

**Informing the Development of More Effective Prevention  
Interventions for Eating Disorders and Disordered Eating**

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## ABSTRACT

Eating disorders, including threshold and subthreshold disordered eating, are prevalent and are associated with elevated mortality rates, impairments across several health dimensions, and reduced quality of life. The development and implementation of effective prevention programs is imperative in order to reduce the burden of this serious mental health problem. Despite recent advances in the eating disorder prevention field, the vast majority of widely disseminated public health interventions are not informed by theory and lack empirical support. Thus there is a need to develop more effective interventions for eating disorders that integrate theory and intervention development.

The first study was a systematic review of the current eating disorder literature, including an examination of the degree to which eating disorder models have informed interventions, and a summary of the variables commonly considered in the most promising models. While an extensive range of theoretical models were identified, few models led to the development of effective interventions. One exception was the dual-pathway model for bulimia nervosa which has informed the gold standard of prevention approaches for eating disorders in young women. Despite the model's success, several critical limitations were identified.

The second study therefore investigated whether this model could be improved, additional temperament factors (i.e., perfectionism, self-efficacy, self-compassion, and difficulties with emotion regulation) were investigated and the model was applied to the full range of disordered eating disturbances and a global measure of eating psychopathology. The model was examined cross-sectionally and longitudinally in a sample of women considered to be at risk of disordered eating ( $N = 167$ ), and sociocultural influences, body dissatisfaction, and difficulties with emotion regulation were identified as potentially robust predictors.

The final study was a randomised controlled trial which specifically targeted emotional regulation through the use of imagery rescripting and postulated increases in self-compassion. We compared two brief interventions, namely imagery rescripting and cognitive dissonance, to a control condition, in terms of reducing disordered eating and modifying risk and protective factors for eating disorders. Change in state variables was examined within the experimental session in a sample of women at risk of disordered eating ( $N = 201$ ;  $M_{age} = 20.18$  years), and support was found for both imagery rescripting and cognitive dissonance interventions compared to the control in increasing self-compassion. Change in trait variables was subsequently examined over the one-week follow-up for those women considered to be at risk of developing an eating disorder ( $N = 107$ ;  $M_{age} = 20.27$  years), and support was found for the imagery rescripting intervention in increasing body image acceptance and self-compassion and reducing disordered eating.

Taken together, these findings identify a number of key variables of pertinence to the development and/or maintenance of disordered eating that ought to be considered in the revision of models that inform the development of future interventions, including those which appear to be most effective for change within prevention research.

## **DECLARATION**

I certify that this thesis does not incorporate without acknowledgment any material previously submitted for a degree or diploma in any university; and that to the best of my knowledge and belief it does not contain any material previously published or written by another person except where due reference is made in the text.

Jamie-Lee Pennesi

BPsychHons

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## **Chapter 1**

### **Overview and Aims of the Research**

## **1.1 Background and Context**

### **1.1.1 Impact of eating disorders and disordered eating.**

Eating disorders are widely recognized as a serious issue that warrants attention, akin to other serious mental health conditions including schizophrenia, bipolar, depression and obsessive-compulsive disorder (Klump, Bulik, Kaye, Treasure, & Tyson, 2009). Eating pathology, including threshold and subthreshold disordered eating, is marked by chronicity, distress, impaired psychosocial functioning and reduced quality of life, and is associated with elevated risk of mortality and suicide (Fichter & Quadflieg, 2016; Stice, Marti, & Rohde, 2013; Winkler et al., 2014). Further, disordered eating increases the risk of developing other health-related problems such as obesity, depression, anxiety, cardiovascular symptoms, chronic fatigue, physical morbidity, infectious diseases, insomnia, and neurological symptoms (Fairweather-Schmidt, Lee, & Wade, 2015; Goldschmidt, Wall, Choo, Becker, & Neumark-Sztainer, 2016; Johnson, Cohen, Kasen, & Brook, 2002; Swanson, Crow, le Grange, Swendsen, & Merikangas, 2011). Treatment is often difficult, with estimates suggesting up to 25% of individuals retain their eating disorder symptoms up to 15 years after treatment (Herzog et al., 1999; Strober, Freeman, & Morrell, 1997). At the service level, eating disorders are also associated with increased health care utilisation and represent a major burden on public health services (Ágh et al., 2016; Mond, Hay, Rodgers, & Owen, 2007).

### **1.1.2 Prevalence of eating disorders and disordered eating.**

Disordered eating and associated problems with weight and shape are prevalent (Allen, Byrne, Oddy, & Crosby, 2013; Goldschmidt et al., 2016; Hay, Mond, Buttner, & Darby, 2008; Stice, Marti, et al., 2013) and have increased in recent years. For example, Hay and colleagues (2008) identified an over two-fold increase in self-reported disordered eating,

including binge eating, purging (i.e., self-induced vomiting and/or laxative or diuretic use), and strict dieting or fasting (i.e., going long periods without or hardly eating) for weight or shape control, in a sample of over 3,000 Australian women and men between 1995 and 2005. Similar increases have been found for binge eating in other cohort population-based studies in the United States (Hudson, Hiripi, Pope, & Kessler, 2007). Estimates suggest that almost one quarter of Australian women aged between 22 and 27 years have experienced disordered eating in the previous 12 months (Wade, Wilksch, & Lee, 2012). Further, up to 15% of females met the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013) criteria for one or more eating disorders by the age of 19 years (Fairweather-Schmidt & Wade, 2014; Wade & O'Shea, 2014). The lifetime prevalence for young females is estimated at 0.8% for anorexia nervosa (AN), 2.6% for bulimia nervosa (BN), 3.0% for binge eating disorder (BED), 2.8% for atypical AN, 4.4% for subthreshold BN, and 3.6% subthreshold BED, with an average episode duration estimated from 3 months for BN to almost 12 months for atypical AN (Stice, Marti, et al., 2013). In addition, there has been increasing awareness of the existence of eating disorders among men (Dakanalis & Riva, 2013; Murray et al., 2017; Tylka, 2011b), with recent epidemiological studies suggesting that males account for approximately one third of adults reporting eating disorder features such as extreme dieting and purging behaviour (Mitchison, Mond, Slewa-Younan, & Hay, 2013). Recent estimates suggest that the lifetime prevalence for young males is estimated at between 0.1% and 0.3% for AN, between 0.1% and 1.6% for BN, and between 0.3% and 2.0% for BED (Murray et al., 2017).

### **1.1.3 Developing prevention approaches.**

Given the increasing prevalence and adverse impacts of disordered eating, and the increased burden for individuals and on the public health system, work in the prevention of disordered eating, especially among those who are at risk, is considered to be an important

endeavour. As noted by Stice (2016), there is a pressing need to identify risk factors that predict the future onset of eating disorders and understand how these risk factors interact within multivariate models to predict the emergence of these conditions, in order to inform the content of future effective prevention programs. This strategy is consistent with the pathway towards the development of complex interventions identified by the Medical Research Council (MRC; Campbell et al., 2000; Craig et al., 2008, 2013; Craig & Petticrew, 2013). This framework, while not specific to eating disorders, has been highly influential and widely cited and offers a process by which to link theory with the development of intervention approaches within the field and develop more effective interventions. It is described that interventions should be developed systematically, using the best available evidence and relevant theory, followed by rigorous testing and evaluation, and finally dissemination and implementation. A comprehensive review of the framework, its rationale, and its various uses, is described in study one (Pennesi & Wade, 2016).

This framework is of particular pertinence to disordered eating and eating disorders, where much attention has been directed towards identifying associated risk factors and influences, and identifying etiological models that describe the mechanism of action (Pennesi & Wade, 2016). However, few models have led to the development of effective interventions, with some exceptions (Fairburn, 2008b; Stice, 2001). One of the most influential theories pertaining to the development of eating disorders, which has resulted in development of a prevention approach, is the dual-pathway model of bulimic pathology proposed by Stice (2001). The model proposes that perceived socio-cultural pressure to be thin (i.e., from family, peers, and the media; Irving, 1990; Stice, Schupak-Neuberg, Shaw, & Stein, 1994; Striegel-Moore, Schreiber, Pike, Wilfley, & Rodin, 1995) promotes both thin-ideal internalisation (i.e., heightened endorsement of the thin-ideal stereotype for women, to the point of modifying one's behaviours in an attempt to reach these standards; Thompson &

Stice, 2001) and body dissatisfaction, which leads to strict dieting and negative affect, which in turn results in the development of bulimic symptoms. This model informed the development of a cognitive dissonance-based intervention (DBI) targeting thin-ideal internalisation through use of cognitive-attitudinal activities such as asking participants to speak and act against the thin ideals of beauty (Stice & Presnell, 2007). Cognitive dissonance theory (Festinger, 1957) is based on the idea that holding cognitions inconsistent with one's beliefs creates psychological discomfort that motivates changes in behaviour and/or attitudes so as to reduce this inconsistency.

DBIs have been shown to be robustly effective prevention programs, resulting in reduced thin-ideal internalisation, body dissatisfaction, dieting, negative affect, and eating pathology, as well as reduced risk for onset of eating disorder symptoms (Halliwell & Diedrichs, 2014; Perez, Becker, & Ramirez, 2010; Stice, Butryn, Rohde, Shaw, & Marti, 2013; Stice, Rohde, Gau, & Shaw, 2009), with effects maintained up to 3 years later (Stice, Marti, Spoor, Presnell, & Shaw, 2008; Stice, Rohde, Shaw, & Gau, 2011). In a meta-analytic review by Stice, Shaw, and Marti (2007), DBIs (e.g., the *Body Project*; Becker & Stice, 2017; Shaw & Stice, 2016) were identified as the most effective targeted interventions to date for girls and women aged 14 years and above. One program that has received global acclaim, having successfully bridged the gap between efficacy and effectiveness research and large-scale dissemination and implementation (Kazdin, 2008), is the *Body Project (BP)* (Becker & Stice, 2017), which has been rolled out in 125 countries to date and reached over 3.5 million girls and young women. Further, the program has shown to be effective in clinician-led, peer-led and internet-delivered formats (Rohde, Stice, Shaw, Gau, & Ohls, 2017; Stice, Rohde, Shaw, & Gau, 2017).

Despite having a significant public health impact, the *BP* is not without its limitations. First, it is a selective prevention approach designed to reduce risk factors and symptoms in

individuals at risk of developing an eating disorder, which is distinct from universal prevention which is designed to prevent growth in risk factors and symptoms over time in all individuals regardless of level of risk (Gordon, 1983; Levine, 2017). Becker (2016) argues that these different (though related) strategies are often both labelled as “prevention” when indeed they are not. Researchers have indicated the need for more effective evidence-based universal programs that reduce the number of new cases (i.e., incidence) at the whole population level in order to achieve meaningful impact and advance population health (Filion & Haines, 2015; Wilksch, 2014; World Health Organization [WHO], 2004). Second, the *BP* does not appear to be uniformly effective (e.g., Atkinson & Wade, 2012), which raises concerns about program effectiveness. For example, Atkinson and Wade (2015) failed to find any significant effects for the *BP* for virtually all eating disorder risk factors. Third, a recent meta-analysis by Watson et al. (2016), identified that several other prevention interventions also show promise (e.g., media literacy; Wade, Davidson, & O’Dea, 2003; Wilksch et al., 2015) and are worthy of further research.

Finally, the model from which the *BP* was developed (Stice, 2001), overlooks influences related to temperament, such as perfectionism, self-efficacy, self-compassion and difficulties with emotion regulation, which have shown to be important in the development and maintenance of disordered (Bardone-Cone et al., 2007; Braun, Park, & Gorin, 2016; Gordon, Denoma, Bardone, Abramson, & Joiner, 2005; Sim & Zeman, 2006) and are considered among existing models of disordered eating (Pennesi & Wade, 2016).

Perfectionism has long been linked to anorexia nervosa, however recently researchers have also identified the role of perfectionism in bulimia nervosa. In his meta-analytic review, Stice (2002) concluded that perfectionism may act as a risk factor for bulimic symptoms and a maintenance factor for general eating pathology, and further that perfectionism may interact with other risk factors such as self-efficacy in producing or maintaining eating pathology

(Vohs, Bardone, Joiner, Abramson, & Heatherton, 1999). It is thought that perfectionism acts as a vulnerability factor which, in the context of an unmet standard (i.e., relating to weight or shape), produces an undesirable discrepancy which generates intense negative affect and aversive self-awareness, which motivates temporary escape through binge eating (Bardone-Cone et al., 2007). Thus perfectionism is considered to be an important variable that ought to be considered in the development of models for disordered eating.

Research has suggested that self-esteem is important in the aetiology and treatment of bulimia nervosa (Gordon et al., 2005). It is thought that low self-esteem makes women more vulnerable to external social pressures and more likely to try to achieve the thin ideal in order to feel more effective (Fairburn, Wilson, & Schleimer, 1993). Further, it is suggested that women with low self-esteem may be more susceptible to self-sabotaging bulimic behaviour (i.e., binge eating) because of their negative expectations for themselves (Vohs et al., 1999). Self-esteem has also been investigated as a prognostic indicator in the treatment of bulimia nervosa (Fairburn, Kirk, O'Connor, Anastasiades, & Cooper, 1987); it is suggested that low self-esteem is associated with less positive treatment outcomes. Recently, it has been suggested that self-efficacy (i.e., a dimension of global self-esteem) is a more relevant component of the construct of interest (i.e., doubt about one's ability to achieve; Gordon et al., 2005), and thus self-efficacy is also considered highly important in this context.

An emerging body of research has suggested a link between self-compassion and eating disorders. A systematic review by Braun et al. (2016) found support for the role of self-compassion as a protective factor against poor body image and eating pathology. It is thought that self-compassion may protect against eating pathology through various pathways, including reducing eating disorder-related outcomes directly, preventing the development of risk factors and/or eating disorder-related outcomes, and interacting with risk factors to interrupt their deleterious effects (Tylka & Kroon Van Diest, 2015). The relevance of self-

compassion is also suggested by the model of clinical perfectionism (Shafran, Cooper, & Fairburn, 2002), where those with high self-compassion are less likely to be self-critical and judge themselves harshly when high standards (i.e., relating to weight or shape) are not met and therefore are less likely to experience negative affect that can result in an increase in disordered eating behaviours. Thus further investigation of self-compassion as a protective factor within models of disordered eating is warranted.

A body of literature now supports the relationship between difficulties with emotion regulation and eating pathology (Gianini, White, & Masheb, 2013; Svaldi, Griepenstroh, Tuschen-Caffier, & Ehring, 2012). In his meta-analytic review, Stice (2002) identified negative emotionality as a major precursor for the development of eating pathology, body dissatisfaction, and calorie intake, suggesting that negative emotionality may work to amplify the effects of other risk factors. Specifically, it is suggested that inadequate coping skills or difficulties coping with adverse emotional states may cause women to become less effective at managing urges to binge (i.e., caused by attempts to restrict their eating) and less able to manage negative affect, thus relying more on disordered eating to regulate emotion (Stice, 1994). While negative affect is considered within the dual-pathway model, the contribution of difficulties with emotion regulation in particular is yet to be investigated. Together, this literature suggests that factors related to temperament (i.e., perfectionism, self-efficacy, self-compassion, and difficulties with emotion regulation) are important factors that ought to be considered within comprehensive models for disordered eating. Thus further work is required in order to examine eating disorder models and variables that make up an effective prevention approach.

## **1.2 Aims of the Current Research**

Despite significant developments in the eating disorder prevention field over the past decade, there remains several areas of concern. The majority of the prevention programs are

designed independently of theoretical consideration (Stice & Shaw, 2004); of twenty prevention studies reviewed by Austin (2000), fewer than half outlined a clear theoretical basis for the chosen preventive approach. Further, many widely disseminated public health interventions lack empirical support, raising concerns about program efficiency and the potential for waste of limited resources (Becker, Plasencia, Kilpela, Briggs, & Stewart, 2014). Thus there remains a pressing need to develop more effective interventions for eating disorders that clearly link theory (i.e. scientific research) and intervention development. The purpose of this thesis is therefore to explore the different linkages described in the MRC (Craig et al., 2008) evaluation framework in order to inform the development of future interventions for eating disorders or disordered eating. Specifically, it sets out to address three key aims:

1. Identify variables of particular pertinence to the development and/or maintenance of disordered eating.
2. Investigate theoretical models to identify key variables that ought to be considered in future interventions.
3. Investigate interventions for disordered eating to identify which variables appear to be most effective for change.

In order to address these aims, a series of studies were conducted. These studies appear as individual chapters, briefly described below.

### **1.3 First Study**

The first study (**Chapter 2**) is a systematic review of the current eating disorder literature. This study utilised the MRC (Craig et al., 2008) evaluation framework to critically examine the usefulness of the theories that have been developed for eating disorders and disordered eating, in terms of theories that have informed interventions, such as prevention and treatment. This study also examined the common variables included

across the most promising models in terms of future implications for the development of interventions for disordered eating. The findings of this study guided the development of subsequent approaches used in this thesis. This study has been published in the *Clinical Psychology Review* (Pennesi & Wade, 2016), and has been cited 9 times in *Scopus* as of 4<sup>th</sup> October 2017.

#### **1.4 Second Study**

The dual-pathway model for bulimia nervosa (Stice, 2001) was identified in study one as one of the front runners in terms of effective interventions, having progressed through the final stages of the MRC (Craig et al., 2008) framework and informed the gold standard of prevention approaches for eating disorders for young women with body image disturbance (e.g., DBI; Stice, Rohde, et al., 2009). Despite this model's success, it overlooks factors related to temperament, which have shown to be important in the development and maintenance of disordered eating and are included across eating disorder models (Pennesi & Wade, 2016), and does not account for the full range of disordered eating symptoms and behaviours, that are also associated with severe impairment (Wade & O'Shea, 2014). Further, longitudinal replications of the model are scarce (e.g., Engler, Crowther, Dalton, & Sanftner, 2006; Stice, 2001; Stice, Shaw, & Nemeroff, 1998).

Thus the second study (**Chapters 3 and 4**) investigated whether the dual-pathway model could be improved. First, we examined the addition of factors related to temperament, including perfectionism, self-efficacy, self-compassion, and difficulties with emotion regulation, which were indicated as key variables in study one. Second, the model was applied to the full spectrum of eating disturbances, including binge eating, fasting, self-induced vomiting, laxative or diuretic misuse, and driven exercise, and a global measure of eating psychopathology. Finally, the model was examined cross-sectionally and longitudinally in a sample of women considered to be at risk of disordered eating ( $N = 167$ ).

**Chapter 3** describes a cross-sectional study. The results of this study suggested that sociocultural influences, body dissatisfaction, and difficulties with emotion regulation may be implicated in the aetiology of eating pathology and that the dual-pathway model may benefit from the inclusion of factors related to temperament (i.e., difficulties with emotion regulation). However, this study utilised cross-sectional data and therefore causation could not be determined. This study was submitted to *Eating Behaviors*, and is currently under review awaiting decision. The version that appears in this thesis is the manuscript as submitted to the journal.

