. Beck Depression Inventory - II (BDI-II) ............................... 97
    5.4.2.13. Eating Disorder Examination Questionnaire (EDEQ) .......... 97
    5.4.2.14. Anxiety Sensitivity Index (ASI-3) .......................................... 98
    5.4.2.15. Compliance to treatment ...................................................... 99
    5.4.2.16. Credibility/Expectancy Questionnaire .................................... 99
  5.4.3. Treatment Protocol ........................................................................... 100
    5.4.3.1. Therapists .............................................................................. 101
  5.4.4. Procedure ........................................................................................... 103
  5.4.5. Design and Statistical Analysis .......................................................... 104
5.5. Results ...................................................................................................... 108
  5.5.1. Data Screening ................................................................................... 108
    5.5.1.1. Missing data ........................................................................... 108
  5.5.2. Demographic and Clinical Characteristics of the Elevated Perfectionism
         Sample (N = 40) ............................................................................. 108
  5.5.3. Treatment Compliance ..................................................................... 111
  5.5.4. Descriptive Statistics for the Elevated Perfectionism Sample (N = 40) ...... 113
  5.5.5. Hypothesis Testing ............................................................................ 115
    5.5.5.1. Perfectionism and Related Construct Outcomes .................. 115
    5.5.5.2. Perfectionism and Related Construct Outcomes at Follow-up. .... 119
    5.5.5.3. Clinically Significant Change on Perfectionism Outcomes ...... 121
    5.5.5.4. Psychopathology Outcomes .................................................... 124
Chapter 6: General Discussion ................................................................. 145
  6.1. Key Findings and Future Directions .............................................. 145
    6.1.1. Limitations of the Thesis....................................................... 148
  6.2. Treatment Accessibility: Evidence-Based Treatment to Evidence-Based Practice .............................................................. 149
    6.2.1. Alternatives to Face-to-Face Interventions .............................. 150
    6.2.2. Transdiagnostic versus Disorder Specific Treatments ............. 151
  6.3. Conclusion .................................................................................... 154

References ............................................................................................ 156

Appendices ............................................................................................ 205
  Appendix A: Participants Starter Package ........................................... 205
  Appendix B: Advertisement .................................................................. 205
  Appendix C: Graphs of the interactions for each Perfectionism outcome variable .. 210
  Appendix D: Graphs of the interactions for each Perfectionism outcome variable at follow-up .................................................. 212
  Appendix E: Graphs of the interactions for each Psychopathology outcome variable .......................................................... 213
  Appendix F: Graphs of the interactions for each Psychopathology outcome variable at follow-up ............................................. 214
Declaration

To the best of my knowledge and belief this thesis contains no material previously published by any other person except where due acknowledgment has been made.

This thesis contains no material that has been accepted for the award of any other degree or diploma in any university.

The research presented and reported in this thesis was conducted in accordance with the National Health and Medical Research Council National Statement on Ethical Conduct in Human Research (2007) – updated March 2014. The proposed research study received human research ethics approval from the Curtin University Human Research Ethics Committee (EC00262), Approval Number # HR 120/2010

Signature:

Date: 11.10.2016
Acknowledgements

I would firstly like to thank my supervisors Dr Sarah Egan, Dr Clare Rees and Dr Robert Kane for their endless encouragement and support throughout this project. Your guidance in the endless meetings, drafts reviews, frantic last minute emails/texts, support at international conferences, has enabled me to build on my research skills and contribute to my education for which I will be forever grateful. I would also like to thank Dr Rebecca Anderson for providing me invaluable feedback on my final draft.

I would acknowledge the participants that volunteered their time to this research. Without their participation and the privilege of being given access to their invaluable data, this study would not have been able to happen. Your dedication has contributed to the improvement of treatment outcomes for the wider community.

I would like to thank my family, friends and work colleagues. You have all been understanding, encouraging, and at times necessarily inpatient with me throughout these last few years. Without a strong family and social network I am confident I would not have got through the last few stages of this process.

Lastly, I would like to thank my husband, Dane Bowler. You have been there for me from the very beginning to the very end of this project. You were able to support me in the very worst of times and were able to laugh, cry and vent with me.

Thank you
List of Tables

Table 1  Demographic and Diagnostic Characteristics of the Mixed Clinical Sample (N = 32) .......................................................... 72
Table 2  Summary of Intercorrelations, Means, Standard Deviations, and Reliabilities for Study Measures (N = 32), .......................... 79
Table 3  Primary Diagnoses in the Sample (N = 40) .................................. 89
Table 4  Weekly Modules, Homework and Corresponding Overcoming Perfectionism Chapters of the Eight Week Treatment Plan .......... 102
Table 5  Formula used to Calculate the Reliable Change Index and Clinically Significant Change ..................................................... 106
Table 6  Clinically Significant and Reliable Change Criteria for Treatment Outcomes According to Jacobson and Truax (1991)........ 108
Table 7  Statistical Group Comparison of Baseline Outcome Variable Means .......................................................................................... 110
Table 8  Baseline Demographic and Clinical Data Comparing Treatment and Control Group ............................................................ 111
Table 9  Self-reported Adherence (%) to the Intervention as a Summary of Modules Read and Exercises Completed ............................... 112
Table 10 Means, Adjusted Means and Standard Deviations for Each Outcome Variable by Time and Group .................................................. 114
Table 11 Results of the Omnibus GLMMs for Each Outcome .................. 116
Table 12 LSD Tests of the Simple Main Effects of Time for the Group x Time Interactions for Perfectionism Variables .......................... 118
Table 13 Means, Adjusted Means and Standard Deviations for the Perfectionism Outcomes in the Treatment Group ....................... 119
Table 14 LSD Tests of the Main Effects of Time for Perfectionism Variables 121
Table 15 Data used to Calculate Reliable Change and Clinical Significance on Perfectionism Outcome Measures ............................... 122
Table 16 The Number (and Percentage) of Participants Falling in Each of the Four Clinical Categories at Post-Treatment on Perfectionism Outcomes .................................................................................. 122
Table 17  The Number (and Percentage) of Participants in the Treatment Group Meeting Clinically Significant Change at Follow-up on Perfectionism Outcomes (n = 17) .............................................................................................................. 124
Table 18  Results of the Omnibus GLMMs for Each Outcome ........................................ 125
Table 19  LSD Tests of the Simple Main Effects of Time for the Group x Time Interactions for Psychopathology Variables .............................................................................. 127
Table 20  Means, Adjusted Means and Standard Deviations for the Psychopathology Outcomes in the Treatment Group ................................................................. 128
Table 21  LSD Tests of the Main Effect of Time for Psychopathology Variables ................................. 129
Table 22  Data used to Calculate Reliable Change and Clinical Significance on Psychopathology Outcome Measures ...................................................................................... 130
Table 23  The Number (and Percentage) of Participants Meeting Clinically Significant Change at Post-Treatment and Follow-Up on Psychopathology Outcomes ................................................................. 130
Table 24  The Number (Percentage) of Participants in the Treatment Group Meeting Clinically Significant Change at Follow-Up on Psychopathology Outcomes (n = 17) .............................................................................................................. 131
Table 25  Primary and Comorbid Diagnosis by Time and Condition for the Mixed Clinical Sample (N = 32) ...................................................................................................................... 133
Table 26  Means and Standard Deviations for Each Disorder Specific Outcome Variable by Time and Group at Baseline ...................................................................................... 135
Table 27  Data used to Calculate Reliable Change and Clinical Significance on Disorder Specific Outcome Measures ...................................................................................................................... 136
Table 28  The Number (Percentage) of Participants Meeting Clinically Significant Change at Post-Treatment on Disorder Specific Outcomes for their Primary Diagnosis .............................................................................................................. 137
## List of Figures

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Figure 1</td>
<td>The maintenance of perfectionism</td>
<td>21</td>
</tr>
<tr>
<td>Figure 2</td>
<td>Preliminary model of the development of perfectionism</td>
<td>37</td>
</tr>
<tr>
<td>Figure 3</td>
<td>Design of the Randomised Controlled Trial</td>
<td>90</td>
</tr>
<tr>
<td>Figure 4</td>
<td>Weekly exercise and readings compliance rates over the 8 week intervention</td>
<td>113</td>
</tr>
<tr>
<td>Acronym</td>
<td>Description</td>
<td></td>
</tr>
<tr>
<td>---------</td>
<td>-------------</td>
<td></td>
</tr>
<tr>
<td>ADIS-IV</td>
<td>Anxiety Disorders Interview Schedule for DSM-IV Disorders</td>
<td></td>
</tr>
<tr>
<td>AN</td>
<td>Anorexia Nervosa</td>
<td></td>
</tr>
<tr>
<td>ASI</td>
<td>Anxiety Sensitivity Index</td>
<td></td>
</tr>
<tr>
<td>BAI</td>
<td>Beck Anxiety Inventory</td>
<td></td>
</tr>
<tr>
<td>BDD</td>
<td>Body Dysmorphic Disorder</td>
<td></td>
</tr>
<tr>
<td>BDI-II</td>
<td>Beck Depression Inventory - 2nd Edition</td>
<td></td>
</tr>
<tr>
<td>BED</td>
<td>Binge Eating Disorder</td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>Body Mass Index</td>
<td></td>
</tr>
<tr>
<td>BN</td>
<td>Bulimia Nervosa</td>
<td></td>
</tr>
<tr>
<td>BPS</td>
<td>Burns Perfectionism Scale</td>
<td></td>
</tr>
<tr>
<td>CBT</td>
<td>Cognitive Behavioural Therapy</td>
<td></td>
</tr>
<tr>
<td>CBT-E</td>
<td>Cognitive Behavioural Therapy - Enhanced for Eating Disorders</td>
<td></td>
</tr>
<tr>
<td>CM</td>
<td>Concern over Mistakes</td>
<td></td>
</tr>
<tr>
<td>CPQ</td>
<td>Clinical Perfectionism Questionnaire</td>
<td></td>
</tr>
<tr>
<td>DA</td>
<td>Doubts about Actions</td>
<td></td>
</tr>
<tr>
<td>DAS-SC</td>
<td>Self Criticism subscale of the Dysfunctional Attitude Scale</td>
<td></td>
</tr>
<tr>
<td>DASS-21</td>
<td>Depression Anxiety and Stress Scale - 21 item version</td>
<td></td>
</tr>
<tr>
<td>DSM-IV</td>
<td>Diagnostic and Statistical Manual of Mental Disorders - Fourth Edition</td>
<td></td>
</tr>
<tr>
<td>DSM5</td>
<td>Diagnostic and Statistical Manual of Mental Disorders - Fifth Edition</td>
<td></td>
</tr>
<tr>
<td>DTEDS-G</td>
<td>Dichotomous Thinking in Eating Disorders Scale-general subscale</td>
<td></td>
</tr>
<tr>
<td>EC</td>
<td>Evaluative Concerns</td>
<td></td>
</tr>
<tr>
<td>EDEQ</td>
<td>Eating Disorders Examination Questionnaire</td>
<td></td>
</tr>
<tr>
<td>EDI-P</td>
<td>Perfectionism subscale of the Eating Disorders Inventory</td>
<td></td>
</tr>
<tr>
<td>EDNOS</td>
<td>Eating Disorders Not Otherwise Specified</td>
<td></td>
</tr>
<tr>
<td>FMPS</td>
<td>Multidimensional Perfectionism Scale by Frost, Marten, Lahart, and Rosenblate (1990)</td>
<td></td>
</tr>
<tr>
<td>FNE</td>
<td>Fear of Negative Evaluation Scale</td>
<td></td>
</tr>
<tr>
<td>Acronym</td>
<td>Description</td>
<td></td>
</tr>
<tr>
<td>---------</td>
<td>-------------</td>
<td></td>
</tr>
<tr>
<td>GAD</td>
<td>Generalised Anxiety Disorder</td>
<td></td>
</tr>
<tr>
<td>GLMM</td>
<td>Generalised Linear Mixed Model</td>
<td></td>
</tr>
<tr>
<td>HMPS</td>
<td>Multidimensional Perfectionism Scale by Hewitt and Flett (1991)</td>
<td></td>
</tr>
<tr>
<td>IAPT</td>
<td>Improving Access to Psychological Therapies</td>
<td></td>
</tr>
<tr>
<td>LSD</td>
<td>Least Significant Difference</td>
<td></td>
</tr>
<tr>
<td>MCAR</td>
<td>Missing Completely at Random</td>
<td></td>
</tr>
<tr>
<td>MECP</td>
<td>Maladaptive Evaluative Concerns Perfectionism</td>
<td></td>
</tr>
<tr>
<td>MINI</td>
<td>Mini International Neuropsychiatric Interview</td>
<td></td>
</tr>
<tr>
<td>MPCI</td>
<td>Multidimensional Perfectionism Cognitions Inventory</td>
<td></td>
</tr>
<tr>
<td>NICE</td>
<td>National Institute for Health and Care</td>
<td></td>
</tr>
<tr>
<td>OCD</td>
<td>Obsessive Compulsive Disorder</td>
<td></td>
</tr>
<tr>
<td>OCI</td>
<td>Obsessive Compulsive Inventory</td>
<td></td>
</tr>
<tr>
<td>OCPD</td>
<td>Obsessive Compulsive Personality Disorder</td>
<td></td>
</tr>
<tr>
<td>PANPS</td>
<td>Positive and Negative Perfectionism Scale</td>
<td></td>
</tr>
<tr>
<td>PC</td>
<td>Parental Criticism</td>
<td></td>
</tr>
<tr>
<td>PCI</td>
<td>Perfectionism Cognitions Inventory</td>
<td></td>
</tr>
<tr>
<td>PE</td>
<td>Parental Expectations</td>
<td></td>
</tr>
<tr>
<td>PS</td>
<td>Personal Standards</td>
<td></td>
</tr>
<tr>
<td>PSP</td>
<td>Personal Standards Perfectionism</td>
<td></td>
</tr>
<tr>
<td>PSWQ</td>
<td>Penn State Worry Questionnaire</td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>Post Traumatic Stress Disorder</td>
<td></td>
</tr>
<tr>
<td>QLES-Q</td>
<td>Quality of Life Enjoyment and Satisfaction Questionnaire</td>
<td></td>
</tr>
<tr>
<td>RCI</td>
<td>Reliable Change Index</td>
<td></td>
</tr>
<tr>
<td>RCT</td>
<td>Randomised Controlled Trial</td>
<td></td>
</tr>
<tr>
<td>SCID</td>
<td>Structured Clinical Interview for DSM-IV</td>
<td></td>
</tr>
<tr>
<td>SEM</td>
<td>Structural Equation Modelling</td>
<td></td>
</tr>
</tbody>
</table>
Abstract
Clinical Perfectionism, as termed by Shafran, Cooper, and Fairburn (2002) is proposed to be a maladaptive construct that maintains psychopathology, with evidence that it can interfere with treatment outcome and result in treatment non-engagement. The Clinical Perfectionism Questionnaire (CPQ) was developed by Fairburn, Cooper and Shafran (2003a) to measure the construct termed by Shafran et al. (2002). However, to date this measure has not been validated in a mixed clinical sample. Study 1 (Chapter 4) assessed the convergent validity of the two factors of the CPQ (Egan et al., 2016) by comparing it to evaluative concerns, a combination of concern over mistakes and doubts about actions subscales, and personal standards subscales of the Frost Multidimensional Perfectionism Scale (Frost et al., 1990), and self-critical perfectionism. The sample consisted of 32 individuals ($M = 34.54$, $SD = 9.71$) with a DSM-IV diagnosis, enrolled in a Randomised Controlled Trial assessing the efficacy of Cognitive Behavioural Therapy (CBT) for clinical perfectionism (Study 2, Chapter 5). Comparing the two factors of the CPQ with a measure of dichotomous thinking, a construct highly related to clinical perfectionism, assessed concurrent validity. There was partial support for convergent and concurrent validity. Internal consistency was adequate for Factor 1 for the current study, however not Factor 2. The results suggest that further evaluation of the two factors of the CPQ is required in a larger mixed clinical sample to fully evaluate its suitability within this population.

It is imperative to evaluate treatments that target underlying factors, such as perfectionism, as current evidence suggest these mechanisms can impact disorder specific treatment. There are reported barriers to accessing treatment therefore alternatives to face-to-face therapies also need to be assessed. It was proposed in this RCT (Study 2) that a transdiagnostic guided self-help cognitive-behavioural approach to targeting clinical perfectionism would reduce perfectionism, as well as symptoms of commonly associated psychopathology: depression, anxiety and stress, and improve an individual’s quality of life. The RCT, was the first to which the author was aware to deliver CBT for clinical perfectionism based on “Overcoming Perfectionism” (Shafran, Egan, & Wade, 2010) in a guided self-help format. The main aims of the RCT included, assessing if the guided self-help intervention reduced perfectionism and an associated construct, dichotomous thinking. The
second aim was to test if a treatment that targets a maintaining mechanism of multiple disorders, clinical perfectionism, also results in the reduction of depression, anxiety, stress and increases in quality of life. Forty participants ($M = 35.43$, $SD = 9.92$) presented with elevated perfectionism and clinical ($n = 32$) and sub clinical ($n = 8$) psychopathology. Several methods of analysis were used to test the hypotheses. Generalised liner mixed models was implemented to compare pre-post-follow-up changes across the treatment and waitlist-control. The intervention was effective at reducing perfectionism as measured by the concern over mistakes (partial $\eta^2 = .29$), personal standards (partial $\eta^2 = .10$) subscales of the FMPS, and self-critical perfectionism (partial $\eta^2 = .27$) and dichotomous thinking at post-treatment (partial $\eta^2 = .17$), with effects maintained at 4-month follow-up. Additionally, whilst not directly targeted in the current study, the treatment significantly reduced symptoms of depression (partial $\eta^2 = 0.10$) and increased quality of life (partial $\eta^2 = 0.14$). No significant changes were observed for clinical perfectionism, as measured by the CPQ, anxiety and stress.

A further aim of the RCT was to assess diagnostic and disorder specific symptom changes for the individuals presenting with a primary DSM-IV diagnosis ($n = 32$). The Anxiety Disorders Interview Schedule for DSM-IV (Brown, Di Nardo, & Barlow, 1994) was implemented at each time point to assess diagnostic status in the mixed clinical sample. Additionally, disorder specific measures corresponding to the participant’s diagnosis were delivered at each assessment point to assess symptom severity. Mixed results were observed in terms of diagnostic changes from pre-treatment to post-treatment. Although a reduction in DSM-IV diagnoses at post-treatment was observed, there was no significant difference in primary diagnostic change between the intervention and control group. The opposite effect was observed when using a dimensional measure of recovery. Primary diagnosis as measured by disorder specific measures, did significantly improve from pre to post-treatment for those in the treatment condition. The intervention was also found to reduce the number of individuals presenting with comorbid psychopathology.

There are reported barriers in translating evidence-based treatment to evidenced-based practice. It was concluded that self-help transdiagnostic interventions could be used to assist in the dissemination of evidence-based therapies. An important future direction for this field is to compare different modes of self-help to face-to-face interventions. Additionally, the comparison of disorder
specific and transdiagnostic interventions is required in a variety of clinical populations, to date this has only been done in eating disorder samples.
Chapter 1: Introduction to Perfectionism

1.1. Overview

The overall aim of this thesis is to further explore the measurement of clinical perfectionism and contribute to the evidence regarding the efficacy of Cognitive Behavioural Therapy (CBT) for clinical perfectionism. First, the psychometric properties of the two factors proposed by Egan et al. (2016) of the Clinical Perfectionism Questionnaire (CPQ) by Fairburn et al. (2003a) will be assessed in a mixed clinical sample ($N = 32$). Second, a Randomised Controlled Trial (RCT) assessing the efficacy of a guided self-help version of CBT for clinical perfectionism will be conducted. The RCT will be divided into two sections; Firstly, a section assessing the intervention effect of CBT for clinical perfectionism on psychopathology in an elevated perfectionism sample ($N = 40$). A section reporting on the diagnostic and clinically significant disorder specific changes for those presenting with a primary diagnoses as classified by the Diagnostic and Statistical Manual of Mental Disorders fourth edition (DSM-IV; American Psychiatric Association, 2000), for a mixed clinical sample ($N = 32$) will then follow. The concluding chapter will review the implications of the findings of the studies together and address key issues in the literature. These studies will make a significant contribution to the literature by providing information regarding the measurement of clinical perfectionism, and the efficacy of a treatment for clinical perfectionism and associated psychopathology.

1.2. What is Perfectionism?

Perfectionism has been identified in the literature since the 1900’s, with several definitions of the construct having emerged over the years. The first acknowledgement by Janet (1898) recognised perfectionists as individuals with fixed ideas. The construct continued to be cited by the likes of Adler (1956) and Freud (1965) whom in their works referred to it as a symptom of neurosis and narcissism. Perfectionism continued to be considered a dysfunctional construct commonly associated with low self-esteem (Horney, 1950), obsessive-compulsive behaviour (Branfman & Bergler, 1955), depression and rigid thinking (Lion, 1942), and medical complications such as gastrointestinal complaints and hypertension (Conn, 1947; Rennie, 1939). The general consensus across the early literature is that
perfectionism was associated with negative processes and dysfunctional psychopathology.

Hamachek (1978) was the first theorist to identify two aspects of perfectionism; normal and neurotic. He separated the two distinct forms by arguing that normal perfectionists receive pleasure from their perfectionistic strivings, whereas neurotic perfectionists suffer. Despite Hamachek’s theory, perfectionism continued to be associated with negative medical consequences (Pacht, 1984) and psychopathology, with Burns (1980a) including a chapter on overcoming perfectionism in his Cognitive-Behavioural self-help manual for depression. Furthermore, perfectionism continued to be associated with personality traits such as neuroticism, as a predictive factor in the development of the construct (Freud, 1965). Little acknowledgement that perfectionism can be associated with positive outcomes was documented until recently.

In the 1990’s, theories began to evolve with recognition of the multidimensional nature of perfectionism and the concurrent development of two widely used multidimensional measures of perfectionism; the Frost Multidimensional Perfectionism Scale (FMPS; Frost, Marten, Lahart, & Rosenblate, 1990) and the Hewitt Multidimensional Perfectionism Scale (HMPS; Hewitt & Flett, 1991). The FMPS consists of six dimensions; concern or fear over mistakes, the setting of high personal standards, perceived parental expectations, parental criticism, doubts about one’s own actions, and organisation. The HMPS consists of three dimensions, including self-oriented perfectionism, other-oriented perfectionism, and socially prescribed perfectionism.

Further evaluation of the multidimensional nature of perfectionism found that the several facets of perfectionism proposed by Frost et al. (1990) and Hewitt and Flett (1991), load onto two distinct factors: positive strivings and maladaptive evaluative concerns (Frost, Heimberg, Holt, Mattia, & Neubauer, 1993). Factor analysis revealed that concern over mistakes, parental criticism, parental expectations, doubts about actions and socially prescribed perfectionism loaded onto maladaptive evaluative concerns and was significantly associated with psychopathology and negative affect. Positive strivings were associated with positive affect and included, personal standards, organisation, self-oriented perfectionism and other-oriented perfectionism, providing support for Frost et al.’s (1990) previous argument that multifaceted perfectionism can be associated with positive and
negative outcomes. These results were supported by the finding of Bieling, Israeli, and Antony (2004) that a conceptual model consisting of two higher order factors of perfectionism; maladaptive evaluative concerns and positive strivings perfectionism, provided a better fit than a unitary approach consisting of FMPS and HMPS subscales. These findings are consistent with Hamachek’s (1978) early theory that perfectionism consists of positive and negative aspects. The definition of perfectionism was changing in the early 1990’s and the construct was beginning to be no longer associated with exclusively negative factors.

The dual process model of positive and negative perfectionism was developed by Slade and Owens (1998) who proposed that positive perfectionism results from high standards being positively reinforced, therefore the individual is encouraged with reward to continue pursuing these standards. They also suggested that pursuing high standards as a means of avoiding negative consequences or punishment, such as self-criticism or a fear of failure, could lead the individual to experience negative perfectionism. Stoeber and Otto (2006) proposed perfectionistic strivings (high personal standards and self-oriented perfectionism) and perfectionistic concerns (high concern over mistakes, doubts about actions and socially prescribed perfectionism). Individuals with high strivings and high concerns are categorised as unhealthy perfectionists, those with low concerns and high strivings are healthy perfectionists and those with low strivings are classified as non-perfectionist. Their review of the literature argued that perfectionistic strivings are associated with positive outcomes such as positive personality traits including extraversion and conscientiousness, greater satisfaction with life, higher self-esteem and greater perceived social support, and reduced attachment anxiety, levels of depression, suicidal ideation, and self-blame.

There is however mixed evidence with regards to the positive benefits of healthy perfectionism in clinical samples. Flett and Hewitt (2006) disagreed with Slade and Owens (1998) notion that perfectionism can be “normal” or “healthy” and highlighted the mixed evidence suggesting that positive perfectionism can be associated with negative effects in clinical samples, in particular eating disorders. Owens and Slade (2008) counter argued Flett and Hewitt’s (2006) response by stating that positive perfectionism is not maladaptive but the measure self-oriented perfectionism, termed a measure of positive perfectionism by several theorists (Klibert, Langhinrichsen-Rohling, & Saito, 2005), can be associated with
maladaptive outcomes. These inconsistencies across the literature lead the authors to highlight that an appropriate measurement of positive perfectionism was required. Whilst many different terms are used; adaptive versus maladaptive, positive versus negative, maladaptive evaluative concerns versus positive strivings, evaluative concerns versus personal standards, normal versus pathological, satisfied versus dissatisfied, healthy versus unhealthy, the general consensus and agreement across the literature is that perfectionism consists of two high order factors (Stoeber & Childs, 2014). The current understanding of perfectionism is consistent with Hamachek’s (1978) early argument that there are “normal” perfectionists and “neurotic” perfectionists. However, there is a new theorized construct called clinical perfectionism, which is proposed to be a further multifaceted dimension of dysfunctional perfectionism highly associated with psychopathology (Shafran et al., 2002).

1.2.1. Construct of Clinical Perfectionism

Shafran et al. (2002) define clinical perfectionism as “the overdependence of self-evaluation on the determined pursuit of personally demanding, self-imposed, standards in at least one highly salient domain, despite adverse consequences”. Shafran and colleagues (2002) model of the maintenance of clinical perfectionism can be seen in Figure 1. The model was developed to identify maintaining mechanisms of the clinically relevant perfectionism that would assist in improving treatment outcome for psychiatric disorders. That is, the aim of developing the model of clinical perfectionism was to advance treatment of the type of perfectionism seen routinely in clinical practice. Shafran et al. (2002) state that central to the maintenance of clinical perfectionism is that an individual bases their self-worth almost exclusively on their pursuit of personally demanding standards which leads to a morbid fear of failure. As a result, the individual with clinical perfectionism sets rigid rules for performance that are judged as being achieved or not through dichotomous thinking. The individual with clinical perfectionism thinks in a dichotomous manner about the attainment of goals. For example, an individual who aims to achieve 90% in an assessment but receives a result of 89% will conclude they are a complete failure.

Increasing literature provides support for the abovementioned pathways defined by Shafran et al. (2002). A qualitative study by Riley and Shafran (2005) revealed that participants with core psychopathology of clinical perfectionism (n =
15) experience self-critical reactions to failure, positive emotional reactions to success, cognitive biases, dichotomous rules and rigidity, engage in avoidance behaviours, and seek escape. Furthermore, additional maintaining mechanisms that were not included in the original model were identified, including safety behaviours, procrastination, fear driven motivation for achieving, and value driven motivation for achieving. These findings provide preliminary support for the proposed maintenance model of clinical perfectionism. However, quantitative methodology is required for empirical validation of the model and the proposed pathways between maintaining mechanisms.

van der Kaap-Deeder et al. (2016) observed that elevated evaluative concerns (EC) perfectionism was associated with greater rumination and less acceptance following experimentally-induced failure of a Tangram Puzzle Task. Additionally, higher avoidance was observed in individuals with elevated EC perfectionism. These findings are consistent with Shafran et al.’s (2002) model which proposed that following failure an individual can become more self-critical, which can lead to adverse consequences such as rumination. In comparison, Kobori, Hayakawa, and Tanno (2009) found that individuals with perfectionism re-appraised their standards and raised their goal after an experimentally induced successes of a task, the Stroop colour-naming test. This supports the pathway of re-appraising standards as insufficiently demanding leading to the re-setting of standards. Supporting the predictions from the Shafran et al.’s (2002) model that perfectionists selectively attend to failure, Howell et al. (2016) observed greater negative attention bias than positive bias in individuals with elevated perfectionism ($n = 31$), compared to individuals with low perfectionism ($n = 25$) on an attention probe task, but only if the stimulus was related to perfectionism.
As dichotomous thinking and self-criticism have been included as factors central to the maintenance of clinical perfectionism (Shafran et al., 2002), they have been included as targets of cognitive-behavioural treatment for clinical perfectionism (Shafran et al., 2010). Egan, Piek, Dyck, and Rees (2007) compared the severity of dichotomous thinking and rigidity in participants diagnosed with a depressive or
anxiety disorder \((n = 40)\), athletes \((n = 111)\), and university students \((n = 101)\). As predicted, the clinical group presented with significantly greater dichotomous thinking, higher levels of negative perfectionism, and rigidity than the athlete and university group. Furthermore, dichotomous thinking and rigidity explained unique variance in positive and negative perfectionism, as measured by the Positive and Negative Perfectionism Scale (PANPS) by Terry-Short, Glynn Owens, Slade, and Dewey (1995). In another study, dichotomous thinking was also evaluated in individuals with eating disorders and explained variance in eating disorder psychopathology beyond that of weight and shape overvaluation (Lethbridge, Watson, Egan, Street, & Nathan, 2011). The findings of these two studies suggest that dichotomous thinking plays an integral part in the psychopathology of depressive, anxiety, and eating disorders.

Self-criticism is strongly correlated with maladaptive dimensions of perfectionism (Grzegorek, Slaney, Franze, & Rice, 2004) and negative outcomes, such as experience of daily hassles, use of avoidance as a coping mechanism, a decrease in the perception of social support, and an increase in negative affect and decrease in positive affect, in community samples (Dunkley, Zuroff, & Blankstein, 2006). Furthermore, it has been argued that the combination of high standards and self-criticism can lead to compulsive exercise behaviours, a proposed developmental risk factor for eating disorders (Taranis & Meyer, 2010). Additionally, further evidence suggests that self-criticism mediates the relationship between high standards and eating disorder psychopathology (Goodwin, Arcelus, Geach, & Meyer, 2014). Dunkley and colleagues (2006) argue that self-criticism can explain the relationship between perfectionism and psychopathology such as depression, anxiety, and eating concerns.

The model of clinical perfectionism has been updated in Shafran, Egan and Wade’s (2010) book titled “Overcoming Perfectionism: A self-help guide using Cognitive Behavioural Techniques”. The revised model is similar to the original, except for the addition of specifying the impact of behaviours in maintaining clinical perfectionism that were implicit in the original model but not specified in the diagram.

1.3. Measurement of Perfectionism

Perfectionism has been reported in the literature to be a predisposing and maintaining factor for a number of disorders, however there is a variety of different
views on how to define the construct (Shafran et al., 2002). Over the years a variety of measurement instruments have been developed to capture the construct of perfectionism as previous literature debated as to whether perfectionism is a multidimensional or single construct (Dunkley, Blankstein, et al., 2006; Hewitt, Flett, Besser, Sherry, & McGee, 2003; Shafran, Cooper, & Fairburn, 2003). Previous uni-dimensional measures of perfectionism have been widely used (Burns, 1980b; Flett, Hewitt, Blankstein, & Gray, 1998; Weissman & Beck, 1978). Frost et al. (1990), and Hewitt and Flett (1991) argue that perfectionism is a multidimensional construct, and these research groups have developed two very widely used measures of multidimensional perfectionism from their theories. Currently, theorists have come to acknowledge that perfectionism consists of two dimensions; maladaptive and adaptive perfectionism (Bieling, Israeli, et al., 2004; Stoeber & Damian, 2014). Whilst there are inconsistencies across the literature on the definition of these two factors, there is a general consensus that they both load onto positive and negative aspects of perfectionism (Terry-Short et al., 1995).

1.3.1. Dysfunctional Attitudes Scale – Self-Criticism

The 15-item self-criticism (DAS-SC) subscale of the Dysfunctional Attitudes Scale (Weissman & Beck, 1978) was one of the earliest measures of perfectionism, although it was not initially designed to measure perfectionism. The Dysfunctional Attitudes Scale was originally designed to measure negative thinking styles, however, Imber and colleagues (1990) factor analysis revealed the 15-item DAS-SC subscale. The validity of the DAS-SC has been assess in clinically depressed (Dunkley, Sanislow, Grilo, & McGlashan, 2004) and non-clinical samples (Dunkley & Kyparissis, 2008). The DAS-SC significantly correlates with symptoms of depression (r = .36) as measured by the Personality Assessment Inventory (Morey, 1991).

1.3.2. Burns Perfectionism Scale

The 10-item Burns Perfectionism Scale (BPS; 1980) was one of the first scales designed to measure a maladaptive form of the construct perfectionism. Burns (1980b) developed the questionnaire by extracting and modifying relevant items from the DAS-SC. Hewitt, Mittelstaedt, and Wollert (1989) established convergent validity of the BPS in a sample of female and male university students (N = 52). Strong correlations were observed between the scale and measures previously used to assess perfectionism, namely, the Attitudes Toward Self-High Standards subscale
(Carver & Ganellen, 1983); \( r = .70 \), and the Irrational Beliefs Test-High Expectations subscale (Jones, 1968a); \( r = .65 \). Discriminant validity was also established with weaker correlations observed between the BPS and discriminant measures; Self-Blame measure (Wollert, Mittelstaedt, Macintosh, Erasmus, & Rawlins, 1986); \( r = .43 \), and the Beck Depression Inventory (BDI: Beck, Ward, Mendelson, Mock, & Erbaugh, 1961); \( r = .36 \). Furthermore, the BPS predicted unique variance in depressed mood following perceived failure of an important task, in a sample of female university students \( (N = 47) \). Also, Hewitt and Dyck (1986) established that the BPS has adequate test re-test reliability \( (r = .63) \) and internal consistency \( (\alpha = .70) \). Despite the BPS being the first unidimensional measure of maladaptive perfectionism, there is limited literature reporting on the psychometric properties of the scale. Further research is required to assess the validity of the scale in clinical samples (Enns & Cox, 2002).

1.3.3. Eating Disorders Inventory Perfectionism subscale

The Eating Disorders Inventory (EDI) by Garner, Olmstead, and Polivy (1983) is a widely used measure of eating disorder psychopathology with extensive literature describing the psychometric properties of the EDI and the EDI-II updated by Garner (1991). The perfectionism subscale of the EDI (EDI-P) is a 6-item self-report measure of perfectionistic cognitions present in eating disorder samples. Approximately 69% of eating disorder studies that have assessed perfectionism have used the EDI-P, however the EDI-P is less commonly used in anxiety disorder and depressive disorder studies (Bardone-Cone, 2007). Bizeul, Sadowsky, and Rigaud (2001) found that a higher score on the EDI-P resulted in a less favourable outcome for participants with Anorexia Nervosa (AN) at 5-10 year follow-up and was significantly associated with illness severity. These findings highlight the need for perfectionism to be assessed in eating disorder samples. Although the EDI-P was designed to measure a single construct of perfectionism, there is increasing debate as to whether it actually is a multidimensional measure. Sherry, Hewitt, Besser, McGee, and Flett (2004) observed a two-factor solution when assessing the factor structure of the EDI-P in a university sample \( (N = 220) \) with three items loading on to self-oriented perfectionism and three items loading onto socially prescribed perfectionism. Lampard, Byrne, McLean, and Fursland (2012) assessed the factor structure of the EDI-P in a sample of 299 females with eating disorders. The authors
confirmed that the EDI-P consists of a two-factor solution with items loading on to socially prescribed and self-oriented perfectionism constructs.

### 1.3.4. Multidimensional Measures of Perfectionism

Prior to the 1990’s perfectionism was measured as a unidimensional construct, however emerging theorists began to argue that perfectionism is likely multidimensional. Whilst there is evidence that perfectionism is strongly related to psychopathology, some theorists argue that aspects of perfectionism can be functional and adaptive (Frost et al., 1990; Hamachek, 1978; Hewitt & Flett, 1991). Based on these arguments, two research groups developed multidimensional measures of perfectionism; the Frost Multidimensional Perfectionism Scale (FMPS: (Frost et al., 1990) and the Hewitt Multidimensional Perfectionism Scale (HMPS: (Hewitt & Flett, 1991).

The FMPS consists of 35 items divided into six subscales measuring different dimensions of perfectionism: concern over mistakes (CM), personal standards (PS), parental expectations (PE), parental criticism (PC), doubts about actions (DA) and organisation. The CM subscale includes items that assess fear of making mistakes, and the perception of negative consequences and self-criticism following perceived failures. Items on the DA subscale measure the individual’s self-doubt in regards to completing a task. Dunkley, Blankstein, et al. (2006) argue that the combination of CM and DA subscales measure EC perfectionism, with several studies comparing EC and other measures of perfectionism (Dickie, Surgenor, Wilson, & McDowall, 2012; Steele, O'Shea, Murdock, & Wade, 2011). PS measures the setting of high standards (Frost et al., 1993). PE and PC assess the perception of parental standards and punishments experienced when the individual was a child. Criticisms of these two subscales are that the items include perception of past experiences and therefore cannot be used to assess treatment outcome as they are not amenable to change. Additionally, theorists argue that these two subscales measure aetiological factors (Rheaume et al., 2000). The organisation subscale is not included in the total FMPS score and includes items that measure neatness and organisation. Frost et al. (1990) acknowledged that the organisation subscale is not considered an integral factor of perfectionism with previous literature acknowledging that it is also a factor associated with functional or positive perfectionism (Chang, Watkins, & Banks, 2004).

Frost and colleagues (1990) reported that the FMPS has good convergent
validity and internal consistency. The CM subscale had strong positive correlations with common measures of perfectionism, namely; BPS ($r = .87$), Jones’s (1968b) Self-Evaluative Scale from the Irrational Beliefs Test (IBT; $r = .61$), and the EDI-P ($r = .57$). The DA and PS subscales had medium to strong positive correlations with the BPS ($r = .47; .53$), Self-Evaluative Scale from the IBT ($r = .31; .53$) and EDI-P ($r = .34; .44$) measures. Recent factor analysis has confirmed that the FMPS could consist of two higher order factors; maladaptive evaluative concerns perfectionism and adaptive perfectionism, also known as PS (Cox, Enns, & Clara, 2002; Stallman & Hurst, 2011). These findings are consistent with Dunkley, Blankstein, et al. (2006) argument that “clinical perfectionism consists of two distinct dimensions. One dimension, tapped by PS perfectionism variables, reflects the determined pursuit of self-imposed standards. The second dimension, tapped by EC perfectionism measures, reflects the extremely vulnerable self-evaluation and critical maintaining pathology of clinical perfectionism” (p.66).