**Chapter 4** describes a longitudinal study that can better inform causality and thus overcomes the limitation of cross-sectional work. The study involved three waves of data collection over a 12-month period. This study provided further support for the key variables indicated in the previous cross-sectional study and further evidence for the role of self-compassion and negative affect in the development of disordered eating, again suggesting that the addition of temperament factors (i.e., difficulties with emotion regulation and self-compassion) may add value to the dual-pathway model. This study was submitted to the *International Journal of Eating Disorders*, and was subsequently rejected as it did not meet the minimum requirements of a longitudinal study i.e., only 34 participants (20.4%) completed all three phases of data collection and the data were not missing completely at random. Despite imputing missing data and utilising analyses to make maximum use of all available data, the reviewers were unanimous that the small number of cases over the three waves limited the ability to produce reliable effects that remain stable over time; it was strongly recommended that we only use the baseline data to conduct a cross-sectional examination of our proposed model. The version that appears in this thesis is the longitudinal analysis originally submitted to the journal.

## **1.5 Third Study**

Emotion regulation was shown to be an important predictor in study two. It is therefore important that approaches to reducing disordered eating address the need to develop skills for effective emotion regulation. One approach likely to be useful is compassion-focussed imagery (Gilbert & Irons, 2005), which is used in imagery rescripting (Wild & Clark, 2011) and has shown to be effective in the treatment of a range of psychological disorders including bulimia nervosa (Cooper, Todd, & Turner, 2007; Ohanian, 2002). However, imagery rescripting has yet to be investigated in eating disorder prevention. An alternative approach is cognitive dissonance, which has been established as an effective approach for reducing body dissatisfaction and eating pathology in young women (e.g., Becker & Stice, 2017; Stice, Butryn, et al., 2013).

Somerville and Cooper (2007) argue that cognitive approaches that rely on rational argument may be less effective at addressing negative self-core beliefs of relevance to eating disorders (such as self-criticism and shame), which have stronger emotional than rational bases, than emotion-focused approaches. This is consistent with the findings in the previous cross-sectional study, which showed difficulties with emotional regulation to be an important addition to the dual-pathway model which has informed the use of cognitive dissonance with disordered eating. In order to identify which approach appeared most promising for further development, the third study (**Chapters 5 and 6**) compared imagery rescripting and cognitive dissonance, to a control condition in terms of reducing disordered eating. We were also interested in the degree to which each intervention modified risk and protective factors for eating disorders, such as body dissatisfaction, negative affect and self-compassion, in order to identify variables that appear to be most effective for change. Notably, at the time of submission of this thesis, this is the first time the use of imagery rescripting has been examined with respect to reducing body dissatisfaction in the prevention of disordered eating.

Change in state variables were examined within the experimental session following a body dissatisfaction induction, in a sample of women at risk of disordered eating ( $N = 201$ ), described in **Chapter 5**. The study involved randomization to one of the three conditions (i.e., imagery rescripting, cognitive dissonance, control), followed by a baseline assessment, body dissatisfaction induction, and brief intervention. This study found support for both the imagery rescripting and cognitive dissonance interventions compared to the control condition, in increasing self-compassion, and both approaches showed promise in reducing negative affect. However, this study only examined brief interventions over the experimental session (i.e., over a 5 minute period); the longer term impact of using such strategies is unclear. Further, while participants were considered to be at increased risk for disordered eating, it is unclear whether these findings would generalize to high risk populations such as body-dissatisfied women. We address these limitations in the subsequent study.

Change in trait variables were examined over the one-week follow-up for body-dissatisfied women considered to be at high risk of developing an eating disorder ( $N = 107$ ), described in **Chapter 6**. The study utilised the same protocol as the previous study however participants were screened for high-risk status before commencing and completed additional home practice and one-week follow-up. The use of home practice was intended to increase exposure to, and practice of, the two approaches to enhance effect sizes for any subsequent changes in trait variables. In the process of examining intervention effectiveness, we developed a quality rating scheme to assess intervention fidelity (i.e., adherence) at home practice; the complete scale is provided in **Appendix A**. This study found support for the imagery rescripting intervention in increasing body image acceptance (i.e., a measure of body image flexibility which has shown to be negatively correlated with body image dissatisfaction; Sandoz, Wilson, Merwin, & Kellum, 2013) and self-compassion and reducing disordered eating at follow-up. This study was submitted to *Behaviour Research and*

*Therapy*, and is currently under review awaiting decision. The version that appears in this thesis is the manuscript as submitted to the journal.

## **1.6 Implication of the Findings**

The final chapter of this thesis (**Chapter 8**) provides a comprehensive discussion, which integrates the overall findings of the three studies included in this thesis. Further, the discussion summarises the key contributions of this thesis within the broader eating disorder field, considers the strengths and limitations of the research, and details the theoretical and clinical implications and directions for future research.

## Chapter 2

### **A Systematic Review of The Existing Models of Disordered Eating: Do They Inform the Development of Effective Interventions?**

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#### **Author Contributions:**

JP led the review design, literature search, results and interpretation, and manuscript preparation.

TW contributed to the review design, results and interpretation, and manuscript preparation.

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## 2.1 Abstract

Despite significant advances in the development of prevention and treatment interventions for eating disorders and disordered eating over the last decade, there still remains a pressing need to develop more effective interventions. In line with the 2008 Medical Research Council (MRC) evaluation framework from the United Kingdom for the development and evaluation of complex interventions to improve health, the development of sound theory is a necessary precursor to the development of effective interventions. The aim of the current review was to identify the existing models for disordered eating and to identify those models which have helped inform the development of interventions for disordered eating. In addition, we examine the variables that most commonly appear across these models, in terms of future implications for the development of interventions for disordered eating. While an extensive range of theoretical models for the development of disordered eating were identified ( $N = 54$ ), only ten (18.5%) had progressed beyond mere description and to the development of interventions that have been evaluated. It is recommended that future work examines whether interventions in eating disorders increase in efficacy when developed in line with theoretical considerations, that initiation of new models gives way to further development of existing models, and that there be greater utilisation of intervention studies to inform the development of theory.

## **2.2 Introduction**

### **2.2.1 The need for the development of effective interventions in eating disorders.**

The prevalence of disordered eating behaviours and associated problems with body, weight, and shape in society is well-documented (Fisher et al., 1995; Holm-Denoma et al., 2005; Hudson et al., 2007; Liberati et al., 2009; Moher et al., 2009; Vohs, Heatherton, & Herrin, 2001). The consequences of disordered eating can often be serious, including elevated mortality and suicide rates (Crow et al., 2009; Crow et al., 2012), impairments across several health dimensions, and quality of life (Thomas, Vartanian, & Brownell, 2009; Wade et al., 2012). Estimates suggest almost one-quarter of young women have experienced disordered eating (including binge eating, purging, and/or fasting) in the previous 12-month period (Wade et al., 2012), supporting the idea that a moderate degree of disordered eating is now normative among young women. In addition, over the last decade there has been increasing awareness of the existence of eating disorders among men (Dakanalis & Riva, 2013; Tylka, 2011b), showing comparable dietary restraint, driven exercise (i.e., exercising in compulsive manner as a means of controlling weight and/or shape) and binge eating rates (Hudson et al., 2007; Lavender, De Young, & Anderson, 2010; Striegel-Moore et al., 2009), as well as levels of clinical impairment (Striegel-Moore, Bedrosian, Wang, & Schwartz, 2012), to women. A recent position paper from the Academy for Eating Disorders (Klump et al., 2009) recognised eating disorders as a serious mental illness warranting attention similar to that of schizophrenia, bipolar disorder, depression, and obsessive-compulsive disorder.

Despite significant advances in the development of prevention (e.g., the *Body Project*; Stice & Presnell, 2007; Stice, Rohde, & Shaw, 2012), and treatment interventions for eating disorders and disordered eating (e.g., enhanced cognitive behaviour therapy [CBT-E]; Fairburn et al., 2009) over the last decade, there remains several sizable gaps with regards to

the provision of effective interventions. For example, in the area of prevention, it is still not clear whether universal prevention approaches have the power to reduce onset of eating disorders. In addition, the vast majority of widely disseminated public health approaches are often developed independently of theoretical considerations and lack empirical support, raising concerns about program effectiveness as well as the potential for waste of resources (Becker et al., 2014). In the area of treatment, there are several notable gaps in our knowledge, including the optimal treatment for adult anorexia nervosa (Bulik, Berkman, Brownley, Sedway, & Lohr, 2007), and knowing which treatment approaches work best for which people (Vall & Wade, 2015). Thus further work is required in the development of effective interventions for disordered eating.

### **2.2.2 The relationship between theory and development of interventions.**

One pathway to the development of effective interventions is through rigorous testing and examination of theoretical models, as described in the Medical Research Council (MRC; Campbell et al., 2000) evaluation framework for developing and evaluating complex interventions to improve health from the United Kingdom. These guidelines have recently been comprehensively revised and updated (Craig et al., 2008, 2013; Craig & Petticrew, 2013). Specifically, the framework is intended to guide: (a) researchers to choose and implement appropriate methods; (b) research funding bodies to understand the limitations on evaluation design; and (c) policy makers, practitioners, and other users of the intervention in the consideration of the available evidence in light of these methodological and practical constraints.

The framework has been highly influential and widely cited. A recent reflection paper by Craig and Petticrew (2013) reported that citations for the MRC's guidelines had increased between 2001 and 2011, suggesting a sustained growth of interest in such guiding frameworks. Furthermore, the guidance is incorporated in advice given to grant applicants in

the United Kingdom (<http://www.nets.nihr.ac.uk/faqs/developing-a-proposal>) and educational materials (e.g., for postdoctoral nursing students involved in cancer research; Senn et al., 2011), and similar guidelines have now been produced for surgical trials (McCulloch et al., 2009), group delivered interventions (Hoddinott, Allan, Avenell, & Britten, 2010), and natural experimental approaches (Craig et al., 2012). A CONSORT extension for complex interventions is now being developed (<http://www.spi.ox.ac.uk/research/site/consort-spi/home.html>). Thus a reasonable body of evidence suggests that researchers have found the guidance useful. In support of the relevance of this framework to the eating disorder field, one meta-analysis has highlighted that more effective interventions are informed by risk factors and theory (Stice & Shaw, 2004). One persistent criticism of eating disorder prevention studies has been the lack of theoretical rationale to guide the content, design and administration of interventions (e.g., Pratt & Woolfenden, 2002); of twenty prevention studies reviewed by Austin (2000), fewer than half clearly outlined a theoretical basis for the chosen preventive approach.

According to the MRC's framework, the process from development to implementation may take a variety of forms. **Figure 2.1** summarises the main stages and the key functions and activities at each stage, where the arrows indicate the main interactions between the phases. Although it is useful to think in terms of stages, they may not follow a linear or even a cyclical sequence. It is recommended that the best practice is to develop interventions systematically. First, *development*, includes: (a) identifying the relevant existing evidence base (e.g., a systematic review); (b) identifying or developing the relevant theory that informs an understanding of the processes of change; and (c) modelling processes and outcomes described in the theory prior to a development of an intervention. Second, *feasibility and piloting* (preparatory phase), includes: (a) testing procedures for feasibility (e.g., piloting and case series); (b) estimating the likely rates of subject recruitment and

retention; and (c) determining appropriate sample sizes. Third, *evaluation*, includes: (a) assessment of effectiveness (see **Table 2.1** for summary of experimental designs for evaluating complex interventions); (b) process evaluation that includes assessment of fidelity and quality of implementation, identification of causal mechanisms, and recognition of contextual factors associated with variances in outcome; and (c) assessment of cost-effectiveness (e.g., economic evaluation and cost estimation). Fourth, *implementation*, includes dissemination of research findings, and implementation and evaluation of the intervention in real world settings, including long-term follow-up.

The present review was conducted using the MRC guidelines to assess the usefulness of theories that have been generated for eating disorders and disordered eating. We were interested in whether the generation of a theory had evolved past the development stage and toward the steps identified in the evaluation framework, including intervention development and evaluation.

## **2.3 Method**

### **2.3.1 Literature search.**

The central aim of the current review was to identify models of disordered eating which have helped inform the development of interventions. A secondary aim was to examine the variables that most commonly appear across these models, in terms of future implications for the development of interventions. To this end, a literature search was conducted by using the PsycINFO database (OvidSP) which covers professional and academic literature across psychology and other related disciplines, including medicine, mental health, nursing, nutrition and dietetics, physiology, and linguistics. Keywords *anorexia nervosa* (Title) OR *bulimia nervosa* (Title) OR *disordered eating* (Title) OR *eating disorders* (Title) OR *bulimic* (Title) OR *eating* (Title) AND *model* (Title) OR *theory* (Title) were used to locate pertinent publications in all journals using an advanced search.

Publications were then inspected for studies meeting the following criteria: (1) published in English in a peer-reviewed journal, or an authored or edited book; (2) made reference to the aetiology and/or development of eating disorders, disordered eating symptoms and/or behaviours; (3) described and/or made reference to models that could be tested (i.e., models identifying no more than six clearly conceptualised and measurable variables in addition to the disordered eating outcome variables which could include bingeing, fasting, dietary restriction, purging, laxative misuse, driven exercise, and/or eating psychopathology); and (4) made reference to models that (i) had been independently investigated by researchers other than the original (developing) research team (i.e., the relationship between at least two independent variables and the outcome variable, as delineated in the model, had been tested), or (ii) informed the development of an intervention that had been pilot tested (i.e., case series), tested for efficacy (i.e., randomised-controlled trials), or tested for effectiveness (i.e., implemented or disseminated in real-life setting). Only those publications meeting *all 4* criteria listed above were included in the present review.

Additional articles from reference lists and extended searches, including those pertinent to the earlier as well as the most recent versions of the disordered eating models, were included in the present literature search, as were those prevention and/or treatment studies informed by such models. Publications were not excluded on the basis of research design, target population (i.e., clinical or non-clinical, male or female, age), evidence type (i.e., cross-sectional, prospective or longitudinal research), or whether the evidence was favourable or not.

The review process was conducted according to the PRISMA statement (Liberati et al., 2009; Moher et al., 2009), as described in **Figure 2.2**. The search of electronic databases resulted in 545 studies listed on January 7, 2015. Additionally, 377 studies were found through other sources. Twelve duplicates were removed. The remaining 910 studies were

then screened. Two hundred and seventy-eight studies were removed after reviewing the publication title and abstract. The remaining 632 studies were evaluated, across which fifty-four disordered eating models were described. Thirty-one of these models (57.4%) across 309 studies had to be eliminated as they did not fulfil eligibility criteria, leaving 323 studies to be included in the present review, across which twenty-three models for disordered eating were described. All screening was conducted by the first author using the inclusion-exclusion criteria described above.

## **2.4 Results**

### **2.4.1 Models that met criteria for inclusion.**

A summary of twenty-three models that met the criteria for inclusion is presented in **Table 2.2**. In line with the processes described in the MRC documents, the empirical evidence for the existing models was collapsed into sections based of the four distinct phases as follows: (1) *modelling process variables (development)*, including testing of the model by the original (developing) research team as well as testing by independent research teams; (2) *pilot testing (feasibility and piloting)*, including case series; (3) *efficacy testing (evaluation)*, including randomised-controlled trials; and (4) *effectiveness testing (implementation)*, including implementation and dissemination of the intervention in real world settings.

### **2.4.2 Models leading to the development of interventions.**

There was much variability in the quality of the existing models for disordered eating. Of the fifty-four models described in the literature, of which twenty-three (42.6%) met criteria for inclusion in this study, less than half (37.0%) had been investigated by researchers other than the original (developing) research team, and a mere ten (18.5%) had progressed beyond description and on to the development of interventions that have been evaluated. A summary of these ten models is presented in **Table 2.3**; seven (13.0%) models met the

criterion for pilot testing (i.e., case series), five (9.3%) models had been tested for efficacy (i.e., randomised-controlled trials), and four (7.4%) models had been tested for effectiveness (i.e., implemented or disseminated in real world settings). Only two (3.7%) models (i.e., the dual-pathway model for bulimia nervosa and the transdiagnostic maintenance model of eating disorders) met criteria for *both* efficacy and effectiveness. Although it is beyond the scope of this paper to conduct a comprehensive review of the ten models that led to the development of an intervention, we provide a brief overview of these models and the intervention approaches that have been informed by them below.

***Acceptance model of intuitive eating.*** The acceptance model of intuitive eating (Augustus-Horvath & Tylka, 2011) was developed in line with emerging evidence of the importance of positive body image, or body appreciation (see Wood-Barcalow, Tylka, & Augustus-Horvath, 2010 for review), and adaptive eating behaviours, or intuitive eating (i.e., trust in, and connection with, internal physiological hunger and satiety cues, and eating in response to these cues; Tribole & Resch, 2003; Tylka, 2006), in the prevention of negative body image and disordered eating. Research has suggested that the intuitive eating is innate, however the likelihood of continuing to engage in this adaptive form of eating is determined by an accepting environment; that is, the extent to which people perceive that their internal self and external body shape and weight are accepted by others, the more likely they are to pay attention to their internal hunger and satiety cues and eat accordingly (Tribole & Resch, 1995). Consistent with this, the model proposes that body acceptance by others helps women to appreciate their body and resist adopting an observer's perspective of their body. This theory led to the development of an intuitive eating education program (e.g., rejecting the diet mentality, or coping with emotions without food) aimed to promote healthy attitudes towards eating in school-aged children (see Healy, Joram, Matvienko, Woolf, & Knesting, 2015 for review). However the program requires further rigorous testing.

***Cognitive model of bulimia nervosa.*** The cognitive model of bulimia nervosa (Cooper, Todd, & Wells, 2009) was developed to reflect recent advances in cognitive theory (see Beck & Freeman, 1990; Padesky & Greenberger, 1995; Wells, 1997; Young, 1990 for review). According to the theory, negative beliefs about the self (e.g., “I’m unlovable”, “I’m a failure”), are expressed as negative automatic thoughts, and typically causes considerable emotional distress. Bingeing or purging takes place to manage the distress experienced when negative self-beliefs are activated, providing a temporary distraction from negative thoughts and emotions. These experiences are typically associated with positive beliefs about eating (e.g., “bingeing will take away my painful feelings”), negative beliefs about the potential consequences (e.g., “I’ll get fat”), and permissive thoughts which make it easier to keep eating, (e.g., “this will be the last time I binge”). This, in turn, reinforces the negative self-appraisals, thus completing the vicious cycle. This model informed the development of a cognitive-based self-help treatment for bulimia nervosa (see Cooper et al., 2009 for review). The manual includes psychoeducation, motivation chapters, orientation to the cognitive model, challenging dysfunctional cognitions, behavioural experiments, and changing maladaptive behaviours, and can be used without assistance (i.e., self-help) or within the framework of guided self-help. To date, there has been one case series evaluation of the guided self-help program (Pritchard, Bergin, & Wade, 2004), which highlights the need for further rigorous evaluation of the treatment.