The 45-item HMPS (Hewitt & Flett, 1991) is another commonly used multidimensional measure of perfectionism. Subscales consist of 15 items each and include self-oriented perfectionism, other-oriented perfectionism and socially prescribed perfectionism. Each subscale includes the setting of unrealistic standards, a “motivation to be perfect” and a focus on mistakes. The self-oriented subscale is when these standards are self-imposed on the individual, where as other-oriented perfectionism is when the individual’s standards are imposed on others. Socially prescribed perfectionism is when there is a belief that people in general are also expecting these standards of others. Bieling, Summerfeldt, Israeli, and Antony (2004) proposed that subscales of the FMPS and HMPS load onto two higher order factors, maladaptive evaluative concerns and positive striving. The authors combined the socially prescribed perfectionism, CM, PC, PE and DA subscales of the HMPS and the FMPS to create the Maladaptive Evaluative Concerns Perfectionism (MECP) measure. Additionally, self-oriented perfectionism, other oriented perfectionism, PS and organisation subscales of the HMPS and FMPS were combined to create a Personal Standards Perfectionism (PSP) measure. Bieling et al. (2004) observed that both factors were related to psychopathology. However, only the maladaptive factor significantly predicted score on psychopathology. Bieling and colleagues (2004) concluded that the two-factor structure provided a better model fit than the original multidimensional structure proposed by FMPS and HMPS.
1.3.5. The Perfectionism Cognitions Inventory

The Perfectionism Cognitions Inventory (PCI) developed by Flett et al. (1998) is a 25-item unidimensional self-report measure of perfectionistic automatic thoughts. The PCI has been described as a “state-like” measure designed to assess the frequency of thoughts and is to be differentiated from “trait-like” stable measures of perfectionism, such as HMPS (Flett, Hewitt, Whelan, & Martin, 2007). The PCI is therefore a measure that is designed to fluctuate with changing perfectionism cognitions. Flett et al. (2007) assessed the psychometric properties of the PCI in a sample of psychiatric inpatients ($n = 258$) and individuals recovering from alcoholism ($n = 80$). The PCI was highly correlated with HMPS subscales ($r = .37 - .63$) and measures of anxiety ($r = .42$) and depression ($r = .48$) in the psychiatric inpatient sample. Additionally, the PCI explained an additional eight percent of the variance in anxiety, and seven percent of the variance in depression, over and above perfectionism as measured by the HMPS. The PCI had excellent internal consistency ($\alpha = .95$) in the recovering alcoholics sample. Similarly, incremental validity was established as the PCI accounted for a significant amount of variance (19%) in depressive scores. The PCI has also been empirically validated for the use in adolescent samples (Flett et al., 2012). Moderate correlations were observed with the PCI and measures child/adolescent perfectionism ($r = .50 - .61$), self-criticism ($r = .38$), dependency ($r = .39$), and general negative automatic thoughts ($r = .46$). Similarly, Flett et al. (2012) confirmed the one-factor structure of the PCI in an adolescent sample ($N = 250$) and reported excellent internal consistency ($\alpha = .91$).

Consistent with previous arguments that perfectionism involves adaptive and maladaptive dimensions, the Multidimensional Perfectionism Cognitions Inventory (MPCI) by Kobori and Tanno (2004) was developed. The 15-item MPCI included three subscales assessing positive and negative aspects of perfectionism: personal standards cognitions, pursuit of perfectionism cognitions, and concern over mistakes cognitions. Stoeber, Kobori, and Brown (2014) assessed the predictive validity of the PCI and MPCI in a sample of university students ($N = 324$). The results showed that the multidimensional instrument predicted more variance in depressive symptomatology and positive and negative affect, than the unidimensional measure. The findings lend support to the theory that perfectionism is multifaceted.
1.3.6. Positive and Negative Perfectionism Scale

The PANPS was developed by Terry-Short et al. (1995) to capture functional and dysfunctional aspects of perfectionism. Prior to the development of the scale the only measure of positive perfectionism was the self-oriented perfectionism dimension from the HMPS. Theorist argued that this dimension was not a true measure of positive perfectionism as the subscale includes negative items of perfectionism and was not originally designed for this purpose (Slade & Owens, 1998).

There are few studies that have examined the factor structure of the PANPS (Haase & Prapavessis, 2004; Haase, Prapavessis, & Owens, 1999, 2002), however, these samples only included athletes and neglected to explore the validity of the PANPS with other measures of perfectionism. Until recently the only studies to date to establish validity of the PANPS have used student samples (Bergman, Nyland, & Burns, 2007; Burns & Fedewa, 2005; Fedewa, Burns, & Gomez, 2005). Egan, Piek, Dyck, and Kane (2011) examined the factor structure and the reliability and validity of the PANPS in a student ($n = 101$), athlete ($n = 111$) and clinical sample ($n = 40$). Egan, Piek, et al. (2011) confirmed the original two-factor solution proposed by Terry-Short et al. (1995), however found that the model was not an acceptable fit (CFI = .825). Moderate to strong correlations were observed between the positive perfectionism subscale and the PS subscale of the FMPS across the student ($r = .61$), athlete ($r = .56$) and clinical ($r = .69$) groups. Furthermore, moderate to strong correlations was observed between the negative perfectionism subscale and the CM ($r = .81, .76, .78$) and DA ($r = .61, .59, .43$) subscale of the FMPS across the three groups. Interestingly, depression as measured by the Beck Depression Inventory 2nd Edition (BDI-II; Beck, Steer, & Brown, 1996) was significantly correlated with both subscales. This is not surprising considering that ‘positive’ perfectionism can be associated with psychopathology in clinical samples (Egan, Wade, & Shafran, 2011). The PANPS is the first measure that has been developed to measure adaptive and maladaptive states of perfectionism identified by several theorists (Hamachek, 1978). However, further examination of the psychometric properties is required for the scale to be considered a reliable and valid measure.

1.3.7. The Clinical Perfectionism Questionnaire

Fairburn, Cooper and Shafran (2003a) developed the CPQ to measure the construct defined by Shafran et al. (2002). A criticism of the literature is that the
perfectionism construct had been defined through the creation of measures, as opposed to a theoretically evolved construct. Shafran et al. (2002) argue that previous measures of perfectionism, such as the FMPS and HMPS do not measure perfectionism, but rather constructs that are highly related to perfectionism. The 12-item CPQ was therefore designed to measure the components of the construct clinical perfectionism, however psychometric validation has been limited. Chang and Sanna (2012) administered the CPQ to 243 university students and compared the CPQ with the HMPS. Weak to moderate positive correlations between the CPQ and the three subscales of the HMPS (self-oriented perfectionism $r = .49$, other-oriented perfectionism $r = .28$, socially prescribed perfectionism, $r = .51$) were observed. They also evaluated the predictive validity of the CPQ and found it explained unique variance in depression, anxiety and stress, in addition to that explained by the HMPS. Although Chang and Sanna’s (2012) finding provides support for the construct validity of the CPQ, they did not report on the factor structure of the CPQ.

Dickie, Surgenor, Wilson, and McDowall (2012) examined the factor structure and the concurrent validity of the CPQ with a university sample ($N = 491$). After items 7 and 8 were removed, Exploratory Factor Analysis (EFA) revealed the remaining 10 items yielded a two-factor solution. The first factor consisted of items 1, 3, 6, 9, 10, and 11. The second factor consisted of items 2, 4, 5, and 12. Internal consistency for Factor 1 and Factor 2 of the CPQ were acceptable ($\alpha = .71; \alpha = .71$). They correlated the two factors of the 10-item CPQ with subscales derived from FMPS, namely, the PS subscale and EC subscale, which is the sum of the CM and DA subscales. They found that Factor 1 had a strong positive correlation with the PS, whilst Factor 2 had a strong positive correlation with the EC subscale.

Stoeber and Damian (2014) extended upon the findings of Dickie et al. (2012) and assessed the proposed two-factor structure of the 12-item CPQ in a university sample ($N = 322$). EFA confirmed a two-factor structure, namely, personal standards and evaluative concerns perfectionism. Positive correlations were observed between the two factors and subscales of the FMPS and HMPS, confirming the factors convergent validity. Further studies can employ confirmatory factor analysis methodology to further establish validity of the two-factor structure.

The findings of Chang and Sanna (2012), Dickie et al. (2012), and Stoeber and Damian (2014) provide support for the validity of the CPQ in a university sample and preliminary evidence for the two-factor structure of the CPQ. However,
these findings cannot be generalised to a community sample. Previous literature has reported that university samples are not representative of the wider community as they often have elevated levels of psychopathology (Stallman, 2010). Furthermore, these results cannot be generalised to clinical populations.

Egan et al. (2016) assessed the validity of the CPQ in two samples. The first study comprised a community sample of 206 participants recruited online. Similar to Dickie and colleagues (2012), Egan et al. (2016) also obtained a two-factor solution. The items that loaded onto each factor were similar to Dickie and colleagues (2012) findings: Factor 1 consisted of items 1, 3, 6, 7, 8, 9, 10, and 11; Factor 2 consisted of items 2, 4, 5, and 12. The two factors appear to be measuring personal standards and evaluative concerns. Factor 1 had a strong positive correlation with PS ($r = .64$) and a moderate positive correlation with EC ($r = .35$). Factor 2 had a moderate positive correlation with PS ($r = .31$) and a strong correlation with EC ($r = .65$). Additionally, Factor 1, Factor 2 and the total CPQ were considered reliable measures ($\alpha = .71$; $\alpha = .63$; $\alpha = .71$).

Egan and colleagues (2016) study also explored the discriminant validity of the two factors of the CPQ with a measure of negative affect, The Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988). Participants presenting with higher negative affect had significantly higher scores on Factor 2 of the CPQ. These results support the construct and discriminative validity of the CPQ and its use in a community sample. However, Chang and Sanna (2012), Dickie and colleagues (2012), Egan and colleagues (2016) and Stoeber and Damian (2014) all recognised the need for the validation of the CPQ in a clinical sample.

Egan and colleagues (2016) second study assessed the validity of the CPQ in a female eating disorder sample ($n = 129$) and a female community sample ($n = 80$). Participants were enrolled in a trial of cognitive behavioural therapy for eating disorders (Fairburn et al., 2009). The aim of the study was to compare the CPQ with clinician ratings of clinical perfectionism. Egan and colleagues (2016) found that the CPQ was significantly correlated with the Eating Disorder Examination – Questionnaire (EDEQ) and there was a significant difference between CPQ scores of the clinical sample and healthy controls. The CPQ was also significantly correlated with clinician’s ratings of clinical perfectionism, providing preliminary evidence of construct validity. Furthermore, adequate internal consistency was observed; clinical sample ($\alpha = 0.82$) and healthy controls ($\alpha = 0.73$), and test-retest reliability was
considered strong in the healthy control sample ($r = .81$). These results of the study suggest that the CPQ has adequate internal consistency in an eating disorders sample, however further assessment of the test-retest reliability and construct validity in an eating disorders clinical sample is required. A limitation of the study was that clinician ratings of perfectionism severity were used as a comparison measure instead of empirically validated gold-standard measure of perfectionism, such as the FMPS. Additionally, cases of AN were excluded due to the inclusion criteria of the RCT that the data was derived from. Therefore, findings can only be interpreted to a Bulimia Nervosa (BN), Binge Eating Disorder (BED) and Eating Disorder Not Otherwise Specified (EDNOS) populations.

Steele, O'Shea, Murdock, and Wade (2011) administered the CPQ to 39 female participants receiving treatment for an eating disorder and found strong positive correlations between the CPQ ($r = .76$) and EC ($r = .73$) and PS ($r = .70$) subscales of the FMPS, and self-criticism as measured by the Depressive Experiences Questionnaire (Blatt, D'Afflitti, & Quinlan, 1976). Additionally, the CPQ was the only measure that accounted for a significant amount of unique variance in depressive symptoms within the eating disorder sample. These findings suggest that the CPQ is an effective tool for measuring depressive psychopathology in an eating disorder sample.


Genetic predisposition and parental rearing have been identified as aetiological factors in the development of perfectionism (Flett, Hewitt, Oliver, & Macdonald, 2002; Tozzi et al., 2004). Literature has identified the association of familial perfectionism in mothers and daughters (Frost, Lahart, & Rosenblate, 1991) however there is debate as to whether this familiar pattern is attributed to nature or nurture. Woodside et al. (2002) concluded that perfectionism can run in families after comparing parents of children with eating disorders with parents of healthy controls on measures of eating disorder psychopathology and perfectionism. Mothers of children with eating disorders showed elevated scores on the drive for thinness, ineffectiveness and interceptive awareness subscales of the EDI, and the CM and PC subscales of the FMPS, compared to healthy controls. Additionally, mothers of children with BN, EDNOS, or AN, had significantly higher scores on the FMPS compared to mothers of children with exclusively restrictive AN and healthy controls. These findings suggest that the prevalence of perfectionistic traits is higher
in mothers of children with eating disorder and provide support for the relationship between parents and children. However, due to limitations of the adopted correlational design clarification on the causal attribute, namely, a genetic factor or behaviour modelled throughout the child’s lifetime cannot be concluded. The question still remains, how much is perfectionism a stable inherited trait versus a learned or modelled behaviour that is amenable to change?

1.4.1. Genetic Predisposition of Perfectionism

Research adopting classical twin designs can assist in disentangling the effects of nature versus nurture by comparing individual’s differences across identical monozygotic and non-identical dizygotic twins (Blokland, Mosing, Verweij, & Medland, 2013). Tozzi et al. (2004) analysed data from the population based Virginian Twin Registry (Kendler & Prescott, 1999) and was the first research available to provided evidence for a genetic predisposition of perfectionism. The sample ($N = 1022$) comprised female monozygotic and dizygotic paired and unpaired twins. After conducting multivariate and univariate twin model analyses Tozzi et al. (2004) concluded that aspects of the FMPS, such as CM, were moderately heritable. Additionally the authors found that CM was central to the construct of perfectionism and highly correlated to PS, a reported advantageous aspect of achievement striving, and this was due to genetic factors. In contrast, the authors found that that correlation between CM and DA was likely due to environmental factors.

Similarly, Wade et al.’s (2008) findings from the Australian Twin Registry (Clifford & Hopper, 1986) provide evidence for perfectionism being a genetic factor in the development of AN. The sample comprised 348 monozygotic and dizygotic female twin pairs. Approximately 10% of the sample met criteria for AN or partial AN. The authors found that AN diagnosis was associated with CM, PS, DA and organisation subscales of the FMPS. Analyses of cross-twin associations revealed that PS and organisation were elevated in non-eating disorder individuals of AN probands, compared to non-eating disorder individuals or controls. These findings suggest that perfectionism can be familial risk factor for the development of AN.

There is mounting evidence for a genetic predisposition to perfectionism in community and eating disorder samples, however there is no available evidence to date of the genetic relationship in samples across psychopathology, despite perfectionism being a maintaining factor for anxiety and depressive disorders (Egan, Wade, et al., 2011). Whilst some theorists will argue that perfectionism is a genetic
factor and not amenable to change, there are other aetiological factors in the development of perfectionism that warrant consideration.

1.4.2. Perfectionism as a Learned Trait

Perfectionism is also likely a learned trait that has been modeled throughout childhood (Flett et al., 2002). Frost et al. (1991) proposed that perfectionism in mothers is related to perfectionism in daughters, however this relationship was not observed between fathers perfectionism and daughters perfectionism. Flett et al. (2002) reviewed several models on the development of perfectionism with increasing empirical support, including, the Social Expectations model, the Social Learning model, the Social Reaction model, and the Anxious Rearing model.

The Social Expectations model proposes that children can develop perfectionism from high (and often unattainable) standards set by their parents. This in turn leads the child’s contingent self-worth to be dependent on the achievement of these standards and can often lead feelings of hopelessness when these standards are not met. The child also learns that parental praise is awarded only when the child does meet parental expectations, thus reinforcing the cycle of seeking approval. Frost et al. (1990) acknowledged this aspect of perfectionism when developing the PE subscale of the FMPS. Whereas Hewitt and Flett’s (1991) socially prescribed subscale of the HMPS extends social expectations from the family to larger societal pressures. Some theorists argue that perfectionism can also develop from a lack of parental guidelines however this has not been empirically validated.

The Social Learning model on the other hand proposes that perfectionism is modeled to children by their parent’s own perfectionistic behaviours and cognitions. Social learning models have been widely empirically validated with research stemming from the work of Bandura and Kupers (1964), with findings suggesting that the child will imitate the standards of adults. Appleton, Hall, and Hill (2010) provide support for the Social Learning and Social Expectations models when assessing the development of perfectionism in a sample of elite junior athletes and their mothers ($n = 302$) and fathers ($n = 259$). The parent’s self-oriented, socially prescribed and other-oriented perfectionism as measured by the HMPS was a significant predictor for the young athletes corresponding sub-types of perfectionism. Furthermore, parental perfectionism as perceived by the child was also found to be a significant predictor of the child’s perfectionism.

The Social Reaction model proposes that perfectionism is adopted as a coping
mechanism of childhood abuse, trauma, or hostile living environments (Flett, Hewitt, & Singer, 1995), including psychological maltreatment. This coping strategy can be used as a means of temporary escape for the child, a mechanism to reduce further humiliation and punishment, or a means of attaining control in an unpredictable environment. A significant limitation of the Social Reaction model is that there is no empirical evidence supporting the theory to date.

The Anxious Rearing model proposes that perfectionism develops in the child from an overly anxious rearing with significant exposure to parental concern, worry and fear about making mistakes (Flett et al., 2002). This is accomplished with the parent frequently communicating to the child that they should be vigilant of situations in which mistakes could likely occur. Mitchell, Broeren, Newall, and Hudson (2013) manipulated maternal perfectionistic behaviours in an experiment to assess the impact it had on their child during a copy task with clinically anxious \((n = 42)\) and non-anxious \((n = 35)\) children. The children were required to copy several figures over three one-minute time periods. The children were told that their scores would be dependent on how accurate their drawing was, how similar to that of their peers, and how long it took them. After the first figure copy task was completed with the mother observing from another room, the children were randomly allocated to either ‘high perfectionistic rearing’ or ‘non perfectionistic rearing’ groups and the task was completed again with the mothers delivering the instructions to the child. Mothers in the high perfectionism group were instructed to focus on the child’s mistakes and highlight the negative consequences of mistakes, whereas mothers in the non-perfectionism condition were asked to encourage the child and their behaviour in a relaxed and calm manner. The Figure task was repeated for a third time with the mothers observing from another room. The authors found that children classified as clinically anxious scored significant higher on pre-treatment perfectionism, as measured by the child self-oriented subscale of the Child and Adolescent Perfectionism Scale (Flett, Hewitt, Boucher, Davidson, & Munro, 2001), and anxiety, as measured by the Spence Child Anxiety Scale - Child (Nauta et al., 2004). Additionally, the mothers of the clinically anxious children scored higher levels on the parent version of the scale and a significant relationship was observed between the mother and child’s perfectionism. Mitchell et al. (2013) also found that children in the high perfectionism group had significantly higher scores on self-oriented perfectionism at post-test, whereas a significant reduction in self-oriented
perfectionism was observed in the children in the non-perfectionism condition. Additionally, the authors also found that maternal perfectionism had a negative effect on their child’s task accuracy. The findings provide support for the Anxious Rearing Model that parental perfectionistic behaviours have a significant impact on their child’s perfectionism and task performance when being evaluated. These results also suggest that non-anxious rearing can significantly reduce a child’s perfectionism.

Whilst Flett et al. (2002) recognize the significant overlap of these social and rearing models, the authors also highlight that there are significant differences. The four different models proposed all relate to different dimensions of parenting. For example, the Social Expectations model would relate to a controlling aspect of parent, whilst the Social Reaction model would relate to a punitive and hostile parent style. Whilst the literature in this area is limited, there is evidence suggesting that parental authoritarianism is associated with socially prescribed perfectionism, maternal permissiveness negatively associated with males other-oriented perfectionism, and paternal permissiveness associated with socially prescribed perfectionism in females (Flett et al., 1995). Additionally, perfectionism has been found to mediate the association between over protection by fathers and depression in men. Lack of care by mothers has been significantly associated with depression, self-criticim, and perfectionism in women (Enns, Cox, & Larsen, 2000).

A limitation of these learned social and rearing models is that it attributes the development of perfectionism exclusively to parental behaviours and rearing. Flett et al. (2002) propose a preliminary model of the development of perfectionism (Figure 1) incorporating a multitude of factors, such as child, parental, and environmental, that can contribute to the development of perfectionism. The model proposes that a child’s insecure attachment style can be associated with socially prescribed perfectionism with highly emotional, fearful and persistent temperaments also playing a significant role in the development of perfectionism. Parental factors include the parents high expectations of their children meeting their standards and goals, the parents own perfectionistic practices and personality, and parenting style, such as the authoritarian parenting style. It is also likely that competitive school and work environments promote a level of perfectionism in children and adults. It is likely that during adolescence peers play a significant role on the development of perfectionism over and above that of parents. Cultural factors could contribute to the development of perfectionism. The current model suggest that perfectionism is a
learned trait and therefore amenable to change with the appropriate intervention. A limitation of the model is that it has not been empirically validated so the above-mentioned pathways cannot yet be confirmed.

In summary, there have been several definitions of perfectionism, with recent literature suggesting that perfectionism consists of two factors; namely evaluative concerns and personal strivings. This has led to a variety of measurements to capture the construct. There is evidence to suggest that perfectionism may be influenced by both genetics and learning. Regardless of its origins, there is a general consensus that perfectionism can be problematic for some individuals, which can have a significant impact on their mental health.
Figure 2. Preliminary model of the development of perfectionism. Adapted from (Flett et al., 2002). Copyright 2002 by the American Psychological Association.
Chapter 2: Perfectionism and Psychopathology

2.1. Perfectionism across Disorders

Perfectionism has been found to be elevated across diagnoses of depression, anxiety disorders, eating disorders and obsessive-compulsive disorder, compared to controls (Egan, Wade, et al., 2011; Egan, Wade, & Shafran, 2012; Maia et al., 2009; Shafran & Mansell, 2001), with evidence suggesting that it can be a predisposing and maintain mechanism of psychopathology. The following chapter will review perfectionism across diagnostic categories and the impact that it can have on disorder specific treatment outcome. Additionally, the chapter details how perfectionism is reported to impact an individual’s quality of life and how the transdiagnostic mechanism can explain the presence of comorbidity in diagnostic samples.

2.1.1. Depressive Disorders

In reviews of the perfectionism literature (Egan, Wade, et al., 2011; Egan et al., 2012; Limburg, Watson, Hagger, & Egan, in press), a number of studies have been cited showing that perfectionism is elevated in individuals with depression, strongly correlated with depressive symptomatology (Sassaroli et al., 2008), and reported to be a significant predictor of depression (O'Connor, Rasmussen, & Hawton, 2010). Theoretical models of depression suggest that perfectionistic self-expectations (Hewitt et al., 2003) are what lead perfectionism to be associated with depressive symptoms. Dunkley, Sanislow, Grilo, and McGlashan (2006) examined the predictive validity of perfectionism and symptoms of depression in 96 participants with a depressive disorder diagnosis. The study was part of a larger multisite, longitudinal study of personality disorders (Gunderson et al., 2000). At 24-month follow up negative social interaction, avoidant coping, and negative perception of social support mediated the relationship between perfectionism, as measured by the DAS-SC, and depressive symptoms three years later. This study shows a clear role for perfectionism in predicting depressive symptoms due to its prospective design.

There is emerging literature that has found that perfectionism is associated with and can impact the severity of postpartum depression (Mazzeo et al., 2006). Gelabert et al. (2012) assessed the prevalence of perfectionism in women with postpartum depression (n = 122) and women without postpartum depression (n = 115). Diagnosis was determined by administering the depression module of the
Structure Clinical Interview for DSM-IV (SCID) developed by First, Spitzer, Gibbon, and Williams (1997), whereas perfectionism was assessed using the Spanish version of the FMPS (Gelabert et al., 2011). The authors found that the postpartum depressed sample had significantly higher CM, PS, PC, and DA, than the control sample. Additionally, CM accounted for a significant amount of variance in depression independent from other psychological and contextual factors, such as neuroticism and stressful life events.

Perfectionism can also be a significant predictor of self-harm (O'Connor et al., 2010) and significantly correlated with past, current and future suicidal thoughts and ideation (Hewitt, Flett, & Weber, 1994). O'Connor (2007) conducted a systematic review of the literature and found 29 papers on perfectionism and suicidality in clinical and community populations. The review concluded that there is a significant association between suicidality and EC perfectionism. Literature has extended upon the relationship of perfectionism and suicidal behaviours and non-suicidal self-harm, and suggest that the construct plays a significant role in maintaining these behaviours and ideation. Rasmussen, Elliott, and O’Connor (2012) assessed the prevalence of perfectionism, as measured by the socially prescribed perfectionism subscale of the HMPS, in a sample of inpatients (N = 125) admitted following a suicide attempt. Significant positive relationships between perfectionism and suicidal thinking and anxiety were observed. Furthermore, perfectionism fully mediated the relationship between a punishment driven motivation and suicidal ideation. Claes, Soenens, Vansteenkiste, and Vandereycken (2012) proposed that evaluative concerns would mediate the relationship between perceived parental criticism and self-harm in a sample of 95 female inpatients diagnosed with an eating disorder. Individuals that engaged in self-harm (n = 35) had significantly greater evaluative concerns and perceived parental concerns than individuals that did not (n = 55). Furthermore, there was a significant association between evaluative concerns and the three subscale of the Self-Injury Questionnaire (Claes, Vandereycken, & Vertommen, 2003); self-punishment (β = .30), self-torturing (β = .30), and a cry for help (β = .36). Perceived parental criticism and a cry for help were significantly associated (β = .29) and the hypothesized mediating relationship was confirmed. These findings highlight the need for appropriate perfectionism assessments to be conducted in populations with increased risk of suicidal ideation or intent and
provide further rational for a treatment for perfectionism. Perfectionism treatment in this client group could potentially reduce the likelihood of attempts at suicide or distressing suicidal thoughts and self-injurious behaviour, however further research is required to determine this.

2.1.2. Anxiety Disorders

Perfectionism is elevated across anxiety disorders (Antony, Purdon, Huta, & Swinson, 1998) with a number of studies reporting on the relationship with social anxiety disorder and panic disorder (Saboonchi, Lundh, & Ost, 1999), and emerging research suggesting that perfectionism is elevated in Generalised Anxiety Disorder (GAD) (Handley, Egan, Kane, & Rees, 2014). The following section will review the literature by diagnosis and report on studies that have identified perfectionism as a predictive and maintaining mechanism of these disorders.

2.1.2.1. Social Anxiety Disorder.

Maladaptive perfectionism is associated with symptoms of social anxiety, with greater socially prescribed perfectionism associated with more negative self-thoughts (Laurenti, Bruch, & Haase, 2008), and is considered a risk factor for the development of psychopathology (DiBartolo et al., 2007). Furthermore, Egan, Wade, et al. (2011) note that perfectionism is also included in one of the leading cognitive-behavioural maintenance models of social anxiety, proposing that individuals assess social situations as being threatening as they have excessively high self-imposed standards for social performance (Clark & Wells, 1995). Several studies have been published reporting on the presence of perfectionism in individuals with social anxiety disorder (Frost, Glossner, & Maxner, 2010).

Saboonchi et al. (1999) compared severity of perfectionism across individuals with social anxiety disorder ($n = 52$), panic disorder ($n = 55$), and non-clinical controls ($n = 113$). Perfectionism was significantly elevated in the social anxiety disorder group compared to the panic disorder and non-clinical group. Furthermore, there were significant correlations between perfectionism and measures of depression and anxiety for the social anxiety disorder group. Rosser, Issakidis, and Peters (2003) explored the association of perfectionism and psychopathology in a sample presenting for treatment for social anxiety disorder ($N = 61$). They observed significant associations between perfectionism, as measured by the CM and DA subscales of the FMPS, and social anxiety. However, the relationship between CM and social anxiety was no longer significant when depression and neuroticism were
controlled for. They also observed significant reductions in CM after seven weeks of group treatment for social anxiety.

The findings of Saboonchi et al. (1999) and Rosser et al. (2003) provide support for the inclusion of perfectionism in the cognitive-behavioural maintenance model of social anxiety. Furthermore, perfectionism could explain presence of comorbidity for individuals diagnosed with a primary social anxiety, as it is associated with symptoms of depression and anxiety in this diagnostic group.

2.1.2.2. Panic with and without Agoraphobia.

Perfectionism is proposed to lead to a hyperawareness or sensitivity of the perception of physiological symptoms, which is associated with panic disorder (Wood, Cano-Vindel, & Salguero, 2015). There are inconsistencies in the literature however, in regards to perfectionism being elevated in individuals with panic disorder. Unlike depression, social anxiety disorder and Obsessive Compulsive Disorder (OCD), Wheeler, Blankstein, Antony, McCabe, and Bieling (2011) found that perfectionism in individuals with panic disorder are comparable to that of community samples. Whilst, Saboonchi et al. (1999) concluded that CM and DA was significantly higher in a social anxiety disorder groups compared to a panic disorder group, they also found that perfectionism was significantly elevated in the panic disorder groups compared to the community sample. The authors therefore concluded that perfectionism plays a greater role in social anxiety than it does in panic disorder. Wheeler et al. (2011) observations are inconsistent with the findings of Iketani et al. (2002b) who compared individuals with panic disorder, with \( n = 59 \) and without \( n = 44 \) agoraphobia, to a non-clinical sample \( n = 35 \). Using the FMPS, the authors found that CM, PS, PC and DA were significantly higher in the panic disorder with agoraphobia group, compared to the panic disorder without agoraphobia and non-clinical groups. Furthermore, perfectionism as measured by the total FMPS score, significantly predicted unique variance in agoraphobia. These findings suggest that perfectionism is elevated in individuals with panic disorder compared to the community and suggest that perfectionism is a factor that can play a role in the development and maintenance of agoraphobia.

Extending upon these findings, Iketani et al. (2002a) assessed the association of perfectionism, panic disorder with and without agoraphobia and comorbidity of personality disorders. Using Stepwise regression analysis the authors found that perfectionism was significantly associated with cluster C personality disorders for
individuals with comorbid panic disorder. Additionally, significant indicators of perfectionism in this diagnostic group were the presence of comorbid avoidant personality disorder and obsessive-compulsive personality disorder. Whilst there is some debate in regards to the severity of perfectionism in panic disorder compared to other diagnoses, there is evidence to suggest that perfectionism is elevated in individuals with panic disorder compared to non-clinical groups.

2.1.2.3. Generalised Anxiety Disorder.

GAD is a debilitating anxiety disorder characterised by excessive and unrelenting worry across several life domains (Barlow, 2008). Literature exploring perfectionism in individuals diagnosed with GAD was scarce until recently, no published studies to date having reported on the association (Egan, Wade, et al., 2011). Jarrett, Black, Rapport, Grills-Taquechel, and Ollendick (2014) interviewed parents of youth meeting diagnostic criteria for GAD \((N = 60)\), as determined by the Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV) Child and Parent versions (Silverman & Albano, 1996). Parents reported greater perfectionism in younger children (7-9 years) than older children (10-13 years) diagnosed with GAD. Whilst this study reported on the differences across the two age groups, no information describing the strength of the relationship between symptomatology and the construct of perfectionism were made. Future research could compare the presentation of youth diagnosed with GAD and non-clinical samples to determine if perfectionism is elevated in this diagnostic group.

Handley et al. (2014) found that perfectionism was significantly associated with pathological worry and depression in 36 individuals diagnosed with GAD, from a larger clinical sample of individuals presenting for treatment of their perfectionism \((N = 42)\). Strong to moderate correlations were observed between pathological worry, as measured by the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) and the CM and PS subscales of the FMPS \((r = .68, .49)\) and the CPQ \((r = .49)\). Furthermore, the CPQ was found to significantly predict unique variance in pathological worry when depression was controlled for. Depression, as measured by the BDI-II was significantly associated with all measures of perfectionism \((CM, r = .49; PS, r = .38; DA, r = .43; CPQ, r = .56)\) and pathological worry \((r = .35)\). The findings of the study confirm hypotheses that perfectionism is a predictive factor in the development of GAD. This was the first study to explore these relationships using a clinical sample. Further research is
required to explore maintaining mechanisms of perfectionism within the GAD diagnostic group as perfection is proposed to maintain other anxiety disorders, such as social anxiety disorder (Egan, Wade, et al., 2011).

2.1.3. Post-Traumatic Stress Disorder.

Despite there being a significant association between maladaptive perfectionism and symptoms of Post-Traumatic Stress Disorder (PTSD) in community samples (Kawamura, Hunt, Frost, & DiBartolo, 2001), literature exploring perfectionism in individuals with PTSD is scarce (Egan, Wade, et al., 2011). Egan, Hattaway, and Kane (2014) assessed the prevalence of perfectionism in 30 individuals that had presented to a sexual assault centre. Diagnostic features of PTSD were determined by a score of ≥ 50 on the Post-Traumatic Stress Checklist (Weathers, Huska, & Keane, 1991), a self-report diagnostic tool of PTSD. Individuals that had experienced their trauma in the previous month were excluded from the study to ensure they did not meet criteria for Acute Stress Disorder. Perfectionism was measured using the CPQ and the CM and PS subscales of the FMPS. Additionally, rumination was assessed, using the Ruminative Response Scale from the Response Styles Questionnaire (Nolen-Hoeksema, 1991), to assess the mediating effect of rumination on the relationship between perfectionism and post-traumatic symptoms. Correlational analyses revealed positive relationships between CM \( (r = .57) \) and CPQ \( (r = .69) \) and post-traumatic symptoms, however this was not the case for PS. Additionally, positive relationships were observed between the three measures of perfectionism and rumination; PS \( (r = .44) \), CM \( (r = .67) \), and CPQ \( (r = .69) \). Further analyses confirmed the author’s predictions that the relationship between perfectionism, as measured by the CPQ, and post-traumatic symptoms were mediated by rumination. This study was the first to which the author is aware to provide evidence that perfectionism is elevated in individual with post-traumatic stress. Although there was no control group in the Egan, Hattaway, et al. (2014) study, the means and standard deviations of the perfectionism measures are elevated compared to previously reported community samples and comparable to that of anxiety disorder samples (Saboonchi et al., 1999). A limitation of their study is the generalizability of the findings to PTSD clinical samples. Whilst an elevated score on the Post-Traumatic Stress Checklist does indicate clinically significant symptoms and a presence of diagnosis, formal diagnosis using a structured and reliable diagnostic measure is required in order for the sample to be determined a clinical
sample. Further research is required using PTSD clinical samples diagnosed with gold standard diagnostic instruments, such as the SCID.

2.1.4. Obsessive Compulsive and Related Disorders

In the DSM-IV OCD was categorised as an anxiety disorder, however it is now been included in a new category of obsessive compulsive and related disorders in the DSM-5 (American Psychiatric Association, 2013). The authors propose that central to these diagnostic categories is the individual experiences of obsessions and compulsions. A number of studies have found that perfectionism is elevated in OCD and related disorders (Buhlmann, Etcoff, & Wilhelm, 2008; Sassaroli et al., 2008). It has also been included in leading theories of OCD (OCCWG, 1997) and Body Dysmorphic Disorder (BDD) (Veale, 2004) as a maintaining mechanism of these disorders.

2.1.4.1. Obsessive Compulsive Disorder.

Perfectionism is elevated in individuals diagnosed with OCD (Sassaroli et al., 2008), with DA higher than in other anxiety disorder or OCD groups (Antony et al., 1998; Buhlmann et al., 2008). Cognitive-behavioural models of OCD suggest that individuals with OCD find the idea of making mistakes and imperfections intolerable (OCCWG, 1997). Dimensions of perfectionism can explain OCD symptom severity as found in Martinelli, Chasson, Wetterneck, Hart, and Bjorgvinsson (2014). The sample included individuals with a primary \( n = 37 \) or secondary \( n = 9 \) diagnosis of OCD and assessed symptoms as measured by the Obsessive Compulsive Inventory (OCI-R) by Foa et al. (2002), and dimensions of perfectionism, as measured by the FMPS. DA was found to predict checking symptoms, whereas the organisation subscale of the FMPS was associated with ordering symptoms. The findings highlight the importance of assessing dimensions of perfectionism in this population as DA has also been linked to poorer treatment outcome (Chik, Whittal, & O’Neill, 2008), which is not surprising considering the strong overlap of OCD symptoms and items included in the DA subscale, as the items from the DA subscale were adapted from a measure of OCD, the Maudsley Obsessive-Compulsive Inventory (Hodgson & Rachman, 1977)

Reuther et al. (2013) explored the relationship between perfectionism, intolerance of uncertainty and severity of obsessive-compulsive symptomatology in 475 college students. The authors found that perfectionism, as measured by the FMPS total score, had a significant indirect effect on OCD symptoms and severity
through the mediating effect of intolerance of uncertainty. These findings support the cognitive model of OCD, which proposes that dysfunctional cognitions contribute to symptom severity (OCCWG, 1997). These findings provide support for the need to treat underlying maintaining mechanisms of psychopathology and provide a possible explanation as to why perfectionism can impede treatment outcome for OCD.

Perfectionism can have a positive or negative effect on individuals within this population. In a sample of individual diagnosed with OCD ($N = 81$) perfectionism, was found to be significantly higher in individuals with a history of suicidal attempts (Kim et al., 2016). This finding suggests that individuals with OCD and elevated perfectionism could be at increased risk of developing suicidal ideation. In contrast, Boisseau, Thompson-Brenner, Pratt, Farchione, and Barlow (2013) observed less risk taking behaviour (gambling task) in females diagnosed with OCD ($n = 19$) similar to that of healthy controls ($n = 21$), compared to females with an eating disorder ($n = 17$). This finding suggests that perfectionism can be adaptive and maladaptive but dependent on the diagnostic population.

2.1.4.2. Body Dysmorphic Disorder.

Perfectionism is elevated in individuals with BDD, however there is only a small amount of literature available supporting this. In the cognitive-behavioural model of BDD perfectionism is proposed to be an “idealised value” which leads to a negative appraisal of self (Veale, 2004). Buhlmann et al. (2008) compared individuals with BDD ($n = 19$) to individuals with OCD ($n = 21$) and to a sample of healthy controls ($n = 21$). The participants were required to rate a series of photographs of faces in terms of their physical attractiveness on a Likert scale from 1-7, with 7 being very attractive. Participants were also required to complete a series of measures, namely, the BDI, the BDD Modification of the Yale-Brown Obsessive Compulsive Scale (Phillips, Hollander, Rasmussen, & Aronowitz, 1997), and the FMPS. As hypothesised, participants with BDD rated their own photograph as less attractive than participants in the OCD and healthy controls. Additionally, participants in the BDD group had significantly higher levels of CM than participants in the control group. However, there was no significant difference observed amongst the two clinical groups. Whilst the BDD group also had significantly higher score on DA than the control groups, the OCD group had significantly greater DA than the BDD group. The findings of the current study suggest that perfectionism is elevated in individuals with BDD relative to that of a clinical OCD sample.
There is emerging literature that suggests perfectionism can be a predictive factor in the development of BDD (Bartsch, 2007; Schieber, Kollei, de Zwaan, Muller, & Martin, 2013). When assessing a sample of Australian university students \((N = 619)\), Bartsch (2007) found that socially prescribed and self-oriented perfectionism, from the HMPS, explained significant and unique variance in dysmorphic concern, as measured by the DYMORphic Concern Questionnaire (Oosthuizen, Lambert, & Castle, 1998). Bartsch’s (2007) findings were confirmed by Schieber et al. (2013) when comparing individuals diagnosed with BDD \((n = 58)\) to a community sample \((n = 2071)\) from a German population survey. Schieber and colleagues (2013) found that perfectionism, as measured by the EDI-P subscale, was not only elevated in the BDD sample compared to the control, but that it also explained unique variance in predicting dysmorphic concerns. The authors concluded that traits such as perfectionism, aesthetic sensitivity and reactivity could predispose the individual to be vulnerable to the development of BDD.