***Cognitive-interpersonal maintenance model of anorexia nervosa.*** The cognitive-interpersonal maintenance model of anorexia nervosa (Schmidt & Treasure, 2006; Treasure & Schmidt, 2013), describes both risk and maintaining factors for anorexia nervosa, including cognitive, socio-emotional, and interpersonal factors. The model proposes that predisposing factors (e.g., obsessive-compulsive features and anxious avoidance, particularly in the context of close relationships), increase vulnerability to anorexia nervosa and

contributes to the maintenance of the disorder, i.e., through fostering pro-anorexic beliefs and behaviours. Furthermore, these traits, paired with eating disorder symptomatology, causes problems with respect to interpersonal relationships, which in turn can serve to maintain the disorder. This model informed the development of the Maudsley model of anorexia nervosa treatment for adults (MANTRA; Schmidt, Wade, & Treasure, 2014), in which both intra- and inter-personal maintaining factors are considered. The therapy addresses the four main maintaining factors in the model, including: (a) a thinking style characterised by rigidity, detail focus, and a fear of making mistakes; (b) an avoidant emotion processing and relational style; (c) positive beliefs about the utility of anorexia for the person; and (d) a response of close others to the illness, e.g., high expressed emotion, or accommodation to the illness. The treatment has shown promise in the treatment of adult women with anorexia nervosa; having received validation in both case series evaluations (Slater, Treasure, Schmidt, Gilchrist, & Wade, 2014; Wade, Treasure, & Schmidt, 2011) and randomised-controlled trials (Lose et al., 2014; Schmidt et al., 2013; Waterman-Collins et al., 2014).

***Dual-pathway model for bulimia nervosa.*** The dual-pathway model for bulimia nervosa (Stice, 2001) is a socio-cultural model of bulimic symptom development in females. According to this model, perceived socio-cultural pressure to be thin promotes both internalisation of the thin-ideal (i.e., heightened endorsement of the thin-ideal stereotype for women, to the point of modifying one's behaviours in an attempt to reach these standards; Thompson & Stice, 2001) and body dissatisfaction, which leads to dieting and negative affect, which then leads to the development of bulimic pathology. This theory has led to development of the gold standard of prevention interventions for bulimic behaviours for young women who experience body dissatisfaction and body image problems (Stice, Chase, Stormer, & Appel, 2001; Stice et al., 2008; Stice, Mazotti, Weibel, & Agras, 2000; Stice, Rohde, et al., 2009; Stice, Rohde, Gau, & Shaw, 2012). The intervention is informed by

cognitive dissonance theory, an approach which utilises Festinger's (1957) theory that holding cognitions inconsistent with one's beliefs creates psychological discomfort that motivates people to alter their behaviour and/or attitudes so as to reduce this inconsistency. In particular, this understanding is used to target the thin-ideal internalisation, by asking young women to argue against this ideal. Change in thin-ideal internalisation has been shown to fully mediate the effects of the program on change in body dissatisfaction and partially mediate changes in bulimic symptoms (Stice, Marti, Rohde, & Shaw, 2011). It is only one of three prevention programs shown to prevent increase of disordered eating in at-risk young women (Becker et al., 2014).

***Functional model of emotion avoidance in anorexia nervosa.*** The functional model of emotion avoidance in anorexia nervosa (Wildes, Ringham, & Marcus, 2010) was developed following various lines of research suggesting that emotion avoidance is pertinent to development and maintenance of anorexia nervosa (see Gale, Holliday, Troop, Serpell, & Treasure, 2006; Nordbø, Espeset, Gulliksen, Skårderud, & Holte, 2006; Serpell, Treasure, Teasdale, & Sullivan, 1999 for review). In particular, the theory proposes that eating disorder psychopathology helps individuals manage comorbid depressive and anxiety symptoms, and that this functional relationship is influenced by emotion avoidance, i.e., the desire to avoid experiencing or expressing physical sensations, thoughts, urges, and behaviours, related to intense emotional states (Wildes et al., 2010). This model helped inform the development of emotion acceptance behavior therapy (EABT; Wildes & Marcus, 2011; Wildes, Marcus, Cheng, McCabe, & Gaskill, 2014) for older adolescents and adults with anorexia nervosa. The treatment approach combines standard behavioural interventions central to the clinical management of anorexia nervosa (e.g., weight monitoring, regular eating, or reduction in eating disorder symptoms) with psychotherapeutic techniques (e.g., mindfulness, acceptance, or being in the present moment), aimed to increase emotion awareness, decrease emotion

avoidance, and encourage participation in valued activities and relationships unrelated to the eating disorder. To date, EABT has received preliminary support with older adolescents and adults with anorexia nervosa, including one case series (Wildes & Marcus, 2011) and one pilot investigation (Wildes et al., 2014); however, further rigorous and independent examination of this treatment approach is warranted.

***Interpersonal model of binge eating.*** The interpersonal model of binge eating (Wilfley, MacKenzie, Welch, Ayers, & Weissman, 2000) was developed in an attempt to integrate two existing risk factor models for binge eating disorder (BED), i.e., the restraint model and the interpersonal vulnerability model (see Wilfley, Pike, & Striegel-Moore, 1997 for reviews), to offer a better prediction for BED than either model alone. The theory originated from research in the depression literature (see Klerman, Weissman, Rounsaville, & Chevron, 1984 for review). The theory proposes that difficulties with social functioning precipitates low self-esteem and negative affect, which in turn triggers binge eating in an attempt to cope with negative feelings. This interpersonal model informed the development of interpersonal psychotherapy for eating disorders (IPT-ED; Jacobs, Welch, & Wilfley, 2004; Rieger et al., 2010), which targets areas of interpersonal functioning (including grief, role transitions, interpersonal role disputes, and interpersonal deficits). IPT-ED is informed by interpersonal psychotherapy (IPT), which was first successfully adapted for the treatment of bulimia nervosa (e.g., Fairburn et al., 1991; Fairburn, Peveler, Jones, Hope, & Doll, 1993) and has also been found effective in a group setting for BED (e.g., Wilfley et al., 1993; Wilfley et al., 2000; Wilfley et al., 2002). Research has demonstrated advantages of IPT over behavioural weight loss treatment and cognitive behavioural therapy (CBT)-based guided self-help for individuals with BED with low self-esteem and high eating disorder psychopathology, e.g., greater effectiveness than behavioural weight loss treatment and CBT-based guided self-help (Wilson, Wilfley, Agras, & Bryson, 2010).

***Model of disordered eating.*** The model of disordered eating (Neumark-Sztainer, Wall, Story, & Perry, 2003) was developed to explain the development of unhealthy weight-control behaviours in adolescents. According to the theory, socio-environmental factors (i.e., family-peer weight-related social norms and teasing behaviours, and family connectedness) influence personal factors (i.e., weight-body concerns, psychological well-being, attitudes related to health and nutrition, and body mass index), which in turn leads to the development of unhealthy weight-control behaviours (e.g., skipping meals, laxative use, or self-induced vomiting; disordered eating behaviours). This theory has led to the development of four primary prevention programs for disordered eating; including three school-based (*Very Important Kids [V.I.K]*; Haines, Neumark-Sztainer, Perry, Hannan, & Levine, 2006; *The Weigh to Eat*; Neumark-Sztainer, Butler, & Palti, 1995; *New Moves*; Neumark-Sztainer et al., 2010) and one community-based intervention (*Free to Be Me*; Neumark-Sztainer, Sherwood, Collier, & Hannan, 2000). *The Weigh to Eat*, *New Moves*, and *Free to Be Me* are based on Bandura's social cognitive theory (see Bandura, 1986 for review) and target socio-environmental factors (e.g., peer support), personal factors (e.g., body image), and behavioural factors (e.g., goal setting), and the interactions between them (Neumark-Sztainer et al., 1995; Neumark-Sztainer et al., 2010; Neumark-Sztainer et al., 2000). *V.I.K.* aims to reduce teasing and unhealthy weight-control behaviours (Haines et al., 2006). Though preliminary support has been found for each of these primary prevention programs, the theory has yet to be investigated by independent research teams, which is imperative in the development and implementation of any effective intervention.

***Multidimensional model of anorexia nervosa.*** The multidimensional model of anorexia nervosa (Lyon et al., 1997) was developed following growing research suggesting that anorexia nervosa is multi-determined (e.g., Martin, 1990; Nagel & Jones, 1992; Nygaard, 1990). The theory proposes that biogenetic factors, individual personality factors (i.e.,

ineffectiveness, low interoceptive awareness, and being a “perfect child” or “model child”), and family characteristics (i.e., low independence), interact to predict the development of anorexia nervosa in adolescent females. This multidimensional approach to anorexia nervosa, has been tested in one case study examination by the original research team with one adolescent (Lyon, Silber, & Atkins, 2005). To date, there have been no further tests of the theory, and while the model has led to the development of a treatment approach, the extant evaluation is insufficient to make any conclusions about the usefulness of this approach. Given a lack of follow-up research over the last 10 years, it is unlikely that such evidence will be emerging.

*Transdiagnostic maintenance model of eating disorders.* The transdiagnostic maintenance model of eating disorders (Fairburn, 2008a; Fairburn, Cooper, & Shafran, 2003) is derived from a longstanding and established theory of eating disorders (see Fairburn, 2008a; Fairburn et al., 2003 for review). The theory and treatment have evolved over time such that it has been adapted to suit all forms of eating disorders, thereby making it “transdiagnostic” in its scope. The most recent iteration of the theory proposes several maintaining factors, including: dietary restraint; mood intolerance; clinical perfectionism; interpersonal problems; core low self-esteem; and over-evaluation of eating, shape and weight, and their control. This latter construct is seen as central to the maintenance of the disorder (the ‘core psychopathology’; Cooper & Fairburn, 1993). While, the theory is concerned with the processes that maintain eating disorder psychopathology rather than those responsible for its initial development, it is recognised that the two may overlap. This theory has led to the development of an “enhanced” form of cognitive behavioural therapy for eating disorders (CBT-E; Fairburn, 2008a) that seeks to address the core maintaining processes of the disordered eating as delineated in the model that are of relevance to the individual, identified in a collaborative case formulation. CBT-E has been extensively tested and

replicated (e.g., Allen, Byrne, & McLean, 2012; Fairburn et al., 2009; Hoiles, Egan, & Kane, 2012; Lampard, Tasca, Balfour, & Bissada, 2013), and is considered the treatment of choice for eating disorders where the body mass index is greater than 17.5 (Fairburn et al., 2015; National Institute for Health and Care Excellence [NICE], 2004; Poulsen et al., 2014). CBT-E has also shown some promise in the treatment of anorexia nervosa in several case series examinations (e.g., Dalle Grave, Calugi, Doll, & Fairburn, 2013; Dalle Grave, Calugi, El Ghoch, Conti, & Fairburn, 2014; Fairburn et al., 2013).

***Tripartite influence model of body dissatisfaction and disordered eating.*** The tripartite influence model of body dissatisfaction and disordered eating (Yamamiya, Shroff, & Thompson, 2008) proposes that sociocultural influences from media, parents, and peers promotes both internalisation of the thin-ideal and social appearance comparison, which ultimately develops into body dissatisfaction, disordered eating, and negative affect (e.g., low self-esteem, or depression). This model has received widespread support with both adolescent and adult female samples (e.g., Keery, van den Berg, & Thompson, 2004; van den Berg, Thompson, Obremski-Brandon, & Coovert, 2002), and modified versions of the model with muscular enhancement behaviours (e.g., muscle-building) as the outcome measure has received some support with adolescent and adult males (e.g., Karazsia & Crowther, 2009; Smolak, Murnen, & Thompson, 2005; Tylka, 2011b; Tylka & Andorka, 2012). An extension of this model, i.e., the biopsychosocial model of body dissatisfaction and disordered eating (see Rodgers, Paxton, & Chabrol, 2009 for review), informed the development of three body image intervention programs for females; including two school-based interventions (*My Life, My Body*; see Heinicke, Paxton, McLean, & Werheim, 2007 for review; *Happy Being Me*; see Richardson & Paxton, 2010 for review) and one for adult women (*Set Your Body Free*; see Gollings & Paxton, 2006 for review).

### **2.4.3 Overlap of independent constructs across the models.**

One way of selecting those variables considered to be important to include in any future models of disordered eating that can inform the development of effective interventions is to consider those variables in the ten existing robust models that have been used to inform interventions (Augustus-Horvath & Tylka, 2011; Cooper et al., 2009; Fairburn, 2008a; Lyon et al., 1997; Neumark-Sztainer et al., 2003; Stice, 2001; Treasure & Schmidt, 2013; Wildes et al., 2010; Wilfley et al., 2000; Yamamiya et al., 2008). An analysis of the overlap among independent variables included in these existing models (presented in **Table 2.3**), along with evidence from risk factor research (Bardone-Cone et al., 2007; Jacobi & Fittig, 2010; Jacobi et al., 2011; Stice, 2002; The McKnight Investigators, 2003), gives some indication as to the core putative risk factors believed to be responsible for the development of disordered eating pathology.

Independent variables were collapsed into main constructs and are summarised in **Table 2.4**. Variables that could not be collapsed were made independent constructs and were therefore not included in the table. Constructs were selected systematically, following independent rating from the first author and a blind rater. Any disagreements that arose were discussed between raters until an agreement could be met. A number of constructs emerged as common risk factors across these models for the development of disordered eating; namely, preoccupation with weight and shape, emotional regulation difficulties, self-esteem deficits, interpersonal issues, and negative affect; and are referred to across four or more of the existing models. In accordance with risk factor research (Jacobi & Fittig, 2010; Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Polivy & Herman, 2002; The McKnight Investigators, 2003), both preoccupation with weight and shape (i.e., weight and shape concerns, thin body preoccupation, body dissatisfaction, body image disturbance, appearance anxiety, and body shame) and negative affect (i.e., affective states, depression, and anxiety), are suggested as two of the single most prominent risk factors and contributors to the

development of eating disorders. A moderate degree of evidence also exists to support the remaining risk factors identified as being in common across these models (see Jacobi & Fittig, 2010; Polivy & Herman, 2002; Shomaker & Furman, 2009; Wang, Peterson, McCormick, & Austin, 2013 for reviews). Given the importance of these risk factors for the development of disordered eating pathology, their inclusion within comprehensive theoretical models for disordered eating may be pivotal in informing the development of future effective interventions for disordered eating.

#### **2.4.4 Models that did not fulfil eligibility criteria.**

The literature search resulted in thirty-one (57.4%) models for disordered eating that did not fulfil eligibility criteria; these models are listed in **Table 2.5**, along with reasons for non-inclusion. It should be noted that all but one of these models has not led to the development of an intervention. Further, of those models excluded, twenty-five (80.6%) either identified more than six independent variables in addition to the outcome variable, or identified variables that were unclear, and twenty-two (71.0%) models had not been independently tested by researchers other than the original (developing) research team.

## **2.5 Discussion**

### **2.5.1 Limitations of current literature.**

While there exists an extensive range of theoretical models describing the development and maintenance of disordered eating, it is clear that few models have led to the development of effective interventions, as described the MRC's evaluation framework (Craig et al., 2008, 2013; Craig & Pettecrew, 2013). The vast majority of theories postulated in the eating disorder field have not moved beyond the first stages (including identifying the relevant risk factors, developing the theory or model, and modelling associations between these factors and the outcome). Only two (3.7%) models met criteria for the last two stages of

the MRC pathway (i.e., evaluation and implementation). It appears that the initiation of theories and models has often been an end in itself, which may impede development in the field, as we seek to engage with a confusing array of models of various but unknown utility. A greater focus on the early stages of model testing may actually discourage progression to the development and testing of interventions, with a case in point being objectification theory (see Noll & Fredrickson, 1998 for review). Despite numerous model testing studies showing strong support for the theory, there has been no progression to the development of an intervention (Tiggemann, 2013), with little integration evident across theoretical and clinical literature in the eating disorder field. It remains unclear as to why the majority of the existing models for disordered eating have not progressed past the initial stages of testing. Clearly moving to the development and testing of an intervention requires many more resources than development and testing of a model, and also requires access to suitable populations which may require multicentre collaboration.

A further major limitation is the nature of the theories used to inform intervention development in the field of eating disorders. The list of risk factors summarised across these theories should not be considered to be the only ones of potential importance in the development of effective interventions. Lack of emphasis on more distal variables is apparent in the models, especially those relating to biogenetic and developmental factors. For example, our increasing understanding of epigenetics may help identify ante- and post-natal environments that are critical to forming temperaments and behaviours that increase risk for the development of eating disorders. Greater integration of model development with developing neurobiological evidence is required in future work.

### **2.5.2 Future directions.**

Three directions of future research are likely to be of benefit to the eating disorder field. First, the MRC guidelines are not necessarily inviolable, but this an empirical question

to be further investigated. While ineffective and atheoretical interventions litter the prevention field (Becker et al., 2014), and evidence suggests that interventions based on identified risk variables and coherent theories are associated with changes of larger effect sizes (Stice, 2002), the importance of this connection has not been tested in the treatment arena. In the current review, exclusion of interventions that were not associated with a testable model means that at least one efficacious eating disorder treatment was not included in the current review, namely family-based therapy (FBT). In fact, it has been noted that FBT is an effective therapy despite the fact that the theoretical model from which most of the empirically based treatments are derived appears flawed (Eisler, 2005). Eisler (2005) has argued that the most relevant theoretical model for FBT is how families organise themselves around a potentially life-threatening problem (i.e., the central role of the symptom in family life, a narrowing of time focus on the here-and-now, restriction of family interaction processes, amplification of unsatisfactory aspects of family functioning, diminished ability to meet family life-cycle needs, and a sense of family helplessness and despair), rather than one relating to the aetiology of the disorder. This confusion has resulted in lack of development of a testable theoretical underpinning FBT, which may disadvantage the further development of the treatment. For instance, while FBT is widely considered the treatment of choice for adolescent anorexia nervosa (NICE; 2004), fewer than half of adolescents undertaking this treatment achieve full remission at 12-month follow-up (Agras et al., 2014; Lock et al., 2010), with no difference in outcome from systemic family therapy which addresses general family processes rather than facilitation of weight gain (Agras et al., 2014). Consideration of other contributing influences in a testable theoretical framework may lead to an improvement in treatment outcomes (Hurst & Zimmer-Gembeck, 2015).

Second, it is questionable whether the development of further new theories within the field of disordered eating research will bring any added benefit and move us closer to the

development of effective interventions for disordered eating. In the case where a new theory is considered of importance to develop, as new information emerges about different risk factors for disordered eating and their relationship to other risk factors, it would be advisable to concurrently plan for studies that can lead to the development and evaluation of an intervention. In other words, where there is an impelling case to develop a new theory, it should be developed in the context of a planned adherence to best practise guidelines as suggested by the MRC, and lead to an iterative testing of the theory in terms of processes and interventions. Alternatively, before developing new theories, researchers should consult the extant literature and consider whether it may be advisable to revise existing theories, in particular keeping in mind the possible utility of biogenetic and developmental factors. Iterative revisions of such theoretical frameworks can also contribute to a “non-ossification” of manualised interventions, such as that observed for the development of CBT for eating disorders (Fairburn, 2008a), allowing greater responsivity to inclusion of new therapeutic elements, or exclusion of elements that no longer have support of the theory or the evidence.