2.1.4.3. Hoarding Disorder.

Hoarding was formally considered a subtype of OCD and was not recognised in DSM-IV. Although hoarding disorder has been included in the DSM-5, further research is required to understand the cognitive processes of an individual presenting with hoarding disorder (Woody, Kellman-McFarlane, & Welsted, 2014). Some theorists argue that perfectionism is a developmental and maintaining mechanism for hoarding disorders (Frost & Hartl, 1996) and suggest that perfectionism, indecision and procrastination are significantly associated with hoarding behaviours (Timpano et al., 2011). However, beyond these reports there is little research acknowledging these associations. Frost and Hartl (1996) included perfectionism in the cognitive behavioural model of compulsive hoarding. They proposed that hoarding evolves from cognitive processing deficits, inabilities to form emotional attachments, behavioural avoidance, and dysfunctional beliefs about possessions. Frost and Hartl (1996) described hoarding as “an indecisiveness behaviour associated with perfectionism” (p. 348). The authors propose that decision making deficits lead the individual to develop a fear of making mistakes which leads to avoidance or procrastination of tasks, such as discarding items. Additionally, deficits, including difficulty with memory, may lead the individual to keep certain objects in order to remember associated events, as making a mistake or forgetting can lead to anxiety, distress and feelings of failure about forgetting. These proposed cycles have led
theorists to hypothesize that perfectionism that leads to behavioural avoidance can be a central factor in hoarding disorder, however there is no empirical evidence to support this theory to date.

Compulsive, impulsive and excessive purchasing or acquiring of items is a central aspect to hoarding disorder. Bose, Burns, and Garretson Folse (2013) proposed that perfectionism would be associated with acquisitive buying, a compulsive behaviour associated with hoarding. The authors interviewed 20 university students’ (i.e., non-acquisitive buyers), and 42 non-university students’ (i.e., acquisitive buyers). Qualitative analysis identified several themes that the participants associated with their purchasing, namely, materialism, variety seeking, self-control, and perfectionism. Furthermore, the individuals identified that perfectionism is a driver for their acquisitive buying. Further quantitative analysis by Bose et al. (2013) using a different sample of shoppers (\(N = 408\)) revealed that perfectionism was significantly elevated in the acquisitive buyers group compared to the mainstream buyers. Further research is required into understanding the role perfectionism plays in hoarding disorder. Mechanisms that propose to maintain the cycle of hoarding and associated behaviours needs to be explored further to improve modest and under researched treatment outcomes (Grisham & Norberg, 2010).

### 2.1.4.4. Trichotillomania.

The DSM-5 defines trichotillomania or hair pulling disorder as the recurrent pulling of one’s hair causing clinically significant distress, with repeated attempts to cease engaging in the behaviour. There is limited research available that reports on the relationship between trichotillomania and perfectionism, however Noble (2013) cited authors that have observed the association in their treatment manuals for the disorder (Keuthen, Stein, & Christenson, 2001; Penzel, 2003). Noble (2013) assessed the association across a sample individuals diagnosed with trichotillomania (\(n = 114\)) and a university sample (\(n = 200\)). The authors proposed that shame would mediate the relationship between perfectionism and trichotillomania and that perfectionism would impact on symptom severity of the disorder. Perfectionism, as measured by the Almost Perfect Scale-Revised (Slaney, Rice, Mobley, Trippi, & Ashby, 2001), was elevated in the clinical sample compared to the university sample and behavioural shame was found to mediate the relationship between maladaptive perfectionism and trichotillomania symptom severity in the clinical sample. It is important to note that there were no mediating relationships for adaptive
perfectionism. The findings suggest that maladaptive perfectionism may need addressing in treatment of trichotillomania as it could be considered a barrier to successful treatment of the disorder. The findings also highlight the importance of differentiating adaptive and maladaptive forms of perfectionism when conceptualising mediating mechanisms of disorders to determine if the form of perfectionism is indeed pathological (Noble, 2013).

2.1.5. Eating Disorders

The eating disorder diagnostic category has undergone significant changes since the introduction of DSM-5 in May 2013. The criterion of amenorrhoea has been removed from AN, weight criterion has become less rigid, and a criterion for BN includes lower frequency binge/purge cycles. Of the most significant change has been the removal of EDNOS as a diagnostic category. Rather, it has been replaced with Other Feeding of Eating Disorder including atypical anorexia and low frequency bulimia nervosa, and Unspecified Feeding or Eating Disorders. Little research is available using DSM-5 criteria, therefore the following literature will include a review of DSM-IV eating disorders, including EDNOS.

Individuals with AN and BN have higher levels of perfectionism than individuals with depression, OCD and healthy controls (Sassaroli et al., 2008). Perfectionism is proposed to impact on achievement striving leading to strict dieting and other weight control behaviour, as outlined in Fairburn, Cooper, and Shafran (2003b) cognitive-behavioural maintenance model of eating disorders. Furthermore, patients with eating disorders and comorbid suicidal ideation/ attempts had significantly greater maladaptive perfectionism than patients without suicidal thoughts (Yamaguchi et al., 2000). Perfectionism has also been identified in the literature as a predictive factor for the development of eating disorders (Fairburn, 2008; Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Stice, 2002). Fairburn, Cooper, Doll, and Welch (1999) aimed to identify risk factors for the development of AN and to also compare these risk factors to risk factors associated with the development of BN and other disorders. The study recruited female participants aged 16-35 years; 67 participants with a history of AN, 102 participants with BN, 102 participants with a variety of disorders and 204 participants without a diagnosis formed a healthy control condition. It was found that reported childhood perfectionism and negative self-evaluation was linked with the later development of an eating disorder.
Perfectionism can remain unchanged following disorder specific treatment for AN. Nilsson, Sundbom, and Hagglof (2008) aimed to explore changes in perfectionism after receiving treatment in the 1980’s. They examined a sample of women (N = 68) who had been diagnosed with AN as a child or adolescent. Nilsson et al. (2008) found that after receiving treatment for AN, levels of perfectionism remained unchanged at 8 and 16 years follow up even though eating disorder symptoms and psychopathology decreased. This finding is concerning as proposed in the transdiagnostic model of eating disorders, clinical perfectionism is a core maintaining mechanism of the psychopathology of eating disorders (Fairburn et al., 2003b). It is believed that for some individuals their eating, weight and shape is a domain in which their perfectionism manifests (Shafran et al., 2002). As perfectionism has been shown to maintain certain individuals eating disorders, it is therefore of fundamental importance to target perfectionism in the treatment of eating disorders (Fairburn, 2008). Enhanced Cognitive Behavioural Therapy (CBT-E) for eating disorders addresses the core psychopathology of eating disorders, who Fairburn et al. (2003b) described as “a dysfunctional system for evaluating self-worth….people with eating disorders judge themselves largely or even exclusively in terms of their eating habits, shape or weight and their ability to control them”. CBT-E also addresses four additional maintaining mechanism specific to certain individuals; clinical perfectionism, core low self-esteem, mood intolerance and interpersonal difficulties (Fairburn, 2008).

Fairburn et al. (2009) conducted a RCT comparing standard CBT and CBT-E for eating disorders. The sample comprised 154 females with a DSM-IV diagnosis of an eating disorder, including BN, EDNOS, and BED. Participants with a Body Mass Index (BMI) <17.5 were excluded from the study, therefore excluding cases of AN. Participants underwent 20 weeks of treatment. Follow up data was collected at 20, 40 and 60 weeks post-treatment. Fairburn et al. found that at 60 weeks post-treatment 61.4% of the sample had eating disorder symptoms and psychopathology within one standard deviation of the community mean, and this result was across both treatments. The findings show that CBT-E was more effective for individuals that had a wider variety of psychopathology; that is clinical perfectionism, core low self-esteem, mood intolerance or interpersonal difficulties, than the focused form of CBT. Fairburn et al. (2009) argued that the focused CBT should be implemented, however for individuals with additional psychopathology, CBT-E should be applied. This is
consistent with Byrne, Fursland, Allen, and Watson (2011) findings that perfectionism can significantly decrease post CBT-E for individuals presenting with elevated perfectionism at pre-treatment. These studies support the notion that by treating additional psychopathology such as perfectionism, treatment outcomes can be improved.

2.1.6. Perfectionism and Treatment Outcome Across Disorders

There is evidence that elevated perfectionism can interfere with treatment outcome across disorders (Chik et al., 2008; Egan, Wade, et al., 2011). Blatt, Zuroff, Bondi, Sanislow, and Pilkonis (1998) examined the impact perfectionism had on 239 participants receiving treatment for a primary diagnosis of major depressive episode. The participants were allocated to one of four treatment conditions; CBT, Interpersonal Therapy, imipramine plus clinical management and a placebo pill plus clinical management. Clinical evaluators, therapists and patients rated their perception of the success of the treatment as well as general functioning, symptoms of depression and therapeutic gain. Blatt et al. (1998) found that elevated pre-treatment scores of perfectionism, as measured by the DAS-SC, impacted on the effectiveness of all four treatments in approximately 67% of the sample. Pre-treatment perfectionism also significantly correlated with patients reporting less change in their depressive symptoms at 18-month follow up, an observation that was consistent with the ratings made by clinical evaluators and therapists. Jacobs et al. (2009) assessed the impact perfectionism had on treatment for adolescents (N = 439) presenting with clinical depression. Adolescents were randomised into one of four 12-week treatment conditions, including CBT, fluoxetine, CBT and fluoxetine combination, and pill placebo. Depression and suicidal ideation were measured using the Children’s Depression Rating Scale (Poznanski & Mokros, 1996) and the Suicidal Ideation Questionnaire (Reynolds, 1987). Perfectionism was assessed with DAS-SC. A significant correlation was observed between baseline perfectionism and baseline depression and suicidal ideation. Additionally, adolescents with higher perfectionism had high scores of depression and suicidal ideation at post-treatment. The authors concluded the perfectionism was a predictor of treatment outcome, with higher perfectionism being associated with high levels of depression across treatment, which impacted suicidal ideation. The results also suggested that perfectionism was a mediator between the treatment and outcome of depression and suicidal ideation. These findings suggest that perfectionism can impede treatment
outcome of adolescents presenting with depression and suicidal ideation. The findings Blatt et al. (1998) and Jacobs et al. (2009) illustrate that the treatment of perfectionism is required in order for individuals to successfully engage in evidence based disorder specific treatments, such as CBT for depression.

Perfectionism has been included in maintenance models of anxiety disorders, such as social anxiety disorder (Egan, Wade, et al., 2011), therefore it make sense that recent literature has found that it can interfere with treatment outcome if not addressed. Lundh and Öst (2001) assessed the efficacy of CBT for social phobia in 24 participants. The FMPS was used to measure levels of perfectionism. At post-treatment all scores on the FMPS had decreased and 75% of the sample had significantly improved. Non-responders to treatment had high levels of perfectionism pre-treatment and whilst they did decrease slightly, their scores were still considered in the clinical range. The authors suggested that individuals with elevated perfectionism should receive a treatment that targets their perfectionism as it could impede upon the treatment for social phobia. Similar findings were observed in Ashbaugh et al. (2007) study that assessed perfectionism in individuals (N = 107) engaging in a 12-session group CBT for social anxiety disorder. As predicted, the authors observed significant pre-post treatment reductions in perfectionism, as measured by the FMPS, however scores were still significantly greater than community norms. Furthermore, larger declines in DA significantly predicted additional post-treatment reductions in social anxiety symptomatology. This finding is significant considering that DA is elevated in social anxiety disorder samples compared to other anxiety disorders (Saboonchi et al., 1999).

Chik et al. (2008) used the FMPS to measure the effect perfectionism has on treatment outcome for individuals with OCD. The sample consisted of 118 participants that were undergoing either cognitive therapy or exposure and response prevention in either an individual or group therapy condition for their primary diagnosis of OCD. Pre-treatment scores on the DA subscale of the FMPS were significantly correlated with OCD symptom severity. Chik et al. (2008) found at post-treatment that OCD symptom severity and DA were associated. Another finding was that DA and CM perfectionism affected treatment response for the individuals in the exposure and response prevention group compared to the cognitive therapy group. The results suggest that cognitive therapy would be a more appropriate treatment for individuals diagnosed with OCD and with elevated DA and CM
perfectionism (Chik et al., 2008). This suggestion is consistent with the findings of Pinto, Liebowitz, Foa, and Simpson (2011), that comorbid Obsessive Compulsive Personality Disorder (OCPD) and in particular perfectionism, interferes with OCD exposure and response prevention treatment and predicted worse outcome. These findings further highlight the importance of accurate case conceptualisation and assessment of pre-treatment perfectionism, to avoid the delivery of an ineffectual disorder specific treatment.

There is a small amount of literature reporting on evidence-based treatments for AN, with recovery rates at approximately 70-75% occurring over 6-11 years (Hay et al., 2014). Considering the length of treatment and the modest likelihood of full remission, it is imperative to identify factors, such as perfectionism, that could impede treatment for this diagnostic group. Sutandar-Pinnock, Woodside, Carter, Olmsted, and Kaplan (2003) observed high levels of perfectionism in patients with AN after receiving inpatient treatment. There was also a significant association between treatment drop out and poor treatment response and levels of perfectionism. The findings highlight the importance of administering assessment tools such as the EDI, which assess eating disorder symptomatology and factors highly related to eating disorders, such as the perfectionism subscale.

2.2. Perfectionism and Quality of Life

The absence of symptomatology is currently the most commonly used indicator of improved treatment outcome across the mental health literature. However, the World Health Organisation (1948) acknowledges that health is “…a state of complete physical, mental and social well-being and not merely the absence of disease” (p. 100). Consequently, emerging research has highlighted the need for a more holistic measure of improvement and an inclusion of positive mental health outcomes, such as an evaluation of quality of life (Swan, Watson, & Nathan, 2009).

Quality of life is significantly more impaired in individuals with anxiety disorders (Barrera & Norton, 2009; Henning, Turk, Mennin, Fresco, & Heimberg, 2007), depressive disorders (Trompenaars, Masthoff, Van Heck, Hodiamont, & De Vries, 2006), eating disorders (Padierna, Quintana, Arostegui, Gonzalez, & Horcajo, 2000), obsessive-compulsive disorders (Didie et al., 2007; Huppert, Simpson, Nissenson, Liebowitz, & Foa, 2009; Phillips, Menard, Fay, & Pagano, 2005), and schizophrenia (Braga, Mendelowicz, Marrocios, & Figueira, 2005), and can be significantly impacted depending on presence of comorbidity (Didie et al., 2007;
Norberg, Diefenbach, & Tolin, 2008; Watson, Swan, & Nathan, 2011). Until recently, limited research was available reporting on the impact of perfectionism on quality of life. Stoeber and Stoeber (2009) observed significant negative associations between life satisfaction, as measured by the Satisfaction with Life Scale (Diener, Emmons, Larsen, & Griffin, 1985) and socially prescribed perfectionism, in a sample of university students \((n = 109)\) and internet users \((n = 289)\). Although these findings provide preliminary evidence that perfectionism has an impact on life satisfaction, further research is required using samples with elevated perfectionism, or clinical perfectionism.

Wong, Chan, and Lau (2010) explored the association of perfectionism, psychopathology and quality of life in a sample of Chinese adults \((N = 146)\) presenting for treatment of depressive symptoms. Using cluster analysis, the participants were divided into three groups; maladaptive perfectionists \((n = 70)\), adaptive perfectionists \((n = 54)\), and non-perfectionists \((n = 20)\). Depression and dysfunctional attitudes were significantly greater in participants with maladaptive perfectionism, however, quality of life, as measured by a Chinese translated version of the Quality of Life Enjoyment and Satisfaction Questionnaire (QLES-Q; Endicott, Nee, Harrison, & Blumenthal, 1993), was comparable across the three groups with mean scores in the clinical range (Ritsner, Kurs, Gibel, Ratner, & Endicott, 2005). Perfectionism, dysfunctional attitudes and depressive symptomatology significantly predicted variance in leisure activity, a subscale of the QLES-Q, and depression predicted significant variance in the social relationships subscale of the QLES-Q. The findings of Wong et al. (2010) provide preliminary evidence for the impact of perfectionism and dysfunctional attitudes on quality of life. This study highlight that further research into this area is required with cross-cultural samples to better understand the impact of perfectionism on quality of life in a Western setting.

Perfectionism does not have an exclusively negative effect on quality of life. Gilman and Ashby (2003) divided a sample of adolescents into adaptive perfectionists \((n = 29)\), maladaptive perfectionists \((n = 17)\) and non-perfectionists \((n = 67)\), as determined by the APS-R. Interestingly, adaptive and maladaptive perfectionists reported significantly greater self-satisfaction scores than non-perfectionists. One potential explanation for these results could be a positive association of perfectionistic behaviours in childhood/adolescents (i.e. good grades, keeping room clean), with parental and societal praise and reward. These findings
highlight the need to assess positive outcomes associated with perfectionism, in order to fully understand the construct of perfectionism (Chang, 2000). It is important to note that whilst life satisfaction and quality of life are significantly related (Frisch, Cornell, Villanueva, & Retzlaff, 1992), they are two distinct constructs with differing measurement instruments.

2.3. Perfectionism as an Explanation for Co-Morbidity

Comorbidity is common amongst individuals presenting with psychiatric diagnoses (Andrews, Henderson, & Hall, 2001; Kessler et al., 2005) and it is likely that the presence of perfectionism can impact on the occurrence of comorbidity. Kaye, Bulik, Thornton, Barbarich, and Masters (2004) observed that perfectionism is higher in women with a current eating disorder and one or more life time comorbid anxiety disorders, than women who present with only one diagnosis. There is evidence to suggest that perfectionism can be a cognitive process shared across diagnoses. Menatti, Weeks, Levinson, and McGowan (2013) observed that maladaptive perfectionism completely mediates the relationship between fear of public scrutiny and bulimic symptomatology in a sample of undergraduate women ($N = 167$), suggesting that perfectionism can be a maintaining factor for comorbid social anxiety and eating disorders. Similarly, Fergus and Wu (2010) found that OCD and GAD share the same cognitive process, such as perfectionism, that maintain psychopathology. Future longitudinal studies looking at the causal links of perfectionism and multiple diagnoses are required as it can assist in the explanation of comorbidity and prevention of psychopathology.

Bieling, Summerfeldt, et al. (2004) explored the association between MEC, PS and comorbidity of disorders in sample ($N = 345$) presenting at a clinic for anxiety disorders. Using the SCID it was determined that the sample included diagnoses of panic disorder with or without agoraphobia, OCD, social phobia and specific phobia. Bieling, Summerfeldt, et al. (2004) screened for principle and additional diagnosis. Participants that had at least two principle diagnoses comprised 65% of the sample, three or more diagnoses 36%, and four or more diagnoses 18%. They found that a significant predictor for comorbidity was MEC. The findings suggest that perfectionism occurs in the psychopathology of a variety of disorders. They argued “the present findings leave open the intriguing possibility that if perfectionism were treated directly, it is possible that the individual would experience symptomatic relief across a number of domains” (p. 199). This argument
has also been posited by Egan, Wade, et al. (2011) who note that it may be more effective for individuals with elevated perfectionism and multiple diagnosis, to receive a treatment that targets their perfectionism, as an underlying maintaining mechanism across disorders.

Wheeler et al. (2011) extended upon the findings of Bieling, Summerfeldt, et al. (2004) by assessing a mixed clinical sample including, social anxiety disorder, panic disorder, OCD, major depressive disorder, and non-clinical controls. There was a significant association between comorbid diagnoses and MEC. Furthermore, when comparing high (2 or more additional diagnoses) and low (0 or 1 comorbid diagnoses), self-critical perfectionism, as measured by the Self-Critical Perfectionism Scale (Blankstein, Harkins, & Jalali, 2008; Harkins, Blankstein, Jalali, Krawaczyk, & Wheeler, 2003), was significantly greater in the high comorbidity group across the diagnostic groups.

2.4. Perfectionism as a Transdiagnostic Process

Harvey, Watkins, Mansell, and Shafran (2004) argued that a transdiagnostic approach has an across-disorders perspective, rather than the disorder focused approach that identifies specific disorders and their risk factors and maintaining factors separately. Harvey et al. (2004) state that “… psychological disorders are more similar than different in terms of the cognitive behavioural processes that maintain them” (p. 23) and argue that cognitive behavioural processes occur on a continuum. A categorical approach to diagnosing does not encapsulate the complexity of the client’s problems. Mansell, Harvey, Watkins, and Shafran (2009) propose that when using a transdiagnostic approach, a diagnosis is not necessary to deliver effective treatment, opposed to a disorder focused approach.

Egan, Wade, et al. (2011) argue that perfectionism is a transdiagnostic process that can explain the comorbidity of a variety of psychopathologies. Mansell et al. (2009) state “…the transdiagnostic approach to CBT hypothesizes that there is a range of cognitive and/or behavioural maintenance processes shared across psychological disorders, that is, processes that are elevated in a wide range of psychological disorders relative to non-psychiatric controls and that causally contribute to the development and/or maintenance of symptoms” (p. 7). There is extensive evidence, as reviewed in this chapter, that suggests that perfectionism is associated with a range of symptomatology, and therefore can share underlying maintain mechanisms across diagnoses. By adopting a transdiagnostic
perspective to perfectionism, treatments can be developed that can be implemented to individuals with diagnoses sharing the same maintaining mechanism, therefore being more effective for individuals with comorbid presentations (Egan, Wade, et al., 2011; Egan et al., 2012; Harvey et al., 2004).

Craske (2012) divided transdiagnostic treatments into two distinct groups. The first group includes treatments that can be applied across all clinical disorders, such as mindfulness-based stress reduction or acceptance and commitment therapy. The second group includes treatment that can be applied across diagnostic categories, such as Barlow’s unified treatment protocol for emotional disorders (Allen, McHugh, & Barlow, 2008) or Fairburn’s (2008) transdiagnostic CBT-E treatment for eating disorders.

McEvoy, Nathan, and Norton (2009) reviewed the literature and concluded that transdiagnostic treatment resulted in decreases in psychopathology and reductions in comorbidity compared to control conditions. Furthermore, preliminary evidence was provided to suggest that transdiagnostic treatments are equally effective to disorder specific interventions. McEvoy et al. (2009) acknowledged that no RCT’s to date have compared transdiagnostic treatment to disorder specific interventions. However, in more recent years literature has emerged that has provided support for the efficacy of transdiagnostic treatments.

Musiat et al. (2014) assessed the efficacy of a cognitive-behavioural transdiagnostic trait focused web-based intervention for university students \( (N = 1047) \). Participants were randomly allocated to the transdiagnostic intervention, or an online control condition that consisted of self-help strategies to manage student life. The transdiagnostic intervention consisted of five modules including: an introduction to CBT, perfectionism, self-esteem, anxiety and worry, and dealing with difficult emotions. At baseline participants with an elevated score on the CM and DA subscales of the FMPS, the Neuroticism subscale of the NEO-Five Factor Inventory (Costa & McCrae, 1992), and Hopelessness subscale of the Substance Use Risk Profile (Woicik, Stewart, Pihl, & Conrad, 2009), were classified as being at high risk of developing a clinical disorder. In comparison to the low risk group, those considered high risk had significantly higher scores of depression, generalised anxiety, and disordered eating, with significantly lower quality of life. There was a significant reduction in depression and generalised anxiety from baseline to 12-week follow-up for participants in the transdiagnostic interaction, however this effect was
not observed for disordered eating. Furthermore, participants classified as high risk had significantly greater reductions in depression and generalised anxiety to those classified as low risk. Perfectionism as measured by the CM and PS subscales of the FMPS also significantly decreased over the two time periods for the individuals receiving the transdiagnostic intervention. These results provide preliminary evidence for the efficacy of a cognitive-behavioural transdiagnostic web-based intervention at reducing disorder specific psychopathology for individuals at high risk of developing these disorders. To extend upon the findings of the current study future research should include an RCT with a pure control condition and a disorder specific comparison intervention to ascertain efficacy of the intervention (Chambless & Hollon, 1998). The study was considered as a preventative intervention for the development of psychological disorders in a university sample. Therefore, further research should implement a similar design in a mixed clinical sample to determine if a transdiagnostic treatment is appropriate across diagnoses.

Egan, Wade, et al. (2011) argue that perfectionism is a transdiagnostic process, and that there is several lines of evidence that suggest that if it were to be targeted directly, then the individual may experience symptom relief across a number of symptom domains. In the example of eating disorders, Fairburn (2008) describes core maintaining factors, such as perfectionism, like the building blocks at the bottom of a house of cards. Ultimately if the building blocks at the bottom of the house of cards; namely perfectionism, were removed the top cards would come tumbling down; namely eating disorder (Shafran et al., 2010). Essentially, Fairburn (2008) indicates that by removing perfectionism, an individual’s eating difficulties may be alleviated as the foundation underlying the eating disorder psychopathology have been removed.

There is a vast amount of literature, detailed in this chapter that provides evidence that perfectionism is associated with a wide array of psychopathology and therefore supports the argument that it is a transdiagnostic process (Egan, Wade, et al., 2011) Consequently, to overcome limitations in the literature, a study is needed that includes participants with a variety of diagnoses; that is depression, anxiety disorders, obsessive compulsive and eating disorders, to test if CBT for perfectionism has efficacy in reducing a number of different psychological symptoms.
Chapter 3: Treatment of Perfectionism

3.1 Cognitive Behavioural Treatment for Clinical Perfectionism

Perfectionism is a predisposing and perpetuating factor in clinical diagnoses (Egan, Wade, et al., 2011). Furthermore, research has shown that perfectionism can maintain disorder specific psychopathology and significantly interfere with treatment outcomes of depressive, anxiety, eating, and obsessive-compulsive disorders (Blatt et al., 1998; Chik et al., 2008; Egan, Wade, et al., 2011; Lundh & Öst, 2001). CBT for clinical perfectionism is a single treatment that can be applied for a variety of disorders when the clinician determines perfectionism to be the primary presenting problem or a barrier to disorder specific change (Egan et al., 2012). The aim of the treatment is to prevent the individual judging their self-worth on the success of meeting personally demanding standards and the resulting self-criticism when these standards are not achieved. Although not a direct aim of the treatment, the individual may change their high standards as a result (Egan, Wade, et al., 2011).

There are several studies in the literature that have evaluated CBT for clinical perfectionism (Egan, Wade, et al., 2011), with a meta-analysis reporting on its efficacy with significant reductions in CM and PS from pre-post intervention (Lloyd, Schmidt, Khondoker, & Tchnaturia, 2015). Shafran, Lee, and Fairburn (2004) investigated the impact of eight sessions of CBT for clinical perfectionism for a client with BED. They reported a reduction in the client’s perfectionism and a reduction in symptoms of her BED and depression at post-treatment and 5-month follow up. The authors hypothesised that the improvements in the eating disorder and depressive symptoms were directly related to the reduction in level of perfectionism as this was identified as the key maintaining variable in the client’s BED. Findings from the study cannot be generalised to a clinical population with BED as it was a single case design.

Glover, Brown, Fairburn, and Shafran (2007) observed nine participants with either depression or an anxiety disorder receiving CBT for perfectionism. Perfectionism was assessed using the FMPS, HMPS and the CPQ. Six participants at post-treatment and follow-up had significantly improved perfectionism scores across the two multidimensional measures and three participants improved across all three perfectionism measures. There was however no reductions in anxiety, as measured by the Beck Anxiety Inventory (BAI) (Beck, Epstein, Brown, & Steer, 1988).
Clinically significant reduction in depression, as measured by the BDI, was observed in three of the nine participants, with only one maintaining the reduction at follow-up. Similarly, Egan and Hine (2008) evaluated the effectiveness of an 8-session CBT for clinical perfectionism in treating four individuals with either a diagnosis of depression or an anxiety disorder. Symptoms of perfectionism decreased across the whole sample with clinically significant reductions on CM for three out of four of the participants. Additionally, three out of the four participants also showed a reduction in symptoms of depression, as measured by the BDI, all of the participants had reductions in anxiety (BAI) whilst in the treatment phase. Two out of the four participants showed an increase in anxiety post-treatment. Whilst Egan and Hine (2008) and Glover et al. (2007) both observed positive improvement in individuals perfectionism and suggest that further RCTs need to be conducted assessing the efficacy of CBT for clinical perfectionism. Interestingly, both studies observed contrasting effects when it came to depression and anxiety, warranting further research into the impact of CBT for clinical perfectionism on psychopathology.

Riley et al. (2007) conducted the first RCT to assess the efficacy of CBT for clinical perfectionism. Riley et al used an 8-week waitlist/control condition and compared this to a perfectionism treatment consisting of 10 sessions over an 8-week period. The treatment consisted of a number of strategies: behavioural experiments, psychoeducation, cognitive restructuring and adopting new cognitions and behaviours to broaden self-focus. The study included 20 individuals with elevated clinical perfectionism as determined by a semi-structured interview, cited by Riley et al. (2007). The majority of the sample (n = 16) also met diagnostic criteria for a range of anxiety disorders such as GAD, social anxiety disorder, OCD, panic with agoraphobia and specific phobia, and major depressive episode, as determined by the SCID. Riley et al. (2007) found that CBT for clinical perfectionism was superior to the control condition in significantly reducing an individual’s clinical perfectionism as determined by the CPQ (d = 1.36) and the semi-structured clinical perfectionism interview (d = 2.05). Additionally, they found that the intervention was effective at reducing symptoms of associated psychopathology, as determined by the BDI and BAI, which were maintained at follow-up. Of the participants that met diagnostic criteria at pre-treatment, 50% of those allocated to the treatment condition no longer met DSM-IV criteria at post-treatment. All of the participants allocated to the waitlist control condition continued to endorse DSM-IV criteria at post-waitlist.
To extend upon Riley et al. (2007) findings, Steele et al. (2013) adopted a single group sequential design to compare psychoeducation and an 8-week cognitive-behavioural group treatment for clinical perfectionism, to a waitlist-control. The sample consisted of 21 participants with and without DSM-IV diagnoses. Diagnoses included major depressive disorder ($n = 4$), social anxiety disorder ($n = 3$), panic disorder ($n = 2$), dysthymia ($n = 2$), generalised anxiety disorder ($n = 2$), and OCD ($n = 1$). Nine of the participants presented with more than one DSM-IV diagnosis with seven of the participants not currently meeting DSM-IV criteria. Five of the non-clinical participants had depression in remission. All participants were allocated to a 4-week waitlist prior to treatment to act as their own control. They were then assigned four chapters from the book titled “Overcoming Perfectionism” (Shafran et al., 2010) for psychoeducation material related to perfectionism. The participants were required to spend four weeks reading the first five chapters of the book that contain information on understanding perfectionism but not strategies for how to overcome perfectionism, and then return to begin CBT for clinical perfectionism in a group treatment setting.

The intervention started with a collaborative formulation of the individual’s maintenance cycle of perfectionism, followed by identifying problem areas and motivation techniques to determine the individual’s readiness for change. The concept of surveys and behavioural experiments were then introduced, which are common strategies used throughout the intervention to address rigid and biased thinking, procrastination, and self-criticism. The intervention also introduced the concept of time management and scheduling pleasant events, practicing self-compassion and relapse prevention. For further details of the treatment content see Shafran et al. (2010) and Egan, Wade, Shafran, and Antony (2014). Two-hour sessions of CBT for clinical perfectionism occurred weekly for eight weeks.

There were significant pre-post treatment reductions in scores on the CPQ ($d = 1.55$), CM ($d = 1.72$) and PS ($d = 1.91$) subscales of the FMPS, self-criticism as measured by the DAS-SC ($d = 1.46$), and negative affect ($d = 1.59$) as measured by the Depression Anxiety Stress Scale (DASS-21; Lovibond & Lovibond, 1995a). The results suggest that group CBT for perfectionism was effective at reducing perfectionism and psychopathology. Importantly, these treatment effects were also maintained at 3-month follow-up with strong effect sizes observed across all of the variables ($ds = 1.55 – 1.93$). None of the participants with a baseline diagnosis of
social anxiety disorder and only 25% with a baseline diagnosis for a depressive disorder met current criteria at 3-month follow-up. However, the 4-week psychoeducation component did not have a significant effect on perfectionism or measures of psychopathology. The intervention was not compared to control group or alternative treatment condition, therefore the effect of non-specific treatment factors were not controlled. The authors acknowledged that an RCT should be conducted with a larger sample size and a pure clinical sample.

There are some limitations of the Riley et al. (2007) and Steele et al. (2013). First, the number of participants was small and only included a mixed anxiety, depressive, and non-clinical sample with mixed diagnostic outcomes. The authors suggest that to extend upon their findings, future studies should aim to recruit a larger number of participants presenting with a range of diagnoses. A rationale was provided to incorporate eating disorders, as clinical perfectionism is a core maintaining mechanism of eating disorder psychopathology (Fairburn et al., 2003b).

Handley, Egan, Kane, and Rees (2015) addressed the limitations of the literature identified by Riley et al. (2007) and Steele et al. (2013) and assessed the efficacy of group CBT for clinical perfectionism in a mixed clinical sample ($N = 42$) comprising anxiety disorders, depressive disorder and eating disorders. Individuals were randomly allocated to the treatment or an 8-week waitlist control condition. Similarly to Steele et al. (2013), the treatment manual was adapted from Shafran et al. (2010) and consisted of 2-hour weekly sessions occurring over eight weeks. There were significant reductions in perfectionism, as measured by the CM and DAS-SC for participants in the treatment condition with large effects observed, $d = 1.23$ and $d = 1.48$ respectively. Furthermore, there were significant reductions for the treatment group in depression ($d = .74$), anxiety ($d = .56$), and dysfunctional eating symptomatology ($d = .30$) as measured by the DASS-21 and EDEQ. The authors also reported a clinically significant pre-post change in perfectionism, as measured by the CM, for participants in the treatment group and 80% of participants with a pre-treatment diagnosis no longer met criteria at 6-month follow-up. The findings suggest that treating a mixed clinical group for clinical perfectionism can reduce perfectionism and symptoms of psychopathology, even though disorder specific symptoms were not directly targeted in the treatment. Although individuals presented with a range of symptomatology, the common underlying factor that was consistent across the group was elevated perfectionism, thereby suggesting that perfectionism is
a transdiagnostic-maintaining factor across disorders and can be treated with a transdiagnostic intervention. As participants were treated in a group setting, this study also provides a rationale for CBT for clinical perfectionism to be administered in alternative formats to traditional face-to-face individual therapy.

### 3.2. Alternatives to Face-to-Face Treatment

The Australian Bureau of Statistics (2007) reported that within the previous 12 months 3.2 million Australians had a psychological disorder. Findings from the National Mental Health Report (2010) suggest that only 38% of adults, adolescents and children that experience psychological disorders sought professional help. These figures suggest that there is a significant proportion of Australian’s mental health needs not being met. Despite CBT being the leading evidence based treatment for a variety of disorders there is evidence emerging that CBT interventions are rarely available and not effectively delivered to the community (Shafran et al., 2009).

A way of reducing the demand on mental health services is by adopting a prevention approach. Research has indicated that prevention programs that focus on perfectionism are effective in reducing perfectionism and psychopathology. Fairweather-Schmidt and Wade (2015) implemented a 2-lesson school-based perfectionism intervention across three primary schools ($N = 125$) with children who had a mean age of 11.6 years. There was a significant reduction in self-oriented perfectionism as measured by the Child and Adolescent Perfectionism Scale (O'Connor, Dixon, & Rasmussen, 2009), hyperactivity and emotional problems. Whilst these results provide support for perfectionism being addressed in primary school aged children, a limitation of the study was the short 4-week follow-up period. Longer follow-up periods are required in prevention studies to measure later development of psychological symptoms. Nehmy and Wade (2015), examined 6-12 month follow-up of adolescents ($N = 688$) across four high schools receiving an 8-lesson intervention targeting unhelpful perfectionism compared to control. At 6-month follow-up reductions in perfectionism, as measured by the DAS-SC, were observed in the intervention condition as well as, self-criticism and negative affect. Additionally, the reductions in perfectionism were maintained at 12-month follow-up. Wliksch, Durbridge, and Wade (2008) also assessed the efficacy of an 8-lesson perfectionism intervention compared to a media literacy program delivered to female adolescents ($N = 127$) from two high schools, to address eating disorder symptoms. A reduction in CM, as measured by the FMPS, and eating disorder symptoms were
greater in the perfectionism intervention than that observed in the media literacy program. Whilst further research is needed in this field, the literature that is available shows support for the utility of prevention approaches focused on perfectionism.

In the United Kingdom (UK) the Improving Access to Psychologies Therapies (IAPT) program was developed as 75% of individuals (45 million people) with anxiety disorders and depression were unable to access psychological treatment. The program aimed to help individuals gain easy access to evidence based psychological therapies, which resulted in funding in the UK being increased substantially due to the recognition of this problem. As there is a high demand for mental health services there is a need to enhance the accessibility and availability to these services at minimal cost (IAPT, 2010). The IAPT (2010) states that to increase access to treatment there is a need for an alternative to face-to-face therapy and distinguish between two types of therapy required. “Low intensity” therapy can be delivered in a self-help format whilst “High intensity” therapy involves face-to-face individual therapy. A stepped-care model can be adopted when determining the appropriateness of the two types of therapy delivery. Low intensity treatments can be offered initially to individuals presenting with mild to moderate difficulties as a first line treatment. If the individuals do not respond to low intensity interventions or their symptoms are deemed to be too severe for low intensity options, they can be referred to high intensity treatment methods. Low intensity therapy can be delivered in several ways, namely; behavioural activation, computerized CBT and group CBT, and CBT in either a pure or guided self-help format (Williams & Martinez, 2008).

According to the adapted Glasgow and Rosen (1978) taxonomy (Newman, Szkodny, Llera, & Przeworski, 2011), self-help can be divided into three categories of delivery; self-administered, no therapist contact throughout the treatment period; minimal contact, irregular unscheduled therapist contact; and guided self-help, regular therapist contact incorporated in the treatment plan. A recent meta-analysis of 38 RCT’s assessing the efficacy of self-help concluded that there was no significant difference in the effect size across the three categories of self-help delivery (guided, \( g = 0.53 \); minimal contact, \( g = 0.55 \); and self-administered, \( g = 0.42 \)) (Farrand & Woodford, 2013). This finding is significant considering previous research reported guided self-help to have superiority to pure self-help (Hirai & Clum, 2006). When looking at the categories separately, Farrand and Woodford (2013) observed medium to large effect sizes when guided self-help was delivered via the telephone (\( g = .91 \),
when participants were diagnosed with a DSM-IV disorder at assessment \( (g = .79) \) including, insomnia \( (g = .94) \), panic disorder \( (g = 1.37) \), recurrent binge eating disorder \( (g = .54) \) and social anxiety \( (g = .74) \), and when participants were recruited from the community setting \( (g = .74) \). Despite the findings that there are minimal differences across categories of self-help, guided self-help is considered the preferred method of treatment delivery (Gyani, Shafran, Layard, & Clark, 2011).