Third, in the spirit of the iterative MRC process, it may be more profitable to focus on further developing interventions using the existing theories for disordered eating. This process should also include the investment in dismantling studies that can identify which elements of interventions are actually required to produce meaningful changes, as well as a greater focus on using intervention studies to test moderation (i.e., which interventions work best for whom) and mediational processes. In this way, information will be generated as to the limitations of these models in informing effective interventions which can then lead to the consideration of which empirically supported risk factors can be used to modify existing theories, with consequent testing of interventions informed by these modifications. A related point, is that special attention should be paid to the interventions that have arisen from the ten robust models reviewed in this paper. In most cases there is a compelling need for

independent replication of these interventions, and comparison between active interventions. This information will, in turn, allow for refinement of models that can become more informative in pushing forward the efficacy and effectiveness of interventions in the eating disorder field.

**Table 2.1***Summary of Experimental Designs for Evaluating Complex Interventions with Descriptions*

Designs	Descriptions
Individually randomised trials	Individuals are randomly allocated to receive either an experimental intervention or an alternative such as standard treatment, a placebo, or remaining on a waiting list. Such trials are sometimes dismissed as inapplicable to complex interventions, but there are many variants of the basic method, & often solutions can be found to the technical & ethical problems associated with randomisation.
Cluster randomised trials	Contamination of the control group, leading to biased estimates of effect size, is often cited as a drawback of randomised trials of population level interventions, but cluster randomisation, widely used in health services research, is one solution. Here, groups such as patients in a GP practice or tenants in a housing scheme are randomly allocated to the experimental or a control intervention.
Stepped wedge designs	The randomised stepped wedge design may be used to overcome practical or ethical objections to experimentally evaluating an intervention for which there is some evidence of effectiveness, or which cannot be made available to the whole population at once. It allows a randomised controlled trial to be conducted without delaying roll-out of the intervention. Eventually, the whole population receives the intervention, but with randomisation built into the phasing of implementation.
Preference trials & Randomised consent trials	Practical or ethical obstacles to randomisation can sometimes be overcome by the use of non-standard designs. Where patients have very strong preferences among treatments, basing treatment allocation on patients' preferences, or randomising patients before seeking consent, may be appropriate.
N-of-1 designs	Conventional trials aim to estimate the average effect of an intervention in a population, & provide little information about within or between person variability in response to interventions, or about the mechanisms by which effective interventions achieve change. N-of-1 trials, in which individuals undergo interventions with the order or scheduling decided at random, can be used to assess between & within person change, & to investigate theoretically predicted mediators of that change.

*Note.* Reproduced with permission from the Medical Research Council (Craig et al., 2008; 2013; also see [www.mrc.ac.uk/complexinterventionsguidance](http://www.mrc.ac.uk/complexinterventionsguidance)).

**Table 2.2***Summary of Models for Disordered Eating with Empirical Evidence*

Developing Theory (Authors) <sup>a</sup>	Modelling Process Variables (Development) <sup>b</sup>	Pilot Testing (Feasibility & Piloting)	Efficacy Testing (Evaluation)	Effectiveness Testing (Implementation)
Acceptance Model of Intuitive Eating (Augustus-Horvath & Tylka, 2011)	✓ Andrew, Tiggemann, and Clark (2015); (Hahn Oh, Wiseman, Hendrickson, Phillips, & Hayden, 2012); Suthendran and Fassnacht (2013)	✗	✗	✓ Healy et al. (2015)
Acquired Preparedness Model of Risk for BN (Combs, Smith, Flory, Simmons, & Hill, 2010)	✓ Combs et al. (2010); Schaumberg and Earleywine (2013) Pearson, Combs, Zapolski, and Smith (2012)	✗	✗	✗
Cognitive Model of BN <sup>c</sup> (Cooper et al., 2009)	✓ Bergin and Wade (2012); Meyer, Leung, Feary, and Mann (2001); Ross and Wade (2004); Sherwood, Crowther, Wills, and Ben-Porath (2000); Stein et al. (2007); Young and Cooper (2013)	✓ Pritchard et al. (2004)	✗	✗
Cognitive-Interpersonal Maintenance Model of AN <sup>d</sup> (Treasure & Schmidt, 2013)	✓	✓ Slater et al. (2014); Wade et al. (2011)	✓ Schmidt et al. (2013); Waterman-Collins et al. (2014); Lose et al. (2014)	✗
Dual-Pathway Model of BN <sup>e, f</sup> (Stice, 2001)	✓ Allen et al. (2012); Austin and Smith (2008); Dakanalis, Timko, Carrà, et al. (2014); Duemm, Adams, and Keating (2003); Engler et al. (2006);	✗	✓ Burton and Stice (2006); Halliwell and Diedrichs (2014); Matusek, Wendt, and Wiseman (2004); Mitchell, Mazzeo, Rausch, and Cooke	✓ Becker, Bull, Schaumberg, Cauble, and Franco (2008); Becker, McDaniel, Bull, Powell, and McIntyre (2012); Becker, Smith, and Ciao

	Evans, Tovee, Boothroyd, and Drewett (2013); Fingeret and Gleaves (2004); Halliwell and Harvey (2006); Holmes, Fuller-Tyszkiewicz, Skouteris, and Broadbent (2014a); Ouwens, van Strien, and van Leeuwe (2009); Pokrajac-Bulian and Ambrosi-Randić (2007); Sivec and Tiggemann (2011); Stice (2001); Stice and Agras (1998); Stice et al. (1998); Stice, Spangler, and Agras (2001); Twamley and Davis (1999); van Strien, Engels, van Leeuwe, and Snoek (2005); Vander Wal, Gibbons, and del Pilar Grazioso (2008); Warren, Castillo, and Gleaves (2010); Warren, Schoen, and Schafer (2010); Wenzel, Weinstock, Vander Wal, and Weaver (2014); White and Halliwell (2010)	(2007); Roehrig, Thompson, Brannick, and van den Berg (2006); Stice, Chase, et al. (2001); Stice et al. (2008); Stice et al. (2000); Stice, Presnell, Gau, and Shaw (2007); Stice, Rohde, Gau, et al. (2012); Stice, Shaw, Burton, and Wade (2006); Stice, Trost, and Chase (2003)	(2006); Becker et al. (2010); Perez et al. (2010); Stice, Butryn, et al. (2013); Stice, Rohde, Durant, and Shaw (2012); Stice, Rohde, et al. (2009)	
Escape Model of Binge Eating (Heatherton, Polivy, Herman, & Baumeister, 1993)	✓	✗	✗	✗
	Holmes, Fuller-Tyszkiewicz, Skouteris, and Broadbent (2014b); Lattimore (2001); Lattimore and Maxwell (2004); Ouwens et al. (2009); Spoor, Bekker, van Strien, and van Heck (2007); Tanofsky-Kraff, Wilfley, and Spurrell (2000); Wallis and			

Functional Model of Emotion Avoidance in AN (Wildes et al., 2010)	Hetherington (2004); Ward and Mann (2000)	✗	✓	✗	✗
Interpersonal Maintenance Model of ED Psychopathology (Arcelus, Haslam, Farrow, & Meyer, 2013)	Raykos, McEvoy, Carter, Fursland, and Nathan (2014); Salerno et al. (2014)	✓	✗	✗	✗
Interpersonal Model of Binge Eating <sup>g</sup> (Wilfley et al., 2000)	Ansell, Grilo, and White (2012); Duchesne et al. (2012); Elliott et al. (2010); Fairburn et al. (1995); Ivanova et al. (2015); Salerno et al. (2014); van Durme, Braet, and Goossens (2015)	✓	✓	✓	✗
Mediational Model of Attachment, Media Internalization, & Body Image Dissatisfaction (Cheng & Mallinckrodt, 2009)	Arroyo and Segrin (2013)	✓	✗	✗	✗
Mediational Model of Parental Bonding, Self-Concept, & Eating Disturbances (Perry, Silvera,	Cella, Iannaccone, and Cotrufo (2014)	✓	✗	✗	✗
	Arcelus et al. (2009)			Agras, Walsh, Fairburn, Wilson, and Kraemer (2000); Fairburn et al. (1991); Fairburn, Jones, Peveler, Hope, and O'Connor (1993); Fairburn, Kirk, O'Connor, and Cooper (1986); Fairburn, Peveler, et al. (1993); Hilbert et al. (2007); McIntosh et al. (2005); Wilfley et al. (1993); Wilfley et al. (2002); Wilson, Fairburn, Agras, Walsh, and Kraemer (2002); Wilson et al. (2010);	

Neilands, Rosenvinge, & Hanssen, 2008)					
Mediational Model of Self-Esteem & Social Problem Solving in AN (Paterson et al., 2011)	✓		✗	✗	✗
Model of Attachment & Social Comparison in ED Symptomatology (Bamford & Halliwell, 2009)	✓		✗	✗	✗
Model of DE (Neumark-Sztainer et al., 2003)	✗		✗	✗	✓ Haines et al. (2006); Neumark-Sztainer et al. (1995); Neumark-Sztainer et al. (2010); Neumark-Sztainer et al. (2000); Neumark-Sztainer et al. (2003)
Model of DE Behaviors (Mazzeo, Mitchell, & Williams, 2008)	✓		✗	✗	✗
Multidimensional Model of AN (Lyon et al., 1997)	✗		✓ (Lyon et al., 2005)	✗	✗
Multidimensional Model of BN (Tobin, Johnson, Steinberg, Staats, & Dennis, 1991)	✓		✗	✗	✗
	Gleaves and Eberenz (1995); Gleaves, Williamson, and Barker (1993); Lowe et al. (1996); Lowe, Gleaves, and Murphy-Eberenz (1998);				

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Multidimensional Model of ED Symptomatology (Phan & Tylka, 2006)	<p>Tiggemann (1994); Williamson, Barker, Bertman, and Gleaves (1995)</p> <p>✓</p> <p>Brannan and Petrie (2008, 2011); Dakanalis, Zanetti, Riva, and Clerici (2013)</p>	✕	✕	✕
Objectification Theory of BN & BD (Noll & Fredrickson, 1998)	<p>✓</p> <p>Augustus-Horvath and Tylka (2009); Brewster et al. (2014); Calogero (2004); Calogero, Davis, and Thompson (2005); Calogero and Thompson (2009); Daniel and Bridges (2010); Daubenmier (2005); Fitzsimmons-Craft and Bardone-Cone (2012); Frederick, Forbes, Grigorian, and Jarcho (2007); Hallsworth, Wade, and Tiggemann (2005); Holmes et al. (2014b); Kim, Seo, and Baek (2014); Kozee and Tylka (2006); Lindner, Tantleff-Dunn, and Jentsch (2012); McKinley (1998, 1999); Mercurio and Rima (2011); Mitchell and Mazzeo (2009); Moradi, Dirks, and Matteson (2005); Moradi and Rottenstein (2007); Muehlenkamp and Saris-Baglana (2002); Myers and Crowther (2008); Peat and Muehlenkamp (2011); Slater and Tiggemann (2002, 2010);</p>	✕	✕	✕

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	Strelan and Hargreaves (2005); Tiggemann and Kuring (2004); Tiggemann and Lynch (2001); Tiggemann and Slater (2001); Tiggemann and Williams (2012); Tylka and Hill (2004); Tylka and Sabik (2010); Wiseman and Moradi (2010)			
Perfectionism Model of Binge Eating (PMOBE; Sherry et al., 2014)	✓	✗	✗	✗
	Boone, Soenens, and Luyten (2014); Watson, Raykos, Street, Fursland, and Nathan (2011)			
Three-Factor Model of BN (Bardone-Cone et al., 2008)	✓	✗	✗	✗
	Bardone-Cone, Abramson, Vohs, Heatherton, and Joiner (2006); Holm-Denoma et al. (2005); Joiner, Heatherton, Rudd, and Schmidt (1997); Minnich, Gordon, Holm-Denoma, and Troop-Gordon (2014); Shaw, Stice, and Springer (2004); Steele, Corsini, and Wade (2007); Tissot and Crowther (2008); Vohs et al. (1999); Vohs, Voelz, et al. (2001); Watson, Steele, Bergin, Fursland, and Wade (2011)			
Transdiagnostic Maintenance Model of AN & BN <sup>h,i</sup> (Fairburn, 2008a)	✓	✓	✓	✓
	Allen et al. (2012); Byrne and McLean (2002); Dakanalis, Carrà, et al. (2014); Dakanalis et al. (2015); Dakanalis, Timko, Carrà, et al. (2014);	Dalle Grave, Calugi, Doll, et al. (2013); Dalle Grave et al. (2014); Fairburn et al. (2013); Schapman-Williams, Lock, and Couturier (2006)	Agras et al. (2000); Dalle Grave, Calugi, Conti, Doll, and Fairburn (2013); Fairburn et al. (2015); Fairburn et al. (2009); Fairburn et al. (1991);	Byrne, Fursland, Allen, and Watson (2011); Knott, Woodward, Hoefkens, and Limbert (2015); Stewart, Carter, Drinkwater,

	Dakanalis, Timko, Zanetti, et al. (2014); Decaluwe and Braet (2005); Fairburn (1981); Friedman and Whisman (1998); Hayaki, Friedman, Whisman, Delinsky, and Brownell (2003); Hoiles et al. (2012); Krause, Robins, and Lynch (2000); Lampard, Byrne, McLean, and Fursland (2011); Lampard et al. (2013); Murray, Rieger, Karlov, and Touyz (2013); Pedlow and Niemeier (2013); Schnitzler, von Ranson, and Wallace (2012); Shanmugam, Jowett, and Meyer (2011); Spangler (2002); Wade and Lowes (2002)		Fairburn, Jones, et al. (1993); Fairburn, Kirk, et al. (1986); Fairburn, Peveler, et al. (1993); Hilbert et al. (2007); McIntosh et al. (2005); Thackwray, Smith, Bodfish, and Meyers (1993); Wilfley et al. (1993); Wilfley et al. (2002); Wilson et al. (2002); Wonderlich et al. (2014)	Hainsworth, and Fairburn (2001)
Tripartite Influence Model of BD & DE <sup>1, k</sup> (Yamamiya et al., 2008)	✓	✗	✓	✗
	Hardit and Hannum (2012); Jones (2004); Jones, Bain, and King (2008); Jones and Crawford (2005); Karazsia and Crowther (2009); McLean, Paxton, and Wertheim (2013); Papp, Urbán, Czeglédi, Babusa, and Túry (2013); Paxton and McLean (2010); Rodgers, Chabrol, and Paxton (2011); Rodgers, Ganchou, Franko, and Chabrol (2012); Rodgers et al. (2009); Rodgers, Paxton, and McLean (2014); Scoffier, Maïano, and d'Arripe-		Bird, Halliwell, Diedrichs, and Harcourt (2013); Gollings and Paxton (2006); Heinicke et al. (2007); McLean, Paxton, and Wertheim (2011); Paxton, McLean, Gollings, Faulkner, and Wertheim (2007); Richardson and Paxton (2010)	

*Note.* RCT's = randomised-controlled trials; AN = anorexia nervosa; BD = body dissatisfaction; BN = bulimia nervosa; DE = disordered eating; ED = eating disorder.

<sup>a</sup> publications refer to most recent versions of the models for disordered eating found in the present literature.

<sup>b</sup> publications refer to testing of the model by independent research teams, as well as prospective and longitudinal testing of the model by the original (developing) research team; some cross-sectional tests of the model by the original research team are referred to in the text.

<sup>c</sup> theory extended by Cooper (2005) to develop the Vulnerability-Stress Model of ED.

<sup>d</sup> also referred to as the Interpersonal Vulnerability Model.

<sup>e</sup> theory extended by Warren, Castillo, et al. (2010) to develop the Sociocultural Model of ED.

<sup>f</sup> theory extended by Slevic and Tiggemann (2011) to develop the Sociocultural Model of DE.

<sup>g</sup> theory extended by Treasure, Corfield, and Cardi (2012) to develop the Three-Phase Model of Social Emotional Functioning in ED (AN).

<sup>h</sup> previously referred to as the Cognitive-Behavioral Model of BN (CB-BN; Fairburn, Cooper, & Cooper, 1986), and the Cognitive-Behavioral Model of AN (CB-AN; Fairburn, Shafran, & Cooper, 1998).

<sup>i</sup> theory extended by Krause et al. (2000) to develop the Mediational Model of Sociotropy, Ambivalence over Emotional Expression and Disordered Eating.

<sup>j</sup> theory extended by Rodgers et al. (2009) to develop the Biopsychosocial Model of BD & DE.

<sup>k</sup> theory extended by Jones and Crawford (2005) to develop the Dual Pathway Model of BD & DE.

**Table 2.3***Summary of Models for Disordered Eating with Independent Variables*

Models (Authors)	Independent Variables
Acceptance Model of Intuitive Eating (Augustus-Horvath & Tylka, 2011)	<ol style="list-style-type: none"> <li>1. social support</li> <li>2. self-objectification</li> <li>3. preoccupation with weight &amp; shape</li> </ol>
Cognitive Model of BN (Cooper et al., 2009)	<ol style="list-style-type: none"> <li>1. cognitive factors</li> <li>2. preoccupation with weight &amp; shape</li> <li>3. emotional regulation difficulties</li> <li>4. negative affect</li> <li>5. developmental factors</li> <li>6. dieting</li> </ol>
Cognitive-Interpersonal Maintenance Model of AN (Treasure & Schmidt, 2013)	<ol style="list-style-type: none"> <li>1. perfectionism</li> <li>2. cognitive factors</li> <li>3. interpersonal issues</li> <li>4. emotional regulation difficulties</li> </ol>
Dual-Pathway Model of BN (Stice, 2001)	<ol style="list-style-type: none"> <li>1. external pressure</li> <li>2. thin-ideal internalisation</li> <li>3. preoccupation with weight &amp; shape</li> <li>4. dieting</li> <li>5. negative affect</li> </ol>
Functional Model of Emotion Avoidance in AN (Wildes et al., 2010)	<ol style="list-style-type: none"> <li>1. negative affect</li> <li>2. emotional regulation difficulties</li> </ol>
Interpersonal Model of Binge Eating (Wilfley et al., 2000)	<ol style="list-style-type: none"> <li>1. interpersonal issues</li> <li>2. self-esteem deficits</li> <li>3. negative affect</li> <li>4. emotional regulation difficulties</li> <li>5. BMI</li> </ol>
Model of DE (Neumark-Sztainer et al., 2003)	<ol style="list-style-type: none"> <li>1. external pressure</li> <li>2. interpersonal issues</li> <li>3. preoccupation with weight &amp; shape</li> <li>4. BMI</li> <li>5. emotional regulation difficulties</li> <li>6. health &amp; nutrition attitudes</li> </ol>
Multidimensional Model of AN (Lyon et al., 1997)	<ol style="list-style-type: none"> <li>1. biogenetic predisposition</li> <li>2. self-esteem deficits</li> <li>3. self-surveillance</li> <li>4. interpersonal issues</li> </ol>
Transdiagnostic Maintenance Model of AN & BN (Fairburn, 2008a)	<ol style="list-style-type: none"> <li>1. perfectionism</li> <li>2. self-esteem deficits</li> <li>3. emotional regulation difficulties</li> <li>4. preoccupation with weight &amp; shape</li> <li>5. interpersonal issues</li> </ol>
Tripartite Influence Model of BD & DE (Yamamiya et al., 2008)	<ol style="list-style-type: none"> <li>1. external pressure</li> <li>2. thin-ideal internalisation</li> <li>3. social comparison</li> <li>4. preoccupation with weight &amp; shape</li> <li>5. self-esteem deficits</li> </ol>

*Note.* AN = anorexia nervosa; BD = body dissatisfaction; BMI = body mass index; BN = bulimia nervosa; DE = disordered eating; ED = eating disorder.