Until recently guided self-help interventions were only considered efficacious for self-presenting community samples with sub-clinical symptomatology (Coull & Morris, 2011) and face-to-face approaches were considered superior to guided self-help for complex clinical samples (NICE, 2013). Cuijpers, Donker, van Straten, Li, and Andersson (2010) found no significant difference between the two methods of treatment delivery at post-treatment and up to one year follow-up when they reviewed 21 RCT’s comparing face-to-face and guided self-help treatment in anxious and depressed clinical samples. Furthermore, no significant differences were observed for the rate of participant drop out. This was surprising considering that previous research has reported drop out to be higher in guided self-help compared to face-to-face interventions. The findings of the study suggest that treatment effects are comparable across the two methods of treatment delivery. However, the authors acknowledged that a limitation of the study was the generalizability of the results to clinical treatment settings the recruitment methods adopted by the majority of the studies \( (n = 17) \) required the participant to self-refer and consent to randomization.

Findings from an initial evaluation of the IAPT program provide a rationale for the use of self-help methods in routine clinical care. Richards and Suckling (2009) report on the treatment outcomes of the program at the Doncaster site in UK. The patients self-referred or were referred by a general practitioner for treatment of anxiety or depression. The site adopted a stepped-care model. Those deemed to have not responded to initially provided low intensity treatment methods, such as guided self-help or computer assisted CBT, were referred to high intensity treatments. By adopting low intensity treatment methods 2794 individuals were assessed and offered treatment at the site in a 12-month period. Recovery rates of 66% and 67% were observed for diagnosis of depression and anxiety. Furthermore, three years after the implementation of the program across the UK 45,000 individuals were reported to have moved off sick pay and benefits, 683,000 individual completed treatment, and
3,400 new therapists were trained in the implementation of NICE interventions (IAPT, 2012).

IAPT (2010) state that CBT can be effectively accessed by the client in an evidence-based self-help format as treatment outcomes are shown to be successful. There is a need to examine the efficacy of both low-intensity and high-intensity therapies, as a way of increasing access. Due to the increase in demand for alternative therapies, a variety of CBT self-help materials, for example eating disorders, have been developed. It is important for these therapies to be trialled to ensure they are appropriate to be delivered to the consumer.

3.2.1 Self-help perfectionism treatment in non-clinical samples

There is increasing evidence that self-help therapies can be effective at reducing a wide array of symptoms. There is evidence to suggest that CBT for perfectionism is effective at reducing perfectionism and symptoms of associated psychopathology in community and clinical samples (Handley et al., 2015; Riley et al., 2007; Steele et al., 2013). As disorder-specific interventions have adopted self-help alternatives, transdiagnostic therapies are following suit. Radhu, Daskalakis, Arpin-Cribbie, Irvine, and Ritvo (2012) investigated the efficacy of a web-based CBT intervention for perfectionism adapted from Arpin-Cribbie et al. (2008) intervention. The study included 47 undergraduate university students with maladaptive perfectionism as determined by the PCI. The study compared the treatment to a waitlist control condition on measures of perfectionism, FMPS and HMPS, and measures of psychopathology. The 12-week web-based CBT for perfectionism was divided into three modules: rediscover clear thinking, learning not to stress yourself out, and bouncing back better. The treatment was designed to address dysfunctional perfectionistic cognitions and behaviours that impact on psychopathology. From pre-treatment to post-treatment there were significant decreases on CM and PCI within the CBT group. Additionally, at post-treatment there was a significant positive correlation between the CM and measures of depression, anxiety, and stress. At post-treatment there were significant decreases on measures of anxiety, helplessness and personal maladjustment for the treatment condition but not for the control condition.

Similarly to Radhu et al. (2012), Arpin-Cribbie, Irvine, and Ritvo (2012) examined the efficacy of a 10-week web-based CBT for perfectionism intervention also adapted from Arpin-Cribbie et al. (2008). The sample consisted of 77 first and
second year university students with elevated scores on the PCI. The students were randomised into three intervention conditions, namely, CBT for perfectionism \((n = 29)\), stress management \((n = 26)\), and a waitlist-control \((n = 22)\). The participants were required to complete measures of perfectionism (namely, the FMPS and HMPS) and psychopathology (namely, The Centre for Epidemiological Studies-Depressed Mood Scale (Radloff, 1987), BAI, Anxiety Sensitivity Index (Beck et al., 1988), and Automatic Thoughts Questionnaire (Hollon & Kendall, 1987). There was a significant post-treatment decrease in scores on all measures of perfectionism for the CBT intervention group and a significant decrease in levels of depression and anxiety sensitivity. There was also a significant decrease in self-orientated perfectionism, CM and anxiety sensitivity scores in the stress management group with no significant change in scores for the waitlist-control condition. Overall, participants in the CBT for perfectionism intervention had a significantly greater reduction in levels of perfectionism than those in the stress management and waitlist-control conditions, however these effects were not observed for symptoms of depression and anxiety. The significant improvement for individuals in the stress management group warrants further consideration. Although the individuals in this group did not receive any cognitive strategies that directly targeted their perfectionism, a significant decrease in perfectionism and associated psychopathology occurred. Therefore, it is possible that the reduction in perfectionism was due to general changes in psychopathology and the learning of cognitive strategies used to decrease stress. The findings of Arpin-Cribbie et al. (2012) and Radhu et al. (2012) suggest that web-based CBT for perfectionism can reduce perfectionism and related psychopathology and provide support for the use of web-based self-help CBT interventions. However, the findings of these studies can only be generalised to a university student populations.

Pleva and Wade (2007) examined CBT for clinical perfectionism self-help in a non-clinical sample. The study consisted of 49 participants with elevated levels of perfectionism as determined by a score of > 84 on the FMPS. Pleva and Wade (2007) compared two formats of the intervention: guided self-help and pure self-help. The therapies were based on a self-help book titled ‘When Perfect Isn’t Good Enough’ by Antony and Swinson (1998). The guided self-help group received eight 50-minute sessions with a Psychologist to review the chapters in the book. The pure self-help condition was only provided with an information sheet and contacted twice via
telephone throughout treatment. Pleva and Wade (2007) found that guided self-help was more effective than pure self-help in reducing perfectionism (as measured by the FMPS) as well as obsessive compulsive and depressive symptoms. These treatment effects were maintained at a 3-month follow up. Limitations of this study include the use of a non-clinical sample, and a failure to identify DSM-IV disorders, thus limiting generalisations that can be made from this study to clinical populations.

### 3.2.2. Self-help perfectionism treatment in clinical samples

There is a large body of literature supporting the use of self-help techniques in the treatment of depression (Vernmark et al., 2010), anxiety disorders (Andersson, Paxling, et al., 2012), eating disorders (Traviss, Heywood-Everett, & Hill, 2011), and obsessive compulsive disorders (Andersson, Enander, et al., 2012). There is also a small body of literature emerging reporting on the outcomes of CBT for clinical perfectionism delivered in a self-help format a variety of diagnostic presentations.

Steele and Wade (2008) compared three types of self-help therapy for 48 individuals meeting DSM-IV criteria for BN. The three conditions consisted of CBT for clinical perfectionism based on Antony and Swinson (1998) book, a traditional treatment of BN using the book ‘Bulimia Nervosa and Binge-Eating’ (Cooper, 1993) and a placebo intervention using mindfulness techniques from a book called ‘Mindfulness Based Cognitive Therapy for Depression’ by Segal, Williams, and Teasdale (2002). Treatment outcomes including, CM, PS, eating disorder psychopathology as measured by the Eating Disorders Examination, depression and anxiety as measured by the DASS and self-esteem as measured by Rosenberg Self Esteem Scale (Rosenberg, 1965), at post treatment and 6-month follow up were comparable across the three groups. Additionally, only 19% of participants had ceased engaging in binge/purge behaviours at six months post-treatment and again this was observed across the three conditions. The findings of this study indicate that a self-help transdiagnostic treatment for perfectionism is comparable to that of a disorder specific intervention for BN. Furthermore, the findings show that individuals in the perfectionism treatment group experienced disorder specific symptom relief even though their disorder specific symptomatology was not been targeted directly. Although the current study was the first RCT to include an eating disorder sample, Steele and Wade (2008) only looked at one diagnostic group, BN, therefore the findings may not generalise across eating disorder diagnoses and cannot be generalised to other diagnostic presentations.
Egan, van Noort, et al. (2014) conducted the first study of which the author is aware that compared a face-to-face and a pure self-help version of CBT for clinical perfectionism, and a waitlist control, in a mixed clinical sample (N = 52). Consistent with previous research (Handley et al., 2015; Steele et al., 2013), the treatment protocol was adapted from Shafran et al. (2010) and consisted of eight weekly sessions for individuals in the face-to-face condition. Participants allocated to the self-help condition received weekly modules delivered online via email delivered book chapters and handouts over an 8-week period. Significant treatment gains were observed in the face-to-face condition, with large reductions in CM (d = 2.11), PS (d = 1.77) and psychopathology (d = 1.16), and improvements in self-esteem (d = 1.16), maintained at 6-month follow-up. There were significant reductions in perfectionism in the pure self-help intervention (CM, d = 0.71; PS, d = .74) however this was not observed for measures of psychopathology. There was a reduction in participants meeting diagnoses from 54% at pre-treatment to 18% at follow-up and participant perfectionism, as measured by CM, improved clinically from pre-treatment to post-treatment with the face-to-face condition being significantly superior to self-help. The study was the largest RCT to assess the efficacy of CBT for clinical perfectionism. The findings provide a rationale for delivering the intervention across diagnoses in an individual format. However, further research in regards to pure self-help is required. Despite the author’s predictions, there was no significant effect on symptoms of eating disorders for either of the conditions. Further trials are needed to look at effectiveness of self-help in a mixed clinical sample including eating disorders. The findings of these studies, that treating perfectionism across a range of disorders not only reduces perfectionism but also the symptoms of different psychopathologies, provide support for the theory that perfectionism is a transdiagnostic process (Egan, Wade, et al., 2011).
Chapter 4: Study 1 - Reliability and Validity of the Clinical Perfectionism Questionnaire in a Mixed Clinical Sample

4.1. Overview

Perfectionism is a transdiagnostic construct with evidence as a predisposing and maintaining factor for many clinical disorders, including anxiety, mood, obsessive-compulsive and eating disorders (Egan, Wade, et al., 2011). One of the unresolved difficulties in the perfectionism literature has been how to best conceptualize the construct of perfectionism in the context of psychopathology (Shafran et al., 2002). There has also been growing recognition that some aspects of perfectionism can be adaptive, fostering productivity and excellence, while other types relate to maladaptive outcomes, including psychopathology, or both (Klibert et al., 2005; Stoeber & Otto, 2006).

Perfectionism theorists largely agree that perfectionism is multidimensional (Frost et al., 1990; Hewitt & Flett, 1991). The earliest and most widely used measures of perfectionism are the FMPS and HMPS. Some theorists argue that subscales of the FMPS and HMPS do not measure the construct of perfectionism, but rather they measure factors that are highly associated with perfectionism (Shafran et al., 2003; Shafran et al., 2002). Shafran et al. (2002) state that only self-oriented perfectionism, PS and CM subscales come close to measuring perfectionism. Shafran and Mansell (2001) highlight the need for clarification of the construct and the development of a measure of that construct, rather than the construct being defined by pre-existing measures.

Shafran and colleagues (2002) define clinical perfectionism as “…the overdependence of self-evaluation on the determined pursuit of personally demanding, self-imposed, standards in at least one highly salient domain, despite adverse consequences” (p. 778). In response, Fairburn et al. (2003a) developed the CPQ to measure the construct defined by Shafran and colleagues (2002). The 12-item CPQ was designed to measure the components of clinical perfectionism. However, there have only been a small number of studies that have examined the factor structure, reliability and validity of the CPQ (Chang & Sanna, 2012; Dickie et al., 2012; Egan et al., 2016; Steele et al., 2011; Stoeber & Damian, 2014). Steele and colleagues (2011) and Egan et al. (2016) identified a need for future research to
assess the validity of the CPQ in a mixed clinical sample, including those with a diagnosis of depressive, anxiety, eating, and obsessive-compulsive disorders.

4.2. Rationale and Aims

Empirically validated measurement instruments are required to identify potential perpetuating factors, such as clinical perfectionism, across disorders. Previous instruments that have been used to evaluate treatment outcome, such as the FMPS and HMPS, are not a direct measure of the construct of clinical perfectionism defined by Shafran et al. (2002). Furthermore, the PE and PC subscales included in these measures assess aspects of perfectionism that are not deemed amenable to change (Ashbaugh et al., 2007; Cox & Enns, 2003; Rice & Aldea, 2006). This raises questions over the use of the FMPS total score in measuring treatment outcome. Fairburn, Cooper and Shafran (2003a) developed the CPQ to measure the construct defined by Shafran et al. (2002), however few studies have assessed the psychometric properties of the scale. Of the available literature the reliability and validity of the CPQ has only been assessed using university samples. The findings of Chang and Sanna (2012), Dickie et al. (2012), and Stoeber and Damian (2014) not only provide support for the validity of the CPQ in a university sample, but also provide preliminary evidence to suggest that the CPQ consists of two factors, EC and PS, however these findings can only be generalised to a university population. Further exploratory analysis is required using clinical samples.

The only studies to date that have assessed the CPQ in a clinical sample have used female participants with an eating disorder and found that the measure consists of two factors (Egan et al., 2016; Steele et al., 2011). Therefore, it is crucial to validate the psychometric properties of the two factors of CPQ in a mixed clinical sample. This study will be the first of which the author is aware to evaluate the psychometric properties of the two factors of the CPQ, established by Egan and colleagues (2016), in a clinical sample with anxiety disorders, obsessive compulsive disorders, depressive disorders and eating disorders.

The primary aim of Study 1 is to assess the reliability and validity of the two factors of the CPQ, identified by Egan et al. (2016), in a mixed clinical sample as clinical perfectionism is proposed to be a maintaining mechanism across a range of psychopathology (Egan, Wade, et al., 2011). The convergent validity of the CPQ will be established by assessing the relationships of the two factors of the CPQ with gold standard measures of clinically relevant perfectionism, EC and PS subscales of...
the FMPS and the self-critical perfectionism subscale of the DAS. Concurrent validity will be established by comparing the two factors with a measure of dichotomous thinking, a construct highly related to clinical perfectionism and included as a factor in the cognitive behavioural maintenance model of clinical perfectionism (Shafran et al., 2002). This will be the first validation study of the CPQ in mixed clinical sample. Confirmatory Factor Analysis of Egan et al. (2016) two factors were beyond the score of the current study due to sample size limitations. This will be a requirement for future research however.

### 4.3. Hypotheses

It was hypothesised that:

**H1.** There will be a significant strong positive correlation, with \( r > .50 \), between the Factor 1 and Factor 2 of the CPQ and the EC and PS subscales of the FMPS and the DAS-SC.

**H2.** There will be a significant moderate positive correlation, with \( r > .30 \), between the Factor 1 and Factor 2 of the CPQ and a measure of dichotomous thinking, namely the Dichotomous Thinking in Eating Disorders Scale-general subscale (DTEDS-G; Bryne, Allen, Dove, Watt, & Nathan, 2008).

**H3.** There will be an acceptable level of internal consistency, where Cronbach’s alpha exceeds .70 for of the two factors of the CPQ.

### 4.4. Method

#### 4.4.1. Participants

The sample consisted of 32 adults (75% female) with mixed DSM-IV psychological disorders participating in baseline measurement for an RCT assessing the efficacy of cognitive behaviour therapy for clinical perfectionism (Chapter 5). Ages ranged from 19 to 57 (\( M = 34.54, SD = 9.71 \)) years. As seen in Table 1 the majority were employed in full-time work (53%), married (41%) and presenting with a primary anxiety disorder diagnosis (72%). The majority of participants presented with comorbidity; = two diagnoses (41%), = three (28%), and \( \geq \) four (6%).

The inclusion criteria for the RCT and hence present study were a stable (3-month) medication regimen if receiving psychotropic medication, lack of concurrent psychotherapy and an elevated score (\( \geq 22 \)) on the CM subscale of the FMPS. A score of \( \geq 22 \) is considered in the clinical range and was derived by computing the mean scores across mixed clinical samples (Shafran & Mansell, 2001). This methodology has been applied in subsequent studies (Egan & Hine, 2008). Inclusion...
criteria for the current study was also a DSM-IV mood, anxiety, or eating disorder diagnosis, however, this was not required for the RCT. Exclusion criteria were high suicide risk, current psychosis, and alcohol/substance dependence ($n = 15$).

Table 1

*Demographic and Diagnostic Characteristics of the Mixed Clinical Sample ($N = 32$)*

<table>
<thead>
<tr>
<th></th>
<th>$n$</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>24</td>
<td>75</td>
</tr>
<tr>
<td>Male</td>
<td>8</td>
<td>25</td>
</tr>
<tr>
<td><strong>Employment status</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Full-time work</td>
<td>17</td>
<td>53</td>
</tr>
<tr>
<td>Student</td>
<td>11</td>
<td>34</td>
</tr>
<tr>
<td>Part-time work</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>No paid work</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td><strong>Relationship</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>13</td>
<td>41</td>
</tr>
<tr>
<td>Single</td>
<td>10</td>
<td>31</td>
</tr>
<tr>
<td>Defacto</td>
<td>9</td>
<td>28</td>
</tr>
<tr>
<td><strong>Primary DSM-IV Diagnosis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Generalized Anxiety Disorder</td>
<td>11</td>
<td>34.4</td>
</tr>
<tr>
<td>Social Phobia</td>
<td>9</td>
<td>28.1</td>
</tr>
<tr>
<td>Major Depression</td>
<td>5</td>
<td>15.6</td>
</tr>
<tr>
<td>Eating Disorder Not Otherwise Specified</td>
<td>2</td>
<td>6.3</td>
</tr>
<tr>
<td>Panic Disorder with and without Agoraphobia</td>
<td>2</td>
<td>6.3</td>
</tr>
<tr>
<td>Bulimia Nervosa</td>
<td>1</td>
<td>3.1</td>
</tr>
<tr>
<td>Dysthymia</td>
<td>1</td>
<td>3.1</td>
</tr>
<tr>
<td>Obsessive-Compulsive Disorder</td>
<td>1</td>
<td>3.1</td>
</tr>
</tbody>
</table>
4.4.2. Measures

4.4.2.1. Mini International Neuropsychiatric Interview-Screen (MINI-Screen; Sheehan & Lecrubier, 2006).

The 21-item Mini International Neuropsychiatric Interview-Screen (MINI-Screen; Sheehan & Lecrubier, 2006) was administered via the telephone to screen for psychopathology. The MINI-Screen assesses initial signs of DSM-IV psychopathology. The participant answers either yes or no to experiencing the symptom. If the participant endorsed a MINI-Screen item, the corresponding module of the ADIS-IV and MINI were administered at the Baseline assessment to determine a DSM-IV diagnosis.

4.4.2.2. Mini International Neuropsychiatric Interview (MINI; Sheehan & Lecrubier, 2009).

The Mini International Neuropsychiatric Interview (MINI; Sheehan & Lecrubier, 2009) is a brief structured interview that assesses 16 DSM-IV disorders. Only four modules of the MINI were administered at the telephone screen to assess the participant’s current suicide risk, symptoms of psychosis and alcohol and substance dependence. Participants respond to questions using a yes/no format (Sheehan & Lecrubier, 2009). Sheehan et al. (1997) found the MINI to have excellent reliability and validity, and stated that it is a useful tool to screen for Axis I disorders in clinical trials. If the participants endorsed the items on the MINI-Screen, they were administered the AN and BN modules of the MINI at the clinical interview.

4.4.2.3. Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; Brown et al., 1994).

The ADIS-IV was administered in the current study to assess diagnostic criteria for each anxiety and depressive disorder, and diagnoses that frequently co-occur with these disorders. The ADIS-IV is a brief version of the ADIS-IV lifetime version by Di Nardo, Brown, and Barlow (1994) and is commonly used to assess current symptomatology across treatment time points (Allen et al., 2008). The ADIS-IV requires the participant to give a rating of the severity and interference of their symptoms using the Hamilton rating scales (Grisham, Brown, & Campbell, 2004). When a client endorsed criteria for more than one diagnosis, Hamilton ratings scales were used to determine the primary diagnosis and comorbid presentations. The
ADIS-IV is reported to have strong reliability and validity (Grisham et al., 2004). Brown, Di Nardo, Lehman, and Campbell (2001) observed good to excellent inter-rater reliability of the ADIS-IV lifetime version across diagnostic categories ($k = .67$ to .86), with the exception of dysthymia ($k = .22$). Furthermore, the ADIS-IV lifetime version is significantly correlated with disorder specific self-report clinical tools for GAD (Gordon & Heimberg, 2011).

4.4.2.4. Clinical Perfectionism Questionnaire (CPQ).

The 12-item CPQ was administered to the participants to assess its reliability and construct validity. The CPQ was developed by Fairburn et al. (2003a) to measure clinical perfectionism over the previous month. Using a 4-point Likert scale (1 = not at all to 4 = all of the time), scores can range from 12 to 48 with a high score indicating a higher level of clinical perfectionism. Prior to summing the items, Items 2 and 8 were reverse scored. There are several studies that have evaluated the CPQ (Chang & Sanna, 2012; Dickie et al., 2012; Egan et al., 2016), however, the CPQ has never been evaluated in a mixed clinical sample. The two factors of the CPQ identified by Egan et al. (2016) will be used in the present study. Factor 1 consists of items 1, 3, 6, 7, 8, 9, 10, and 11. Factor 2 contains items 2, 4, 5, 12.

4.4.2.5. Frost Multidimensional Perfectionism Scale (FMPS; Frost et al., 1990).

The 35-item FMPS (reviewed in Chapter 1) is divided into six subscales measuring different dimensions of perfectionism: concern over mistakes, personal standards, parental expectations, parental criticism, doubts about actions and organisation. To assess the construct validity of the CPQ previous studies have used the PS subscale and the EC subscale, that is, the sum of CM and DA (Dickie et al., 2012; Egan et al., 2016; Steele et al., 2011). Therefore, to remain consistent with previous research the 13-item EC subscale, and 7-item PS subscale of the FMPS was used in the current study to assess convergent validity. Using a 5-point Likert scale (1 = strongly disagree, to 5 = strongly agree), the participants rate their agreement with the corresponding statement. Subscale scores are derived by summing the item scores within each subscale, with a higher score indicating higher levels of perfectionism. Psychometric properties of the FMPS have been reported in Chapter 1 and will not be repeated here. In the current study, the Cronbach’s alpha for the EC subscale was acceptable ($\alpha = .86$). As the PS subscales consisted of less than 10 items, a Cronbach’s alpha of approximately .6 is deemed acceptable (Loewenthal,
Therefore, the internal consistency of the PS subscale \( (\alpha = .58) \) was adequate for the current study however was lower than other studies (Frost et al., 1990).

**4.4.2.6. The Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978).**

The self-criticism subscale of the DAS was used to measure self-critical perfectionism and was administered in the current study to assess construct validity. The DAS consists of 40 items, with 15 of them loading on the DAS-SC subscale. Participants rate their self-criticism using a 7-point Likert scale. The items are summed to get a total DAS-SC score. Higher scores indicate higher levels of self-criticism. The psychometric properties of the DAS-SC have been reviewed in Chapter 1 and will not be repeated here. The internal consistency of the DAS-SC for the current study was acceptable \( (\alpha = .90) \).

**4.4.2.7. The Dichotomous Thinking in Eating Disorders Scale (DTEDS; Bryne et al., 2008).**

The general subscale of the DTEDS was designed to measure change in the individual’s dichotomous thinking and used in the current study to determine concurrent validity. The DTEDS is an updated version the Dichotomous Thinking Scale (Bryne, Cooper, & Fairburn, 2004). The DTEDS is an 11-item self-report measure that uses a 4-point Likert scale response format. Confirmatory Factor Analysis has yielded a two-factor solution consisting of four items relating to food, eating, weight and dieting and seven items relating to a more general measure of dichotomous thinking (Byrne et al., 2008). For the purpose of this study the seven general items were used. These items were used in other studies that aimed to measure general areas of dichotomous thinking using the original Dichotomous Thinking Scale (Egan et al., 2007). The DTEDS-G significantly correlated with the DASS-21 depression scores \( (r = .52; \text{Byrne et al., 2008}) \) and had acceptable internal consistency \( (\alpha = .78) \) for the current study.

**4.4.3. Procedure**

This study was granted approval from the Curtin University Human Research Ethics Committee (HR 120/2010) and participants provided written informed consent. The participants were recruited from a mail out to clinical psychologists, mental health practitioners and general practitioners in the Perth metropolitan area. Participants recruited from mental health professionals were temporarily discharged from concurrent therapy for the time of the study period i.e. intake – follow-up, to ensure the treatment effects could be attributed to CBT for clinical perfectionism.
Advertisements were also distributed to Perth metropolitan universities and to members of a triathlon club. The participants were provided with a participant starters package (see Appendix A) containing an information sheet, consent form and a screening questionnaire; namely the CM subscale of FMPS. Upon the return of a signed consent form and a score of $\geq 22$ on the CM subscale of the FMPS, participants were telephoned and screened for eligibility using the MINI-Screen. Four MINI modules; Suicidality, Alcohol Dependence/Abuse, Substance Dependence/Abuse (Non-Alcohol), and Psychotic Disorders and Mood Disorder with Psychotic Features, were administered at the telephone screen to assess exclusion criteria. A score of $\geq 22$ on the CM subscale of the FMPS is considered in the clinical range and was derived by computing the mean scores across mixed clinical samples (Shafran & Mansell, 2001) and has been used in subsequent studies (Egan & Hine, 2008). Participants attended a clinical interview, conducted by a Clinical Psychology Masters trainee. If the participant endorsed a MINI-Screen item on the telephone, the corresponding module of the ADIS-IV or MINI was administered at a clinical interview to determine diagnosis. The AN and BN modules of the MINI were administered at the clinical interview if the participants endorsed the corresponding items on the MINI-Screen on the telephone. The Clinical Psychology Masters trainee administering the assessment instruments were trained in the administration of the MINI and ADIS-IV, and supervised by experienced Clinical Psychologists via videotaped supervision of their clinical interview sessions to arrive at consensus in the diagnoses. Participants completed the CPQ, EC and PS subscales of the FMPS, DAS-SC and DTEDS-G at the time of the clinical interview.

4.5. Results

4.5.1. Data Screening

Prior to analysis, the data were screened as per Tabachnick and Fidell (2007) recommendations. Upon visual inspect of the frequencies no out of range values were observed. Therefore there were no obvious errors in data entry. Additionally, missing data, univariate and multivariate outliers, normality, linearity, multicollinearity and singularity were assessed.

4.5.1.1. Missing data

Missing values analysis revealed some missing items scores. Expectation maximisation was used to identify and replace the missing data. Expectation
maximisation was used because the missing data appeared to be occurring in a random pattern, as indicated by the non-significant Little’s Missing Completely At Random (MCAR) statistic (Tabachnick & Fidell, 2007). Missing values were replaced for items on the CPQ (Items 3 and 11) and the EC and PS subscales of the FMPS (Items 4, 12, 16, 19, 24, 28, 32, 33). There were no missing item values for the DAS-SC or DTEDS-G.

4.5.1.2. Outliers

Boxplots were used to assess univariate outliers for all of the variables. Visual inspection of the box plots revealed outliers in the CPQ (case 26), and the DAS-SC subscale score (case 6). Tabachnick and Fidell (2007) do not consider an outlier to be a threat if it is within 3.29 standard deviations above or below the mean score and therefore does not need to be changed. As both of the outliers were within 3.29 standard deviations from the mean they were not changed.

Multivariate outliers were assessed for the 12 CPQ items, and the CPQ, EC and PS subscales of the FMPS, DAS-SC, and DTEDS-G. Two separate analyses were conducted. Allen and Bennett (2008) state that “…a maximum Mahalanobis distance larger than the critical chi-square ($\chi^2$) value for $df = k$ at $\alpha = .001$ indicates the presence of one or more multivariate outliers” (p. 182). The critical ($\chi^2$) value for $df = 12$ at $\alpha = .001$ is 32.909. As the Mahalanobis Distance is 18.560, multivariate outliers among the items of the CPQ were not considered a threat. Additionally, the critical ($\chi^2$) value for $df = 6$ at $\alpha = .001$ is 22.458. As the Mahalanobis Distance 13.042, multivariate outliers among the variables in the correlational analysis were not considered a threat.

4.5.2. Assumption Testing for Correlational Analysis

The assumption of normality was assessed for each of the variables used to determine the validity of the CPQ. The Shapiro-Wilk statistic revealed that normality was not violated for any of the variables. A visual inspection of the scatterplots of the CPQ against the variables in the analysis confirmed that the relationship between these variables were reasonably linear.

4.5.3. Hypothesis Testing

A priori power analysis determined that a sample size of at least 29 would be required to reject the null hypothesis under the conditions of a large correlation ($r = .5$), power of .8, alpha of .05, and two-tailed test. Pearson’s correlation coefficients were used to assess the convergent and concurrent validity of the two factors of the
CPQ. Cohen’s (1988) criteria, >.50 strong, .30 to .49 moderate, and .10 to .29 weak, was used to evaluate the size of the correlations. The intercorrelations, means, standard deviations and reliabilities of the study measures are reported in Table 2.

4.5.3.1. Convergent validity.

Convergent validity was examined by observing the correlations of the Factor 1 and Factor 2 of the CPQ with three commonly used measures of perfectionism, namely the EC and the PS subscales of the FMPS, and the self-critical perfectionism subscale of the DAS. To account for multiple testing, a Bonferroni-adjusted alpha for the three perfectionism measures was applied and calculated to be .017 (.05/ three perfectionism measures)

A significant moderate positive correlation was observed between Factor 1 and the PS subscale of the FMPS ($r = .464, p = .008$). There was no significant correlation between Factor 1 and EC ($r = .379, p = .033$) subscale of the FMPS and DAS-SC ($r = .339, p = .058$). No significant correlations were observed between Factor 2 of the CPQ and any of the perfectionism measures (EC, $r = .253, p = .163$; PS, $r = .133, p = .467$; DAS-SC, $r = .361, p = .043$). Partial evidence of convergent validity was established (H1), as strong positive correlation between Factor 1 and PS measures were observed.

4.5.3.2. Concurrent validity.

The DTEDS-G were used to assess the concurrent validity of the CPQ, as dichotomous thinking have been proposed to maintain the cycle of clinical perfectionism (Egan, Wade, et al., 2011). The conventional alpha level of .05 was retained for the current analysis, as there was no multiple testing. No significant relationship was observed between Factor 1 of the CPQ and DTEDS-G ($r = .171, p = .349$). However, there was a moderate positive correlation observed between Factor 2 and DTEDS-G ($r = .356, p = .046$). The results provide partial support for the hypothesis (H2) that Factor 2 of the CPQ moderately correlated with a construct highly related to perfectionism; namely, dichotomous thinking.
Table 2
Summary of Intercorrelations, Means, Standard Deviations, and Reliabilities for Study Measures (N = 32).

<table>
<thead>
<tr>
<th></th>
<th>CPQ</th>
<th>Factor 1</th>
<th>Factor 2</th>
<th>EC</th>
<th>PS</th>
<th>DAS-SC</th>
<th>DTEDS-G</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPQ</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factor 1</td>
<td>.879**</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factor 2</td>
<td>.584**</td>
<td>.126</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EC</td>
<td>.431*</td>
<td>.379*</td>
<td>.253</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PS</td>
<td>.443*</td>
<td>.464**</td>
<td>.133</td>
<td>.426*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SC</td>
<td>.451**</td>
<td>.339</td>
<td>.361*</td>
<td>.758**</td>
<td>.267</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DT</td>
<td>.311</td>
<td>.171</td>
<td>.356*</td>
<td>.241</td>
<td>.003</td>
<td>.538**</td>
<td>-</td>
</tr>
<tr>
<td>M</td>
<td>30.24</td>
<td>19.61</td>
<td>10.63</td>
<td>45.44</td>
<td>28.99</td>
<td>62.72</td>
<td>2.54</td>
</tr>
<tr>
<td>SD</td>
<td>4.69</td>
<td>3.84</td>
<td>2.25</td>
<td>7.58</td>
<td>3.09</td>
<td>14.11</td>
<td>.65</td>
</tr>
<tr>
<td>α</td>
<td>.68</td>
<td>.71</td>
<td>.63</td>
<td>.86</td>
<td>.58</td>
<td>.90</td>
<td>.78</td>
</tr>
</tbody>
</table>

Note. CPQ = Clinical Perfectionism Questionnaire, EC = Evaluative Concerns subscale of the FMPS, PS = Personal Standards subscale of the FMPS, DAS-SC = Self Criticism subscale of the DAS, DTEDS-G = General subscale of the DTEDS. *p < .05 **p < .01.

4.6. Discussion

The aim of the study was to assess the reliability and validity of the CPQ in a mixed clinical sample. Convergent and concurrent validity with related measures was partially established. Internal consistency was acceptable for Factor 1 but not for Factor 2. These findings are lower than reliability estimates in the literature for the total CPQ score in community (α = .72 - .83) and eating disorder (α = .82 - .83) samples (Chang & Sanna, 2012; Egan et al., 2016; Steele et al., 2011).

Convergent validity was determined by assessing the correlation between the two factors of the CPQ and the EC and PS subscales of the FMPS and the self-critical perfectionism as measured by the DAS-SC. Factor 1 results did not support
the hypotheses that there is a strong correlation between EC, PS or self-critical perfectionism. Moderate correlations were observed between Factor 1 and PS. This is consistent with previous literature (Dickie et al., 2012; Egan et al., 2016). Di Bartolo, Frost, Chang, LaSota, and Grills (2004) state that “… it is not high personal standards per se that are related to psychopathology, but rather that high personal standards are associated with psychopathology only when meeting those standards is used to define self-worth” (p. 243). Previous literature has reported that the combination of EC and PS is maladaptive in clinical samples (Bardone-Cone et al., 2007; Egan, Wade, et al., 2011; Steele et al., 2011). There were no significant relationships observed between the three measures of perfectionism and Factor 2 of the CPQ. These non-significant findings are inconsistent with Egan et al.’s (2016) study. An explanation for this could be due to the small sample size ($N = 32$) leading to the analysis being underpowered. At the revised alpha level we cannot conclude that the small - moderate effects observed with the non-significant findings is an association beyond chance.

Concurrent validity was evaluated with a measure of theoretically related construct proposed to maintain the cycle of clinical perfectionism; namely, dichotomous thinking. The results partially supported the proposed hypotheses; with Factor 2 of the CPQ significantly moderately correlated with dichotomous thinking. There was no significant association with Factor 1. The significant finding is consistent with previous research which has found that dichotomous thinking is related to perfectionism in mixed clinical groups and eating disorders (Egan et al., 2007; Egan et al., 2016; Lethbridge et al., 2011; Zucker et al., 2011).

A significant strength of the current study is that is the first to which the author is aware to assess the psychometric properties of the two factors of the CPQ in a mixed clinical sample. Previous studies that have assessed the psychometric properties of the CPQ have been conducted either using community or female eating disorder samples (Chang & Sanna, 2012; Dickie et al., 2012; Egan et al., 2016; Steele et al., 2011). A limitation of the current study is that the sample size was small for psychometric evaluation, therefore the findings should be interpreted with caution.

The results of the study provide partial support for the two factors of the CPQ. Further research is needed with larger mixed clinical samples as the findings of this study are in contrast to previous research which has found good psychometric
properties for the CPQ (Chang & Sanna, 2012; Dickie et al., 2012; Egan et al., 2016; Steele et al., 2011). Future research should examine if the CPQ has better utility in evaluating change in clinical perfectionism than existing measures of perfectionism such as the FMPS. Having a valid measure of clinical perfectionism for use in a clinical population is very important as it has been demonstrated to be a transdiagnostic factor that maintains a range of psychopathologies (Egan, Wade, et al., 2011). In summary, the CPQ appears to be a promising measure of the construct of clinical perfectionism. As there was partial support for the use of the CPQ, the measure will be used in the following study (Chapter 5) to assess change in perfectionism across the three time points in the RCT. Additional measures will also be used to assess change i.e. FMPS, and DAS-SC.
Chapter 5: Study 2. A Randomised Controlled Trial of Cognitive Behavioural Guided Self-Help Therapy for Clinical Perfectionism

5.1. Overview

The following chapter report the findings from a RCT assessing the efficacy of a guided self-help version of CBT for clinical perfectionism. This study will assess change in measures of perfectionism and psychopathology. Participants presenting with a range of DSM-IV psychopathology ($n = 32$) as well as participants not meeting criteria for a primary DSM-IV diagnosis ($n = 8$) will be included in the analysis and will be described as an elevated perfectionism sample. Diagnostic and clinically significant disorder-specific changes in the individuals presenting with DSM-IV diagnoses ($n = 32$) after they have received the intervention will also be assessed. In the second series of analyses assessing diagnostic change, the sample will be described as a mixed clinical sample.

As mentioned previously, there are a number of studies providing evidence that perfectionism interferes with treatment outcome (Blatt et al., 1998; Chik et al., 2008; Kyrios, Hordern, & Fassnacht, 2015). Harvey et al. (2004) state that by adopting a transdiagnostic perspective a single treatment can be developed for a variety of disorders by targeting a core maintaining mechanism across those disorders. This supports the rationale that targeting perfectionism can help reduce a significant risk and maintaining factor across a range of disorders (Egan, Wade, et al., 2011). This study proposes that a transdiagnostic guided self-help cognitive-behavioural approach to targeting perfectionism will not only reduce symptoms of perfectionism, but also symptoms of commonly associated psychopathology such as depression, anxiety and stress, and improve an individual’s quality of life. By removing the maintaining mechanism of perfectionism across a number of psychological disorders these disorders will be reduced, even though the treatment does not directly target the symptoms of these disorders. This ultimately may enhance treatment outcome across a number of psychological disorders at the same time. This transdiagnostic approach may potentially reduce the symptoms of individuals who did not respond to previous treatments as the underlying cause and maintaining mechanism is being addressed.
As discussed in Chapter 3, Guided self-help has been referred to as a low intensity intervention (IAPT, 2010) and considered secondary to face-to-face therapy for the treatment of individuals with complex clinical presentations of high severity of symptoms and comorbidity. As such, the National Institute for Health and Care (NICE, 2013) have recommended that self-help be administered to individuals with low-moderate symptomatology. Despite this, emerging literature suggest that guided self-help therapies produce similar treatment outcomes to that of face-to-face therapy (Cuijpers et al., 2010) and can result in clinically meaningful change in symptoms and diagnostic outcomes. Several RCTs assessing the efficacy of guided self-help interventions have reported moderate to large treatment effects (Farrand & Woodford, 2013) and diagnostic changes in anxiety, depressive, and eating disorder clinical samples (Haug, Nordgreen, Öst, & Havik, 2012; Traviss et al., 2011; Vernmark et al., 2010). Furthermore, there is an increasing evidence base for the efficacy of CBT for perfectionism in producing clinically meaningful changes and diagnostic shifts.