**Table 2.4***Summary of Main Constructs with Independent Variables*

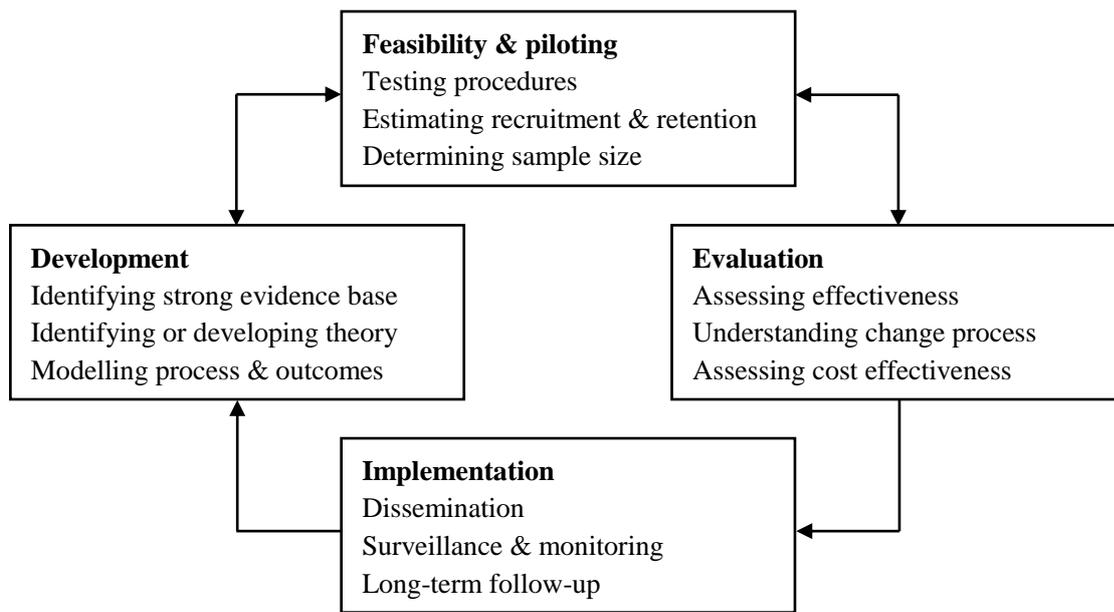
Constructs	Independent Variables
preoccupation with weight & shape self-esteem deficits	weight & shape concern; body image disturbance; body dissatisfaction; appearance anxiety; body shame low self-esteem; dysphoria; ineffectiveness; low self-efficacy; poor self-concept; aversive self-awareness
emotional regulation difficulties	mood intolerance; emotional distress; emotional dysregulation; emotional avoidance; emotional eating; poor psychological well-being; affective instability
interpersonal issues	interpersonal problems; family functioning; family connectedness; social dependency; response from close others
negative affect	depression; anxiety; affective states
thin-ideal internalisation	endorsement of the thin-ideal
external pressure	pressure to be thin; pressure to diet; family-peer weight norms & teasing; media, parental, & peer influences
perfectionism	cognitive rigidity
cognitive factors	negative self-belief; negative automatic thoughts; permissive thoughts; maladaptive cognitions; pro-anorectic beliefs; negative beliefs about eating; positive beliefs about eating
dieting	dietary restraint; unhealthy weight control behaviours
self-surveillance	poor interoceptive awareness
self-objectification	body-surveillance
body mass index	weight; weight fluctuation
social comparison	appearance comparison
social support	perceived unconditional acceptance; body acceptance by others
developmental factors	early trauma; early negative experiences
biogenetic predisposition	family history

*Note.* Variables that could not be collapsed were made independent constructs and are therefore not included in this table (i.e., health and nutrition attitudes).

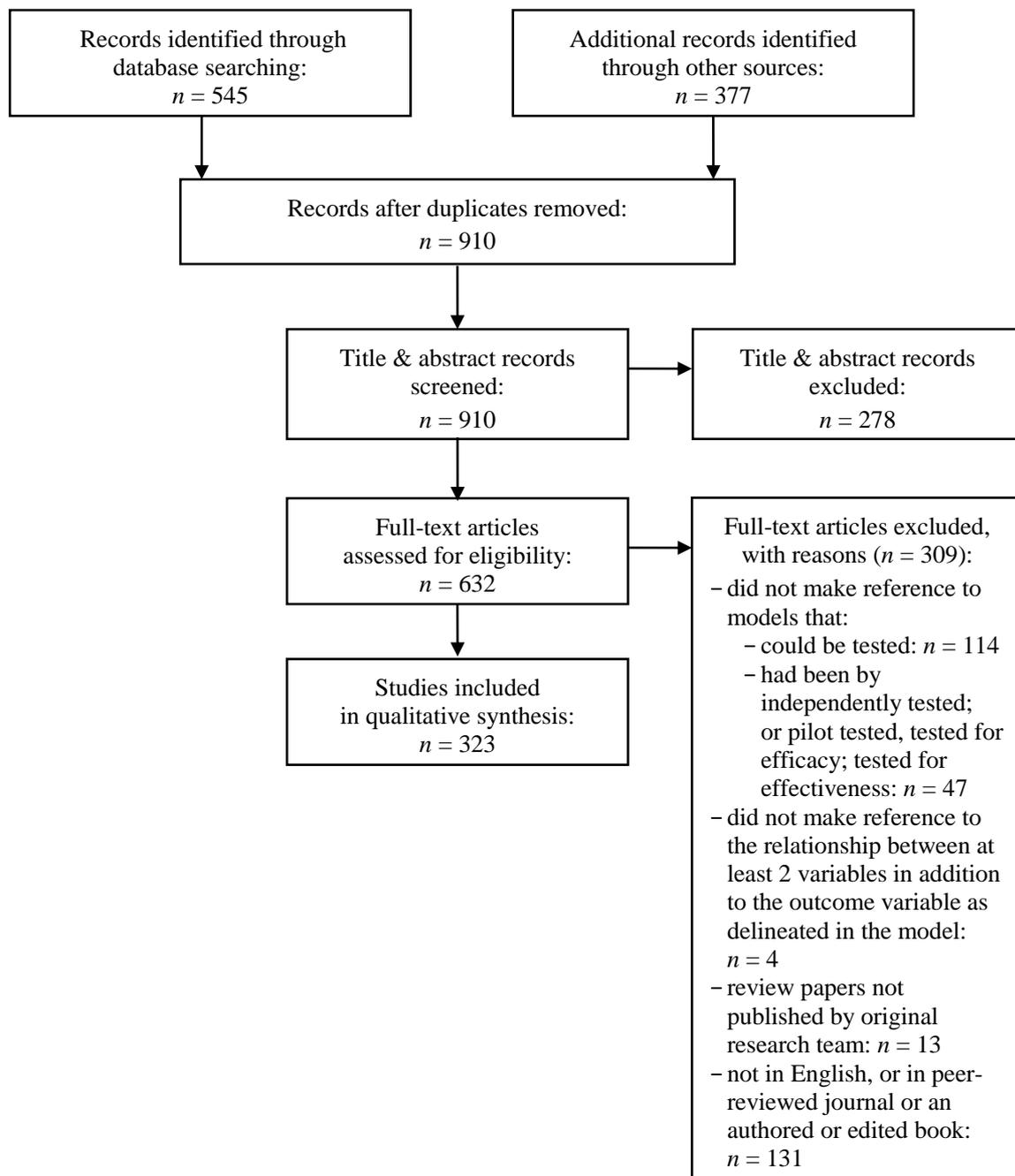
**Table 2.5***Summary of Models for Disordered Eating That Did Not Fulfil Inclusion Criteria with Reason(s)*

Models (Authors)	Reason(s) for Non-Inclusion
Aetiological Model of AN (Herpertz-Dahlmann, Seitz, & Konrad, 2011)	2; 3
Affect Regulation Model of Binge Eating (Hawkins & Clement, 1984)	1; 3
Attachment Insecurity Model of ED Psychopathology (Tasca et al., 2006)	3
Biopsychosocial Model of AN (Lucas, 1981)	1; 2; 3
Biopsychosocial Model of DE (Ricciardelli, McCabe, Holt, & Finemore, 2003)	1; 3
Biopsychosocial Model of ED (Southgate, Tchanturia, & Treasure, 2005)	1; 2; 3
Boundary Model of Eating Behavior (BN; Herman & Polivy, 1984)	1; 3
Boundary-Control Model of AN (Blank et al., 2002)	1; 2; 3
Cognitive-Social Learning Model of BN (Wilson, 1989)	1; 2; 3
Cumulative Risk Model of BN (Pike, 1995)	1; 2; 3
Dual-Process Family Model of Eating Pathology (Leung, Schwartzman, & Steiger, 1996)	1; 3
Elaborated Sociocultural Model of DE (Fitzsimmons-Craft et al., 2014)	2; 3
Emotion Regulation Model of Binge Eating (Leehr et al., 2015)	1; 2; 3
Etiological Model of BN (Petrie, Galli, Greenleaf, Reel, & Carter, 2014)	1; 2; 3
Five-Way Model of Emotion-Induced Changes in Eating (Macht, 2008)	2; 3
General Model of ED (Levine & Smolak, 1992)	1; 2
Goal Conflict Model of Eating (Stroebe, 2008)	2; 3
Integrated Model of ED Symptomatology (Kiang & Harter, 2006)	1; 2; 3
Integrative Model of Eating Psychopathology (Pinto-Gouveia, Ferreira, & Duarte, 2014)	1; 2; 3
Integrative Risk Model of BN (Pearson, Riley, Davis, & Smith, 2014)	1; 2; 3
Interdisciplinary 'Telescope' Model of ED (Barber, 1998)	1; 2; 3
Mediational Model of Autonomy, Self-Esteem & ED Attitudes & Behaviors (Frederick & Grow, 1996)	2; 3
Mediational Model of Social Physique Anxiety & ED Behaviors (Frederick & Morrison, 1998)	1; 3
Model of DE (Pike & Rodin, 1991)	1; 3
Model of Eating Behaviour (Grunert, 1989)	1; 2; 3
Model of Excessive Exercise in AN (Davis, Katzman, & Kirsh, 1999)	1; 3
Multivariate Risk Model of Eating Disturbances (Maharaj, Rodin, Olmsted, Connolly, & Daneman, 2003)	1; 3
SPAARS Model of ED Behaviours (Fox et al., 2013)	1; 3
Spiral Model of Dietary Restraint (Heatherton & Polivy, 1992)	1; 3
Tripartite Model of EDs (Markey, 2004)	1; 3
Two-Factor Model of DE (Garner, Olmsted, Polivy, & Garfinkel, 1984)	1; 3

*Note.* AN = anorexia nervosa; BN = bulimia nervosa; DE = disordered eating; ED = eating disorder; SPAARS = Schematic Propositional Analogical Associative Representation System. 1 = model could not be tested (i.e., either the model identified more than six variables in addition to the disordered eating outcome variable or the variables identified were unclear); 2 = model had not been independently investigated by researchers other than the original (developing) research team (i.e., the relationship between at least two independent variables and the outcome variable, as delineated in the model, had not been tested); 3 = model had not informed the development of an intervention that had been pilot tested, tested for efficacy, or tested for effectiveness.



**Figure 2.1** Key elements of the Medical Research Council's (MRC) evaluation framework. Figure reproduced with permission from the Medical Research Council (Craig et al., 2008; Craig et al., 2013; also see [www.mrc.ac.uk/complexinterventionsguidance](http://www.mrc.ac.uk/complexinterventionsguidance)).



**Figure 2.2** PRISMA flow chart for publication selection. Figure taken from Liberati et al. (2009) and Moher et al. (2009).

## Chapter 3

### **A Revised Dual-Pathway Model for Disordered Eating: The Importance of Emotion Regulation**

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#### **Author Contributions:**

JP led the study design, recruitment, data collection, statistical analyses, results and interpretation, and manuscript preparation. TW contributed to the study design, statistical analyses, results and interpretation, and manuscript preparation.

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This chapter has been submitted to *Eating Behaviors*, and at the time of submission of this thesis, was under review. The version that appears here is the manuscript as submitted to the journal.

### **3.1 Abstract**

The aim of the study was to examine and extend the dual-pathway model of bulimic pathology to include additional factors related to temperament in a sample of young women at risk of disordered eating. Self-report questionnaires were completed by 167 undergraduate females and a latent profile analysis differentiated two classes; a ‘non-disordered eating’ group and a ‘disordered eating’ group, the latter characterized by higher disordered eating psychopathology and higher prevalence of disordered eating behaviours. Sociocultural influences, body dissatisfaction, and difficulties with emotion regulation distinguished between groups. A cross-sectional mediation model where difficulties with emotion regulation contributed to the impact of sociocultural influences, which in turn contributed to body dissatisfaction, predicted class membership better than a model that omitted emotion regulation. Similar results were found for mediation models using a continuous outcome measure of disordered eating psychopathology. These findings suggest that the dual-pathway model may benefit from the inclusion of factors related to temperament (i.e., difficulties with emotion regulation).

## 3.2 Introduction

While an extensive range of theoretical models describing the development and maintenance of disordered eating exist (Pennesi & Wade, 2016), few have led to the development of effective interventions. One exception is dual-pathway model of bulimic pathology (Stice, 2001) which has informed the gold standard of prevention approaches for eating disorders among at-risk young women (Stice, Rohde, et al., 2009). According to this model, perceived sociocultural pressure to be thin promotes internalisation of the thin-ideal, and body dissatisfaction, which leads to dieting and negative affect, which in turn results in bulimic pathology.

Despite the model's success, two major limitations are identified. First, the model overlooks influences related to temperament, such as perfectionism, self-efficacy, self-compassion, and difficulties with emotion regulation, which are implicated in the development and maintenance of disordered eating (Bardone-Cone et al., 2007; Braun et al., 2016; Gordon et al., 2005; Sim & Zeman, 2006) and are considered among existing eating disorder models (Pennesi & Wade, 2016). Second, the model only predicts growth of bulimic symptoms and does not account for all disordered eating behaviours, including fasting and driven exercise, which are also associated with significant impairment (Wade & O'Shea, 2014). Wade and colleagues (2012) found patterns of disordered eating even at subclinical levels had a significant adverse impact on quality of life up to 9 years later. Thus the dual-pathway model as it is currently conceptualised has limited applicability.

Accordingly, the current research aims to examine and extend the dual-pathway model in two ways (**Figure 3.1**). First, additional factors related to temperament, including perfectionism, self-efficacy, self-compassion, and difficulties with emotion regulation, will be investigated. Second, the model will be applied to the full spectrum of eating disturbances. It was hypothesised that perfectionism, self-efficacy, self-compassion, and difficulties with

emotion regulation, would predict disordered eating in addition to existing factors included in the dual-pathway model (i.e., sociocultural pressure to be thin, thin-ideal internalisation, body dissatisfaction, and negative affect). Further, it was hypothesised that these additional variables would promote sociocultural pressure to be thin and thin-ideal internalisation, which would promote body dissatisfaction, which would lead to negative affect, which in turn would result in disordered eating. This mediational hypothesis was consistent with the dual-pathway model. Dieting, which was originally included in the dual-pathway model, could not be investigated as the measurement items were already included in our measure of eating psychopathology.

### **3.3 Method**

#### **3.3.1 Participants.**

Participants were female undergraduate students. Given that disordered eating occurs predominantly among females (Hudson et al., 2007; Liechty & Lee, 2013), and that early adulthood poses risk for the emergence of new eating disorders (Favaro, Caregaro, Tenconi, Bosello, & Santonastaso, 2009; Kessler et al., 2013), this sample was considered to be at greater risk for disordered eating.

In order to obtain a more homogenous sample with respect to the relation between actual body size and body dissatisfaction, participants with a body mass index (BMI) of  $\geq 30$  (i.e., the criteria for obesity; World Health Organization [WHO], 2006;  $n = 14$ ,  $M = 34.31$ ,  $SD = 4.58$ ) were excluded. The final sample included 167 females aged 17 to 25 years ( $M_{age} = 19.20$ ,  $SD = 1.63$ ) with a mean BMI in the normal range (i.e., BMI 18.5–24.99; WHO, 2006;  $M = 22.04$ ,  $SD = 2.99$ ). The majority of participants (85.6%) self-reported as Caucasian.

#### **3.3.2 Procedures.**

The study was presented as an investigation of issues relating to different eating styles in women. Participants completed online self-report questionnaires in a research laboratory and earned course credits for participation. The Flinders University Social and Behavioural Research Ethics Committee approved this study.

### **3.3.3 Measures.**

In each case, higher scores indicated higher levels of the construct.

***Disordered eating.*** Disordered eating behaviours from the past 28 days were assessed using the diagnostic items from the Eating Disorder Examination–Questionnaire (EDE-Q; Fairburn & Beglin, 1994). The EDE-Q has demonstrated moderate to high convergent validity with the Eating Disorder Examination (EDE; Berg, Peterson, Frazier, & Crow, 2011; Fairburn & Cooper, 1993), and has demonstrated the ability to distinguish between eating disorder and non-eating disorder cases (Mond, Hay, Rodgers, Owen, & Beumont, 2004).

Disordered eating psychopathology was assessed using 22 items from the Weight Concern, Shape Concern, Eating Concern, and Dietary Restraint subscales of the EDE-Q which make up the global score. The global score is commonly used as an indicator of outcome (recovery) in treatment studies (Bardone-Cone et al., 2010; Fairburn et al., 2009). The EDE-Q subscales have demonstrated high internal consistency ( $\alpha = .70-.93$ ) and test-retest reliability (1 to 14-day interval,  $\rho_1 = .66-.94$ ; Berg, Peterson, Frazier, & Crow, 2012; Peterson et al., 2007), and the global score has demonstrated strong internal consistency ( $\alpha = .95$ ; Kelly, Carter, Zuroff, & Borairi, 2013) and high convergent validity with the EDE global score (Mond et al., 2004). In the current study the Cronbach's alpha ranged from .78 to .91 for the EDE-Q subscales, and was .95 for the global score.