5.2. Rationale and Aims

Perfectionism is a maintaining mechanism of psychopathology and has been proposed to significantly interfere with disorder specific treatment, resulting in poor treatment outcomes and non-engagement (Blatt et al., 1998; Chik et al., 2008; Jacobs et al., 2009; Lundh & Öst, 2001). Psychological disorders have been listed as one of the top five non-communicable diseases with a global cost reported to be $2.5 trillion in 2010. This figure is expected to rise to over $6 trillion by 2030 (Bloom et al., 2011). It is therefore imperative to evaluate treatments that target underlying factors, such as perfectionism, that could be impacting current evidence based treatments. Furthermore, there is increasing evidence to suggest that transdiagnostic treatments can alleviate disorder specific symptomatology such as depression and anxiety (McEvoy et al., 2009). Mansell et al. (2009) argue that a transdiagnostic approach to treatment will be more efficient at reducing symptoms of psychopathology than treating each disorder separately. Disorder specific interventions, are currently the most evidence based recommendation and according to the NICE guidelines, CBT is currently the most recommended treatment across several diagnostic categories i.e., GAD (Cuijpers et al., 2014), or CBT-E for adult eating disorders (Fairburn et al., 2003b). It is likely that cognitive behavioural techniques such as behavioural
experiments, cognitive restructuring, and thought diaries, can be applied across a range of disorders to address underlying psychopathology that is maintaining disorder specific symptomatology. Whilst there is preliminary evidence for transdiagnostic treatments (McEvoy et al., 2009), there is a gap in the literature as no study to date has compared transdiagnostic and disorder specific interventions in a sample presenting with a range of diagnoses. There are however, few evidence-based protocols to guide clinicians when clients present with comorbid diagnoses (Craske et al., 2007; Egan et al., 2012). This is concerning considering that approximately 38% of individuals with psychological disorders in Australia meet criteria for two or more diagnoses at one time (from Australian Bureau of Statistics 2007). Therefore, we need to be able to provide evidence for treatments that address comorbidity, such as CBT for clinical perfectionism (Egan et al., 2012).

Due to the high demand for clinical services there is a need to find evidence-based alternatives to face-to-face therapies. Previous research has found support for the use of guided self-help interventions in community samples with sub-clinical symptomatology. However, there are mixed findings in regards to the efficacy of guided self-help interventions in clinical samples (Coull & Morris, 2011; Cuijpers et al., 2010). Further research is required before guided self-help interventions can be deemed efficacious in reducing symptomatology across clinical disorders. Additionally, Jacobson and Truax (1991) state “…conventional statistical comparisons between groups tell us very little about the efficacy of psychotherapy” (p. 12). To further explore the impact of the treatment on the individual clinically significant outcomes need to be reported. Clinicians implementing evidence-based treatments also want to know about the impact the treatment has on the individuals.

The current study will contribute to the increasing literature providing support for guided self-help interventions to be used in complex clinical samples to not only shift symptoms, but also clinically significant changes. Previous research assessing the self-help perfectionism interventions have used Antony and Swinson (1998) “When Perfect isn’t Good Enough” (Pleva & Wade, 2007; Steele & Wade, 2008). This will be the first study, to which the author is aware to deliver CBT for clinical perfectionism based on “Overcoming Perfectionism” (Shafran et al., 2010) in a guided self-help format. This is important as Shafran et al. (2010) intervention is based on the theoretical maintenance model of clinical perfectionism (Shafran et al., 2002) unlike Antony and Swinson (1998). “Overcoming Perfectionism” has been
delivered in a pure self-help form; with results showing the intervention does reduce perfectionism however the pure self-help version was not effective in comparison to the face to face version of treatment which reduced perfectionism but also psychological symptoms (Egan, van Noort, et al., 2014). Further, it has been argued that pure self-help interventions are not likely to be as powerful as a guided self-help version (Gyani et al., 2011).

Treatment studies for anxiety disorders (Hofmann, Wu, & Boettcher, 2014), eating disorders (Jenkins, Hoste, Meyer, & Blissett, 2011; Watson, Allen, Fursland, Byrne, & Nathan, 2012), and depressive disorders (Swan et al., 2009) have included quality of life measures in assessing treatment outcome in addition to measures of psychopathology. Few treatment trials of perfectionism have used quality of life as a treatment outcome. It is important for treatment studies to include quality of life measures as Egan, Wade, et al. (2011) report that an outcome of CBT for clinical perfectionism is an improvement in quality of life. The authors highlight that this is an indirect effect through the reduction of high standards. The small amount of studies evaluating CBT for clinical perfectionism has used quality of life as a measure of treatment outcome (Egan, van Noort, et al., 2014; Handley et al., 2015). Studies that have included the measure found that quality of life increases from pre-treatment to post-treatment for those receiving CBT for clinical perfectionism intervention relative to that of controls (Egan, van Noort, et al., 2014; Handley et al., 2015). Participants not meeting full DSM criteria will also be included in the study as perfectionism is not exclusively associated with clinical samples. Perfectionism can significantly impact general community samples and lead to difficulties in relationships (Habke, Hewitt, & Flett, 1999), increase blood pressure (Albert, Rice, & Caffee, 2014), result in higher rates of burnout within the workforce (Phillip, Egan, & Kane, 2012) and greater severity of symptoms for those that suffer chronic fatigue syndrome (Kempke et al., 2011). As perfectionism is a risk factor for the development of psychopathology, it is also likely that the treatment could serve as a preventative intervention, although this cannot be assessed in the current study.

The main aim of the current study is to compare change in perfectionism and related constructs in individuals that have received guided self-help CBT for clinical perfectionism, to an 8-week waitlist-control group. A mechanisms proposed to maintain clinical perfectionism will be explored, dichotomous thinking. This construct is deemed important to the maintenance of clinical perfectionism and
modules of CBT for clinical perfectionism treatment are dedicated to overcoming the factor. Dichotomous thinking have been targeted in perfectionism treatments with an entire module dedicated to the construct, however no research to date has examined their role in the maintenance of clinical perfectionism proposed by Shafran et al. (2002) and later adapted by Shafran et al. (2010). Dichotomous thinking will be measured in the current study to explore this relationship and to assess impact on treatment. The study proposes to address a gap in the current perfectionism literature by conducting a RCT with a mixed diagnostic cohort consisting of anxiety disorders, depression, eating disorders, obsessive-compulsive disorders and a sub-clinical community sample. The primary aim of the study is to test whether treating clinical perfectionism reduces perfectionism and therefore the removal of a transdiagnostic maintaining mechanism across disorders, as evident by a reduction in clinical diagnoses and the presence of comorbidity. Furthermore, the study aims to examine if guided self-help is an effective form of treatment for clinical perfectionism. A secondary aim is to test whether a treatment that targets a maintaining mechanism of multiple disorders, clinical perfectionism, also results in the reduction of the severity of the symptoms of that disorder, whilst not targeting the disorder specifically. This will be evident by changes in measures of depression, anxiety, stress and quality of life. This study will contribute to the understanding of the construct of perfectionism and its impact on treatment.

A further aim of the current study is to examine whether a guided self-help CBT intervention for clinical perfectionism is effective at reducing DSM-IV diagnoses and producing clinically significant change in disorder specific symptoms. Specifically, this study aims to assess the diagnostic changes from pre-treatment to post-treatment for the treatment and control groups and endeavours to extended upon the findings of Egan, van Noort, et al. (2014) that CBT for clinical perfectionism does produce clinically meaningful change. Disorder specific symptomatic changes will also be assessed from pre-post treatment using disorder specific measures relevant to the individuals endorsed diagnosis or diagnoses. Given that perfectionism has been shown to be a predictive factor and maintaining mechanism of several disorders, it is to be likely that the current sample will present with complex clinical characteristics, such as the presence of more than one diagnosis. Therefore, percentages of comorbidity amongst the sample will be assessed to see if the
intervention is effective at reducing the number of participants meeting more than one DSM-IV diagnosis.

This study offers an alternate view to the way we currently treat depressive, anxiety, eating and obsessive-compulsive disorders, which is predominately disorder specific. Furthermore, the treatment will likely improve the individuals presenting with sub-clinical symptoms quality of life and general psychopathology by potentially removing a factor that could be a risk for the development of later psychopathology.

5.3. Hypotheses

5.3.1. Effects of the Treatment on Perfectionism and Related Constructs

H1. a) At post-treatment CBT for clinical perfectionism will be significantly superior to the control group in decreasing clinical perfectionism (self-help>wait-list).

b) The significant pre-post changes in clinical perfectionism will be maintained at 4-month follow-up.

c) The significant pre-post improvements in clinical perfectionism for individuals in the CBT for clinical perfectionism group will be reliable and clinically significant.

H2. a) At post-treatment CBT for clinical perfectionism will be significantly superior to the control group in decreasing concern of mistakes (self-help>wait-list).

b) The significant pre-post changes in concern of mistakes will be maintained at 4-month follow-up.

c) The significant pre-post improvements in concern of mistakes for individuals in the CBT for clinical perfectionism group will be reliable and clinically significant.

H3. a) At post-treatment CBT for clinical perfectionism will be significantly superior to the control group in decreasing personal standards (self-help>wait-list).

b) The significant pre-post changes in personal standards will be maintained at 4-month follow-up.

c) The significant pre-post improvements in personal standards for individuals in the CBT for clinical perfectionism group will be reliable and clinically significant.
H4. a) At post-treatment CBT for clinical perfectionism will be significantly superior to the control group in decreasing **self-critical perfectionism** (self-help>wait-list).

b) The significant pre-post changes in **self-critical perfectionism** will be maintained at 4-month follow-up.

c) The significant pre-post improvements in **self-criticism** for individuals in the CBT for clinical perfectionism group will be reliable and clinically significant.

H5. a) At post-treatment CBT for clinical perfectionism will be significantly superior to the control group in decreasing **dichotomous thinking** (self-help>wait-list).

b) The significant pre-post changes in **dichotomous thinking** will be maintained at 4-month follow-up.

c) The significant pre-post improvements in **dichotomous thinking** for individuals in the CBT for clinical perfectionism group will be reliable and clinically significant.

5.3.2. Effects of the Treatment on Psychopathology

H6. a) At post-treatment CBT for clinical perfectionism will be significantly superior to the control group in decreasing **symptoms of depression** (self-help>wait-list).

b) The significant pre-post changes in **depression** will be maintained at 4-month follow-up.

c) The significant pre-post improvements in **depression** for individuals in the CBT for clinical perfectionism group will be reliable and clinically significant.

H7. a) At post-treatment CBT for clinical perfectionism will be significantly superior to the control group in decreasing **symptoms of anxiety** (self-help>wait-list).

b) The significant pre-post changes in **anxiety** will be maintained at 4-month follow-up.

c) The significant pre-post improvements in **anxiety** for individuals in the CBT for clinical perfectionism group will be reliable and clinically significant.
H8. a) At post-treatment CBT for clinical perfectionism will be significantly superior to the control group in decreasing symptoms of stress (self-help>wait-list).

b) The significant pre-post changes in stress will be maintained at 4-month follow-up.

c) The significant pre-post improvements in stress for individuals in the CBT for clinical perfectionism group will be reliable and clinically significant.

H9. a) At post-treatment CBT for clinical perfectionism will be significantly superior to the control group in increasing quality of life (self-help>wait-list).

b) The significant pre-post changes in quality of life will be maintained at 4-month follow-up.

c) The significant pre-post improvements in quality of life for individuals in the CBT for clinical perfectionism group will be reliable and clinically significant.

5.3.4. Effect of the Treatment on DSM-IV Diagnoses

H10 a) At post-treatment, CBT for clinical perfectionism will be significantly superior to the control group in decreasing the number of participants meeting diagnostic criteria for a primary diagnosis of an anxiety disorder, depression, or eating disorder.

b) At post-treatment, CBT for clinical perfectionism will be significantly superior to the control group in decreasing the number of participants meeting diagnostic criteria for comorbid diagnoses of an anxiety disorder, depression, or eating disorder.

5.3.5. Effects of the Treatment on Reliable and Clinically Significant Change in Disorder-Specific Symptoms

H11. The pre-post improvement in disorder specific symptoms for individuals in the CBT for clinical perfectionism group will be reliable and clinically significant and superior to that of the control group.

5.4. Method

5.4.1. Participants

A total of 77 participants expressed interest in the study, after screening and baseline assessment 40 participants with elevated perfectionism were included in the
current study as seen in Figure 3.

**Figure 3.** Design of the RCT

The sample consisted mostly of females (70%) with ages ranging from 19 to 57 years ($M = 35.43, SD = 9.92$). The majority of the sample was married or in a defacto relationship (65%) and either in full or part-time employment (62.5%). The primary
diagnoses of the sample are reported in Table 3. It can be seen from this table that 80% of the sample met DSM-IV diagnostic criteria, with 55% \( (n = 22) \) of participants meeting the criteria for a primary anxiety disorder diagnosis, 15% \( (n = 6) \) for a depressive disorder, 7.5% \( (n = 3) \) for an eating disorder and 2.5% for an obsessive compulsive disorder. Seven participants presented with symptoms of depression and anxiety but did not meet current DSM-IV diagnostic criteria.

Table 3

*Primary Diagnoses in the Sample (\( N = 40 \))*

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of diagnoses</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Generalised Anxiety Disorder (GAD)</td>
<td>11</td>
<td>27.5%</td>
</tr>
<tr>
<td>Social Phobia</td>
<td>9</td>
<td>22.5%</td>
</tr>
<tr>
<td>Major Depression</td>
<td>5</td>
<td>12.5%</td>
</tr>
<tr>
<td>Eating Disorder Not Otherwise Specified (EDNOS)</td>
<td>2</td>
<td>5%</td>
</tr>
<tr>
<td>Panic Disorder with and without Agoraphobia</td>
<td>2</td>
<td>5%</td>
</tr>
<tr>
<td>Bulimia Nervosa</td>
<td>1</td>
<td>2.5%</td>
</tr>
<tr>
<td>Dysthymia</td>
<td>1</td>
<td>2.5%</td>
</tr>
<tr>
<td>Obsessive-Compulsive Disorder (OCD)</td>
<td>1</td>
<td>2.5%</td>
</tr>
<tr>
<td>No diagnosis</td>
<td>8</td>
<td>20%</td>
</tr>
</tbody>
</table>

**5.4.1.1. Recruitment and sampling method.**

Non-probability purposive sampling methods were used to recruit participants into the study. This method was ‘selected’ as individuals with elevated clinical perfectionism were needed. Advertisements (see Appendix B) were distributed to local psychologists, psychiatrists, general practitioners and public or private mental health practitioners who may have had waiting lists and could refer their clients to the study. The advertisements were also distributed to counselling services at four
local Universities and an email list from a triathlon club. Participants recruited from mental health professionals were not receiving concurrent therapy during the study. The participants referred from mental health professionals were discharged from the referring clinician or treatment put on hold for the study period. If an individual was to commence therapy throughout the trial period their data was excluded from subsequent time points. If this occurred throughout the intervention period the full 8-week intervention was still provided but subsequent data not included in analysis. The advertisements stated the selection criteria and provided a number of examples of elevated perfectionism. The study was also advertised throughout state and national online and print newspapers.

5.4.1.2. Inclusion criteria.

The participant needed to meet a number of criteria in order to be eligible for the present study:

1) Age 18 years of age or older.
2) Must have an elevated score (≥22) on the CM subscale of the FMPS*
3) An agreement not to undergo other psychological treatments through the course of the study, i.e., from the initial assessment to the 4-month follow up period.
4) If on antidepressant medication, must have been stabilised on this medication for at least three months prior to the baseline assessment.
5) If on antidepressant medication, an agreement not to alter/change the medication and the dosage for the duration of the study i.e. from the initial assessment to the 4-month follow up period.

* A score of ≥22 on the CM subscale of the FMPS is considered to be in the clinical range. This score was derived by computing the mean scores across mixed clinical samples (Shafran & Mansell, 2001) and has been used in subsequent studies (Egan & Hine, 2008).

5.4.1.3. Exclusion criteria.

A number of exclusion criteria were used to not only limit the influence of external factors i.e. co-occurring alternative treatment, on the treatment effect but to also safeguard participants who could have been in a high risk category, i.e. co-occurring psychosis, high risk suicidal ideation, high risk of malnutrition leading to cognitive compromise (BMI < 17.5), and allocated to the waitlist condition. Alcohol
and substance dependence was listed as an exclusion criterion to limit disruption to therapeutic engagement in the context of frequent alcohol/drug using behaviour.

1) DSM-IV diagnosis of schizophrenia/psychosis*.
2) Serious suicide ideation*.
3) Alcohol/Substance dependence**.
4) Anyone meeting the DSM-IV criteria for Anorexia Nervosa and/or a seriously low body weight (BMI < 17.5)***.
5) Anyone currently in psychological therapy.

* Suicidal ideation and symptoms of Schizophrenia/Psychosis were screened via telephone using the corresponding modules of the MINI.
** Alcohol/Substance dependence was screened via telephone using the MINI-Screen. If the participant answered yes to either item then the appropriate module of the MINI was administered to clarify diagnosis.
*** Self-reported height and weight were requested at the telephone screen to determine BMI.

5.4.2. Measures

5.4.2.1. Mini International Neuropsychiatric Interview-Screen (MINI-Screen; Sheehan & Lecrubier, 2006).

The MINI-Screen is a 21-item diagnostic tool to screen for DSM-IV psychopathology. The MINI-Screen was used to assess initial DSM-IV psychopathology via the telephone. The MINI Screen was described in Chapter 4.

5.4.2.2. Mini International Neuropsychiatric Interview (MINI; Sheehan & Lecrubier, 2009).

The MINI is a brief structured interview that assesses 16 DSM-IV disorders. The Eating Disorders module of the MINI was administered in the current study to assess DSM-IV diagnostic criteria. The AN or BN module was only administered if the participant endorsed eating disorder symptomatology on the MINI-Screen. The MINI was described in Chapter 4.

5.4.2.3. Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; Brown et al., 1994).

The ADIS-IV was used in the current study to assess diagnostic criteria for each anxiety disorder, depressive disorder, and diagnoses that frequently co-occur with anxiety and depressive disorders. The ADIS-IV and its psychometric properties were described in Chapter 4.
5.4.2.4. Clinical Perfectionism Questionnaire (CPQ; Fairburn, et al., 2003a).

The 12-item CPQ (Fairburn et al., 2003a) was evaluated in Chapter 4 and was used to measure clinical perfectionism in the current study. The psychometric properties of the CPQ have been explained in Chapter 1. Despite emerging literature suggesting that the CPQ contains two factors (Egan et al., 2016), the CPQ total will be used in the current studies align with other treatment studies assessing change in clinical perfectionism (Handley et al., 2015; Riley et al., 2007; Steele et al., 2013). Cronbach’s alpha of the CPQ was low (.68) therefore the results should be interpreted with this in mind. This was a lower alpha level than the .8 or greater found in other studies (Chang & Sanna, 2012; Egan et al., 2016; Steele et al., 2011).

5.4.2.5. Frost Multidimensional Perfectionism Scale (FMPS; Frost et al., 1990).

The FMPS consists of 35 items divided into six subscales measuring different dimensions of perfectionism: Concern over mistakes, personal standards, parental expectations, parental criticism, doubts about actions and organisation. The FMPS was described in Chapter 1. The internal consistency was acceptable for four of the subscales used to measure multidimensional perfectionism in the current study; CM ($\alpha = .84$), PE ($\alpha = .87$), PC ($\alpha = .89$), and DA ($\alpha = .77$). The internal consistency of the PS subscale ($\alpha = .67$) was considered inadequate by Tabachnick and Fidell (2007) recommendations. This is inconsistent with previous studies that have reported excellent reliability of the PS subscale (Frost et al., 1990). Loewenthal (2001) however concludes that for scales with less than 10 items, such as the 7-item PS subscale, a Cronbach’s alpha of .6 will generally suffice.

5.4.2.6. The Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978).

The self-criticism subscale of the DAS was used to measure self-critical perfectionism. The DAS consists of 40 items, with 15 of them loading on the DAS-SC subscale derived from factor analysis by Imber et al. (1990). The DAS-SC subscale was described in detail in Chapter 1. The internal consistency of the DAS-SC for the current study was acceptable ($\alpha = .89$).
5.4.2.7. The Dichotomous Thinking in Eating Disorders Scale (DTEDS; Bryne et al., 2008).

The DTEDS is an 11-item updated version of the Dichotomous Thinking Scale. The DTEDS-G subscale was described in Chapter 4. The DTEDS-G had acceptable internal consistency ($\alpha = .80$) for the current study.

5.4.2.8. Depression Anxiety Stress Scales - 21 (DASS-21-Lovibond & Lovibond, 1995).

The DASS-21 is a 21-item measure developed from the original 42-item DASS (1995a). The DASS-21 consists of three subscales that assess the severity of an individual’s depression, anxiety and stress over the previous week. The DASS-21 instructs the individual to rate their symptoms on a 4-point Likert scale (Lovibond & Lovibond, 1995a). The seven items within each subscale are summed with a higher score indicating a more severe experience of the symptom (Lovibond & Lovibond, 1995a). The DASS-21 is suitable for determining the severity of symptoms in a clinical sample. The three subscales are significantly inter-correlated; depression and anxiety ($r = .55$) depression and stress ($r = .62$), and anxiety and stress ($r = .72$), and correlated with the four subscales of the Mental Health Questionnaire (-.22 to -.67) (Ng et al., 2007). In the current study, the Cronbach’s alpha for each subscale was acceptable; depression ($\alpha = .81$), anxiety ($\alpha = .72$) and stress ($\alpha = .82$).

5.4.2.9. The Quality of Life, Enjoyment and Satisfaction Questionnaire-18 (QLES-Q; Ritsner, Kurs, Gibel, Ratner, & Endicott, 2005).

Previous research has shown that quality life as measured by the QLES-Q is significantly impaired in clinical samples (Watson et al., 2011). Clinical trials have a tendency to report exclusively on the absence of disorder and/or symptom specific outcomes; however, emerging research is highlighting the need for a more holistic measure of improvement and an inclusion of positive mental health outcomes (Swan et al., 2009).

QLES-Q is an 18-item self-report measure adapted from Endicott et al. (1993) quality of life measure. The measure consists of five subscales, namely, physical health, subjective feelings, leisure time activity, social relationships, and satisfaction with medication (one item). Subscale scores are computed by averaging the scores of all items within the subscale. A total score is computed by averaging the scores across the 18 items. If the medication satisfaction item is not applicable,
i.e., if the participant does not use medication, then the total score is averaged across the 17 items. Scores range from 1 = “not at all/never” to 5 = “frequently/all the time” with a high score indicating a greater quality of life and enjoyment satisfaction.

Ritsner et al. (2005) found the QLES-Q can successfully discriminate between healthy and clinical populations, has good test-retest reliability ($r = .83$) and shows a strong correlation ($r = .93$) with the original quality of life measure by Endicott et al. (1993). The internal consistency of the QLES-Q was acceptable for the current study ($\alpha = .90$).

5.4.2.10. Penn State Worry Questionnaire (PSWQ; Stoeber & Bittencourt, 1998).

The PSWQ was used to assess clinical change in participants diagnosed with GAD. The version of the PSWQ that was adapted by Stoeber and Bittencourt (1998) from the Meyer et al. (1990) lifetime version to include a weekly assessment of worry, and was used in the current study. The adapted PSWQ consists of 15 items that are summed to compute a total score of worry. Items are scored using a 7-point Likert scale with from 0 “Never” to 6 “Almost always”. Items 1, 3, 8, 10, and 11 require reverse scoring. Scores can range from 0-90 with a high score indicating a more severe level of worry. The PSWQ has been reported to strongly correlate ($r = .63$) with The Worry Domains Questionnaire by Tallis, Eysenck, and Mathews (1992) and is able to capture changes in worry throughout treatment (Stoeber & Bittencourt, 1998). Several studies have used the adapted PSWQ in treatment trials to assess change in symptoms of worry (Geraghty, Wood, & Hyland, 2010; Rufer, Moergeli, Moritz, Drabe, & Weidt, 2014; von Känel et al., 2005); however, no clinical cut offs have been derived for the measure and no community norms are currently available. The reliability of the PSWQ in the current study was adequate ($\alpha = .90$).

5.4.2.11. Fear of Negative Evaluation Scale – Brief version (FNE-B; Leary, 1983).

The brief version of the Fear of Negative Evaluation Scale (FNE-B) is a 12-item measure of social anxiety adapted from the 30-item Fear of Negative Evaluation Scale by Watson and Friend (1969). The FNE-B was used in the current study to assess clinical change in individuals diagnosed with social anxiety disorder. Item responses are recorded on a 5-point Likert scale from 1 “Not at all characteristic of me” to 5 “Extremely characteristic of me”. Consistent with methodology suggested
by Collins, Westra, Dozois, and Stewart (2005), reverse scored items (2, 4, 7, and 10) were positively phrased. Scores are computed by summing the items and can range from 12-60 with a higher score indicating a more severe level of social anxiety. The FNE-B is strongly correlated \((r = .56)\) with measures of social anxiety, can effectively discriminate between individuals with social phobia, individuals with panic disorder and community samples, and is sensitive enough to detect treatment changes in individuals with social anxiety disorder (Collins et al., 2005). The reliability of the FNE-B in the current sample was deemed acceptable \((\alpha = .93)\).

**5.4.2.12 Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996).**

The BDI-II is a 21-item commonly used measure of depressive symptomatology, adapted from the original BDI (Beck & Steer, 1987). The BDI-II was used to assess clinical change in participants diagnosed with a DSM-IV depressive disorder. Items consist of a 4-point Likert scale ranging from 0 “I don’t have any thoughts of killing myself” to 3 “I would kill myself if I had the chance”. To assess the severity of the participant’s symptoms item scores are summed to compute a total BDI-II score from 0-63. Clinical cut of scores range from 0-13 indicating minimal depression, 14-19 indicating mild depression, 20-28 indicating moderate depression, and 29-63 indicating severe depression. The validity of the BDI-II has been assessed in clinical inpatient and outpatient, and community samples (Cole, Grossman, Prilliman, & Hunsaker, 2003; Dozois, Dobson, & Ahnberg, 1998; Kung et al., 2013; Longwell & Truax, 2005; Steer, Ball, Ranieri, & Beck, 1997) and is strongly correlated to the original BDI \((r = .93)\) in an outpatient university sample (Dozois et al., 1998). Additionally, Brouwer, Meijer, and Zevalkink (2013) confirmed the one-dimensional factor structure of the BDI-II on an outpatient clinical sample \((N = 1520)\). The reliability of the BDI-II in the current study was acceptable \((\alpha = .86)\).

**5.4.2.13. Eating Disorder Examination Questionnaire (EDEQ; Fairburn & Beglin, 1994).**

To measure the participant’s change in eating thoughts and behaviours over the duration of the treatment the 28-item EDEQ was used. The EDEQ is a self-report version of the structured clinical interview, the Eating Disorder Examination (Fairburn & Cooper, 1993) used in clinical settings to diagnose individuals with eating disorders (Fairburn, 2008). Twenty-two items in the EDEQ are divided into four subscales that address the core psychopathology of eating disorders; restraint,
eating concerns, shape concerns and weight concerns (Fairburn, 2008). All items are measured using a 7-point Likert scale; however, the response format is different for each question. To generate a total EDEQ score, the items on the subscale are summed and divided by the amount of items within each subscale. The subscale total scores are then summed and divided by four (Fairburn, 2008). Scores for the total EDEQ can range from 0-6 with a higher score indicating higher eating disorder psychopathology with previous literature reporting a score ≥4 in the clinical range (Fairburn, 2008). The remaining five items are concerned with dysfunctional eating behaviours and are used to diagnose according to the DSM-IV. The subscales of the EDEQ have a strong positive correlation with subscales of the Eating Disorder Examination interview, therefore the EDEQ is a useful tool in measuring an individual’s eating psychopathology (Mond, Hay, Rodgers, Owen, & Beumont, 2004). The EDEQ is reliable in the current sample with an alpha coefficient of .84. Additionally, the subscales are deemed reliable; Weight Concerns (α = .78), Shape Concerns (α = .85), Eating Concerns (α = .70), and Restraint (α = .81).

5.4.2.14. Anxiety Sensitivity Index (ASI-3; Taylor et al., 2007).

The third version of the Anxiety Sensitivity Index (ASI-3) is an 18-item measure of physical, cognitive and social concerns associated with panic disorder. The ASI-3 is a revised version of the Anxiety Sensitivity Scale developed by Reiss, Peterson, Gursky, and McNally (1986) and was used in the current study to assess clinical change in symptoms of DSM-IV panic disorder. Items are scored using a 5-point Likert scale from 0 “Very little” to 4 “Very much”. The items are partitioned into three subscales; physical concerns (Items 3, 4, 7, 8, 12, and 15), cognitive concerns (items 2, 5, 10, 14, 16, and 18), and social concerns (items 1, 6, 9, 11, 13, and 17) and then summed to give subscale scores and a total score ranging from 0 – 72 with a higher score indicating a greater severity in symptoms. Wheaton, Deacon, McGrath, Berman, and Abramowitz (2012) confirmed the three-factor structure of the ASI-3 and found significant correlations with measures of anxiety and panic (r = .35-.48). Cronbach’s alphas in the current study were acceptable for physical concerns (α = .91), cognitive concerns (α = .82), and social concerns (α = .71), and the total score (α = .87).
5.4.2.14. Obsessive Compulsive Inventory – Revised (OCI-R; Foa et al., 2002).

The revised version of the Obsessive Compulsive Inventory (OCI-R) is an 18-item version of the Foa, Kozak, Salkovskis, Coles, and Amir (1998) 42-item Obsessive Compulsive Inventory. The OCI-R consists of six subscales - namely: Washing (items 5, 11, and 17), Obsessing (items 6, 12, and 18), Hoarding (items 1, 7, and 13), Ordering (items 3, 9, and 15), Checking (items 2, 8, and 14), and Neutralising (items 4, 10, and 16), and was used to assess clinical change in individuals diagnosed with OCD. Items are scored on a 5-point Likert scale from 0 “Not at all” to 4 “Extremely”. Items are summed to compute subscale scores and a total score of obsessive and compulsive symptomatology ranging from 0 – 72. The OCI-R has been used to assess changes in obsessive compulsive behaviours and cognitions in several treatment trials (Andersson, Enander, et al., 2012; Russell et al., 2013; Storch et al., 2008) with a total score of ≥21 reportedly an indicator of symptoms in the clinical range (Foa et al., 2002). Abramowitz and Deacon (2006) confirmed the six-factor structure of the OCI-R in a mixed clinical sample, found that it is a suitable measure to distinguish OCD from anxiety disorders, and is significantly correlated to other measures of obsessive compulsive symptoms ($r = .21-.47$). The Cronbach’s alpha for the six subscales ranged from .75-.94 in the current study; however the total score was not of an acceptable alpha (.58).

5.4.2.15. Compliance to treatment.

To measure the participant’s compliance in completing the readings, six short questions were asked each week. The Feedback Questionnaire was attached to each participant’s workbook to be completed after each weekly module. Items include; “How much of the readings/exercises did you read/complete?”, “On average, how much time did you spend on the readings/exercises?”, “How would you rate the usefulness of the readings so far?”, “The reading was easy to read?”. These questions have been derived from Thiels, Schmidt, Troop, Treasure, and Garthe (2001) who evaluated participant compliance with a self-help book for treating BN.

5.4.2.16. Credibility/Expectancy Questionnaire (Devilly & Borkovec, 2000).

The 6-item Credibility Expectancy Questionnaire was used to measure the participant’s treatment credibility and expectancy and administered with the pre-treatment questionnaires. The Credibility Expectancy Questionnaire consists of two subscales, credibility and expectancy, a 9-point Likert scale for Items 1-3 and 5, and
an 11-point Likert scale ranging from 0% to 100% for Items 4 and 6. Items 4 and 6 are converted to the linear equivalent of 1-9 to compute the subscale score. Scores can range from 3 to 27 with a higher score indicating greater credibility of treatment expectancy. Devilly and Borkovec (2000) report acceptable internal consistency for the credibility ($\alpha = .79$) and expectancy ($\alpha = .81$) scales, and test-retest reliability over one week (credibility $r = .75$, expectancy $r = .82$). However, the internal consistency for the credibility subscale in the current study was unacceptable ($\alpha = .39$), therefore it was excluded from subsequent analyses. The internal consistency for the expectancy subscale was acceptable for use in the current study ($\alpha = .87$).

5.4.3. Treatment Protocol

Eight CBT for clinical perfectionism modules were developed and based on chapters from the book “Overcoming perfectionism: A self help guide using cognitive behavioural techniques” (Shafran et al., 2010). Each participant was provided a copy of the book and a workbook containing weekly module information, assigned worksheets and a compliance to treatment measures, at the start of the treatment period. Each week the participants were required to complete designated readings and assigned homework tasks. The 8-week intervention started with a collaborative formulation of the individual’s maintenance cycle of perfectionism, followed by identifying problem areas and motivation techniques to determine the individual’s readiness for change. The concept of surveys and behavioural experiments are then introduced, which are common strategies used throughout the intervention to address rigid and biased thinking, procrastination, and self-criticism. The intervention also introduces the concept of time management and scheduling pleasant events, practicing self-compassion and relapse prevention. An outline of the chapters and weekly modules can be seen in Table 4.

It was recommended that the participant complete the chapters in the privacy of their home on an allocated day of the week. The therapist scheduled weekly telephone contacts with the participant throughout the treatment to assess progress and to ensure they were keeping up to date with the chapters. Each contact was no longer than 15 minutes and was scheduled at a time convenient for the participant. The participant was instructed at the clinical interview that the purpose of the calls was to review the previous week’s module and that content unrelated to that module was not to be discussed.
The participants allocated to the waitlist control condition were advised that there was an 8-week waitlist until they received treatment. High-risk suicidal participants were excluded from the study using the Suicidality MINI module, therefore participants that were asked to wait for eight weeks were not deemed to be at risk. Another assessment was scheduled with the participant after eight weeks and the participant was instructed that they would not have contact with the therapist at this time. At the Time 2 assessment session the participants were provided with the treatment detailed above.

5.4.3.1. Therapists.

The screening, assessment sessions and weekly telephone contact were conducted by the author (Clinical Psychology Masters trainee). The trainee received weekly supervision and specialist training in delivering CBT for clinical perfectionism in an individual setting by Dr Sarah Egan, one of three authors (the others being Roz Shafran and Tracey Wade) who developed CBT for clinical perfectionism (Shafran et al., 2010). Additionally, the trainee had experience in delivering CBT for clinical perfectionism in a group treatment format prior to the first baseline assessment.

The decision was made to use only one therapist as the use of multiple therapists introduces a variety of problems; including therapist effects associated with, for instance therapist differences in empathy and warmth and compliance with the treatment protocol. The cost of hiring an experienced therapist would have exceeded the budget of the project. It was decided that a single therapist approach would be better to ensure consistency and adherence to the treatment protocol.
Table 4

*Weekly Modules, Homework and Corresponding Overcoming Perfectionism Chapters of the 8-Week Treatment Plan*

<table>
<thead>
<tr>
<th>Week</th>
<th>Module</th>
<th>Chapter</th>
<th>Homework</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-treatment readings</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>The First Steps and The Cost of Changing</td>
<td>5, 6</td>
<td>Maintenance cycle, pros and cons of change</td>
</tr>
<tr>
<td>2</td>
<td>Identifying Problem Areas and Psychoeducation</td>
<td>7.1, 7.2</td>
<td>Self-monitoring</td>
</tr>
<tr>
<td>3</td>
<td>Surveys and Behavioural Experiments</td>
<td>7.3, 7.4</td>
<td>Survey, behavioural experiment</td>
</tr>
<tr>
<td>4</td>
<td>All or Nothing Thinking</td>
<td>7.5</td>
<td>Behavioural experiment, continuums</td>
</tr>
<tr>
<td>5</td>
<td>Noticing the Positive and Changing Thinking Styles</td>
<td>7.6, 7.7</td>
<td>Thought diary, record of positive comments</td>
</tr>
<tr>
<td>6</td>
<td>Procrastination, Problem Solving, Time Management and Pleasant Events</td>
<td>7.8, 7.9</td>
<td>Self-monitoring procrastination, flashcards, chunking, time management schedules</td>
</tr>
<tr>
<td>7</td>
<td>Self-criticism and Compassion</td>
<td>8</td>
<td>Self-critical and compassionate thoughts diary</td>
</tr>
<tr>
<td>8</td>
<td>Self-evaluation and Freedom</td>
<td>9, 10</td>
<td>Future goals</td>
</tr>
</tbody>
</table>
5.4.4. Procedure

Ethics approval was obtained from Curtin University’s Human Research and Ethics Committee (HR 120/2010) prior to advertising and recruiting participants. Following this, information and advertisements were distributed. An information sheet, consent form and a screening questionnaire, the CM subscale from the FMPS were distributed to the participants who expressed interest in the study. Upon the return of a signed consent form and a score of ≥22 on the CM subscale of the FMPS, participants were telephoned and screened for eligibility using the MINI-Screen. The Suicidality, Psychotic Disorders and Alcohol/Substance dependence modules of the MINI were also administered to determine eligibility for the study. Participants ineligible for the study because of high suicide risk, psychosis or alcohol/substance dependence were referred to the appropriate services. Eligible participants were asked to attend the Curtin Psychology and Speech Clinic, a clinical psychology postgraduate training centre, for a clinical interview. The clinical interview was used to discuss the participant’s presenting problems and determine a diagnosis using relevant modules from the ADIS-IV and AN and BN module from the MINI. The Clinical Psychology Masters trainee administering the assessment instruments were trained in the administration of the MINI and ADIS-IV, and supervised by experienced Clinical Psychologists via videotaped supervision of their clinical interview sessions to arrive at consensus in the diagnoses. Participants completed the CPQ, EC and PS subscales of the FMPS, DAS-SC and DTEDS-G at the time of the clinical interview. Measurements were collected at the clinical interview (baseline assessment for both groups), post-waitlist (control group), post-treatment (intervention group) and at a 4-month follow-up (intervention group). Primary outcome measures including the CPQ, CM and PS subscales of the FMPS, DAS-SC, DTEDS-G, DASS-21, and the QLES-Q were completed in the Curtin Psychology and Speech Pathology Clinic waiting room prior to the clinical interview. In addition to the primary outcome measure, disorder specific measures were implemented. After primary diagnosis was prescribed at the intake clinical interview, the corresponding disorder specific measure i.e. BDI-II for depressive disorders, was administered. This process was repeated at each assessment time point; baseline assessment for both groups, post-waitlist (control group), post-treatment (intervention group) and at a 4-month follow-up (intervention group).
At the baseline assessment (Time 1) eligible participants were randomly allocated into one of two groups: guided self-help CBT for clinical perfectionism, or the 8-week waitlist control. Post-treatment and post-waitlist (Time 2) measures were administered upon completion of the eight weeks of treatment and the eight weeks waitlist. The waitlist control group commenced guided self-help CBT for clinical perfectionism at week nine. Additionally, the treated waitlist control participants were required to complete post-treatment measures (Time 3). All of the participants were required to complete follow-up measures four months post treatment (Time 4).

5.4.5. Design and Statistical Analysis

The study adopted a randomised, treatment control design with measurements at baseline (Time 1) and post-treatment/post-waitlist (Time 2). The RCT compared guided self-help CBT for clinical perfectionism with a waitlist/control group and conformed to CONSORT guidelines (Moher, Schulz, & Altman, 2001). Participants were randomly allocated to either of the two conditions using a simple random allocation procedure. Random Allocation Software version 1.0 developed by Saghaei (2004) was used to generate the lists. The number of groups and number of participants per group were entered into the system, that is, two groups with 20 participants per group. Each participant was then allocated a 2-digit numerical code with a randomly allocated treatment condition.

Several methods of analysis were used to test the hypotheses. The Generalised Liner Mixed Model (GLMM), as implemented through SPSS version 21.0 GENLINMIXED procedure, was used to compare pre-post changes in the treatment group to pre-post changes in the waitlist control group. For this analysis, participant was treated as a nominal random effect, group (treatment versus waitlist control) was treated as a fixed nominal effect, and time (pre-test, post-test) was treated as an ordinal fixed effect.

A second GLMM was used to determine whether any significant intervention effects found in the first GLMM were maintained at the 4-month follow-up. For this analysis, participant was treated as a nominal random effect and time (pre-test, post-test, follow-up) was treated as an ordinal fixed effect. Due to the control group not having a control follow-up time period the group factor was dropped from the second GLMM.

Alternative methods of analysing behavioural change across time, such as repeated measures analysis of variance, require normality, homogeneity of variance,
and (when there are more than two assessments as in the second GLMM) sphericity (Tabachnick & Fidell, 2001). The GLMM robust statistics option will control for violations of normality and homogeneity of variance and violations of sphericity can be accommodated by changing the covariance matrix from the default of compound symmetry to autoregressive (Maronna, Martin, & Yobai, 2006). Additionally, GLMM is less sensitive to participant attrition because it does not rely on participants providing data at every assessment point. The GLMM maximum likelihood procedure is a full information estimation procedure that uses all the data present at each assessment point, which reduces sampling bias associated with participant attrition and optimises statistical power (Elobeid et al., 2009).

In order to optimise the likelihood of convergence, a separate GLMM analysis was run for each of the nine outcome variables. Analysing each outcome independently of the others would have inflated the familywise error rate. Therefore, the per-test alpha needed to be corrected to control the inflation. In order to conserve statistical power, alpha corrections were applied within groups of conceptually related outcomes rather than across the entire set of outcomes (Klockars, Hancock, & McAweeney, 1995). The bonferroni-adjusted alpha for the targeted perfectionism outcomes was calculated to be .001 (.05/ five outcome variables) and .0125 (.05/ four outcome variables) for the non-targeted psychopathology outcomes. All other tests were performed at the conventional alpha level of .05.

Previous studies that have assessed the efficacy of CBT for clinical perfectionism have found large effect sizes on measures of perfectionism ($d = 1.36$-$1.90$) with small sample sizes (Riley et al., 2007; Steele et al., 2013). In the first GLMM, the intervention effect is embodied in the Group x Time interaction effect and interaction effects are generally small to moderate in size. Therefore, a moderate Group x Time interaction effect was predicted for the current study. An a priori power analysis was conducted to determine the sample size required for an 80% probability of capturing a moderate Group x Time interaction ($f^2 = .3$) at the bonferroni adjusted alpha level of .01. G*Power 3.1 (Faul, Erdfelder, Lang, & Buchner, 2009) computed a sample size of 38 (19 per group). It was deemed sufficient to recruit 19 participants per group; because GLMM minimises the impact of subject attrition on statistical power, there were no additional participants recruited to compensate for attrition. If the first GLMM were sufficiently powered, then the second (less complex) GLMM would also be sufficiently powered.
Computing effect sizes and analysing clinically significant change supplemented statistical hypothesis testing. Pre and post-treatment intervention effect sizes were determined by calculating Cohen’s $d$ (GLMM1) for the intervention group and the corresponding pre-post changes for the control group. Additionally, pre, post and follow-up effect sizes were calculated for the intervention group. Cohen’s $d$ cannot be used to measure interaction effects. Therefore, Partial eta squared was calculated using the following formula partial $\eta^2 = F/(F+df2)$. The conventions for partial $\eta^2$ were used; .01 = small, .06 = moderate, .14+ = large. Cohen’s $d$ (GLMM2) was calculated for Post-hoc Least Significant Difference (LSD) tests, conducted to locate the source of the significant interactions (i.e. pre-post, post-follow-up, pre-follow-up changes for the intervention). Cohen’s $d$ was estimated from the LSD t-values using the following formula, $d = t/sqrt(N)$. Conventions for Cohen’s $d$ were used, .2 = small, .5 = moderate, .8 = large.

Clinically significant and reliable change indices were used to determine whether the treatment produced a clinically important change on primary outcome measures, as well as disorder specific outcome measurements. Jacobson and Truax (1991) methodology was used to assess the reliability and clinical significance of each participant’s pre to post-treatment and pre to follow-up change scores.

Before behavioural change can be clinically significant it has to be statistically reliable. Statistical reliability is assessed with the Reliable Change Index (RCI; Jacobson, Follette, & Revenstorf, 1986). The RCI is the degree to which the person changes on the outcome variable divided by the standard error of difference between the pre and post-test scores. When the absolute value of the RCI exceeds 1.96 has argued that this value can be reduced in some situations, it is likely that the pre-post change score reflects a real or reliable change. The formula for computing RCI can be seen in Table 5.

Table 5

*Formula used to Calculate the Reliable Change Index and Clinically Significant Change*

<table>
<thead>
<tr>
<th>Formulae</th>
<th>Criteria $c$</th>
<th>Reliable Change</th>
<th>$S_E$</th>
<th>$S_{diff}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$c = \frac{S_0M_1 + S_1M_0}{S_0 + S_1}$</td>
<td>$RC = \frac{x_1 - x_2}{S_{diff}}$</td>
<td>$S_1\sqrt{(1-rel)}$</td>
<td>$\sqrt{2(S_E)^2}$</td>
<td></td>
</tr>
</tbody>
</table>
Jacobson et al. (1986) provided three cut off criteria to determine the reliability and clinical significance of a participant’s change score. Criterion a requires that the post-intervention score falls two standard deviations outside that of the mean of the clinical population in the direction of the community population. Criterion b requires that the score falls within two standard deviations of the mean from the community population. Criterion c is the midpoint between the mean of the clinical and community population; a score that falls closer to the community mean is classified as representing a clinically significant change. Criterion c is deemed the most appropriate one to use when means and standard deviations of the clinical and community samples are available. The formula for calculating Criteria c can also be seen in Table 5. Criteria c was adopted for all outcome measures as it is the least arbitrary and all clinical and community normative data were available except for DTEDS-G and the adapted PSWQ. Criteria b was implemented for DTEDS-G as community normative data were not published. The following formula was used to calculate criteria $b = M_0 - 2S_1$. Criteria a was implemented for the adapted PSWQ as neither community nor clinical data were available. The following formula was used $a = M_1 + 2s_1$.

Absolute RCIs less than or equal to 1.96 indicate no change. Absolute RCIs greater than 1.96 may fall into one of three clinical categories. A classification of “recovered” is appropriate when the score crosses from the clinical range to the community range; a classification of “improved” is used when the score moves towards the community mean but still falls within the clinical range; and a classification of “deteriorated” is used when the score moves away from the community mean.

Pearson’s chi-square tests of contingencies will be used to assess the difference between the treatment and control condition in rates of clinically significant and reliable improvement from pre to post-treatment. Phi conventions will be used to determine effect size; .00 and < .10 = negligible association, .10 and < .20 = weak association, .20 and < .40 = moderate association, .40 and < .60 = relatively strong association, .60 and < .80 = strong association, .80 and < 1.00 = very strong association.
Table 6

*Clinically Significant and Reliable Change Criteria for Treatment Outcomes According to Jacobson and Truax (1991)*

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Clinically Significant Change</th>
<th>RCI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recovered</td>
<td>Yes</td>
<td>≥ + 1.96</td>
</tr>
<tr>
<td>Improved</td>
<td>No</td>
<td>≥ + 1.96</td>
</tr>
<tr>
<td>Unchanged</td>
<td>No</td>
<td>≤ ± 1.96</td>
</tr>
<tr>
<td>Deteriorated</td>
<td>No</td>
<td>≥ - 1.96</td>
</tr>
</tbody>
</table>

*Note:* RCI = Reliable Change Index.

5.5. Results

5.5.1. Data Screening

Prior to analysis, the data were screened as per Tabachnick and Fidell (2007) recommendations. Upon visual inspection of all variables in the analysis, no out of range values were observed and therefore there were no obvious errors in data entry. The GLMM ‘robust statistics’ was invoked to accommodate any violations of normality and homogeneity of variance; and violations of sphericity were accommodated by changing the covariance matrix from the default of ‘compound symmetry’ to ‘autoregressive’.

5.5.1.1. Missing data

Missing values analysis revealed missing items scores. Expectation maximisation was used to identify and replace the missing data for missing item scores only. Expectation maximisation was used because the missing data appeared to be occurring in a random pattern, as indicated by the non-significant Little’s MCAR statistic (Tabachnick & Fidell, 2007). Missing values were replaced for items on all of the outcome measures.

5.5.2. Demographic and Clinical Characteristics of the Elevated Perfectionism Sample (N = 40)

Baseline demographic and clinical characteristics were analysed to determine group equivalence as defined by statistically non-significant differences between groups (*p* > .05). An independent samples *t*-test was used to compare the groups in terms of age and treatment expectations, as measured by the Credibility Expectancy Questionnaire. The results of the group comparisons are reported in Table 7. The Shapiro-Wilk test was non-significant for both age and treatment expectation,
indicating that the assumption of normality was not violated for either of these variables. Levene’s test was also non-significant for both variables, indicating homogeneity of variance across the two groups. The t-tests were statistically non-significant indicating that age and treatment expectancy were comparable across the two groups. Pearson’s chi-square tests of contingencies were used to compare the two groups in terms of the categorical demographic variables, namely, gender, marital status, occupation and diagnostic category. Except for gender, there were no significant differences between the two groups on the categorical demographic or diagnostic variables, suggesting that the groups were comparable on these variables. As gender was not correlated with the measure of perfectionism and psychopathology, it was not controlled for in subsequent analyses.

Additionally, t-tests were conducted to evaluate the difference between baseline means of the outcome variables CPQ, CM, PS, DAS-SC, DTEDS-G, depression, anxiety, stress, and QLES-Q. The Shapiro-Wilk statistic was significant only for depression and anxiety. However, the Shapiro-Wilk statistic can be sensitive to small departures from normality that has no impact on the reliability of the statistical tests. Histograms were therefore inspected and showed reasonably normal distributions. With the exception of DTEDS-G, all outcomes yielded a non-significant Levene’s test indicating homogeneity of variance across the two groups. The inferential statistics reported in Table 8 indicate that there were no significant differences between the two groups at baseline on any of the outcome measures, suggesting that the two groups were comparable on these measures.
Table 7

Baseline Demographic and Clinical Data Comparing Treatment and Control Group for the Elevated Perfectionism Sample (N = 40)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Treatment (n = 20)</th>
<th>Control (n = 20)</th>
<th>Group Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>n</td>
</tr>
<tr>
<td>Age</td>
<td>33.56</td>
<td>8.84</td>
<td>20</td>
</tr>
<tr>
<td>Treatment expectancy</td>
<td>16.00</td>
<td>5.13</td>
<td>16</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>11</td>
<td>55</td>
<td>17</td>
</tr>
<tr>
<td>Male</td>
<td>9</td>
<td>45</td>
<td>3</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>7</td>
<td>35</td>
<td>9</td>
</tr>
<tr>
<td>Defacto</td>
<td>5</td>
<td>25</td>
<td>5</td>
</tr>
<tr>
<td>Single</td>
<td>8</td>
<td>40</td>
<td>6</td>
</tr>
<tr>
<td>Work status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Full/part time work</td>
<td>13</td>
<td>65</td>
<td>12</td>
</tr>
<tr>
<td>Full/part time student</td>
<td>6</td>
<td>30</td>
<td>6</td>
</tr>
<tr>
<td>Homemaker</td>
<td>1</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Diagnosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive Disorder</td>
<td>3</td>
<td>15</td>
<td>3</td>
</tr>
<tr>
<td>Anxiety Disorder</td>
<td>11</td>
<td>55</td>
<td>12</td>
</tr>
<tr>
<td>Eating Disorder</td>
<td>2</td>
<td>10</td>
<td>1</td>
</tr>
<tr>
<td>No diagnosis</td>
<td>4</td>
<td>20</td>
<td>3</td>
</tr>
</tbody>
</table>

$p < .05$
Table 8

Statistical Group Comparison of Baseline Outcome Variable Means

<table>
<thead>
<tr>
<th>Variable</th>
<th>t-statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPQ</td>
<td>( t(38) = 0.473, p = .639 )</td>
</tr>
<tr>
<td>CM</td>
<td>( t(38) = 0.457, p = .650 )</td>
</tr>
<tr>
<td>PS</td>
<td>( t(38) = 0.302, p = .764 )</td>
</tr>
<tr>
<td>DAS-SC</td>
<td>( t(38) = 0.498, p = .621 )</td>
</tr>
<tr>
<td>DTEDS-G</td>
<td>( t(31) = 0.715, p = .480 )</td>
</tr>
<tr>
<td>Depression</td>
<td>( t(38) = 0.800, p = .429 )</td>
</tr>
<tr>
<td>Anxiety</td>
<td>( t(38) = 1.152, p = .256 )</td>
</tr>
<tr>
<td>Stress</td>
<td>( t(38) = 0.739, p = .464 )</td>
</tr>
<tr>
<td>QLES-Q</td>
<td>( t(38) = 1.527, p = .135 )</td>
</tr>
</tbody>
</table>

Note: CPQ = Clinical Perfectionism Questionnaire; CM = concern over mistakes subscale of the Frosts Multidimensional Perfectionism Scale (FMPS); PS = personal standards subscale of the FMPS; DAS-SC = Dysfunctional Attitudes Scale – self-criticism subscale; DTEDS-G = Dichotomous Thinking in Eating Disorders Scale – general subscale; QLES-Q = Quality of Life Enjoyment and Satisfaction Questionnaire.

5.5.3. Treatment Compliance

Treatment compliance was measured at the end of each module. Table 9 displays results of the participant’s compliance in completing the weekly readings and exercises. A graphical display can be seen in Figure 3.
**Table 9**
Self-reported Adherence (%) to the Intervention as a Summary of Modules Read and Exercises Completed

<table>
<thead>
<tr>
<th>Module</th>
<th>Readings</th>
<th></th>
<th></th>
<th>Exercises</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Completed all</td>
<td>Completed part</td>
<td>Did not complete</td>
<td>Completed all</td>
<td>Completed part</td>
<td>Did not complete</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n (%)</td>
<td>n (%)</td>
<td>n (%)</td>
<td>n (%)</td>
<td>n (%)</td>
<td>n (%)</td>
<td></td>
</tr>
<tr>
<td>Pre-treatment readings</td>
<td>12 (70.6)</td>
<td>2 (11.8)</td>
<td>5 (29.4)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>14 (87.5)</td>
<td>1 (6.3)</td>
<td>1 (6.3)</td>
<td>11 (73.3)</td>
<td>4 (26.7)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>12 (75.0)</td>
<td>4 (25.0)</td>
<td>0</td>
<td>2 (12.5)</td>
<td>13 (81.3)</td>
<td>1 (6.3)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>13 (86.7)</td>
<td>2 (13.3)</td>
<td>0</td>
<td>4 (28.6)</td>
<td>5 (35.7)</td>
<td>5 (35.7)</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>14 (100)</td>
<td>0</td>
<td>0</td>
<td>4 (26.7)</td>
<td>7 (46.7)</td>
<td>4 (26.7)</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>13 (92.9)</td>
<td>1 (7.1)</td>
<td>0</td>
<td>6 (42.9)</td>
<td>6 (42.9)</td>
<td>2 (14.3)</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>7 (50.0)</td>
<td>6 (42.9)</td>
<td>1 (7.1)</td>
<td>0</td>
<td>7 (58.3)</td>
<td>5 (41.7)</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>12 (85.7)</td>
<td>1 (7.1)</td>
<td>1 (7.1)</td>
<td>2 (16.7)</td>
<td>9 (75.0)</td>
<td>1 (8.3)</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>11 (78.6)</td>
<td>2 (14.3)</td>
<td>1 (7.1)</td>
<td>3 (23.1)</td>
<td>7 (53.9)</td>
<td>3 (23.1)</td>
<td></td>
</tr>
</tbody>
</table>

*Note: Module numbers are defined as follows: Pre-treatment readings = Introduction: Understanding clinical perfectionism, 1 = Individualised maintenance cycle of clinical perfectionism, 2 = Self-monitoring and psychoeducation, 3 = Surveys and behavioural experiments, 4 = Challenging dichotomous thinking via behavioural experiments and continuums, 5 = Challenging cognitive biases, 6 = Procrastination, time management, and pleasant events, 7 = Self-criticism and self-compassion, 8 = Self-evaluation and relapse prevention.*
5.5.4. Descriptive Statistics for the elevated perfectionism sample (N = 40)

The means and standard deviations of all of the measures from pre-treatment to post-treatment are reported in Table 10. Perfectionism outcome pre-test means for the intervention and control groups were within a clinical range (i.e., one standard deviation from their clinical normative means) on the CPQ ($M = 28.53, SD = 6.23$, Egan et al., 2013), the CM ($M = 26.7, SD = 7.6$; Saboonchi, Lund, & Ost, 1999), the DAS-SC ($M = 47.28, SD = 17.75$; Dunkley et al., 2004), and the DTEDS-G ($M = 2.77, SD = 0.76$; Bryne et al., 2008). However, the PS ($M = 21.8, SD = 6.0$; Saboonchi et al.) pre-test mean score was greater than one standard deviation above the clinical mean, suggesting that the current sample had higher than average personal standards. Psychopathology outcome pre-test means for the intervention and control groups were within clinical range for all subscales of the DASS-21 (Lovibond & Lovibond, 1995b), depression ($M = 10.63, SD = 9.3$), anxiety ($M = 10.90, SD = 8.12$) and stress ($M = 21.1, SD = 11.15$) and the QLES-Q ($M = 3.4, SD = 0.8$; Ritsner et al., 2005).
Table 10

Means, Adjusted Means and Standard Deviations for Each Outcome Variable by Time and Group

<table>
<thead>
<tr>
<th></th>
<th>Treatment</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-Treatment ( n = 20 )</td>
<td>Post-Treatment ( n = 17 )</td>
<td>Pre-Treatment ( n = 20 )</td>
<td>Post-Treatment ( n = 18 )</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>( M )</td>
<td>( SD )</td>
<td>( M ) (adjusted)</td>
<td>( SD )</td>
<td>( M )</td>
<td>( SD )</td>
<td>( M ) (adjusted)</td>
<td>( SD )</td>
<td></td>
</tr>
<tr>
<td>CPQ</td>
<td>29.80</td>
<td>4.58</td>
<td>25.88 (26.05)</td>
<td>4.17</td>
<td>30.49</td>
<td>4.61</td>
<td>29.37 (29.63)</td>
<td>4.20</td>
<td></td>
</tr>
<tr>
<td>CM</td>
<td>30.65</td>
<td>5.63</td>
<td>22.65 (23.10)</td>
<td>4.12</td>
<td>31.45</td>
<td>5.44</td>
<td>32.23 (32.35)</td>
<td>6.24</td>
<td></td>
</tr>
<tr>
<td>PS</td>
<td>29.18</td>
<td>3.59</td>
<td>25.24 (25.29)</td>
<td>3.58</td>
<td>28.86</td>
<td>3.11</td>
<td>28.02 (28.30)</td>
<td>3.31</td>
<td></td>
</tr>
<tr>
<td>DAS-SC</td>
<td>61.50</td>
<td>13.23</td>
<td>43.94 (44.13)</td>
<td>11.22</td>
<td>63.65</td>
<td>14.04</td>
<td>65.72 (65.62)</td>
<td>15.06</td>
<td></td>
</tr>
<tr>
<td>DTEDS-G</td>
<td>2.49</td>
<td>.49</td>
<td>1.90 (1.88)</td>
<td>.62</td>
<td>2.64</td>
<td>.80</td>
<td>2.69 (2.71)</td>
<td>.68</td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>9.70</td>
<td>6.13</td>
<td>5.88 (5.59)</td>
<td>4.44</td>
<td>11.54</td>
<td>8.27</td>
<td>13.66 (14.23)</td>
<td>9.25</td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>8.10</td>
<td>5.82</td>
<td>4.35 (4.27)</td>
<td>4.37</td>
<td>10.70</td>
<td>8.24</td>
<td>9.15 (9.37)</td>
<td>7.63</td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>21.30</td>
<td>8.47</td>
<td>14.94 (14.78)</td>
<td>8.55</td>
<td>23.33</td>
<td>8.92</td>
<td>22.57 (23.20)</td>
<td>9.80</td>
<td></td>
</tr>
<tr>
<td>QLES-Q</td>
<td>3.45</td>
<td>.55</td>
<td>3.74 (3.80)</td>
<td>.26</td>
<td>3.76</td>
<td>.73</td>
<td>3.62 (3.60)</td>
<td>.76</td>
<td></td>
</tr>
</tbody>
</table>

Note: CPQ = Clinical Perfectionism Questionnaire; CM = concern over mistakes subscale of the Frost Multidimensional Subscale (FMPS); PS = personal standards subscale of the FMPS; DAS-SC = self-criticism subscale of the Dysfunctional Attitudes Scale; DTEDS-G = Dichotomous Thinking in Eating Disorders Scale – general subscale; QLES-Q = Quality Life Enjoyment Satisfaction Questionnaire.
5.5.5. Hypothesis Testing

5.5.5.1. Perfectionism and Related Construct Outcomes.

The relationships between the fixed effects (group, time, Group x Time) and the targeted outcomes (CPQ, CM, PS, DAS-SC and DTEDS-G) were analysed with a GLMM (see Table 11). There was a 12.5% rate of attrition from pre-post treatment across the intervention and control condition. Hypotheses 1-5 predicted a significant Group x Time interaction for all outcome variables. See Appendix C for graphs of the interactions for each outcome variable. At the Bonferroni adjusted alpha level of .01, the Group x Time interactions were significant for four of the outcomes thereby comprising the interpretation of the main effects of group and time (CM: $F[1,69] = 28.35, p < .001$, partial $\eta^2 = .29$; PS: $F[1,69] = 8.01, p = .006$, partial $\eta^2 = .10$; DAS-SC: $F[1,70] = 26.42, p < .001$, partial $\eta^2 = .27$; DTEDS-G: $F[1,71] = 14.60, p < .001$, partial $\eta^2 = .17$). There was a non-significant Group x Time interaction for the CPQ ($F[1,71] = 4.33, p = .041$, partial $\eta^2 = .06$), therefore each of the two main effects for CPQ can be interpreted independently of one another. The main effect of time was significant ($F[1,71] = 10.98, p = .001$, partial $\eta^2 = .13$), however, the main effect for group was not ($F[1,71] = 3.04, p = .058$, partial $\eta^2 = .04$). These results indicate that the treatment and control groups showed a significant decrease in CPQ over time, and that the rate of decrease was the same for both groups.
Table 11

Results of the Omnibus GLMMs for Each Outcome

<table>
<thead>
<tr>
<th>Source</th>
<th>Num df</th>
<th>Den df</th>
<th>F-value</th>
<th>p-value</th>
<th>Partial η²</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPQ</td>
<td>Group</td>
<td>1</td>
<td>71</td>
<td>3.04</td>
<td>.058</td>
</tr>
<tr>
<td></td>
<td>Time</td>
<td>1</td>
<td>71</td>
<td>10.98</td>
<td>.001*</td>
</tr>
<tr>
<td></td>
<td>Group x</td>
<td>1</td>
<td>71</td>
<td>4.33</td>
<td>.041</td>
</tr>
<tr>
<td></td>
<td>Time</td>
<td></td>
<td></td>
<td></td>
<td>.06</td>
</tr>
<tr>
<td>CM</td>
<td>Group</td>
<td>1</td>
<td>69</td>
<td>10.16</td>
<td>.002*</td>
</tr>
<tr>
<td></td>
<td>Time</td>
<td>1</td>
<td>69</td>
<td>17.48</td>
<td>.000*</td>
</tr>
<tr>
<td></td>
<td>Group x</td>
<td>1</td>
<td>69</td>
<td>28.35</td>
<td>.000*</td>
</tr>
<tr>
<td></td>
<td>Time</td>
<td></td>
<td></td>
<td></td>
<td>.29</td>
</tr>
<tr>
<td>PS</td>
<td>Group</td>
<td>1</td>
<td>69</td>
<td>2.01</td>
<td>.161</td>
</tr>
<tr>
<td></td>
<td>Time</td>
<td>1</td>
<td>69</td>
<td>14.41</td>
<td>.000*</td>
</tr>
<tr>
<td></td>
<td>Group x</td>
<td>1</td>
<td>69</td>
<td>8.01</td>
<td>.006*</td>
</tr>
<tr>
<td></td>
<td>Time</td>
<td></td>
<td></td>
<td></td>
<td>.10</td>
</tr>
<tr>
<td>DAS-SC</td>
<td>Group</td>
<td>1</td>
<td>70</td>
<td>9.75</td>
<td>.003*</td>
</tr>
<tr>
<td></td>
<td>Time</td>
<td>1</td>
<td>70</td>
<td>263.23</td>
<td>.000*</td>
</tr>
<tr>
<td></td>
<td>Group x</td>
<td>1</td>
<td>70</td>
<td>26.42</td>
<td>.000*</td>
</tr>
<tr>
<td></td>
<td>Time</td>
<td></td>
<td></td>
<td></td>
<td>.27</td>
</tr>
<tr>
<td>DTEDS-G</td>
<td>Group</td>
<td>1</td>
<td>71</td>
<td>6.67</td>
<td>.012*</td>
</tr>
<tr>
<td></td>
<td>Time</td>
<td>1</td>
<td>71</td>
<td>8.78</td>
<td>.004*</td>
</tr>
<tr>
<td></td>
<td>Group x</td>
<td>1</td>
<td>71</td>
<td>14.60</td>
<td>.000*</td>
</tr>
</tbody>
</table>

Note: CPQ = Clinical Perfectionism Questionnaire; CM = concern over mistakes subscale of the Frost Multidimensional Subscale (FMPS); PS = personal standards subscale of the FMPS; DAS-SC = self-criticism subscale of the Dysfunctional Attitudes Scale; DTEDS-G = Dichotomous Thinking in Eating Disorders Scale – general subscale.

*p < Bonferroni correct alpha-level of .01

Conventions for Partial η² are: .01 = small; .06 = moderate; .14+ = large

Post-hoc LSD tests were conducted to locate the source of the significant interactions for CM, PS, DAS-SC, and DTEDS-G. LSDs were also calculated for the CPQ so that comparisons of effect sizes could be made, even though the intervention was not significant at the adjusted Bonferroni corrected alpha level. LSD contrasts conducted on the simple main effects of time indicated that the treatment group showed a significant pre-post decrease on CM perfectionism (p < .001, d = 1.60) (see Table 12). In contrast, the control group showed no significant pre-post change (p = .417, d = 0.195). Additionally, there was a significant pre-post decrease in PS perfectionism for the treatment group (p < .001, d = 1.12, but no change for the control group (p = .492, d = 0.12). There was a significant pre-post decrease in DAS-
SC perfectionism for the treatment group \((p < .001, d = 1.60)\), and a difference for the control group \((p = .035, d = 0.51)\). Finally, the treatment group showed a significant pre-post decrease in DTEDS-G \((p < .001, d = 1.20)\). In contrast, the control group showed no significant pre-post change \((p = .566, d = 0.14)\). The results provide support for Hypotheses 2-5a that CBT for clinical perfectionism will be associated with greater pre-post improvements on CM, PS, DAS-SC, and DTEDS-G, compared to the wait-list control group. All four of the significant targeted perfectionism variables showed large effect sizes for the treatment condition \((d = 1.12 – 1.60)\). A large effect size was also observed on the CPQ \((d = 0.97)\).

The hypothesis that the intervention will be associated with greater pre-post improvement for the CPQ was not supported (Hypothesis 1a).
Table 12

Least Significance Difference (LSD) Tests of the Simple Main Effects of Time for the Group x Time Interactions for Perfectionism Variables

<table>
<thead>
<tr>
<th></th>
<th>Treatment</th>
<th></th>
<th>Control</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$t$</td>
<td>$df$</td>
<td>$Contrast$</td>
<td>$Std.$</td>
</tr>
<tr>
<td></td>
<td>estimate</td>
<td>error</td>
<td>CI</td>
<td>value</td>
</tr>
<tr>
<td>CPQ</td>
<td>4.078</td>
<td>71</td>
<td>3.752</td>
<td>0.920</td>
</tr>
<tr>
<td>CM</td>
<td>6.648</td>
<td>69</td>
<td>7.555</td>
<td>1.137</td>
</tr>
<tr>
<td>PS</td>
<td>4.634</td>
<td>69</td>
<td>3.894</td>
<td>0.840</td>
</tr>
<tr>
<td>DAS-SC</td>
<td>6.714</td>
<td>70</td>
<td>17.369</td>
<td>2.587</td>
</tr>
<tr>
<td>DTEEDS-</td>
<td>5.071</td>
<td>71</td>
<td>0.603</td>
<td>0.119</td>
</tr>
</tbody>
</table>

Note: CPQ = Clinical Perfectionism Questionnaire; CM = concern over mistakes subscale of the Frost Multidimensional Subscale (FMPS); PS = personal standards subscale of the FMPS; DAS-SC = self-criticism subscale of the Dysfunctional Attitudes Scale; DTEEDS-G = Dichotomous Thinking in Eating Disorders Scale – general subscale.

*** $p < .001$.

Conventions for Cohen’s $d$ are: $.2$ = small; $.5$ = moderate; $.8$ = large.
5.5.5.2. Perfectionism and Related Construct Outcomes at Follow-up.

A GLMM comparing pre-treatment, post-treatment and follow-up scores for the treatment group was used to test Hypotheses 2-5b. There was a 15% rate of attrition from pre-treatment to follow-up for the intervention condition. The hypotheses predicted that the intervention effect would be maintained at 4-month follow-up; see Table 13 for means and standard deviations and Appendix D for interactions of each perfectionism outcome variable. CPQ was not included in follow-up analyses as there was a non-significant Group x Time intervention effect at post-treatment.

The main effect for time was significant across the four perfectionism outcome variables (CM: $F[2,51] = 16.039, p < .001$, partial $\eta^2 = 0.24$; PS: $F[2,51] = 11.553, p < .001$, $d = 0.19$; DAS-SC: $F[2,51] = 17.829, p < .001$, partial $\eta^2 = 0.26$; DTEDS-G: $F[2,51] = 17.223, p < .001$, partial $\eta^2 = 0.25$).

Table 13
Means, Adjusted Means and Standard Deviations for the Perfectionism Outcomes in the Treatment Group

<table>
<thead>
<tr>
<th></th>
<th>Pre-Treatment ($n = 20$)</th>
<th>Post-Treatment ($n = 17$)</th>
<th>Follow-up ($n = 17$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$ (adjusted mean)</td>
</tr>
<tr>
<td>CM</td>
<td>30.65</td>
<td>5.63</td>
<td>22.65 (22.95)</td>
</tr>
<tr>
<td>PS</td>
<td>29.18</td>
<td>3.59</td>
<td>25.24 (25.32)</td>
</tr>
<tr>
<td>DAS-SC</td>
<td>61.50</td>
<td>13.23</td>
<td>43.94 (44.01)</td>
</tr>
<tr>
<td>DTEDS-G</td>
<td>2.49</td>
<td>0.49</td>
<td>1.90 (1.90)</td>
</tr>
</tbody>
</table>

Note: CM = concern over mistakes subscale of the Frost Multidimensional Subscale (FMPS); PS = personal standards subscale of the FMPS; DAS-SC = self-criticism subscale of the Dysfunctional Attitudes Scale; DTEDS-G = Dichotomous Thinking in Eating Disorders Scale-general.

In order to locate the source of the interactions for the four significant perfectionism outcomes, post-hoc LSD contrasts were conducted across the main effect of time (see Table 14); the contrasts indicated a significant pre-post decrease
across all outcomes (CM: $p < .001$, $d = 1.513$; PS: $p < .001$, $d = 1.223$; DAS-SC $p < .001$, $d = 1.54$; DTEDS-G: $p < .001$, $d = 1.398$). There was a non-significant post to follow-up change (CM: $p = .375$, $d = 0.251$; PS: $p = .539$, $d = 0.173$; DAS-SC: $p = .582$, $d = 0.155$; DTEDS-G: $p = .914$, $d = 0.028$) across the four outcomes. However, a significant pre to follow-up decrease on CM, PS, DAS-SC, and (CM: $p < .001$, $d = 1.56$, $p < .001$, $d = 1.334$; PS: $p < .001$, $d = 1.64$; DAS-SC: $p < .001$, $d = 1.526$) was observed suggesting maintenance of the intervention effects at follow-up. These results support Hypothesis 2-5b that the significant pre-post improvements on CM, PS, DAS-SC and DTEDS-G, for the treatment group will be maintained at the 4-month follow-up.
Table 14

Least Significance Difference (LSD) Tests of the Main Effects of Time for Perfectionism Variables

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Time</th>
<th>t</th>
<th>df</th>
<th>Estimate</th>
<th>Std. Error</th>
<th>95% CI</th>
<th>Adj. p-value</th>
<th>Cohen’s d</th>
</tr>
</thead>
<tbody>
<tr>
<td>CM</td>
<td>Pre-Post</td>
<td>5.402</td>
<td>51</td>
<td>7.721</td>
<td>1.429</td>
<td>4.420, 11.022</td>
<td>&lt;.001***</td>
<td>1.51</td>
</tr>
<tr>
<td></td>
<td>Post-FU</td>
<td>0.895</td>
<td>51</td>
<td>0.636</td>
<td>0.711</td>
<td>-0.791, 2.064</td>
<td>.375</td>
<td>0.25</td>
</tr>
<tr>
<td></td>
<td>Pre-FU</td>
<td>5.570</td>
<td>51</td>
<td>8.357</td>
<td>1.500</td>
<td>4.643, 12.071</td>
<td>&lt;.001***</td>
<td>1.56</td>
</tr>
<tr>
<td>PS</td>
<td>Pre-Post</td>
<td>4.367</td>
<td>51</td>
<td>3.885</td>
<td>0.889</td>
<td>1.8350, 5.939</td>
<td>&lt;.001***</td>
<td>1.22</td>
</tr>
<tr>
<td></td>
<td>Post-FU</td>
<td>0.618</td>
<td>51</td>
<td>0.307</td>
<td>0.496</td>
<td>-0.690, 1.303</td>
<td>.539</td>
<td>0.17</td>
</tr>
<tr>
<td></td>
<td>Pre-FU</td>
<td>4.762</td>
<td>51</td>
<td>4.191</td>
<td>0.880</td>
<td>2.012, 6.370</td>
<td>&lt;.001***</td>
<td>1.33</td>
</tr>
<tr>
<td>DAS-SC</td>
<td>Pre-Post</td>
<td>5.500</td>
<td>51</td>
<td>17.396</td>
<td>3.163</td>
<td>10.091, 24.700</td>
<td>&lt;.001***</td>
<td>1.54</td>
</tr>
<tr>
<td></td>
<td>Post-FU</td>
<td>0.553</td>
<td>51</td>
<td>1.028</td>
<td>1.857</td>
<td>-2.701, 4.756</td>
<td>.582</td>
<td>0.15</td>
</tr>
<tr>
<td></td>
<td>Pre-FU</td>
<td>5.855</td>
<td>51</td>
<td>18.423</td>
<td>3.147</td>
<td>10.634, 26.213</td>
<td>&lt;.001***</td>
<td>1.64</td>
</tr>
<tr>
<td>DTEDS-G</td>
<td>Pre-Post</td>
<td>4.992</td>
<td>51</td>
<td>0.601</td>
<td>0.120</td>
<td>0.323, 0.879</td>
<td>&lt;.001***</td>
<td>1.34</td>
</tr>
<tr>
<td></td>
<td>Post-FU</td>
<td>0.108</td>
<td>51</td>
<td>0.011</td>
<td>0.108</td>
<td>-0.200, 0.222</td>
<td>.914</td>
<td>0.03</td>
</tr>
<tr>
<td></td>
<td>Pre-FU</td>
<td>5.449</td>
<td>51</td>
<td>0.612</td>
<td>0.112</td>
<td>0.334, 0.891</td>
<td>&lt;.001***</td>
<td>1.53</td>
</tr>
</tbody>
</table>

Note: CM = concern over mistakes subscale of the Frost Multidimensional Subscale (FMPS); PS = personal standards subscale of the FMPS; DAS-SC = self-criticism subscale of the Dysfunctional Attitudes Scale; DTEDS-G = Dichotomous Thinking in Eating Disorders Scale – general subscale.

*** p < .001.

Conventions for Cohen’s d are: .2 = small; .5 = moderate; .8 = large.

5.5.5.3. Clinically Significant Change on Perfectionism Outcomes.

RCI scores were calculated for each participants pre-post test score across the five perfectionism outcome variables. Clinical change was computed for those with an RCI score greater than an absolute value of 1.96. Normative data were available for CPQ, CM, PS, and DAS-SC, therefore Jacobson and Truax (1991) Criteria c was
used. Criteria \( b \) was used to assess DTEDS-G, as no community norms were available. Normative data can be seen in Table 15.

Table 15

Data used to Calculate Reliable Change and Clinical Significance on Perfectionism

Outcome Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>Population</th>
<th>( M )</th>
<th>( SD )</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPQ(^a)</td>
<td>Clinical</td>
<td>28.53</td>
<td>6.23</td>
<td>.646</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>24.17</td>
<td>4.47</td>
<td></td>
</tr>
<tr>
<td>CM(^b)</td>
<td>Clinical</td>
<td>26.7</td>
<td>7.6</td>
<td>.837</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>19.7</td>
<td>6.4</td>
<td></td>
</tr>
<tr>
<td>PS(^b)</td>
<td>Clinical</td>
<td>21.8</td>
<td>6</td>
<td>.672</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>20.5</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>DAS-SC(^c)</td>
<td>Clinical</td>
<td>47.28</td>
<td>17.75</td>
<td>.892</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>38.81</td>
<td>15.15</td>
<td></td>
</tr>
<tr>
<td>DTEDS-G(^d)</td>
<td>Clinical</td>
<td>2.77</td>
<td>0.76</td>
<td>.803</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

Note: CPQ = Clinical Perfectionism Questionnaire; CM = concern over mistakes subscale of the Frost Multidimensional Subscale (FMPS); PS = personal standards subscale of the FMPS; DAS-SC = self-criticism subscale of the Dysfunctional Attitudes Scale; DTEDS-G = Dichotomous Thinking in Eating Disorders Scale – general subscale. Reliability scores from the current study were used.