***Perfectionism.*** This was measured using 16 items from the Concern over Mistakes and Personal Standards subscales of the Frost Multidimensional Perfectionism Scale (FMPS;

Frost, Marten, Lahart, & Rosenblate, 1990). These subscales were positively correlated ( $r = .597$ ) and were therefore combined to make a single ‘perfectionism’ variable. Items were scored on 5-point scales. Both subscales have demonstrated high internal consistency ( $\alpha = .77-.93$ ) and high correlations with other measures of perfectionism (Bardone-Cone et al., 2008; Frost et al., 1990). In the current study the Cronbach’s alpha was .93.

***Body dissatisfaction.*** This was measured as current weight – ideal weight. Current weight was measured by the researcher.

***Body mass index (BMI).*** This was measured as weight / height<sup>2</sup>. Height was based on self-report data but a tape measure was provided to measure accurate height if unknown.

***Self-efficacy.*** This was measured using the 17-item General Self-Efficacy subscale of the Self-Efficacy Scale (Sherer et al., 1982). Items were scored on 5-point scales. This subscale has shown good internal consistency ( $\alpha = .71-.89$ ) and validity (Bardone-Cone et al., 2008; Sherer et al., 1982). In the current study the Cronbach’s alpha was .87.

***Difficulties with emotion regulation.*** This was assessed using the 36-item Difficulties in Emotion Regulation Scale (Gratz & Roemer, 2004). Items were scored on 5-point scales. This scale has excellent internal consistency ( $\alpha = .93-.96$ ), good test-retest reliability (4 to 8-week interval,  $\rho_1 = .88$ ), and adequate construct validity (Gratz & Roemer, 2004; Manuel & Wade, 2013). In the current study the Cronbach’s alpha was .94.

***Self-compassion.*** This was measured using the 12-item Self-Compassion Scale (Raes, Pommier, Neff, & Van Gucht, 2011). Items were scored on 5-point scales. The scale has demonstrated high internal consistency ( $\alpha = .92$ ) and test-retest reliability (3-week interval,  $\rho_1 = .93$ ), and has shown to be positively correlated with similar measures of social connectedness and emotional intelligence (Neff, 2003a). In the current study the Cronbach’s alpha was .83.

***Sociocultural influences.*** This was measured using 16 items from the Pressures and Internalization-General subscales of the Sociocultural Attitudes Towards Appearance Questionnaire-3 (Thompson, van den Berg, Roehrig, Guarda, & Heinberg, 2004). Items were scored on 5-point scales. Both subscales have demonstrated high internal consistency in community and disordered eating samples ( $\alpha = .92-.97$ ), and good convergent validity with similar subscales of internalization and drive for thinness ( $r = .38-.58$ ; Calogero, Davis, & Thompson, 2004). In the current study the Cronbach's alpha was .95.

***Negative affect.*** This was measured using 14 items from the Depression and Anxiety subscales of the Depression Anxiety Stress Scale (Henry & Crawford, 2005). Items were scored on 4-point scales. Both subscales have demonstrated high internal consistency ( $\alpha = .90-.95$ ) and good convergent validity with independent measures of depression and anxiety (Henry & Crawford, 2005). In the current study the Cronbach's alpha was .92.

### **3.3.4 Statistical analyses.**

All analyses used the *Statistical Package for the Social Sciences (SPSS)* version 23 unless otherwise specified.

***Latent classes.*** Latent Profile Analysis (LPA; Collins & Lanza, 2010) was employed to investigate different latent subgroups (classes). LPA is an extension of latent class analysis that can accommodate continuous, ordinal and categorical indicator variables to identify subgroups of people who exhibit similar patterns of behaviour. LPA is based on *within-class (or conditional) independence* (Lazarsfeld & Henry, 1968), whereby classes are created such that (within-class) indicator variables are statistically independent (uncorrelated). Disordered eating psychopathology (i.e., EDE-Q subscales) and disordered eating behaviours (i.e., purging/laxative misuse, driven exercise/fasting) were used to derive latent classes.

In the process of determining the number of latent classes, available theory, existing research findings, clinical knowledge, and statistical fit indices were considered. In determining model selection (Ram & Grimm, 2009), two issues are considered: (1) the optimal number of latent classes; and (2) the type and extent of differences between and within those classes. The former is determined using relative fit *information criteria* including Akaike Information Criteria (AIC), Bayesian Information Criteria (BIC), and sample-size Adjusted BIC (ABIC). Better-fitting models generally produce lower absolute values (Nylund, Asparouhov, & Muthén, 2007). Models can also be evaluated by classification precision (*entropy*), where higher values are indicative of greater precision (where 1.0 represents perfect classification) and identify the preferred model (Muthén, 2004). Finally, comparisons are made with *likelihood ratio tests* including Vuong-Lo-Mendell-Rubin likelihood ratio test (VLMR-LRT) and Adjusted Lo-Mendell-Rubin likelihood ratio test (Adjusted LRT), where classes ('C') are compared with an analogous model with one fewer classes ('C-1'). Analyses were undertaken with *Mplus* version 7.31.

***Predictors of class membership.*** Consistent with an open science approach to interpreting significance (Cumming & Calin-Jageman, 2016), mean differences between identified classes were investigated using a between-group effect size (Cohen's *d*) with 95% confidence intervals (CI). If the CI is entirely above or below zero, the null hypothesis is rejected and the difference between groups is considered to be statistically significant (Hayes, 2009). Those statistically significant univariate risk factors were then subsequently entered into a multivariate binary logistic regression model to determine the factors independently differentiating class membership.

***Mediational pathways.*** Mediational pathways were tested to predict class membership and disordered eating psychopathology respectively. Mediation analysis is typically used to describe the way in which one variable (predictor) influences another

variable (outcome) through its effect on one or more mediator variables. Mediation was tested using PROCESS macro (Hayes, 2013). Among its other functions, this computational tool allows for simultaneous multivariate analysis and uses bootstrapping to investigate models with multiple mediators. The indirect (or mediating) effects were tested through corrected bootstrapping, which is recommended in mediation analysis with small to moderate sample sizes. Bootstrap analysis is one of the more powerful methods for detecting indirect effects and has improved accuracy of CIs and the best Type I error rates compared to alternative resampling methods (Hayes, 2009; MacKinnon, Lockwood, & Williams, 2004). Bootstrapping with 10000 resamples was used to generate 95% CIs to determine the significance of the indirect effect. As is recommended by Hayes (2013) we did not test preconditions and avoid interpreting the results in terms of *complete* or *partial* mediation.

### **3.4 Results**

#### **3.4.1 Preliminary analyses.**

Variables were normally distributed with the exception of negative affect and disordered eating behaviours (positively skewed), and sociocultural influences (negatively skewed). The square root and reflected square root of negative affect and sociocultural influences were used in all analyses respectively (Tabachnick & Fidell, 2013).

#### **3.4.2 Descriptives.**

Means and standard deviations are shown in **Table 3.1**. The majority of participants (85.6%) wanted to reduce their current body weight, and some (7.2%) wanted a higher body weight. The majority of participants (80.8%) reported engaging in some form of disordered eating behaviour, specifically bingeing (62.9%), driven exercise/fasting (61.7%), and purging/laxative misuse (4.8%).

#### **3.4.3 Associations between variables.**

Correlations (**Table 3.1**) were all in the expected direction. Our measure of body dissatisfaction was highly correlated with BMI, and therefore we included body dissatisfaction but not BMI in all analyses. Body dissatisfaction was also originally included in the dual-pathway model, and therefore this variable was retained. While high correlations between some variables were present, a decision was made to keep these variables separate in order to compare our results to previous studies. However, the most highly correlated scales (Pressures and Internalization-General subscales) were combined to make a single ‘sociocultural influences’ variable; **Figure 3.2** presents the updated configuration of variables.

#### **3.4.4 Latent classes.**

A LPA specifying 1 through to 6 latent classes was performed (**Table 3.2**). First, AIC, BIC and ABIC criteria incrementally decreased with increasing numbers of latent classes. Second, increasing the number of classes resulted in increasing accuracy of group membership classification (*entropy*) initially (i.e., models C2 through C4) before deteriorating (i.e., models C5 through C6), with classification confidence lowest for the 6-class model. Third, *likelihood ratio tests* (i.e., VLMR-LRT, Adjusted LRT) were used to compare each model with an analogous model with one fewer class. The C-1 model could not be rejected in favour of the C model in all instances (i.e., models for C3 through C6) except for the C2 model which reached significance. Across these indicators, it was decided that the 2-class model was most appropriate for these data.

The largest class (LP1;  $n = 111$ , 66.5%) was characterized by lower disordered eating psychopathology across all EDE-Q subscales and lower prevalence of disordered eating behaviours, termed the ‘non-disordered eating’ group. LP2 ( $n = 56$ , 33.5%) was characterized by higher levels of disordered eating psychopathology and higher prevalence of disordered eating behaviours, termed the ‘disordered eating’ group. **Table 3.3** presents a summary of

comparisons between these two latent profiles. All variables were significant predictors of group membership in the expected directions, with the exception of perfectionism.

Significant predictors were entered into a multivariate model (**Table 3.4**), where sociocultural influences, body dissatisfaction, and difficulties with emotion regulation, were shown to uniquely discriminate between classes.

### **3.4.5 Mediation pathways.**

Those risk factors uniquely discriminating class membership were included in the investigation of four mediation models to predict class and eating disordered psychopathology respectively. In Model 1 (**Figure 3.3**), consistent with the proposed revised dual-pathway model, and incorporating variables included in the multivariate model, sociocultural influences and body dissatisfaction were explored as mediators (in serial) of the relationship between difficulties with emotion regulation and class. In Model 2 (**Figure 3.4**), consistent with the original dual-pathway model, difficulties with emotion regulation was omitted. In Model 3 and Model 4 (**Figure 3.5** and **Figure 3.6** respectively), the same mediation models above were investigated with disordered eating psychopathology as the outcome variable. The indirect effect (mediational pathway) was statistically significant for all models (**Table 3.5**). Variance explained and model results indicated that Model 1 was superior to Model 2, with an extra 7-10% of variance of group membership explained with the inclusion of emotion regulation on top of 32-47% already explained. A similar pattern of results was found for Model 3 and Model 4, with an additional 7% of the variance of disordered eating psychopathology explained with the inclusion of emotion regulation on top of 50% already explained.

## **3.5 Discussion**

### **3.5.1 Summary of findings.**

The aim of the present research was to investigate the inclusion of temperament variables in the dual-pathway model across the full spectrum of eating disturbances. Two latent classes were identified across disordered eating psychopathology and disordered eating behaviour indicators: the ‘non-disordered eating’ group, and the ‘disordered eating’ group. Sociocultural influences, body dissatisfaction, and difficulties with emotion regulation uniquely distinguished group membership. Cross-sectional mediation models with disordered eating measured dichotomously and continuously showed that greater difficulties with emotion regulation was associated with higher levels of sociocultural influences, which was associated with higher body dissatisfaction, which in turn was associated with greater likelihood of belonging to the ‘disordered eating’ group. When difficulties with emotion regulation was removed and the model was re-tested, variance explained and model results worsened, suggesting that the incorporation of difficulties with emotion regulation resulted in a better explanation of disordered eating.

The present findings add to the growing literature suggesting that difficulties with emotion regulation may be implicated in the aetiology of eating pathology (Gianini et al., 2013; Svaldi et al., 2012). In his meta-analytic review, Stice (2002) identified negative emotionality as a major precursor for the development of eating pathology, body dissatisfaction, and calorie intake, suggesting that negative emotionality may amplify the effects of other risk factors. It is suggested that inadequate coping skills or difficulties coping with adverse emotional states may cause women to become less effective at managing urges to binge (i.e., caused by attempts to restrict their eating) and less able to manage negative affect, thus relying more on disordered eating to regulate emotion (Stice, 1994). While negative affect is considered within the dual-pathway model, the contribution of difficulties with emotion regulation in particular is yet to be investigated.

In the current research negative affect was not found to have a unique relationship with disordered eating after accounting for difficulties with emotion regulation. This is consistent with research showing that emotion regulation difficulties explains unique variance in eating pathology beyond the contribution of other factors already included in the dual-pathway model such as negative affect (Gianini et al., 2013; Whiteside et al., 2007). This offers an important clarification of the aspects of negative affect that need to be targeted in interventions.

The dual-pathway model has resulted in the development of effective prevention approaches with young women who are body dissatisfied (e.g., dissonance-based prevention programs; Stice, Rohde, et al., 2009), but the current findings indicate that emotion regulation strategies should also be considered in the development of future prevention approaches for disordered eating. For example, programs could teach women more adaptive ways of coping with negative emotions, including strategies aimed at reducing or tolerating arousal such as exposure and response prevention or other self-management strategies. Such emotion regulation strategies are shown to be more effective than cognitive strategies in situations where individuals are under extreme emotional arousal (Fruzzetti, Crook, & Erikson, 2008). There is considerable evidence supporting the use of such strategies in eating disorder treatment, in particular dialectical behaviour therapy (DBT; Linehan, 1993), in which the central focus is emotion regulation. While typically applied to individuals with borderline personality disorder or suicidal behaviours, studies have demonstrated the effectiveness of DBT in bulimia nervosa (Safer, Telch, & Agras, 2001) and binge eating disorder (Telch, Agras, & Linehan, 2001). Furthermore, more recent adaptations of CBT for eating disorders have incorporated an emphasis on emotion regulation (e.g., Fairburn, 2008a). Further, research in the prevention field has suggested that mindfulness-based acceptance approaches may be used to target those at risk of emotion regulation difficulties and disordered eating

(e.g., Atkinson & Wade, 2012). Future prevention efforts would benefit from further exploration of the use of emotion regulation strategies to reduce the risk of disordered eating.

### **3.5.2 Limitations of current research.**

The results of the study should be interpreted in the context of several limitations. First, this study utilised cross-sectional data, therefore causation is unable to be established. Longitudinal or experimental work is required to inform causality. Second, disordered eating behaviours were measured using self-report items from the EDE-Q, a standardised measure with acceptable psychometrics (Berg et al., 2011, 2012); however the use of structured interviews may improve predictive validity. While the use of confidential self-report procedures may encourage honest responding (Lavender & Anderson, 2009), use of interview data would increase the strength of findings. Third, body dissatisfaction was measured as the discrepancy between current and ideal weight, which may be more accurately a weight dissatisfaction construct; the use of a standardised measure of body dissatisfaction may improve model fit further. Fourth, the present model focuses on the aetiology of disordered eating among non-obese (i.e., BMI <30) undergraduate females aged between 17 and 25 years, and is unable to inform the development of disordered eating in other groups (e.g., younger/older women, men, the wider community).

### **3.5.3 Future directions.**

These findings have several implications for future research. Whereas the present research focused on predicting the full spectrum of eating disturbances, future research might investigate whether the predictors of diagnostic levels of eating pathology differ. Second, while this study adds to the existing literature examining the dual-pathway model, few independent researchers have replicated the complete model longitudinally, and given the current debate about reproducibility in science (Open Science Collaboration, 2015), it is

important that future researchers address this gap in order to inform the development of future effective interventions. Third, researchers should consider testing the model with the inclusion of factors related to temperament (i.e., difficulties with emotion regulation) as well as additional risk factors implicated in the development of disordered eating that are not considered within the current model.

**Table 3.1***Summary of Descriptive Statistics and Correlations between Variables*

Variable	Mean (SD)	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Sociocultural influences	1.64 (.26)	–									
2. BMI	22.04 (2.99)	.128	–								
3. Body dissatisfaction	4.40 (5.00)	<b>.307**</b>	<b>.793**</b>	–							
4. Perfectionism	2.99 (.78)	<b>.209**</b>	.049	.015	–						
5. Self-efficacy	3.34 (.53)	<b>-.266**</b>	-.050	-.137	-.090	–					
6. Negative affect	.72 (.37)	<b>.316**</b>	.067	.110	.071	<b>-.411**</b>	–				
7. Difficulties with emotion regulation	2.58 (.66)	<b>.357**</b>	.138	<b>.249**</b>	.130	<b>-.579**</b>	<b>.663**</b>	–			
8. Self-compassion	2.80 (.57)	<b>-.481**</b>	-.134	<b>-.179*</b>	<b>-.185*</b>	<b>.496**</b>	<b>-.610**</b>	<b>-.726**</b>	–		
9. EDE-Q global	2.19 (1.30)	<b>.595**</b>	<b>.407**</b>	<b>.545**</b>	.063	<b>-.351**</b>	<b>.375**</b>	<b>.507**</b>	<b>-.470**</b>	–	
10. Purging/laxative misuse	.30 (2.33)	.149	.036	.090	<b>.196*</b>	-.095	<b>.231**</b>	<b>.247**</b>	<b>-.210**</b>	<b>.261**</b>	–
11. Driven exercise/fasting	6.23 (8.32)	<b>.398**</b>	<b>.205**</b>	<b>.313**</b>	.150	-.118	<b>.248**</b>	<b>.300**</b>	<b>-.311**</b>	<b>.566**</b>	<b>.251**</b>

*Note.* SD = standard deviation; BMI = body mass index; EDE-Q = Eating Disorder Examination–Questionnaire.  $N = 167$ , \* $p < .05$ , \*\* $p < .001$ . Significant values are in **bold**.

**Table 3.2***Information Criteria for Latent Profile Modelling with 1-6 Latent Classes*

Classes	Fit statistics					
	No. of free parameters	AIC	BIC	ABIC	Entropy	LRT <sup>§1</sup> <i>p</i>
N <sub>c=1</sub>	12	4320.987	4358.403	4320.409	–	–
N <sub>c=2</sub>	19	3880.882	3940.124	3879.967	.947	<b>.040</b>
N <sub>c=3</sub>	26	3573.998	3655.066	3572.746	.966	.368
N <sub>c=4</sub>	33	3279.821	3382.715	3278.232	.972	.231
N <sub>c=5</sub>	40	3187.623	3312.343	3185.697	.958	.334
N <sub>c=6</sub>	47	3071.662	3218.208	3069.399	.934	.589

*Note.* AIC = Akaike information criteria; BIC = Bayesian information criteria; ABIC = Adjusted BIC; LRT = likelihood ratio tests; N<sub>c</sub> = number of latent classes. Significant values are in **bold**.

<sup>1</sup> LRT<sup>§</sup> (e.g., Vuong-Lo-Mendell-Rubin likelihood ratio tests; Lo-Mendell-Rubin adjusted LRT test) quantify specific comparisons between the model of interest and a model with one fewer class, C-1.

**Table 3.3***Summary of Comparison between Latent Profiles (LP)*

Variable	LP1	LP2	ES <sup>1</sup> (95% CI)
	Mean (SD)	Mean (SD)	
<i>Variables used to derive latent classes</i>			
EDE-Q weight concern	1.50 (.96)	4.27 (.83)	<b>-3.02 (-3.17, -2.56)</b>
EDE-Q shape concern	2.01 (.95)	4.73 (.85)	<b>-2.96 (-3.42, -2.51)</b>
EDE-Q eating concern	.60 (.53)	2.68 (1.16)	<b>-2.61 (-3.03, -2.18)</b>
EDE-Q dietary restraint	.95 (.77)	2.66 (1.32)	<b>-1.73 (-2.10, -1.36)</b>
Purging/laxative misuse	.00 (.00)	.89 (3.99)	<b>-.39 (-.71, -.06)</b>
Driven exercise/fasting	3.31 (5.29)	12.04 (10.07)	<b>-1.21 (-1.55, -.86)</b>
<i>Variables used to validate latent classes</i>			
BMI	21.39 (2.94)	23.31 (2.70)	<b>-.67 (-1.00, -.34)</b>
Sociocultural influences	1.55 (.24)	1.82 (.20)	<b>-1.19 (-1.53, -.84)</b>
Body dissatisfaction	2.85 (4.60)	7.47 (4.34)	<b>-1.02 (-1.36, -.68)</b>
Perfectionism	2.97 (.77)	3.03 (.82)	-.08 (-.40, .25)
Self-efficacy	3.46 (.54)	3.11 (.43)	<b>.69 (.36, 1.02)</b>
Negative affect	.64 (.33)	.89 (.39)	<b>-.71 (-1.04, -.38)</b>
Difficulties with emotion regulation	2.37 (.57)	3.00 (.60)	<b>-1.09 (-1.43, -.74)</b>
Self-compassion	2.97 (.53)	2.50 (.51)	<b>.90 (.56, 1.23)</b>

*Note.* LP1 = latent profile 1; LP2 = latent profile 2; SD = standard deviation; ES = effect size (Cohen's *d*); CI = confidence interval; BMI = body mass index; EDE-Q = Eating Disorder Examination–Questionnaire. LP1 (*n* = 111); LP2 (*n* = 56). **Bolded** ES indicates significant difference between groups.

<sup>1</sup> Indicates between-group ES.

**Table 3.4***Summary of Multivariate Statistics of Predictors with Class*

Variable	Wald	<i>p</i>	Exp <i>B</i> (95% CI)
Sociocultural influences	15.314	<.001	<b>4.044 (2.009, 8.143)</b>
Body dissatisfaction	13.660	<.001	<b>3.085 (1.698, 5.607)</b>
Self-efficacy	.799	.371	.786 (.464, 1.333)
Negative affect	.146	.703	1.110 (.649, 1.899)
Difficulties with emotion regulation	6.741	.009	<b>2.856 (1.293, 6.306)</b>
Self-compassion	.356	.551	1.259 (.591, 2.685)

*Note.* Exp = exponent; CI = confidence interval. Significant values are in **bold**.

**Table 3.5***Testing Predicted Mediation Pathways*

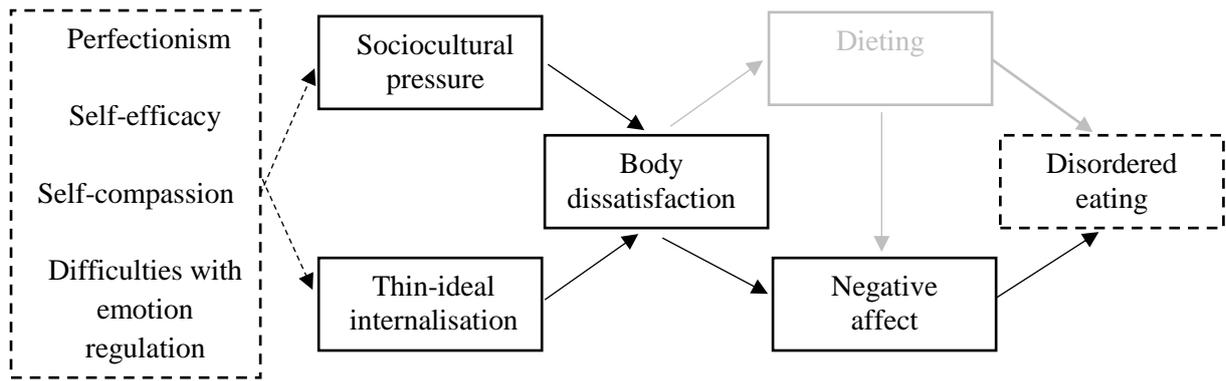
Model	Model Fit Statistics					Indirect Effect		
	-2LL <sup>1</sup>	Model LL <sup>2</sup>	McFadden <sup>3</sup>	CoxSnell <sup>3</sup>	Nagelkrk <sup>3</sup>	Effect	SE	Boot 95% CI
Model 1								
Difficulties with emotion regulation → sociocultural influences → body dissatisfaction → class	125.355	86.880	.410	.408	.565	<b>1.140</b>	<b>.379</b>	<b>.564, 1.919</b>
Model 2								
Sociocultural influences → body dissatisfaction → class	144.016	68.218	.321	.337	.467	<b>1.281</b>	<b>.556</b>	<b>.541, 2.677</b>
Model Summary								
	R square	F	df1	df2	p	Indirect Effect		
						Effect	SE	Boot 95% CI
Model 3								
Difficulties with emotion regulation → sociocultural influences → body dissatisfaction → disordered eating psychopathology	.564	69.961	3	162	<.001	<b>.445</b>	<b>.097</b>	<b>.260, .644</b>
Model 4								
Sociocultural influences → body dissatisfaction → disordered eating psychopathology	.496	80.282	2	163	<.001	<b>.613</b>	<b>.155</b>	<b>.349, .964</b>

*Note.* -2LL = -2 log likelihood; Model LL = model log likelihood; McFadden = McFadden R<sup>2</sup>; CoxSnell = Cox & Snell R<sup>2</sup>; Nagelkrk = Nagelkerke R<sup>2</sup>; SE = bootstrap standard error; Boot 95% CI = bootstrap 95% confidence interval; *df* = degrees of freedom. Significant indirect effects are in **bold**.

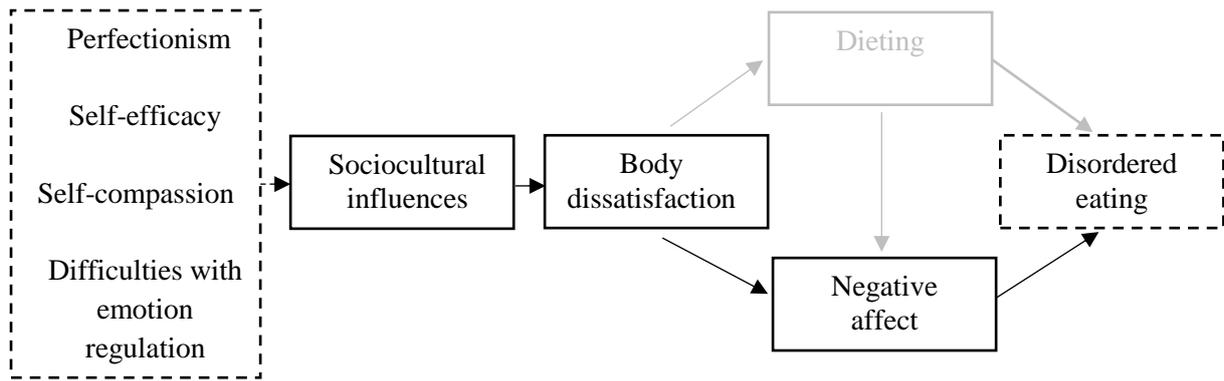
<sup>1</sup> Indicates measure of model deviance or how poorly the model predicts decisions (i.e., the smaller the statistic the better the model).

<sup>2</sup> Indicates measure of the difference in likelihood between the estimated model and the model with no predictors.

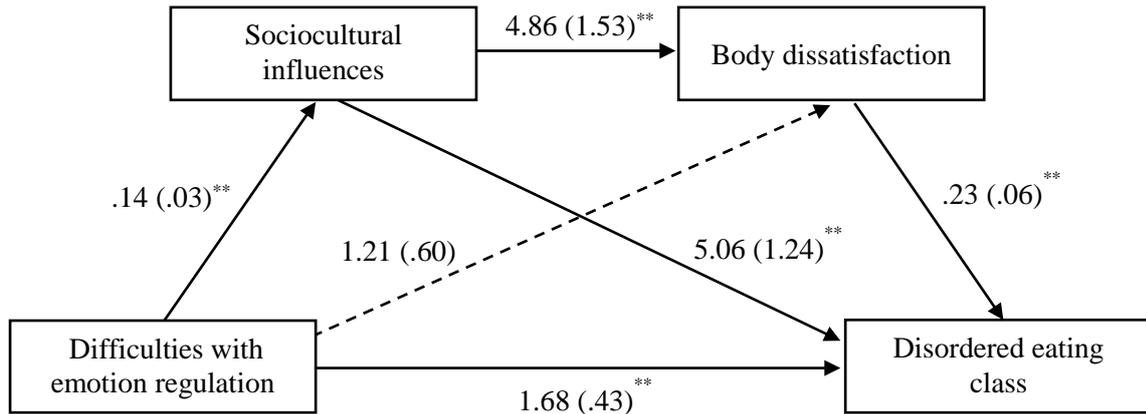
<sup>3</sup> Pseudo R<sup>2</sup> (analogous to R<sup>2</sup> in linear regression) indicates measure of substantive significance of the model.



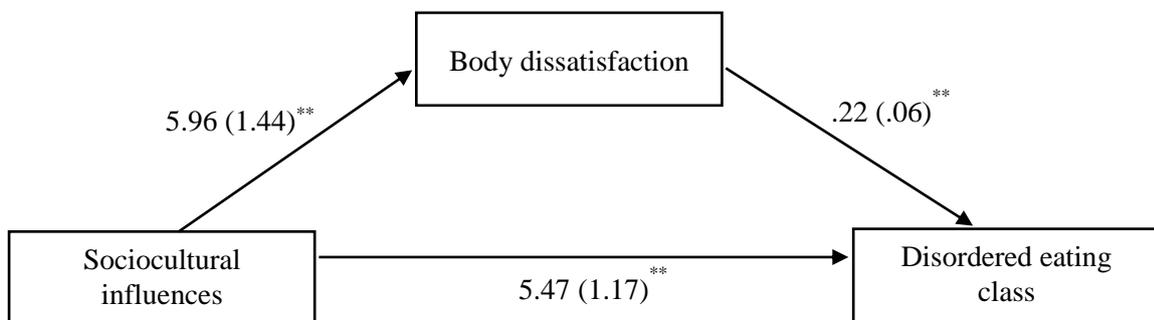
**Figure 3.1** Theoretical components of the current revised dual-pathway model of disordered eating. Solid lines represent the original dual-pathway model as most recently conceptualised in Stice (2001). Broken lines represent the proposed revisions to the model. Washed-out lines represent pathways that were not investigated.



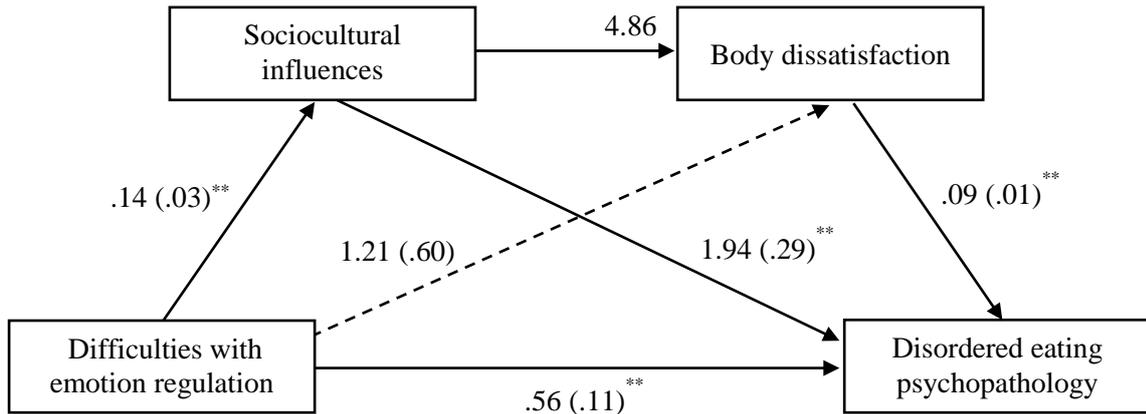
**Figure 3.2** Theoretical components of the current revised dual-pathway model of disordered eating, with Sociocultural pressure and Thin-ideal internalisation combined to make single Sociocultural influences variable. Solid lines represent the original dual-pathway model (Stice, 2001), with the exception of Sociocultural influences. Broken lines represent the proposed revisions to the model. Washed-out lines represent pathways that were not investigated.



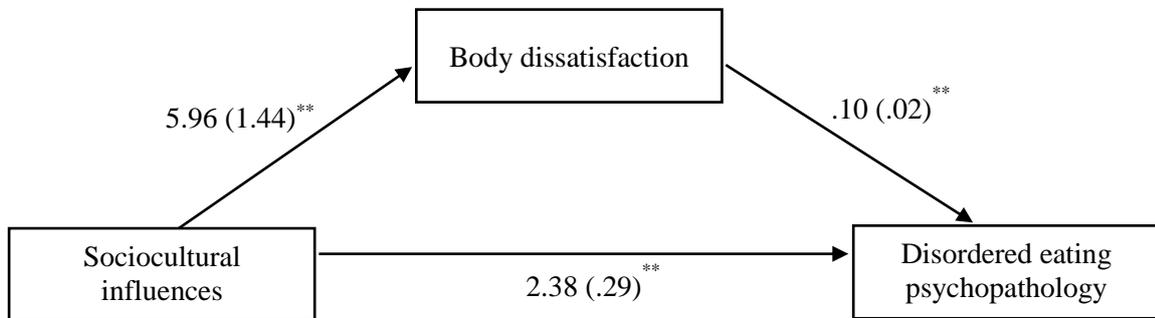
**Figure 3.3** Multiple mediation analysis with unstandardized path coefficients (and standard errors) and the estimates of the direct effect of difficulties with emotion regulation on class. Significant pathways are indicated with a solid line. \* $p < .05$ ; \*\* $p < .01$ .



**Figure 3.4** Simple mediation analysis with unstandardized path coefficients (and standard errors) and the estimates of the direct effect of sociocultural influences on class. Significant pathways are indicated with a solid line. \* $p < .05$ ; \*\* $p < .01$ .



**Figure 3.5** Multiple mediation analysis with unstandardized path coefficients (and standard errors) and the estimates of the direct effect of difficulties with emotion regulation on disordered eating psychopathology. Significant pathways are indicated with a solid line. \* $p < .05$ ; \*\* $p < .01$ .



**Figure 3.6** Simple mediation analysis with unstandardized path coefficients (and standard errors) and the estimates of the direct effect of sociocultural influences on disordered eating psychopathology. Significant pathways are indicated with a solid line. \* $p < .05$ ; \*\* $p < .01$ .

## Chapter 4

### **A Revised Dual-Pathway Model for Disordered Eating: A Longitudinal Study Implicating Self-Compassion and Emotion Regulation**

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#### **Author Contributions:**

JP led the study design, recruitment, data collection, statistical analyses, results and interpretation, and manuscript preparation. TW contributed to the study design, statistical analyses, results and interpretation, and manuscript preparation.

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This chapter was submitted and subsequently rejected from the *International Journal of Eating Disorders* as it did not meet the minimum requirements of a longitudinal study. The version that appears here is the manuscript as originally submitted to the journal.

#### **4.1 Abstract**

The aim of the present study was to examine and extend the variables included in the dual-pathway model in a longitudinal study of at-risk young women. Additional factors related to temperament, including perfectionism, self-efficacy, difficulties with emotion regulation, and self-compassion, were investigated. One hundred and sixty-seven undergraduate females were tested across three waves of data collection over a 12-month period. Two latent classes were identified, including one group characterised by low and ascending disordered eating psychopathology and one group characterised by high and decreasing disordered eating psychopathology over time, with baseline sociocultural influences, body dissatisfaction, and difficulties with emotion regulation, distinguishing group membership. Mediational pathways were also investigated, and a significant pathway was found between self-compassion, negative affect and disordered eating psychopathology. Together, these findings suggest that these factors play an important role in the development of disordered eating, and the dual-pathway model may benefit from the inclusion of factors related to temperament (i.e., difficulties with emotion regulation and self-compassion). Further, these findings suggest that prevention approaches for disordered eating should consider the role of temperament.

## 4.2 Introduction

Disordered eating behaviours and associated problems are prevalent among women (Hudson et al., 2007), with serious consequences, including increased mortality and suicide rates (Crow et al., 2012), impairment across several health dimensions, and decreased quality of life (Wade et al., 2012). There has been growing interest in identifying and understanding the putative risk factors, and identifying explanatory models that describe the mechanism of action, in order to inform the development of prevention strategies. However, while an extensive range of theoretical models exist (Pennesi & Wade, 2016), few researchers have developed and tested integrative etiological models across the full spectrum of eating disturbances (Fairburn et al., 2003; Stice, 2001). In addition, there has been a reliance on correlational testing; few studies have investigated disordered eating growth over time (e.g., Engler et al., 2006; Stice, 2001).

One exception is dual-pathway model of bulimic pathology (Stice, 2001) which has informed the gold standard of prevention approaches for eating disorders among young women (Stice, Rohde, et al., 2009). According to this model, perceived sociocultural pressure to be thin promotes internalisation of the thin-ideal and body dissatisfaction, which leads to dieting and negative affect, which in turn results in bulimic pathology. Despite the model's success, the model overlooks influences related to temperament which have shown to be important in the development and maintenance of disordered eating (Jacobi & Fittig, 2010; Pennesi & Wade, 2016). Further, the model neglects to account for the full spectrum of disordered eating behaviours, namely fasting and driven exercise, that also have a serious adverse impact (Wade & O'Shea, 2014), and longitudinal replications of the whole (Rohde, Stice, & Marti, 2015; Stice, 2001), and partial model (Dakanalis, Timko, Carrà, et al., 2014; Engler et al., 2006) are scarce.

The current research aims to examine and extend the dual-pathway model (Stice, 2001) in three ways. First, additional factors related to temperament, including perfectionism, self-efficacy, self-compassion, and difficulties with emotion regulation, will be investigated. Second, the model will be applied to the full spectrum of eating disturbances, as well as a global measure of eating psychopathology. Finally, the model will be examined longitudinally. It was hypothesised that perfectionism, self-efficacy, self-compassion, and difficulties with emotion regulation, would predict disordered eating in addition to existing factors included in the dual-pathway model (i.e., sociocultural pressure to be thin, thin-ideal internalisation, body dissatisfaction, and negative affect). Further, it was also hypothesised that negative affect would mediate the relationships between a variety of baseline variables (i.e., sociocultural pressure to be thin, thin-ideal internalisation, perfectionism, self-efficacy, difficulties with emotion regulation, self-compassion, and body dissatisfaction) and disordered eating. This mediational hypothesis was consistent with the latter part of the dual-pathway model which posits that body dissatisfaction promotes negative affect because one's appearance is considered to be one of the most important evaluative dimensions for women in society, and this ultimately leads to disordered eating (Stice, 1994, 2001).