\(^a\) Community and clinical normative data were derived from Egan et al. (2016)

\(^b\) Community and clinical normative data were derived from Saboonchi et al. (1999)

\(^c\) Community normative data were derived from Dunkley and Kyparissis (2008), whilst clinical normative data were derived from Dunkley et al. (2004)

\(^d\) Clinical normative data were derived from Byrne et al. (2008)

The Reliable and Clinical Change Generator (ClinTools, 2008), an online computer program, was used to generate reliable and clinical change scores. As seen in Table 16, the majority of the participants in the intervention condition were classified as improved or recovered at post-treatment on the CM and DAS-SC, whereas the majority of the participants remained unchanged on measures CPQ, PS, and DTEDS-G.
Table 16

*The Number (and Percentage) of Participants Falling in Each of the Four Clinical Categories at Post-Treatment on Perfectionism Outcomes*

<table>
<thead>
<tr>
<th></th>
<th>CPQ</th>
<th>CM</th>
<th>PS</th>
<th>DAS-SC</th>
<th>DTEDS-G</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Treatment (n = 17)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recovered</td>
<td>1 (5.9)</td>
<td>9 (52.9)</td>
<td>1 (5.9)</td>
<td>6 (35.3)</td>
<td>1 (5.9)</td>
</tr>
<tr>
<td>Improved</td>
<td>0</td>
<td>1 (5.9)</td>
<td>4 (23.5)</td>
<td>4 (23.5)</td>
<td>4 (23.5)</td>
</tr>
<tr>
<td>Unchanged</td>
<td>16 (94.1)</td>
<td>7 (41.2)</td>
<td>12 (70.6)</td>
<td>7 (41.2)</td>
<td>12 (70.6)</td>
</tr>
<tr>
<td>Deteriorated</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Control (n = 18)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recovered</td>
<td>1 (5.5)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Improved</td>
<td>0</td>
<td>0</td>
<td>1 (5.5)</td>
<td>0</td>
<td>1 (5.5)</td>
</tr>
<tr>
<td>Unchanged</td>
<td>16 (88.9)</td>
<td>18 (100)</td>
<td>17 (94.5)</td>
<td>14 (77.8)</td>
<td>15 (83.3)</td>
</tr>
<tr>
<td>Deteriorated</td>
<td>1 (5.5)</td>
<td>0</td>
<td>0</td>
<td>4 (22.2)</td>
<td>2 (11.1)</td>
</tr>
</tbody>
</table>

*Note:* CPQ = Clinical Perfectionism Questionnaire; CM = concern over mistakes subscale of the Frost Multidimensional Subscale (FMPS); PS = personal standards subscale of the FMPS; DAS-SC = self-criticism subscale of the Dysfunctional Attitudes Scale; DTEDS-G = Dichotomous Thinking in Eating Disorders Scale – general subscale.

Pearson’s chi-square tests of contingencies were used to evaluate whether treatment is related to clinically significant change from pre-treatment to post-treatment. The difference in rates of improvement from pre to post-treatment between the treatment and the control condition was significant for DAS-SC ($\chi^2 [1, N = 35] = 4.782, p = .029, \phi = .37$), however there was a non-significant difference between rates of improvement for CM ($\chi^2 [1, N = 35] = 1.09, p = .296, \phi = .18$), PS ($\chi^2 [1, N = 35] = 2.31, p = .129, \phi = .26$) and DTEDS-G ($\chi^2 [1, N = 35] = 2.31, p = .129, \phi = .26$). Pre-post-treatment recovery rates between the treatment and control group were significant for CM ($\chi^2 [1, N = 35] = 12.83, p < .001, \phi = .61$) and DASS (6 $\chi^2 [1, N = 35] = 7.67, p = .006, \phi = .47$), however there was a non-significant difference for PS ($\chi^2 [1, N = 35] = 1.09, p = .296, \phi = .18$), CPQ ($\chi^2 [1, N = 35] = 0.002, p = .967, \phi = .01$) and DTEDS-G ($\chi^2 [1, N = 35] = 1.09, p = .296, \phi = .18$). These findings partially support Hypotheses 2-5c that the pre-post improvement on
perfectionism and related measures in the treatment group will be reliable and clinically significant.

There was no control group at 4-month follow-up, therefore comparisons cannot be made across the treatment and control group. As seen in Table 17, the majority of the participants in the treatment condition were classified as clinically improved or recovered at 4-month follow-up on the measures CM, DAS-SC, and DTEDS-G.

### Table 17

The Number (and Percentage) of Participants in the Treatment Group Meeting Clinically Significant Change at Follow-up on Perfectionism Outcomes (n = 17)

<table>
<thead>
<tr>
<th></th>
<th>CPQ</th>
<th>CM</th>
<th>PS</th>
<th>DAS-SC</th>
<th>DTEDS-G</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recovered</td>
<td>7 (41.2)</td>
<td>7 (41.2)</td>
<td>1 (5.9)</td>
<td>6 (35.3)</td>
<td>1 (5.9)</td>
</tr>
<tr>
<td>Improved</td>
<td>1 (5.9)</td>
<td>2 (11.8)</td>
<td>6 (35.3)</td>
<td>5 (29.4)</td>
<td>8 (47.05)</td>
</tr>
<tr>
<td>Unchanged</td>
<td>9 (52.9)</td>
<td>8 (47)</td>
<td>10 (58.8)</td>
<td>6 (35.3)</td>
<td>8 (47.05)</td>
</tr>
<tr>
<td>Deteriorated</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Note: CPQ = Clinical Perfectionism Questionnaire; CM = concern over mistakes subscale of the Frosts Multidimensional Perfectionism Scale (FMPS); PS = personal standards subscale of the FMPS; DAS-SC = Dysfunctional Attitudes Scale – self-criticism subscale; DTEDS-G = Dichotomous Thinking in Eating Disorders Scale – general subscale.

5.5.5.4. Psychopathology Outcomes.

The relationships between the fixed effects (group, time, Group x Time) and the non-targeted psychopathology outcomes (depression, anxiety, stress, and QLES-Q) were analysed with a series of GLMMs (see Table 18). There was a 12.5% rate of attrition from pre-post treatment across the intervention and control condition. Hypotheses 6-9a predicted a significant Group x Time interaction for all psychopathology outcome variables. See Appendix E for graphs of the interactions for each outcome variable. At the Bonferroni adjusted alpha level of .0125, the Group x Time interaction was significant for depression and QLES-Q (depression: $F[1,71] = 7.64, p = .007, \text{partial } \eta^2 = .10$; QLES-Q: $F[1,71] = 11.36, p = .001, \text{partial } \eta^2 = .14$) thereby compromising the interpretation of the main effects of group and
time for these outcomes. There was a non-significant Group x Time interaction for anxiety ($F[1,71] = 1.83, p = .180$, partial $\eta^2 = .03$) and stress ($F[1,71] = 3.97, p = .050$, partial $\eta^2 = .05$), therefore each of the two main effects can be interpreted independently of one another. The main effect for time was significant ($F[1,71] = 7.81, p = .007$, partial $\eta^2 = 0.10$) for anxiety, however, the main effect for group was not ($F[1,71] = 4.22, p = .044$, partial $\eta^2 = 0.06$). These results indicate that the treatment and control groups showed a significant decrease in anxiety over time, and that the rate of decrease was the same for both groups.

The main effect for time was not significant ($F[1,71] = 4.31, p = .037$, partial $\eta^2 = 0.06$) for stress. Additionally, the main effect for group was not significant ($F[1,71] = 3.97, p = .050$, partial $\eta^2 = 0.05$). These results suggest no change in stress from pre-treatment to post-treatment for either of the groups.

Table 18

Results of the Omnibus GLMMs for Each Outcome

<table>
<thead>
<tr>
<th>Source</th>
<th>Numerator $df$</th>
<th>Denomenator $df$</th>
<th>$F$-value</th>
<th>$p$-value</th>
<th>Partial $\eta^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>Group 1</td>
<td>71</td>
<td>5.54</td>
<td>.021</td>
<td>.07</td>
</tr>
<tr>
<td></td>
<td>Time 1</td>
<td>71</td>
<td>0.33</td>
<td>.565</td>
<td>.005</td>
</tr>
<tr>
<td></td>
<td>Group x Time 1</td>
<td>71</td>
<td>7.64</td>
<td>.007*</td>
<td>.10</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Group 1</td>
<td>71</td>
<td>4.22</td>
<td>.044</td>
<td>.06</td>
</tr>
<tr>
<td></td>
<td>Time 1</td>
<td>71</td>
<td>7.81</td>
<td>.007*</td>
<td>.10</td>
</tr>
<tr>
<td></td>
<td>Group x Time 1</td>
<td>71</td>
<td>1.83</td>
<td>.180</td>
<td>.03</td>
</tr>
<tr>
<td>Stress</td>
<td>Group 1</td>
<td>71</td>
<td>4.54</td>
<td>.037</td>
<td>.06</td>
</tr>
<tr>
<td></td>
<td>Time 1</td>
<td>71</td>
<td>4.31</td>
<td>.042</td>
<td>.06</td>
</tr>
<tr>
<td></td>
<td>Group x Time 1</td>
<td>71</td>
<td>3.97</td>
<td>.050</td>
<td>.05</td>
</tr>
<tr>
<td>QLES-Q</td>
<td>Group 1</td>
<td>71</td>
<td>0.12</td>
<td>.731</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>Time 1</td>
<td>71</td>
<td>1.65</td>
<td>.203</td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>Group x Time 1</td>
<td>71</td>
<td>11.36</td>
<td>.001*</td>
<td>.14</td>
</tr>
</tbody>
</table>

Note: QLES-Q = Quality Life Enjoyment Satisfaction Questionnaire.
* $p <$ Bonferroni correct alpha-level of 0.0125
Conventions for Partial $\eta^2$ are: .01 = small, .06 = moderate, .14+ = large

Post-hoc LSD contrasts conducted on the simple main effects of time indicated that the treatment group showed a significant pre-post decrease in depression ($p = .022, d = 0.55$) (see Table 19). In contrast, the control group showed no significant pre-post change ($p = .122, d = 0.37$). Additionally, there was a
significant pre-post increase in QLES-Q for the treatment group \((p = .003, d = 0.731)\), but no change for the control group \((p = .116, d = 0.38)\).

LSD contrasts were also conducted for the anxiety and stress so that comparisons of effect sizes could be made, even though the intervention was not significant at the adjusted Bonferroni corrected alpha level. Moderate effect sizes were observed for anxiety \((d = 0.58)\) and stress \((d = 0.67)\), respectively. The effect size estimates are consistent with predictions as effects for all variables were in directions that indicated improvements.

The results support Hypotheses 6a and 9a that CBT for clinical perfectionism will be associated with a significantly greater pre-post change with a significantly greater pre-post improvement on measures of depression and QLES-Q, compared to the wait-list control group. However, the hypothesis that the intervention will be associated with greater pre-post improvement for anxiety and stress was not supported (Hypotheses 7a and 8a).
Table 19

*Least Significance Difference (LSD) Tests of the Simple Main Effects of Time for the Group x Time Interactions for Psychopathology Variables*

| Outcome | Treatment | | | | Control | | |
|---------|-----------|-------|---------|--------|---------|-------|---------|-------|
|         | $t$  | df | Contrast estimate | Std. error | 95% CI | Adj. $p$-value | $D$ | $t$ | df | Contrast estimate | Std. error | 95% CI | Adj. $p$-value | $d$ |
| Depression | 2.337 | 71 | 4.106 | 1.757 | 0.603, 7.609 | .022* | 0.55 | -1.563 | 71 | -2.686 | 1.718 | -6.112, 0.740 | .122 | 0.37 |
| Anxiety | 2.438 | 71 | 3.828 | 1.570 | 0.697, 6.959 | .017* | 0.58 | 1.371 | 71 | 1.330 | 0.970 | 3.264 | .175 | 0.33 |
| Stress | 2.845 | 71 | 6.516 | 2.291 | 1.948, 11.083 | .006** | 0.67 | 0.059 | 71 | 0.132 | 2.240 | -4.335, 4.599 | .953 | 0.01 |
| QLES-Q | -3.079 | 71 | -0.349 | 0.113 | -0.574, -0.123 | .003** | 0.73 | 1.592 | 71 | 0.156 | 0.098 | -0.039, 0.352 | .116 | 0.38 |

*Note:* QLES-Q = Quality Life Enjoyment Satisfaction Questionnaire.

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

Conventions for Cohen’s $d$ are: .2 = small, .5 = moderate, .8 = large
5.5.5.5. Psychopathology Outcomes at Follow-up.

Hypotheses 6b and 9b were tested with GLMMs comparing pre-treatment, post-treatment and follow-up psychopathology outcome scores for the treatment group. There was a 15% rate of attrition from pre-treatment to follow-up for the intervention condition. The hypothesis predicted that the intervention effect would be maintained at 4-month follow-up (see Table 20 for means and standard deviations, and Appendix F for interactions of each psychopathology outcome variable). Anxiety and stress was not included in follow-up analyses as there were non-significant Group x Time interactions for these outcomes.

The main effect for time was significant for the two psychopathology outcome variables of depression ($F[2,51] = 8.339, p = .001$, partial $\eta^2 = 0.14$) and QLES-Q ($F[2,51] = 10.266, p < .001$, partial $\eta^2 = 0.17$).

Table 20
Means, Adjusted Means and Standard Deviations for the Psychopathology Outcomes in the Treatment Group

<table>
<thead>
<tr>
<th></th>
<th>Pre-Treatment $(n = 20)$</th>
<th>Post-Treatment $(n = 17)$</th>
<th>Follow-up $(n = 17)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>$M = 9.70$ $SD = 6.13$</td>
<td>$M (adjusted mean) = 5.88 (5.66)$ $SD = 4.44$</td>
<td>$M (adjusted mean) = 4.12 (3.92)$ $SD = 4.03$</td>
</tr>
<tr>
<td>QLES-Q</td>
<td>$M = 3.45$ $SD = 0.55$</td>
<td>$M (adjusted mean) = 3.74 (3.78)$ $SD = 0.26$</td>
<td>$M (adjusted mean) = 3.98 (4.01)$ $SD = 0.44$</td>
</tr>
</tbody>
</table>

*Note: QLES-Q = Quality of Life Enjoyment Satisfaction Questionnaire.*

LSD post-hoc contrasts were conducted across the main effect of time in order to locate the source of the significant interactions for the two psychopathology outcomes (see Table 21 for statistics). The contrasts indicated a significant pre-post decrease on Depression and a significant increase on QLES-Q ($p = .018, d = 0.76$; $p = .010, d = .82$). There was a non-significant post to follow-up change for depression ($p = .150, d = 0.41$), however a significant increase for QLES-Q ($p = .017, d = .694$) across the two outcomes. A significant pre to follow-up decrease on depression and increase on QLES-Q ($p < .001, d = 1.14$; $p < .001, d = 1.27$) was observed suggesting maintenance of the intervention effects at follow-up. These results support
Hypotheses 6b and 9b that the significant pre-post improvement for the treatment group will be maintained at the 4-month follow-up.

Table 21

Least Significance Difference (LSD) Tests of the Main Effect of Time for Psychopathology Variables

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Estimate</th>
<th>Std. Error</th>
<th>95% CI</th>
<th>Adj. p-value</th>
<th>Cohen’s d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-Post</td>
<td>2.713</td>
<td>3.992</td>
<td>1.472</td>
<td>0.594, 7.391</td>
<td>.018*</td>
</tr>
<tr>
<td>Post-FU</td>
<td>1.461</td>
<td>1.735</td>
<td>1.187</td>
<td>-0.648, 4.118</td>
<td>.150</td>
</tr>
<tr>
<td>Pre-FU</td>
<td>4.084</td>
<td>5.727</td>
<td>1.402</td>
<td>2.256, 9.199</td>
<td>&lt;.001***</td>
</tr>
<tr>
<td>QLES-Q</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-Post</td>
<td>-2.934</td>
<td>-0.329</td>
<td>0.112</td>
<td>-0.588, -0.070</td>
<td>.010*</td>
</tr>
<tr>
<td>Post-FU</td>
<td>-2.479</td>
<td>-0.236</td>
<td>0.095</td>
<td>-0.427, -0.045</td>
<td>.017*</td>
</tr>
<tr>
<td>Pre-FU</td>
<td>-4.526</td>
<td>-0.565</td>
<td>0.125</td>
<td>-0.874, -0.256</td>
<td>&lt;.001***</td>
</tr>
</tbody>
</table>

Note: QLES-Q = Quality Life Enjoyment Satisfaction Questionnaire.

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

Conventions for Cohen’s d are: .2 = small, .5 = moderate, .8 = large

5.5.5.6. Clinically Significant Change on Psychopathology Outcomes.

Pre-post RCI scores were calculated for each of the participants across the four psychopathology outcome variables. Clinical change was computed for those with RCI scores greater than an absolute value of 1.96. Normative data were available for all measures, therefore Jacobson and Truax (1991) criteria c was used. Normative data can be seen in Table 22.

The Reliable and Clinical Change Generator (ClinTools, 2008) was used to generate reliable and clinical change scores. As seen in Table 23, the majority of the participants in the intervention condition were classified unchanged at post-treatment across all psychopathology measures.
Table 22

*Data used to Calculate Reliable Change and Clinical Significance on Psychopathology Outcome Measures*

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Population</th>
<th>M</th>
<th>SD</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>Clinical</td>
<td>10.65</td>
<td>9.3</td>
<td>.808</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>6.34</td>
<td>6.97</td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>Clinical</td>
<td>10.90</td>
<td>8.12</td>
<td>.724</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>4.7</td>
<td>4.91</td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>Clinical</td>
<td>21.1</td>
<td>11.15</td>
<td>.824</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>10.11</td>
<td>7.91</td>
<td></td>
</tr>
<tr>
<td>QLES-Q</td>
<td>Clinical</td>
<td>3.4</td>
<td>.8</td>
<td>.900</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>4.2</td>
<td>.4</td>
<td></td>
</tr>
</tbody>
</table>

*Note:* QLES-Q = Quality Life Enjoyment Satisfaction Questionnaire. Reliability scores from the current study were used.  
*Clinical and community normative data were derived from* Lovibond and Lovibond (1995b)  
*b Clinical and community normative data were derived from Ritsner et al. (2005)*

Table 23

*The Number (and Percentage) of Participants Meeting Clinically Significant Change at Post-Treatment and Follow-Up on Psychopathology Outcomes*

<table>
<thead>
<tr>
<th></th>
<th>Depression</th>
<th>Anxiety</th>
<th>Stress</th>
<th>QLES-Q</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Treatment (n = 17)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recovered</td>
<td>2 (11.7)</td>
<td>3 (17.6)</td>
<td>5 (29.4)</td>
<td>1 (5.9)</td>
</tr>
<tr>
<td>Improved</td>
<td>1 (5.9)</td>
<td>0</td>
<td>0</td>
<td>5 (29.4)</td>
</tr>
<tr>
<td>Unchanged</td>
<td>14 (82.4)</td>
<td>14 (82.4)</td>
<td>11 (64.7)</td>
<td>10 (58.8)</td>
</tr>
<tr>
<td>Deteriorated</td>
<td>0</td>
<td>0</td>
<td>1 (5.9)</td>
<td>1 (5.9)</td>
</tr>
<tr>
<td><strong>Control (n = 18)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recovered</td>
<td>0</td>
<td>1 (5.6)</td>
<td>1 (5.6)</td>
<td>1 (5.6)</td>
</tr>
<tr>
<td>Improved</td>
<td>0</td>
<td>0</td>
<td>1 (5.6)</td>
<td>0</td>
</tr>
<tr>
<td>Unchanged</td>
<td>16 (88.9)</td>
<td>17 (94.4)</td>
<td>14 (77.8)</td>
<td>15 (83.3)</td>
</tr>
<tr>
<td>Deteriorated</td>
<td>2 (11.1)</td>
<td>0</td>
<td>2 (11.1)</td>
<td>2 (11.1)</td>
</tr>
</tbody>
</table>

*Note:* QLES-Q = Quality of Life Enjoyment Satisfaction Questionnaire.
Pearson’s chi-square tests of contingencies were used to evaluate whether treatment is related to clinically significant change from pre-treatment to post-treatment. The difference in rates of improvement from pre to post-treatment between the treatment and the control condition was significant for QLES-Q ($\chi^2 [1, N = 35] = 6.18, p = .013, \phi = 0.42$), however there was a non-significant difference between rates of improvement for depression ($\chi^2 [1, N = 35] = 1.09, p = .296, \phi = 0.18$) and stress ($\chi^2 [1, N = 35] = 0.97, p = .324, \phi = 0.17$). Pre-post treatment recovery rates between the treatment and control group were not significant across all measures of psychopathology, namely, depression ($\chi^2 [1, N = 35] = 2.25, p = .134, \phi = 0.25$), anxiety ($\chi^2 [1, N = 35] = 1.26, p = .261, \phi = 0.19$), stress ($\chi^2 [1, N = 35] = 3.50, p = .061, \phi = 0.32$) and QLES-Q ($\chi^2 [1, N = 35] = .002, p = .97, \phi = 0.01$). These findings partially support Hypotheses 6-9c that the pre-post improvement on psychopathology measures in the treatment group will be reliable and clinically significant.

There was no control group at 4-month follow-up, therefore comparisons cannot be made across the treatment and control group. As seen in Table 24, the majority of the participants in the treatment condition were classified as unchanged across depression, anxiety, stress, and QLES-Q.

Table 24

<table>
<thead>
<tr>
<th></th>
<th>Depression</th>
<th>Anxiety</th>
<th>Stress</th>
<th>QLES-Q</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recovered</td>
<td>3 (17.6)</td>
<td>2 (11.8)</td>
<td>7 (41.2)</td>
<td>5 (29.4)</td>
</tr>
<tr>
<td>Improved</td>
<td>0</td>
<td>0</td>
<td>1 (5.9)</td>
<td>1 (5.9)</td>
</tr>
<tr>
<td>Unchanged</td>
<td>14 (82.4)</td>
<td>15 (88.2)</td>
<td>9 (52.9)</td>
<td>11 (64.7)</td>
</tr>
<tr>
<td>Deteriorated</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

*Note: QLES-Q = Quality Life Enjoyment Satisfaction Questionnaire.*
5.5.6. Demographic and Clinical Characteristics of the Mixed Clinical Sample (N = 32)

The majority of the clinical sample were female (n = 24), married or in a defacto relationship (n = 22), engaged in full time work (n = 17), and with an age range from 19 to 57 (M = 34.54, SD = 9.71). Sixty-three percent (n = 20) of the sample reported receiving treatment prior to the baseline assessment. A total of four participants reported taking medication at the time of the baseline assessment, including antidepressants (n = 2), anxiety (n = 1) and sleeping medication (n = 1).

Diagnoses were determined with the ADIS-IV and the relevant modules of the MINI. The primary diagnoses of the sample at pre and post-treatment for the treatment and control are reported in Table 25. Due to the design of the study, there was no follow-up diagnostic assessment for the control condition. Therefore 4-month follow-up diagnoses are only reported for the intervention group.

The majority of the sample had multiple diagnoses; 40.6% of the sample had at least two diagnoses, 28.1% had at least three diagnoses, and 6.3% had at least four diagnoses; participants (25%) had only one current diagnosis.

A Fisher’s Exact Test was used to evaluate whether the treatment condition, CBT for clinical perfectionism, is related to recovery of DSM-IV diagnoses at post-treatment. This method of analysis was chosen as some cells in the contingency table had expected frequencies less than five. The test was statistically non-significant, (p = .153, ϕ = 0.31). Although there were a smaller proportion of participants meeting DSM-IV diagnostic criteria at post-treatment in the treatment condition than the control condition, this result was not statistically significant. This finding does not support Hypothesis 10a.

Pearson’s chi-square tests of contingencies were also used to evaluate whether treatment is related to recovery of comorbid DSM-IV disorders at pre-treatment and at post-treatment. First the chi-square revealed that the treatment and control groups did not differ significantly at pre-treatment (χ² [1, N = 32] = 1.247, p = .264, ϕ = 0.20) suggesting group equivalence. However, the chi-square test at post-treatment was statistically significant with a moderate effect size (χ² [1, N = 28] = 5.320, p = .021, ϕ = 0.44). The participants in the treatment group were significantly less like to present with DSM-IV comorbid disorders at post-treatment than the control group, supporting Hypothesis 10b.
Table 25

*Primary and Comorbid Diagnosis by Time and Condition for the Mixed Clinical Sample (N = 32)*

<table>
<thead>
<tr>
<th></th>
<th>Treatment</th>
<th></th>
<th></th>
<th></th>
<th>Control</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre (n = 16)</td>
<td>Post (n = 16)</td>
<td>FU (n = 12)</td>
<td>Pre (n = 16)</td>
<td>Post (n = 16)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>n %</td>
<td>n %</td>
<td>n %</td>
<td>n %</td>
<td>n %</td>
<td>n %</td>
<td>n %</td>
</tr>
<tr>
<td>Primary Diagnosis</td>
<td>GAD</td>
<td>6</td>
<td>37.5</td>
<td>4</td>
<td>25</td>
<td>2</td>
<td>16.7</td>
</tr>
<tr>
<td></td>
<td>Social Phobia</td>
<td>4</td>
<td>25</td>
<td>4</td>
<td>25</td>
<td>2</td>
<td>12.5</td>
</tr>
<tr>
<td></td>
<td>Major Depression</td>
<td>3</td>
<td>18.8</td>
<td>2</td>
<td>12.5</td>
<td>0</td>
<td>6 -</td>
</tr>
<tr>
<td></td>
<td>EDNOS</td>
<td>1</td>
<td>6.3</td>
<td>1</td>
<td>6.3</td>
<td>0 -</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Bulimia Nervosa</td>
<td>1</td>
<td>6.3</td>
<td>0 -</td>
<td>0 -</td>
<td>0 -</td>
<td>0 -</td>
</tr>
<tr>
<td></td>
<td>OCD</td>
<td>1</td>
<td>6.3</td>
<td>0 -</td>
<td>0 -</td>
<td>0 -</td>
<td>0 -</td>
</tr>
<tr>
<td></td>
<td>Panic Disorder</td>
<td>0 -</td>
<td>0 -</td>
<td>0 -</td>
<td>0 -</td>
<td>2</td>
<td>12.5</td>
</tr>
<tr>
<td></td>
<td>Dysthymia</td>
<td>0 -</td>
<td>0 -</td>
<td>0 -</td>
<td>0 -</td>
<td>1</td>
<td>6.3</td>
</tr>
<tr>
<td></td>
<td>No diagnosis</td>
<td>0 -</td>
<td>4</td>
<td>25</td>
<td>8</td>
<td>66.7</td>
<td>0 -</td>
</tr>
<tr>
<td>Comorbid Diagnosis</td>
<td>1 x comorbidity</td>
<td>7</td>
<td>43.8</td>
<td>2</td>
<td>10.5</td>
<td>0 -</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>2 x comorbidity</td>
<td>3</td>
<td>18.8</td>
<td>0 -</td>
<td>0 -</td>
<td>0 -</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>≥ 3 x comorbidity</td>
<td>1</td>
<td>6.3</td>
<td>0 -</td>
<td>0 -</td>
<td>0 -</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>No comorbidity</td>
<td>5</td>
<td>31.3</td>
<td>14</td>
<td>87.5</td>
<td>0 -</td>
<td>3</td>
</tr>
</tbody>
</table>

*Note: GAD = Generalised Anxiety Disorder; EDNOS = Eating Disorder Not Otherwise Specified; OCD = Obsessive Compulsive Disorder.*
5.5.7. Descriptive Statistics for the Mixed Clinical Sample (N = 32)

The means and standard deviations of the disorder specific measures from pre-treatment to post-treatment are reported in Table 26. Disorder specific outcome pre-test means for the intervention and control groups were within the clinical range (i.e., one standard deviation from their reported clinical means) on the PSWQ ($M = 60.93$, $SD = 9.47$; Morgan, 2011); the FNE-B ($M = 51.5$, $SD = 7.3$; Collins et al., 2005); the BDI-II ($M = 27.55$, $SD = 9.75$; Titov et al., 2011); and the EDEQ ($M = 4.02$, $SD = 1.28$; Aardoom et al., 2012). The pre-treatment intervention mean for the OCI-R ($M = 28.01$, $SD = 13.53$; Foa et al., 2002) and the pre-treatment control mean for the ASI-3 ($M = 32.6$, $SD = 14.3$; Taylor et al., 2007) were within the clinical range.
Table 26
Means and Standard Deviations for Each Disorder Specific Outcome Variable by Time and Group at Baseline

<table>
<thead>
<tr>
<th></th>
<th>Pre-Treatment</th>
<th>Post-Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>M</td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSWQ</td>
<td>6</td>
<td>58.50</td>
</tr>
<tr>
<td>FNE-B</td>
<td>4</td>
<td>51.75</td>
</tr>
<tr>
<td>BDI-II</td>
<td>3</td>
<td>24.67</td>
</tr>
<tr>
<td>EDEQ</td>
<td>2</td>
<td>2.94</td>
</tr>
<tr>
<td>OCI-R</td>
<td>1</td>
<td>34</td>
</tr>
<tr>
<td>ASI-3</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSWQ</td>
<td>5</td>
<td>65.40</td>
</tr>
<tr>
<td>FNE-B</td>
<td>4</td>
<td>50.25</td>
</tr>
<tr>
<td>BDI-II</td>
<td>2</td>
<td>18.50</td>
</tr>
<tr>
<td>EDEQ</td>
<td>2</td>
<td>4.76</td>
</tr>
<tr>
<td>OCI-R</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>ASI-3</td>
<td>2</td>
<td>37.50</td>
</tr>
</tbody>
</table>

Notes: PSWQ = Penn State Worry Questionnaire; FNE-B = Fear of Negative Evaluation scale – Brief; BDI-II = Beck Depressive Inventory 2nd Edition; EDEQ = Eating Disorder Examination Questionnaire; ASI-3 = Anxiety Sensitivity Index 3rd Edition; OCI-R = Obsessive Compulsive Inventory-Revised.

5.5.8. Clinically Significant Change in Disorder Specific Symptomatology.

Pre-post RCI scores were calculated for each participant for the relevant disorder specific measure. Clinical change was computed for those with an RCI score greater than 1.96. Clinical and community data were available for FNE-B, BDI-II,
EDEQ and ASI-3; therefore, Jacobson and Truax (1991) Criteria \( c \) was used. There were no current community norms available for the adapted version of the PSWQ, therefore criteria \( a \) was used. Normative data can be seen in Table 27. OCI-R could not be assessed, as no post-treatment data were available due to treatment dropout.

Table 27
**Data used to Calculate Reliable Change and Clinical Significance on Disorder Specific Outcome Measures.**

<table>
<thead>
<tr>
<th>Outcome Measure</th>
<th>Population</th>
<th>Reference</th>
<th>( M )</th>
<th>( SD )</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSWQ</td>
<td>Clinical</td>
<td>(Morgan, 2011)</td>
<td>60.93</td>
<td>9.47</td>
<td>.906</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>FNE-B</td>
<td>Clinical</td>
<td>(Collins et al., 2005)</td>
<td>51.5</td>
<td>7.3</td>
<td>.926</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>29.2</td>
<td>8.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI-II</td>
<td>Clinical</td>
<td>(Titov et al., 2011)</td>
<td>27.55</td>
<td>9.75</td>
<td>.857</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>(Roelofs et al., 2013)</td>
<td>10.6</td>
<td>10.9</td>
<td></td>
</tr>
<tr>
<td>EDEQ</td>
<td>Clinical</td>
<td>(Aardoom, Dingemans, Slof Op't Landt, &amp; Van Furth, 2012)</td>
<td>4.02</td>
<td>1.28</td>
<td>.841</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>.93</td>
<td>.86</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASI-3</td>
<td>Clinical</td>
<td>(Taylor et al., 2007)</td>
<td>32.6</td>
<td>14.3</td>
<td>.784</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>12.8</td>
<td>10.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>OCI-R</td>
<td>Clinical</td>
<td>(Foa et al., 2002)</td>
<td>28.01</td>
<td>13.53</td>
<td>.900</td>
</tr>
<tr>
<td></td>
<td>Community</td>
<td>18.82</td>
<td>11.1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Notes: PSWQ = Penn State Worry Questionnaire; FNE-B = Fear of Negative Evaluation scale – Brief; BDI-II = Beck Depressive Inventory 2nd Edition; EDEQ = Eating Disorder examination Questionnaire; ASI-3 = Anxiety Sensitivity Index 3rd Edition; OCI-R = Obsessive Compulsive Inventory-Revised.*
The Reliable and Clinical Change Generator (ClinTools, 2008), was used to generate reliable and clinical change scores for the participants disorder specific measure for their corresponding primary diagnosis. As seen in Table 28, 50% \((n = 6)\) of the participants in the intervention condition were classified as improved or recovered at post-treatment compared to 7.7% \((n = 1)\) in the control condition.

Table 28

<table>
<thead>
<tr>
<th></th>
<th>PSWQ</th>
<th>FNE-B</th>
<th>BDII</th>
<th>EDEQ</th>
<th>ASI-3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Treatment ((n = 12))</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recovered</td>
<td>2 (50)</td>
<td>1 (25)</td>
<td>1 (33.3)</td>
<td>-</td>
<td>-</td>
<td>4 (33.3)</td>
</tr>
<tr>
<td>Improved</td>
<td>1 (25)</td>
<td>0</td>
<td>1 (33.3)</td>
<td>-</td>
<td>-</td>
<td>2 (16.7)</td>
</tr>
<tr>
<td>Unchanged</td>
<td>0</td>
<td>3 (75)</td>
<td>1 (33.3)</td>
<td>1 (100)</td>
<td>-</td>
<td>5 (41.7)</td>
</tr>
<tr>
<td>Deteriorated</td>
<td>1 (25)</td>
<td>0</td>
<td>0</td>
<td>-</td>
<td>-</td>
<td>1 (8.3)</td>
</tr>
<tr>
<td><strong>Control ((n = 13))</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recovered</td>
<td>1 (20)</td>
<td>0</td>
<td>0</td>
<td>-</td>
<td>0</td>
<td>1 (7.7)</td>
</tr>
<tr>
<td>Improved</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>-</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Unchanged</td>
<td>4 (80)</td>
<td>4 (100)</td>
<td>1 (100)</td>
<td>1 (100)</td>
<td>2 (100)</td>
<td>12 (92.3)</td>
</tr>
<tr>
<td>Deteriorated</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>-</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

*Notes:* PSWQ = Penn State Worry Questionnaire; FNE-B = Fear of Negative Evaluation scale – Brief; BDII = Beck Depressive Inventory 2nd Edition; EDEQ = Eating Disorder examination Questionnaire; ASI-3 = Anxiety Sensitivity Index 3rd Edition.

A Pearson’s chi-square test of contingencies (with \(\alpha = .05\)) was used to evaluate whether the treatment is related to clinically significant improvement and recovery on disorder specific measures for the corresponding primary diagnosis. Because some cells in the contingency table had expected frequencies less than five, Fisher’s Exact Test was used. The difference in rates of clinically significant
improvement and recovery from pre-treatment to post-treatment between the treatment and control group was significant ($p = .030, \phi = 0.47$). The participants in the treatment condition were significantly more likely to have clinical improvement and recovery than the participants in the control condition. This result provides support for Hypothesis 11.

5.5.6. Discussion

The primary aim of the study was to assess the effect of the intervention on measures of perfectionism and a related cognitive construct; dichotomous thinking. A further aim was to assess the diagnostic changes and disorder specific symptoms in the individuals presenting with DSM-IV diagnoses. The current study sought to describe the complexity of the individuals presenting for treatment and provide efficacy for a guided-self intervention in reducing complex primary presentations and comorbidity.

CBT for clinical perfectionism was effective at reducing symptoms of perfectionism as assessed through CM (partial $\eta^2 = .29$), PS (partial $\eta^2 = .10$), and self-criticism (partial $\eta^2 = .27$), with large effects observed. This finding is consistent with previous research supporting the efficacy of the CBT intervention delivered in a face-to-face individual (Riley et al., 2007) and group format (Handley et al., 2015; Steele et al., 2013), and self-help (Arpin-Cribbie et al., 2012; Pleva & Wade, 2007; Steele & Wade, 2008). These, significant effects were maintained at the 4-month follow-up assessment with large effects observed (CM, partial $\eta^2 = .24$; PS, partial $\eta^2 = .19$; DAS-SC, partial $\eta^2 = .26$). Additionally, clinically significant change was observed for the significant clinical perfectionism measures, meaning that the individuals in the treatment group presented with perfectionism closer to the community population at the end of their treatment.

Contrary to predictions and the literature (Handley et al., 2015; Riley et al., 2007; Steele et al., 2013), the intervention was not effective at reducing symptoms of clinical perfectionism as measured by the CPQ, despite finding reductions in alternate measures of perfectionism i.e. CM and PS subscales of the FMPS. This finding is significant as the intervention is designed to address clinical perfectionism, defined by Shafran et al (2002). Interestingly, the treatment significantly reduced levels of clinical perfectionism from pre-treatment to post-treatment, however this effect was also observed in the control group as well. Therefore, the reduction in
clinical perfectionism cannot be attributed to the treatment and these findings do not provide evidence for the efficacy of treatment in reducing clinical perfectionism as measured by the CPQ. One possible explanation for these findings is the poor evidence of psychometric properties of the CPQ in this sample (see Chapter 4, Study 1). Whilst the findings of Study 1 only provide preliminary evidence for the suitability of the CPQ, previous literature has found strong support for its psychometric properties (Chang & Sanna, 2012; Dickie et al., 2012; Egan et al., 2016; Steele et al., 2011). Another possible explanation for this non-significant finding could be the low self-reported adherence to the treatment modules and assigned homework, therefore leading to less exposure and practice of necessary techniques designed to address Shafran et al.’s (2002) construct, clinical perfectionism. This finding is not surprising considering that greater engagement in homework compliance leads to greater symptoms improvement for cognitive-behavioural interventions (Mausbach, Moore, Roesch, Cardenas, & Patterson, 2010).

Consistent with predictions, the treatment was effective at reducing dichotomous thinking at post-treatment (partial $\eta^2 = .17$) with a maintained effect at 4-month follow-up (partial $\eta^2 = .25$). This finding illustrated that the treatment was able to target a construct highly related to clinical perfectionism (Egan et al., 2007) and included as a key maintaining mechanism in the CBT maintenance model of clinical perfectionism (Shafran et al., 2010). This has important clinical implications as dichotomous thinking has also been associated with the development and maintenance of psychopathology, such as eating disorders (Lethbridge et al., 2011), and depressive and anxiety disorders (Egan et al., 2007), and is identified as a factor that can be associated with treatment relapse (Teasdale et al., 2001).

The findings of the current study also provide evidence for the secondary hypothesis, that CBT for clinical perfectionism will reduce symptoms of psychopathology. Although not directly targeted in the current study, the treatment significantly reduced symptoms of depression (partial $\eta^2 = 0.10$) and increased the participant’s quality of life (partial $\eta^2 = 0.14$), which was maintained at 4-month follow-up (depression, partial $\eta^2 = 0.14$; QLES-Q, partial $\eta^2 = 0.17$). Again, this is consistent with the findings of previous studies that have shown that associated psychopathology can be reduced with CBT for clinical perfectionism (Arpin-Cribbie et al., 2012; Pleva & Wade, 2007; Riley et al., 2007; Steele & Wade, 2008; Steele et al., 2013). Additionally, quality of life significantly improved from post-treatment to
follow-up suggesting that the intervention effect was continuing despite there being no active treatment across the four month time period. Previous literature has also observed this (Handley et al., 2015). It is possible that participants may need time to implement the strategies into their life that had been introduced in the 8-week treatment period, and therefore additional benefits take longer to take effect. The participants in the current study were encouraged to continue using the techniques that they had learnt in the previous eight weeks and were provided with a copy of all the material, making it easier to revise treatment techniques. Therefore, consistent with predictions the treatment effects were maintained at follow-up.

The intervention was not effective at reducing symptoms of anxiety and stress, similar to other studies (Arpin-Cribbie et al., 2012; Riley et al., 2007; Steele et al., 2013). The results suggest that symptoms of anxiety significantly reduced from pre-treatment to post-treatment for the intervention and waitlist control. Similar to this observed effect for the CPQ, as there was no significant difference across the two groups, the effect cannot be attributed to the treatment. Furthermore, no significant decrease in stress was observed across either of the treatment conditions. Interestingly, stress was observed to reduce in the waitlist control group but not the intervention group. It is possible that knowledge that treatment will commence in eight weeks may reduce the participant’s levels of anxiety or stress. The reduction in these symptoms could further explain the reduction in clinical perfectionism, as measured by the CPQ, from pre-post treatment, considering the strong association of perfectionism and anxiety (Frost & DiBartolo, 2002). A lack of significant differences in pre to post-treatment anxiety and stress has been observed in other studies. Steele and Wade (2008) observed no significant change in anxiety from pre-treatment to post-treatment across a guided self-help CBT intervention for perfectionism, a guided self-help CBT intervention for BN, and a placebo, mindfulness based cognitive therapy for depression. Arpin-Cribbie et al. (2012) found no significant changes from pre-treatment to post-treatment when anxiety was measured using the BAI. Furthermore, Arpin-Cribbie et al. (2012) found that anxiety scores remained in the clinical range at post-treatment. Additionally, Radhu et al. (2012) found no significant change in anxiety or stress from pre-treatment to post-treatment when comparing a web-based CBT intervention for perfectionism with a waitlist control.
Clinical perfectionism treatment delivered via self-help format may not be powerful enough to shift anxiety or stress. It is likely that more intensive therapist intervention may be required to shift this symptomatology, especially considering that challenging avoidance or safety behaviours is often required in those that experience anxiety (Barlow, 2008). The need for further therapist intervention is also evident by the reduction in rates of treatment exercise compliance from week one through to week eight. Whilst the readings modules were mostly completed throughout the intervention, the exercises that challenged the individual’s perfectionism were increasingly not completed by some participants, a recognised disadvantage of self-help approaches. These individuals would likely benefit from a treatment approach with a greater rate of therapist input through the intervention, especially considering that compliance in homework activities is associated with treatment outcome for CBT interventions (Westra, Dozois, & Marcus, 2007). There is support for the reduction of anxiety and stress when CBT for clinical perfectionism is delivered in a face-to-face format (Handley et al., 2015; Riley et al., 2007; Steele et al., 2013). This argument is supported by the findings of Egan, van Noort, et al. (2014), who found that pure self-help CBT for clinical perfectionism was not effective at reducing psychopathology; depression, anxiety and stress, compared to the face-to-face equivalent treatment. These findings suggest that it is not the treatment content per se that is not able to shift anxiety and stress, but rather the method of delivery.

As anticipated, the current sample presented with a significant rate of comorbidity (61.5%) and symptom severity, with scores on disorder specific measures within the clinical normative ranges. These presentations are similar to that of other studies that have found that individuals with perfectionism have complex symptomatology (Bieling, Summerfeldt, et al., 2004). Mixed results were observed in terms of diagnostic changes from pre-treatment to post-treatment. Although a reduction in DSM-IV diagnoses at post-treatment was observed, there was no significant difference in primary diagnostic change between the intervention and control group. A lack of literature in the area of self-help treatments for perfectionism limits explanation of this finding however this result is inconsistent with disorder specific self-help treatment trials for anxiety disorders (Lucock et al., 2008; Paxling et al., 2011). This could be attributed to the sample size and the small numbers of participants per diagnostic group i.e. one individual diagnosed with
OCD. It could also be that the self-help transdiagnostic treatment was not powerful enough to produce diagnostic shifts in the individuals, despite the reduction in depressive symptoms and increase in quality of life. Again, it is likely that guided self-help may not be powerful enough to shift primary diagnosis. It could be that the low intensity treatment delivery of guided self-help could limit treatment outcome in this complex clinical sample, given the sample had multiple comorbidities with fairly severe levels of symptoms despite a large proportion of the participants reporting that they had previously received treatment. A high intensity version of the intervention could result in greater diagnostic shift and anxiety symptom reduction. Supporting this argument is the findings of Egan, van Noort, et al. (2014), where 71% of individuals in their unguided self-help for perfectionism low intensity sample still met diagnostic criteria at post-treatment. This is in comparison to only 30% in the face-to-face high intensity version of the intervention. These findings support the notion of a stepped-care model, where by individuals that do not respond to low intensity interventions, can be offered high intensity alternatives.

Interestingly, the hypothesis that the intervention will be superior to the control group in decreasing the number participants presenting with comorbid diagnoses at post-treatment was confirmed. This finding is not surprising considering a key maintaining mechanism, clinical perfectionism, of anxiety, depressive and eating disorders had been targeted in the current treatment. These results support the use of a transdiagnostic treatment and Bieling et al.‘s (2004) argument that “…if perfectionism were treated directly, it is possible that the individual would experience symptomatic relief across a number of domains” (p.199). Therefore it may be more effective for individuals with elevated perfectionism and multiple diagnoses, to receive a treatment that targets their perfectionism; the underlying maintaining mechanism across disorder specific symptomatology.

Whilst there was no significant difference when using a categorical measure of recovery (i.e. the absence of primary DSM-IV diagnosis), the opposite effect was observed when using a dimensional measure of recovery. Primary diagnosis as measured by disorder specific measures, did significantly improve from pre to post-treatment for those in the treatment condition, with rates comparable to that of other studies. Paxling et al. (2011) observed clinically significant change in 42% of individuals receiving self-help treatment for GAD, whereas Riley et al. (2007) observed 75% of individuals improving after receiving treatment for clinical
perfectionism. The findings of the current study extend upon the results of Riley et al. (2007) and provides efficacy that a transdiagnostic self-help cognitive behavioural treatment for clinical perfectionism can effectively produce disorder specific clinical change in symptoms even though the treatment does not directly address disorder specific symptomatology.

Despite the strengths of the study; the use of an evidence based treatment manual and a sample presenting with a range of clinical characteristics, there are several limitations that warrant consideration. First, due to ethical considerations and not wanting participants presenting with clinical diagnoses to wait for up to six months, there was no pure control condition at follow-up. We therefore cannot conclude that the intervention effects were sustained at 4-month follow-up due to the treatment. Whilst this is undesirable, Chambless and Hollon (1998) acknowledge that this is a common outcome in clinical trials and conclusions about treatment efficacy can still be made.

Delivery of the whole treatment material at the start of the 8-week treatment period was a further limitation. This was done as the treatment was delivered via a hard copy book. The participants were instructed that due to the study and a recommendation provided by the authors to not read this manual all at once and to stick to the eight weeks guide that was provided by the clinician. This was reiterated to the participant each week by the therapist, although the therapist could not determine if these suggestions were adhered to. Previous research has looked at the efficacy of online CBT for clinical perfectionism, enabling the therapist to control the delivery of weekly modules (Egan, van Noort, et al., 2014). Additionally, there was no adherence to treatment used, meaning the therapist could only rely on the participants self-report of adherence to completed modules. The therapist implementing the treatment was the same therapist performing the diagnostic clinical interviews at each time point (the first author, KH). This is acknowledged as a limitation of the study as there was no blind assessment of individuals in the intervention and control group.

The findings of the study contribute to the evidence base suggesting that CBT for clinical perfectionism is an efficacious treatment across disorders. The intervention not only reduced symptoms of clinical perfectionism and constructs related to perfectionism such as dichotomous thinking, but also depression, despite the intervention not targeting these symptoms directly. The intervention was also
found to significantly improve quality of life. Despite guided self-help being termed a low intensity intervention that is secondary to face-to-face treatments, the results of the current study show that it has efficacy in reducing comorbidity and psychological severity of symptoms across clinical presentations. It is possible that low intensity intervention may not be efficacious for all clinical presentations so a stepped-care model should be considered. Whilst CBT for clinical perfectionism is not the most evidence based treatment intervention for individual diagnoses, the findings support the use of the treatment in reducing comorbidity diagnostic presentations and removing a maintaining mechanism of psychopathology, as well as reducing the severity of disorder specific symptoms. These findings support the use of a CBT guided self-help intervention for clinical perfectionism and demonstrate its efficacy at producing clinically significant change in treatment outcomes. The following chapter will detail the clinical implications of these findings and instruct on the appropriate delivery of the intervention.
Chapter 6: General Discussion

The following chapter will outline the major findings of the current research in regards to the assessment and treatment of clinical perfectionism. Areas of future research and how the findings can be translated into future practice will also be discussed.

6.1. Key Findings and Future Directions

Emerging literature suggests that the measure consists of two factors including evaluative concerns/perfectionistic concerns and personal standards/perfectionistic strivings (Dickie et al., 2012; Egan et al., 2016; Stoeber & Damian, 2014). Only one previous study has examined the factor structure of the CPQ in a clinical sample and this was an eating disorder only sample, which found evidence for this two-factor structure (Egan et al., 2016). There have not been any other studies to date which have examined the factor structure of the CPQ in other psychological disorders, therefore it is currently unknown if the two dimensions are relevant in a clinical population.

Study 1 (Chapter 4) assessed the psychometric properties of the two factors of the CPQ determined by Egan et al. (2016), in a mixed clinical sample (N = 32). Internal consistency was acceptable for Factor 1 but not for Factor 2, which is inconsistent with previous studies (Chang & Sanna, 2012; Dickie et al., 2012; Egan et al., 2016; Steele et al., 2011; Stoeber & Damian, 2014). The Factor 1 of the CPQ was found to significantly relate to PS subscale of the FMPS, whereas Factor 2 did not correlate to any of the measures of perfectionism. Due to the small sample size factor analysis was beyond the scope of Study 1. Therefore, it would be beneficial for future research to assess the factor structure of the CPQ in a mixed clinical sample to test the proposed factor structure of previous studies. Further research is required to extend upon the preliminary findings that the CPQ is an appropriate measure for use in clinical samples with a range of psychological disorders. Whilst previous research has validated the CPQ in university samples with sub-clinical symptomatology (Chang & Sanna, 2012; Dickie et al., 2012; Stoeber & Damian, 2014), only two other studies to date have assessed the psychometric properties of the CPQ in clinical samples and both of these were in eating disorder only samples (Egan et al., 2016; Steele et al., 2011). Whilst the findings of Egan et al. (2016) and Steele et al. (2011) did provide evidence for the reliability and validity of the CPQ,
these findings can only be generalised to eating disorders samples. Study 1 is the first to which the author is aware to assess the psychometric properties of the two factors of the CPQ in a mixed clinical sample. Clinical perfectionism has been proposed to be a transdiagnostic factor that maintains various psychopathology including eating disorders, as well as anxiety and depressive disorders (Egan, Wade, et al., 2011). It is therefore important for future research to assess the psychometric properties of the CPQ across all diagnostic groups.

Egan, Wade, et al. (2014) note that an imperative step in the assessment of client suitability for perfectionism treatment is to undergo a comprehensive assessment. The CPQ was developed by Shafran et al. (2002) to measure the construct of clinical perfectionism and therefore is a tool that can assist in assessment and formulation in clinical practice, however, it needs to be validated in clinical samples more widely to determine its suitability. Furthermore, the purpose of the CPQ is to provide a measure that is sensitive to change in treatment of perfectionism, which has shown promise in a number of trials to date (see Egan and colleagues (2011) for a review). The predictive validity of the CPQ also needs to be explored to see if it can predict treatment outcome. This has yet to be done in the field and is an important direction for future research.

In Study 2 (Chapter 5) an 8-week cognitive-behavioural guided self-help intervention for clinical perfectionism based on Shafran, Egan, and Wade’s (2010) self-help book was compared to a wait-list control. The study assessed changes in measures of perfectionism and psychopathology and explored the mediating effect of perfectionism on psychopathology in an elevated perfectionism sample (N = 40). Primary and comorbid diagnosis, and disorder specific symptomatology, for those presenting with DSM-IV diagnoses (N = 32), was also assessed from pre to post treatment.

The treatment reduced perfectionism, as measured by the CM and PS subscales of the FMPS and self-critical perfectionism as measured by the DAS-SC, dichotomous thinking, depression and increased quality of life. Furthermore, these treatment effects were maintained at four months follow-up.

The intervention was not effective at changing symptoms of anxiety and stress or clinical perfectionism, as measured by the CPQ. Interestingly CPQ and anxiety did significantly reduce from pre-post treatment but this effect was observed across the intervention and control condition. Several possible explanations are
offered that may explain this effect. First, as this finding was inconsistent with previous research that assessed the intervention in a face-to-face format (Egan, van Noort, et al., 2014; Handley et al., 2015; Riley et al., 2007; Steele et al., 2013), it is possible that the self-help format may not be powerful enough to shift these symptoms and additional therapist intervention is required. This interpretation is consistent with the findings of Egan van Noort, et al. (2014) where an unguided self-help version of the treatment was also found to not be effective in changing anxiety and stress, despite the face-to-face version being effective. It is also possible that an absence of significant reductions in the CPQ could be attributed to measurement error, as Study 1 (Chapter 3) only provided very preliminary psychometric support for the CPQ in this sample. Participant adherence to modules and assigned homework, was offered as an explanation for non-significant findings, as well as the reduction in an individual’s anxiety whilst waiting impending treatment.

Despite guided self-help being termed a “low intensity” intervention that is secondary to face-to-face treatments for the use in complex clinical samples, the results of the current study show that it can be effective at reducing perfectionism and some, but not all, associated psychopathology. Guided self-help therapies enable a greater distribution of therapeutic intervention to the community and individuals that may not have access to face-to-face individual therapy. A recommendation for future research is to compare different modes of self-help to face-to-face interventions. Whilst Egan, van Noort, et al. (2014) compared pure self-help to face-to-face CBT for clinical perfectionism, there is no other research to date that has compared the guided self-help version to differing modes of treatment delivery. A guided self-help version of the intervention needs to be compared with other treatment delivery modes to assess the hypothesis that the increased therapist interaction can result in reductions of greater reductions of symptoms.

The current study compared the transdiagnostic intervention to a waitlist control. Whilst this design was able to determine that the treatment did result in a significant improvement in outcomes, conclusions about its superiority over alternative treatments cannot be made. The RCT conducted by Steele and Wade (2008) is the only study to date comparing a transdiagnostic intervention for perfectionism with a disorder specific active treatment, namely CBT for BN. The outcomes were comparable across the two interventions, suggesting that the disorder specific intervention was not superior to the transdiagnostic one. However,
importantly, there was larger effect sizes found for reduction of associated anxious and depressive symptoms in the eating disorder sample for perfectionism treatment compared to CBT for BN in the eating disorder population (Steele & Wade, 2008). These results however are only generalizable to eating disorder populations. To the authors knowledge there are no published studies comparing CBT for clinical perfectionism with other disorder specific active treatments in anxiety or depressive disorder samples. As the results of the current study, and other RCTs (Egan, van Noort, et al., 2014; Handley et al., 2015; Riley et al., 2007), perfectionism treatment does result in improvement, the next step for research is to compare the transdiagnostic intervention with the most evidence based intervention for eating disorders, anxiety disorders and depression.

An additional aim of the RCT was to see if the treatment was effective at reducing the occurrence of diagnoses and presence of comorbidity, as perfectionism has been identified as a factor maintaining a variety of diagnoses. Contrasting results were observed. When using a categorical measure of recovery (i.e., the absence of diagnosis), there was no significant difference between the treatment group and the control at post-treatment. This is in contrast to previous research which has found a significant reduction in categorical diagnoses with perfectionism treatment compared to control (Egan, van Noort, et al., 2014). However, when using a dimensional measure of recovery (i.e., clinically significant change on disorder specific measures), there were significant improvements from pre to post-treatment for those in the treatment condition. These results also contradict the findings reported in Study 2 that anxiety and stress as measured by the DASS-21 did not significantly improve after treatment. Although disorder specific symptomatology was not directly targeted in the treatment, there were clinically significant reductions in disorder specific psychopathology. These findings further support the notion that perfectionism is a maintaining mechanism across psychopathologies and can explain the presence of comorbidity (Egan, Wade, et al., 2011).

6.1.1. Limitations of the Thesis

Limitations of the specific studies have been addressed in Chapters 4 and 5, however two general limitations of the thesis are worth noting. First, due to the design of the RCT, there was no pure control condition at 4-month follow-up. This was due to ethical reasons and not wanting to ask clients presenting with complex clinical presentations to potentially wait 6-months for active treatment (8-week
waitlist and then 4-month follow-up). This means that the intervention and control condition could not be compared at follow-up to see if the significant differences observed between the groups were maintained.

A further limitation was the absence of an alternative comparison treatment, and therefore limits in making claims of efficacy i.e., that the intervention was superior to a disorder specific intervention. Whilst the gold standard RCT design contains the targeted intervention, alternative treatment, and control, Chambless and Hollon (1998) acknowledge that this is a common dilemma in clinical trials and conclusions about treatment efficacy can still be made.

A final overall limitation was the sample size and rate of attrition. A priori power analysis determined that 38 individuals would need to be recruited to the study \( n = 19 \) per condition, as anticipated effects were large and the adopted analysis, GLMM, would account for participant attrition. Whilst the attrition rate of 12.5\% at post-treatment, and 32.5\% at 4-month follow-up are relatively high, the rates are comparable to that of other self-help CBT interventions for perfectionism (Pleva & Wade, 2007; Radhu et al., 2012).

### 6.2. Treatment Accessibility: Evidence-Based Treatment to Evidence-Based Practice

The findings of the RCT provide support for CBT for clinical perfectionism being an efficacious treatment and therefore increasing the current evidence base for the intervention. Despite this, a concern in the field is that evidence-based treatment is not necessarily translating to evidence-base practice. Barlow (2008) acknowledges that evidence-based practice is fundamental in the delivery of quality treatment across a range of disorders. However, emerging research suggest that there are several barriers to the implementation of evidence-based treatment protocols (Shafran et al., 2009). Gyani, Shafran, Myles, and Rose (2014) assessed 736 psychologists in the United Kingdom and found that therapists are significantly more likely to base their clinical decisions on personal experience with clients, individual and peer supervision, reviewing case studies, clinical observations and outcome measures, than empirical evidence from clinical trial research. Whilst we do have evidence-based practice guidelines (e.g., NICE guidelines), research informs us that this is not being translated to the clinical setting. Shafran et al. (2009) reviewed
several barriers to the dissemination of evidence based CBT in clinical care and provided several key recommendations including;

“…a need for more research on efficient ways of disseminating treatment procedures… Methods to establish which patients would benefit from lower intensity interventions and which require more face-to-face contact are required” (p. 907).

6.2.1. Alternatives to Face-to-Face Interventions

Alternative ways of efficiently disseminating evidence-based treatments need to be researched. Alternatives to face-to-face interventions are proving increasingly popular as demand for psychological services increase (IAPT, 2010) and self-help formats of treatment delivery are leading the forefront. The Australian government has identified that an individualised approach to mental health is required as individuals “fall through the cracks’ of the current system. The National Blueprint of Mental Health Services (2015) outlines that investment in mental health technologies is required over the next three years to target this concern. There are two main advantages of adopting a self-help approach. First, they are time efficient for the therapist, therefore reducing cost of treatment to the individual. This is a significant benefit as cost could be a barrier in access to psychological treatment. In Australia, it is estimated that treatment for eating and obsessive compulsive disorders on average cost the individual $21 565 in 2009-2010 (Butterfly Foundation for Eating Disorders, 2012). This is obviously not sustainable for some individuals in the community. The second main benefit of adopting a self-help approach to treatment delivery is that it allows for a great dissemination of evidence-based treatments to the community. This could be because therapy is not limited to standard business hours that enable consumers to engage in treatment and hours convenient for them and at a location of their choosing. It is also likely that self-help interventions will appeal to individuals that do not perceive their symptoms warrant intervention from a therapist per se but are of reasonable concern. Self-help interventions may also enable greater access to treatment for individuals experiencing shame or embarrassment about their psychological concerns and may be reluctant to attend a practice or clinic in person.

However, advantages of self-help interventions are also coupled with a number of disadvantages or limitations. There is a vast amount of literature publicly available on the internet that is not evidence based, therefore making it challenging for the individual to choose the most appropriate treatment for their needs.
Furthermore, the individual may lack the necessary insight into their current concerns or not diagnose their current symptoms accurately and then spend time engaging in an intervention that is not in their best interest or addresses their psychopathology. These limitations can be overcome by adopting a guided self-help approach. This argument provides further rationale for adopting guided self-help interventions to be empirically validated in RCTs. Most face-to-face interventions addressing a wide range of diagnoses have been translated into a guided self-help format, and now with the results from the current research there is preliminary evidence to support the efficacy of a guided self-help clinical perfectionism treatment. Guided self-help is the recommended approach as it will enable a trained mental health professional to conduct a comprehensive assessment of the client’s needs in order to recommend an appropriate intervention. A further benefit to a guided approach is reducing treatment drop out. Meta-analyses comparing the difference between treatment outcome of face-to-face and guided self-help interventions for depression and anxiety and found no difference in rates of treatment drop out across the two methods of treatment delivery (Cuijpers et al., 2010; Hirai & Clum, 2006). These findings challenge previous arguments that self-help treatments have higher levels of dropouts compared to that of the more traditional means of treatment delivery (Rosen, 1987).

The limitations of self-help interventions can be overcome with minimal therapist contact, such as a phone and email contact, or infrequent sessions throughout the intervention. Primary health settings would benefit from adopting guided self-help strategies as an alternative to face-to-face interventions that are costly in time and money for the individual and community. Time efficient therapies also enable the practitioner to condense their client contact time enabling them time to engage in further training and supervision, processes identified by Shafran et al. (2009) as impacting the dissemination of evidence-based therapies.

6.2.2. Transdiagnostic versus Disorder Specific Treatments

Transdiagnostic therapies can also assist the delivery of evidence-based practice in clinical settings. As it currently stands, CBT for clinical perfectionism is not the most evidence-based intervention for specific disorders as there is a vast amount of empirical support for disorder specific approaches (e.g., see www.nice.org.uk for specific recommendations). This may change in the coming years as there is increasing support demonstrating that CBT for clinical perfectionism is efficacious in reducing symptoms of disorders (Egan, van Noort, et al., 2014;
Handley et al., 2015; Riley et al., 2007; Steele et al., 2013). Whilst a comparison of a disorder specific and perfectionism treatment has been done in BN samples (Steele & Wade, 2008) to date there has been no such comparison in obsessive compulsive, anxiety and depressive disorder samples. Until there is further empirical support for transdiagnostic therapies the most evidence based approach is to treat the individual disorder. This is problematic for therapists prescribing treatment protocols when individuals present with multiple diagnoses. As seen in the current study, a significant amount (62%) of clients presenting to treatment have comorbidity. This is also common amongst clinical practice (Andrews et al., 2001; Kessler et al., 2005), however there are few evidence based recommendations for managing comorbidity (Craske et al., 2007). This could be a further reason why evidence based treatments are not being used in clinical practice. In order to overcome this barrier to evidence based dissemination we need to be able to provide evidence for treatments that target comorbidity. Transdiagnostic treatment can help address comorbidity (Egan, Wade, et al., 2011). It is possible that the use of shared and collaborative transdiagnostic formulations can be a therapeutic tool and provide psychoeducation to the client about the function of their perfectionism, potentially leading to an improvement in treatment outcome, for example, perfectionism maintaining their excessively high social standards and reinforcing social anxiety, which leads to symptoms depression and isolation. However, at present the therapist is advised to distinguish between primary and secondary diagnoses and then apply the appropriate interventions sequentially (Craske et al., 2007). For example, someone with a primary presentation of OCD and secondary depression, the most evidence based approach would be for the therapist to administer exposure and response prevention (Lack, 2012), followed by another CBT or Interpersonal Therapy intervention for the depression (Parker & Fletcher, 2007). The current method of treating individual diagnoses is not time efficient however appropriate in some cases. In other cases when underlying mechanisms (e.g., clinical perfectionism, or low self-esteem) is maintaining the psychopathology it may be more appropriate to use transdiagnostic interventions instead.

In their treatment manual “Cognitive Behavioural Treatment of Perfectionism”, Egan, Wade, et al. (2014) have provided guidelines to clinicians for the implementation of CBT for clinical perfectionism. Just because a client presents with clinical perfectionism, it does not necessarily mean that CBT for clinical
perfectionism is the most appropriate treatment to proceed with. The authors first describe that an integral part of deciding on the appropriateness of the intervention is to conduct a thorough formulation of the clients presenting problems using a functional analysis. This is because the recommendation to proceed with the intervention or not is dependent on the function of the individual’s perfectionism and their psychopathology (i.e., did their perfectionism cause the psychopathology or did their perfectionism develop as a consequence of their psychopathology). If it is determined that perfectionism is exclusively the presenting problem, it is recommended to proceed with the transdiagnostic treatment. In clinical practice however it is more likely that a client presents with numerous psychopathology and presenting concerns.

There is evidence to suggest that transdiagnostic processes can negatively impact on disorder specific treatment outcome and can result in treatment dropout (Sutandar-Pinnock et al., 2003). If the individuals’ perfectionism is a maintaining factor for the primary presenting problem, such as depression or anxiety, Egan, Wade, et al. (2014) recommend considering the implementation of CBT for clinical perfectionism instead of the disorder specific intervention to eliminate a maintaining factor of the presenting disorders. A transdiagnostic approach in this case can treat underlying maintain mechanisms of the diagnosis (e.g., perfectionism, low self-esteem), within a 12-week time frame. By removing a maintaining mechanism across disorders it can be treated more cost and time effectively than treating each disorder individually (Bieling, Summerfeldt, et al., 2004). The authors note that this is to be carefully considered as the evidence base as it currently stands would recommend implementing the treatment for depression or anxiety. It is this clinical dilemma that highlights the importance of conducting RCTs comparing the current most evidence-based disorder specific treatments and transdiagnostic interventions.

If perfectionism has been identified as a predisposing factor and has led to the development of the presenting problem it is recommended to implement the most evidence-based disorder specific intervention and then consider commencing CBT for clinical perfectionism to assist with relapse prevention (Egan, Wade, et al., 2014). Egan, Wade, et al. (2014) also advise using the disorder specific intervention if the individual’s perfectionism has developed as a result of the primary presenting problem. In this case, theoretically, once the psychopathology has improved the
perfectionism will not be a concern, therefore it would not be appropriate to implement CBT for clinical perfectionism (Egan, Wade, et al., 2014).

Shafran et al. (2009) report that a further barrier to the dissemination of evidence-based treatments could be that therapists are not adequately trained or have the time to be trained in the vast amount of necessary treatment protocols. To offer the most evidence-based practice clinicians must be trained in a wide variety of treatment protocols, for example CBT-E for bulimia nervosa, exposure and response prevention for OCD, and CBT for social anxiety disorder. It could be argued that in order for therapist to be appropriately trained in treatment protocols it would be more effective to be highly skilled in transdiagnostic treatments that can be applied across disorders, then overwhelmed by several disorder specific treatment protocols. Transdiagnostic treatments are easy to administer and clinicians can be trained more easily than if learning several disorder specific treatment protocols (Egan, Wade, et al., 2014). Transdiagnostic CBT includes several common techniques used in disorder specific treatments, including behavioural experiments, cognitive restructuring, and surveys.

6.3. Conclusion

To conclude, this research found that CBT for clinical perfectionism delivered in a guided self-help format was effective at reducing perfectionism and associated psychopathology, whilst improving quality of life. Literature pertaining to clinical perfectionism is currently in its infancy compared to other presenting concerns, such as the depression field. The evidence base is building for CBT for perfectionism with numerous RCTs now published, and meta-analyses attesting to the efficacy of the treatment in reducing perfectionism and psychological symptoms (Lloyd et al., 2015). Whilst transdiagnostic therapies are not currently the most evidence-based approach for treatment of specific disorders in comparison to the large evidence base that is existing for disorder specific treatments (see NICE guidelines), they do offer a solution to the dissemination of psychological therapies for those not otherwise able to access treatment.

To extend upon the findings of the current research, future studies should compare CBT for clinical perfectionism with another active treatment, such as a disorder specific intervention, and a control. Disorder specific approaches to treatment are currently the recommended most evidence-based practice. RCTs, such as in the current research, need to be conducted to contribute to the growing evidence
that transdiagnostic interventions are efficacious. The use of a transdiagnostic intervention also means that multiple comorbidities can be treated consecutively resulting in a more time and cost efficient alternative for the individual, clinician, and treatment providers, than disorder specific interventions. Ultimately the aim of this research into treatments such as CBT for perfectionism and other transdiagnostic approaches and self-help formats is to improve the efficacy and dissemination of evidence based treatments for psychological disorders to the wider population throughout the world.
References


disorder. *Journal of Anxiety Disorders*, 22(3), 540-547. doi: 10.1016/j.janxdis.2007.05.004


functioning in obsessive-compulsive disorder and body dysmorphic disorder.


distress and the frequency of perfectionistic thinking. *Journal of Personality
and Social Psychology, 75*(5), 1363-1381. doi: 10.1037/0022-3514.75.5.1363
Child-Adolescent Perfectionism Scale: Development, validation, and
association with adjustment.
(2012). Perfectionistic automatic thoughts and psychological distress in
adolescents: An analysis of the perfectionism cognitions inventory. *Journal
of Rational-Emotive & Cognitive-Behavior Therapy, 30*(2), 91-104. doi:
10.1007/s10942-011-0131-7
Hewitt (Eds.), *Perfectionism: Theory, research, and treatment*. Washington,
styles. *Individual Psychology: Journal of Adlerian Theory, Research &
Practice, 51*(1), 50-60.
Cognitions Inventory: Psychometric properties and associations with distress
and deficits in cognitive self-management. *Journal of Rational-Emotive &
Foa, E. B., Huppert, J. D., Leiberg, S., Langner, R., Kichic, R., Hajcak, G., &
Salkovskis, P. M. (2002). The Obsessive-Compulsive Inventory:


Iketani, T., Kiriike, N., Stein, M., Nagao, K., Nagata, T., Minamikawa, N., . . .


Iketani, T., Kiriike, N., Stein, M. B., Nagao, K., Nagata, T., Minamikawa, N., . . .


Jacobs, R. H., Silva, S. G., Reinecke, M. A., Curry, J. F., Ginsburg, G. S.,


obsessive-compulsive disorder. *Bulletin of the Menninger Clinic, 78*(2), 140-159. doi: 10.1521/bumc.2014.78.2.140


Noble, C. L. (2013). The relationships among multidimensional perfectionism, shame and trichotillomania symptom severity. *Dissertation Abstracts*


distress. *Journal of Clinical Psychiatry, 72*(6), 780-786. doi: 10.4088/JCP.10m06380


individualized e-mail therapy: A randomized trial of two versions of CBT for major depression. *Behaviour Research and Therapy, 48*(5), 368-376. doi: 10.1016/j.brat.2010.01.005


*PCL-C for DSM-IV*. Boston: National Center for PTSD - Behavioral Science Division.


*Behavioural and Cognitive Psychotherapy*, 36(6), 675-683. doi: 10.1017/S1352465808004864


Appendices

Appendix A: Participants Starter Package

Treatment for Clinical Perfectionism

The questions in this booklet are designed to determine if you have elevated perfectionism. You will first see an information sheet and consent form attached. If you agree to take part in the study, please sign and return the consent form and the completed questionnaire. Please keep the information sheet for yourself.

Thank you for your time
Information Sheet
Cognitive Behavioural Therapy for Clinical Perfectionism

You have been invited to participate in a project that will be exploring the efficacy of a treatment for Clinical Perfectionism. Previous studies have shown that perfectionism can lead to anxiety disorders and depression. This study aims to see if treating perfectionism leads to a reduction in symptoms of perfectionism, anxiety and depression.

Voluntary participation
Your participation in this study is completely voluntary, so if you do not want to participate in this study you do not have to. If you decide to take part in the study but later change your mind, you have the right to withdraw at anytime. There will be no negative consequences if you withdraw and you don’t have to explain why. You can choose to continue to receive treatment, or be referred elsewhere.

Purpose of the research
The purpose of this study is to assess the efficacy of Cognitive Behavioural Therapy for Clinical Perfectionism. Participants will attend a clinical interview at the Curtin University Psychology and Speech Clinic, Bentley Campus. They will then be given a self-help book titled “Overcoming Perfectionism: A self help guide using cognitive behavioural techniques” to be read weekly for 8 weeks. Each participant will be asked to fill out questionnaires before and after the 8 week period. They will receive a call from a therapist each week to see how they are going and discuss any problems that may arise.

Some participants will be asked to wait for 8 weeks to receive their treatment.

What does the study involve?
1) First, if you are interested in participating in the study you will read and sign the consent form, you must be over 18 years of age. You will also complete the questionnaire attached and return both of them using the reply paid envelope.
2) Second, you will be contacted by a Clinical Psychologist Trainee, via telephone. You will be asked a number of questions to see if you are suitable to receive the treatment that this study offers. This call will take approximately 15-20 minutes. If you are eligible you will be asked to come into the Curtin University Psychology and Speech Clinic for a clinical interview. If you are not eligible for the study you will be provided with some appropriate referrals.
3) The clinical interview will take approximately 90 minutes. We will talk about some of the problems you are experiencing at the moment.
4) After this you will be randomly allocated the treatment for perfectionism or an 8 week waitlist group. After the 8 weeks participants will then receive the treatment.
5) You will be required to be available to complete the readings, exercises and talk on the phone throughout the 8 weeks of treatment.
6) Throughout the treatments you will be asked to complete a number of questionnaires. You will complete one questionnaire package before your treatment and one after your treatment.

7) Four months after you have finished treatment you will be sent a final questionnaire package and required to come into the Curtin University Psychology and Speech Clinic to see how you have been going.

**Potential risks**
Treating perfectionism may reduce unhelpful aspects of perfectionism but also symptoms of anxiety and depression. However, there is no guarantee that you will benefit from the treatment. You have the right to withdraw from the study at any time.

**Alternative Treatments and Medications**
During the treatment and up to four months after the treatment, we encourage you to not receive any other form of Cognitive Behavioural Therapy, for example visiting another psychologist. If you are on any antidepressant medication we ask that you must be stable on this medication for at least three months. We also ask that if you are on antidepressant medication, to agree not to alter/change the medication and the dosage for the duration of the study. That is, from the clinical interview to the 4 month follow up period.

**Confidentiality**
All records containing personal information that we collect will be kept strictly confidential and will be locked in a file at Curtin University for 5 years. Each participant will be allocated a three digit code and that will be used to identify your data. No names will be attached to your questionnaires. No one external to the study will be able to see any of the information we will collect. If the research is published, no individuals will be identified.

All of the clinical interviews will be recorded on a DVD. This is so I can receive feedback about my performance as a Clinical Psychologist Trainee from my Supervisor, Dr Sarah Egan. All DVDs will be destroyed after this process.

**Further Information:**
Should you require any further information please contact myself Kimberley Hoiles on (08) 9266 3436 or email kimberley.hoiles@postgrad.curtin.edu.au or my supervisor Dr Sarah Egan on (08) 9266 2367 or email s.egan@curtin.edu.au

*This study has been approved by the Curtin University Human Research Ethics Committee (Approval Number HR 120/2010). The Committee is comprised of members of the public, academics, lawyers, doctors and pastoral carers. Its main role is to protect participants. If needed, verification of approval can be obtained either by writing to the Curtin University Human Research Ethics Committee, c/- Office of Research and Development, Curtin University of Technology, GPO Box U1987, Perth, 6845 or by telephoning 9266 2784 or by emailing hrec@curtin.edu.au.*
Consent Form

Please return this page

I, …………………………………………

Have fully read the above information sheet and consent to be part of the study.

- I understand that I will receive a self-help book that treats perfectionism. I may also be asked to wait 8 weeks until I receive a self-help book.
- I understand that this allocation is random and I can’t choose if I have to wait 8 weeks or not.
- I understand that I will need to complete some questionnaires before and after the treatment.
- I agree not to pursue alternate Cognitive Behavioural Therapy until I have finished the treatment and up to 4 months post treatment.
- If I am taking antidepressant medications, I agree that I have been stable on my medication for the past 3 months. I also agree to not change/alter my medication and dosage until I finish the study.
- I understand that my personal information will be kept completely confidential and if the research was to be published I will not be able to be identified.
- I understand that my data will be retained for 5 years in a locked cabinet at the Curtin University Psychology and Speech Clinic.
- I understand that my clinical interview will be recorded on DVD but these will be kept in a locked cabinet in the Curtin University Psychology and Speech Clinic and destroyed after the study has finished.
- I understand that there is a chance that I may not benefit from this treatment.
- I understand that I am able to withdraw from the study at any stage without having to give a reason and that by withdrawing I may continue to receive treatment or be referred elsewhere for treatment.

Signature …………………………………………… Date …../....../......
Appendix B: Advertisement

Does everything have to be perfect?

Always feel like you could do better?

Do you strive for excessively high standards, despite negative consequences such as anxiety and depression? Are you excessively self-critical about your achievements?

If you answered yes to the above questions, then it is possible that you are suffering from Clinical Perfectionism.

We invite you to take part in a study that will be assessing the efficacy of Cognitive Behavioural Therapy (CBT) for Clinical Perfectionism. Studies suggest that CBT can be an effective treatment for Clinical Perfectionism and for Anxiety and Depression. This study will be assessing a guided self-help version of CBT for Clinical Perfectionism.

We will be testing to see if CBT for clinical perfectionism is not only effective at reducing levels of perfectionism, but associated symptoms such as depression and anxiety as well. After screening for suitability, you will be booked in for an individual assessment with a Clinical Psychology Trainee at the Curtin University Psychology and Speech Clinic.

For more information please ring Kimberley Hoiles at the Curtin University Psychology and Speech Clinic on 9266 3436 or email kimberley.hoiles@postgrad.curtin.edu.au You must be at least 18 years old to participate. There is no cost involved for you.
Appendix C: Graphs of the interactions for each Perfectionism outcome variable

Clinical Perfectionism (CPQ)  Concern over Mistakes (CM)

![Graph of Clinical Perfectionism (CPQ)](image1)
![Graph of Concern over Mistakes (CM)](image2)

Personal Standards (PS)  Self-Criticism (SC)

![Graph of Personal Standards (PS)](image3)
![Graph of Self-Criticism (SC)](image4)
Dichotomous Thinking (DT)

Figure 6. Mean perfectionism outcome scores at pre-treatment and post-treatment for each group. Error bars, which represent 95% confidence intervals, are offset horizontally to make them visible.
Appendix D: Graphs of the interactions for each Perfectionism outcome variable at follow-up

<table>
<thead>
<tr>
<th>Concern over Mistakes (CM)</th>
<th>Personal Standards (PS)</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image1" alt="Graph CM" /></td>
<td><img src="image2" alt="Graph PS" /></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Self-Criticism (SC)</th>
<th>Dichotomous Thinking (DT)</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image3" alt="Graph SC" /></td>
<td><img src="image4" alt="Graph DT" /></td>
</tr>
</tbody>
</table>

**Figure 7.** Mean perfectionism outcome scores at pre-treatment, post-treatment and follow-up for the treatment group. Error bars, which represent 95% confidence intervals, are offset horizontally to make them visible.
Appendix E: Graphs of the interactions for each Psychopathology outcome variable

Depression

Anxiety

Stress

Quality of Life (QLESQ)

Figure 8. Mean psychopathology outcome scores at pre-treatment and post-treatment for each condition. Error bars, which represent 95% confidence intervals, are offset horizontally to make them visible.
Appendix F: Graphs of the interactions for each Psychopathology outcome variable at follow-up

**Depression**

**Quality of Life (QLESQ)**

*Figure 9.* Mean psychopathology outcome scores at pre-treatment, post-treatment and follow-up for the treatment group. Error bars, which represent 95% confidence intervals, are offset horizontally to make them visible.