### **4.3 Method**

#### **4.3.1 Participants.**

Participants were 167 females between 17 and 25 years of age recruited from first year Psychology undergraduates. In order to obtain a more homogenous sample, participants with a body mass index (BMI) of  $\geq 30$  (i.e., the criteria for morbid obesity,  $n = 14$ ; World Health Organization [WHO], 2006) were excluded. The majority of participants (85.6%) self-reported as Caucasian, with the next largest groups being Asian (1.8%) and African (1.2%).

#### **4.3.2 Procedures.**

The study was presented as an investigation of issues relating to different eating styles in women. The study involved three phases of data collection over 12 months; participants were tested at baseline (Time 1; T1), 6-month follow-up (T2), and 12-month follow-up (T3). The mean time between T1 and T2 was 5.4 months ( $SD = 0.8$  months), and between T2 and T3 was 6.2 months ( $SD = 0.5$  months). Participants earned course credits for research participation at T1, were offered additional course credits or \$15 at T2, and were offered \$20 gift vouchers at T3.

Participants were asked to complete an online self-report questionnaire battery. Data collection was conducted at Flinders University at T1 and T2, and online at T3. Questionnaires were completed online using *Survey Monkey*. Participants who gave consent to be contacted following T1 ( $n = 156, 93.4\%$ ) were invited to participate at T2, and again at T3. The present study was approved by the Flinders University Social and Behavioural Research Ethics Committee.

#### **4.3.3 Participant flow.**

**Figure 4.1** shows the flow of participants. Of the 167 eligible participants, 63 (37.7%) completed T2, and 66 (39.5%) completed T3; 34 (20.4%) completed all three phases of data collection. Of the original sample, 11 (6.6%) participants withdrew from the study following T1, and 1 (0.6%) participant withdrew following T2.

#### **4.3.4 Measures.**

***Disordered eating behaviours.*** Disordered eating behaviours were assessed using the Eating Disorder Examination–Questionnaire (EDE-Q; Fairburn & Beglin, 1994). The EDE-Q has demonstrated high convergent validity with the compensatory behaviour items of the Eating Disorder Examination (EDE; Berg et al., 2011; Fairburn & Cooper, 1993), and moderate convergent validity with the objective binge eating items (Berg et al., 2011).

**Disordered eating psychopathology.** Disordered eating psychopathology was assessed using the 22-item EDE-Q global score. The global score is commonly used as an indicator of outcome (i.e., recovery) in treatment studies (Bardone-Cone et al., 2010; Fairburn et al., 2009). The EDE-Q subscales have demonstrated high internal consistency ( $\alpha = .70-.93$ ) and test-retest reliability (1 to 14-day interval,  $\rho_1 = .66-.94$ ; Berg et al., 2012; Peterson et al., 2007), and the global score has demonstrated strong internal consistency ( $\alpha = .95$ ; Kelly et al., 2013) and high convergent validity with the EDE global score ( $r = .84$ ; Mond et al., 2004). In the current study internal consistency was .95 at each time point, and the intra-class correlation coefficient was .86.

**Perfectionism.** Perfectionism was measured using 16 items from the Concern over Mistakes and Personal Standards subscales of the Frost Multidimensional Perfectionism Scale (FMPS; Frost et al., 1990). These subscales were positively correlated ( $r = .597$ ) and were therefore combined to make a single ‘perfectionism’ variable. Items were scored on 5-point Likert scales, with higher scores indicating higher perfectionism. The FMPS subscales have demonstrated high consistency ( $\alpha = .77-.93$ ) and high correlations with other measures of perfectionism (Bardone-Cone et al., 2008; Frost et al., 1990). In the current study internal consistency ranged from .92 to .94 and the intra-class correlation coefficient was .93.

**Body dissatisfaction.** Body dissatisfaction was measured as current weight – ideal weight. Current weight was measured at T1 and T2, and based on self-report at T3. Self-reported weight has been found to be moderately correlated with confederate measured weight ( $r = -.51$ ; Attie & Brooks-Gunn, 1989). The intra-class correlation coefficient in the current study was .95.

**Body mass index (BMI).** BMI was measured as weight (kg) / height<sup>2</sup> (m<sup>2</sup>). Height was based on self-report data at T1 but a tape measure was provided to measure accurate height if unknown.

***Self-efficacy.*** Self-efficacy was measured using the 17-item General Self-Efficacy subscale (GSES) of the Self-Efficacy Scale (Sherer et al., 1982), focusing on three areas: willingness to initiate behaviour; willingness to make an effort to complete the behaviour; and persistence when faced with adversity (Sherer et al., 1982). Items were scored on 5-point Likert scales, with higher scores indicating higher self-efficacy. The GSES has shown good internal consistency ( $\alpha = .71-.89$ ) and validity (Bardone-Cone et al., 2008; Sherer et al., 1982). In the current study internal consistency ranged from .87 to .93, and the intra-class correlation coefficient was .92.

***Difficulties with emotion regulation.*** Difficulties with emotion regulation were assessed using the 30-item version of the Difficulties in Emotion Regulation Scale (DERS; Cooper, O'Shea, Atkinson, & Wade, 2014; Gratz & Roemer, 2004). The DERS assesses emotion regulation difficulties across six subscales, namely: non-acceptance of emotional responses; difficulties engaging in goal-directed behaviour; impulse control difficulties; lack of emotional awareness; limited access to emotion regulation strategies; and lack of emotion clarity. Items were scored on 5-point Likert scales, with higher scores representing greater difficulty with emotion regulation. The DERS has excellent internal consistency ( $\alpha = .93-.96$ ), high test-retest reliability (4 to 8-week interval;  $\rho_1 = .88$ ), and acceptable validity (Gratz & Roemer, 2004; Manuel & Wade, 2013). In the current study internal consistency ranged from .94 to .96, and the intra-class correlation coefficient was .94.

***Self-compassion.*** Self-compassion was measured using the 12-item version of the Self-Compassion Scale (Neff, 2003a; Raes et al., 2011). Items were scored on 5-point Likert scales, with higher scores indicating higher self-compassion. The scale has demonstrated high internal consistency ( $\alpha = .92$ ) and test-retest reliability (3-week interval,  $\rho_1 = .93$ ), and has shown to be positively correlated with similar measures (Neff, 2003a). In the current study internal consistency ranged from .83 to .92, and the intra-class correlation coefficient was .94.

***Sociocultural influences.*** Sociocultural influences was measured using 16 items from the Pressures and Internalization-General subscales of the Sociocultural Attitudes Towards Appearance Questionnaire-3 (SATAQ-3; Thompson et al., 2004). The Pressures subscale measures the extent to which one feels pressure from the media to strive for cultural ideals of beauty and engage in potentially dangerous behaviours in order to change their appearance, while the Internalization-General subscale assesses the extent to which one idealises, endorses, and compares oneself to media figures. Items were scored on 5-point Likert scales, with higher scores indicating higher sociocultural influences. Both subscales have demonstrated high internal consistency in community ( $\alpha = .92-.96$ ; Thompson et al., 2004) and disordered eating samples ( $\alpha = .93-.97$ ; Calogero et al., 2004), and good convergent validity with similar subscales ( $r = .38-.58$ ; Calogero et al., 2004). In the current study internal consistency ranged from .91 to .96, and the intra-class correlation coefficient was .86.

***Negative affect.*** Negative affect was measured using 14 items from the Depression and Anxiety subscales of the Depression Anxiety Stress Scale-21 (DASS-21; Henry & Crawford, 2005; Lovibond & Lovibond, 1995). Items were scored on 4-point Likert scales, with higher scores indicating higher psychopathology. Both subscales have demonstrated high internal consistency ( $\alpha = .90-.95$ ) and reliability ( $r = .82-.94$ ), and good convergent validity with validated measures of depression and anxiety (Antony et al., 1998; Henry & Crawford, 2005). In the current study internal consistency ranged from .92 to .93, and the intra-class correlation coefficient was .90.

#### **4.3.5 Statistical analyses.**

All analyses used the *Statistical Package for the Social Sciences (SPSS)* version 23 unless otherwise specified.

**Missing observations.** Mean item baseline differences between participants who provided data at one time point only (i.e., T1) and participants who provided data at multiple time points were investigated using independent samples *t*-tests with an alpha of .05. Effect sizes for between group differences were calculated using Cohen's *d*:  $M$  (provided data at one time point only) –  $M$  (provided data at multiple time points) / pooled *SD*, where .2 = small, .5 = medium, and .8 = large.

**Outcome variables over time.** Analyses were conducted using Linear Mixed Models (LMM), which allows use of all available data by employing Maximum Likelihood (ML) estimation which assumes that the data is missing at random. Two types of LMM were used: (1) unconditional means models, to determine whether there was significant variance; and (2) unconditional growth models, to determine whether significant variance was explained by time.

Growth Mixture Modelling (GMM or Latent Class Analysis [LCA]), was employed to investigate different trajectories over time. LCA employs ML estimation and identifies multiple latent (unobserved) subgroups (classes). Available theory, existing research findings, clinical knowledge, and statistical fit indices, all contribute when determining the number of classes. In determining model selection and interpretation (Ram & Grimm, 2009), two issues are considered: (1) the optimal number of latent classes; and (2) the type and extent of differences between and within those classes. The former is determined using relative fit *information criteria* including the Akaike Information Criteria (AIC), Bayesian Information Criteria (BIC), and sample-size Adjusted BIC (ABIC). Better-fitting models generally produce lower absolute values (Nylund et al., 2007). Models can also be evaluated by classification precision or *entropy*, where higher values (where 1.0 represents perfect classification, with high entropy values >.8) are indicative of greater precision, and identify the preferred model (Muthén, 2004). Finally, comparisons are made with *likelihood ratio*

*tests* including Vuong-Lo-Mendell-Rubin likelihood ratio test (VLMR-LRT) and Adjusted Lo-Mendell-Rubin likelihood ratio test (Adjusted LRT), where classes ('C') are compared with an analogous model with one fewer classes ('C-1'). Analyses were undertaken with *Mplus* (PC) version 7.31.

Mean differences between identified classes were investigated using logistic regressions, while mean differences within identified classes were investigated using paired samples *t*-tests. All analyses used an alpha of .05. Missing data were replaced using the expectation-maximization (EM) algorithm.

***Model testing.*** Following Boone et al. (2014), path analysis using *Mplus* was employed to test mediational pathways. As recommended by Hayes (2013), we did not test preconditions. Multiple imputation was used to estimate missing values using Bayesian analysis (Rubin, 1987). Ten imputed data sets were specified and the parameter estimates were averaged over the set of 10 analyses. This method does not allow for analysis of the indirect effect, so this was tested for each of the 10 imputed datasets. Model results were generated through corrected bootstrapping with 1000 resamples. Baseline variables related to the dual-pathway model (i.e., body dissatisfaction and sociocultural influences), as well as additional proposed variables related to temperament (i.e., perfectionism, self-efficacy, self-compassion, and difficulties with emotion regulation), were entered as the predictor. Negative affect at T2 was entered as the mediator. Disordered eating psychopathology at T3 was entered as the outcome. Covariates included the mediator and the outcome variable at T1.

## **4.4 Results**

### **4.4.1 Preliminary analyses.**

Skewness for each variable was within the accepted range with the exception of negative affect and disordered eating psychopathology, which were positively skewed, and

sociocultural influences, which was negatively skewed. The square root and reflected square root of negative affect and sociocultural influences were used for all waves in all analyses respectively (Tabachnick & Fidell, 2013). Disordered eating psychopathology was only mildly skewed and did not improve with transformation and was therefore left untransformed.

#### **4.4.2 Descriptives.**

Means and standard deviations are shown in **Table 4.1**. At T1 10.2% met criterion for underweight status and the majority of participants (85.6% at T1, 87.3% at T2, and 77.3% at T3) wanted to reduce their current body weight, with some (7.2% at T1, 9.5% at T2, and 9.1% at T3) wanting a higher body weight. The majority of participants (80.8% at T1, 76.2% at T2, and 66.7% at T3) reported engaging in some form of disordered eating behaviour. Almost a third of participants (33.5% at T1, 33.3% at T2, and 21.5% at T3) reported eating disorder psychopathology above the clinical cut-off (Mond, Hay, Rodgers, & Owen, 2006).

#### **4.4.3 Associations between baseline variables.**

Correlations are presented in **Table 4.2**, all in the expected direction. Our measure of body dissatisfaction was highly correlated with BMI ( $r = .793$ ), and therefore we included body dissatisfaction but not BMI in all multivariate analyses. While high correlations between some variables were present, ranging from  $r = -.579$  to  $r = -.726$ , a decision was made to keep these variables separate in order to compare our results to previous studies in the area. However, the most highly correlated scales (i.e., Pressures and Internalization-General subscales of the SATAQ-3,  $r = .768$ ) were combined to make a single ‘sociocultural influences’ variable.

#### **4.4.4 Missing observations.**

Mean item baseline differences between participants who provided data at one time point only ( $n = 72$ , 43.1%) and participants who provided data at multiple time points ( $n = 95$ , 56.9%) were compared (**Table 4.3**). The two groups did not differ significantly on any baseline variables with the exception of body dissatisfaction; where participants who provided data at one time point only reported significantly higher body dissatisfaction scores. Therefore, the data were not missing completely at random.

#### **4.4.5 Outcome variables over time.**

The unconditional means model showed that only disordered eating psychopathology showed significant variance over time,  $t(164.002) = 22.758$ ,  $p < .001$ . Hence, disordered eating behaviours were not investigated as an outcome variable. The unconditional growth model suggested that variance in disordered eating psychopathology was not explained by time,  $t(91.482) = .763$ ,  $p = .447$  i.e., there was no significant change over time.

In order to better understand the trajectory of disordered eating psychopathology over time, we used LCA to examine five models (i.e., specifying 1 through to 5 latent classes) across the three waves of data collection, presented in **Table 4.4**. Both AIC and ABIC criteria incrementally decreased with increasing numbers of latent classes, while BIC criteria incrementally decreased (i.e., from *C1* through *C2*) and increased (i.e., from *C3* through *C5*). Second, increasing the number of classes resulted in deteriorating accuracy of group membership classification (*entropy*), where classification confidence was lowest for the 5-class model. Third, *likelihood ratio tests* (i.e., VLMR-LRT, and Adjusted LRT) were used to compare each model with an analogous model with one fewer class. Results indicated that the *C-1* model could not be rejected in favour of the *C* model in all instances (i.e., models for *C3* through *C5*) except for the *C2* model. Current recommendations (Ram & Grimm, 2009) encourage model selection driven by all available fit indices as well as domain specific theory

and existing research findings. Across these indicators it was decided that the 2-class model was most appropriate for these data, as illustrated in **Figure 4.2**.

Class 1 ( $n = 112$ , 67.1%) was characterized by low and significantly ascending disordered eating psychopathology (Cohen's  $d = 0.74$ ), termed *Low increasing psychopathology*, and Class 2 ( $n = 55$ , 32.9%) was characterized by high and decreasing disordered eating psychopathology that approached significance (Cohen's  $d = 1.09$ ), termed *High decreasing psychopathology*. There was a significant difference in disordered eating psychopathology between the groups at each wave with a respective Cohens  $d$  of 3.55 (T1), 2.72 (T2) and 1.35 (T3).

All baseline variables were significant predictors of group membership in the expected directions ( $p < .001$ ), with the exception of perfectionism ( $p = .587$ ). Significant predictors were entered into a multivariate model where sociocultural influences (OR = 243.276, 95% CI = 16.135–3667.910), body dissatisfaction (OR = 1.262, 95% CI = 1.118–1.425), and difficulties with emotion regulation (OR = 3.564, 95% CI = 1.079–11.772) were shown to uniquely discriminate the two classes.

#### **4.4.6 Model testing.**

Negative affect was explored as a mediator of the relationships between six baseline variables (i.e., body dissatisfaction, sociocultural influences, perfectionism, self-efficacy, difficulties with emotion regulation, and self-compassion) and disordered eating psychopathology (**Table 4.5**). Only the relationship between baseline self-compassion and disordered eating psychopathology was mediated by negative affect, shown in **Figure 4.3**. Individual path coefficients show that lower baseline levels of self-compassion were associated with higher negative affect at T2, which was associated with greater disordered eating psychopathology at T3. The indirect path was significant but the direct path (direct

effect) from self-compassion to disordered eating psychopathology was not. As recommended by Hayes (2013), we do not interpret the present findings in terms of *complete* or *partial* mediation.

#### **4.5 Discussion**

The aim of the present research was to longitudinally investigate the inclusion of temperament variables in the dual-pathway model of bulimic pathology across the full spectrum of eating disturbances and using a global measure of eating psychopathology of transdiagnostic relevance. The present findings add to the growing body of literature suggesting that factors related to temperament (i.e., difficulties with emotion regulation and self-compassion) are implicated in the aetiology of eating pathology.

Two latent classes were identified for disordered eating psychopathology, including one group categorised by low and ascending psychopathology and one group characterised by high and decreasing psychopathology over time. This pattern of results is interesting in light of previous work on trajectories of disordered eating outcomes (Fairweather-Schmidt & Wade, 2016; Rodgers, McLean, Marques, Dunstan, & Paxton, 2015; Rohde et al., 2015). For example, Fairweather-Schmidt and Wade (2016) identified trajectories of three latent classes of EDE global scores over time among adolescent females, including low static, escalating (increasing), and attenuating (decreasing). Further, Rodgers et al. (2015) identified trajectories of four different latent classes of body dissatisfaction (i.e., as measured by the weight and shape subscales of the EDE-Q) over time among a sample of early adolescent females, including low, moderate decreasing, moderate increasing and high. These different patterns may be due to differences in the age of participants (i.e., adolescents versus adults), or could represent regression to the mean in the present study. Further research should investigate this further.

Further analysis revealed that baseline sociocultural influences, body dissatisfaction, and difficulties with emotion regulation, uniquely distinguished group membership. A body of literature supports the relationship between difficulties with emotion regulation and eating pathology (Gianini et al., 2013; Svaldi et al., 2012). It is suggested that difficulties coping with adverse emotional states can result in disordered eating, which may become a habitual way of coping with, and managing, difficult emotions. Research has shown that emotion regulation difficulties explains unique variance in eating pathology beyond the contribution of negative affect (Gianini et al., 2013; Whiteside et al., 2007). Whereas negative affect is already included in the dual-pathway model and is already targeted in prevention programs for eating disorders (e.g., dissonance-based prevention programs; see Stice, Rohde, et al., 2009 for overview), the contribution of difficulties with emotion regulation is yet to be investigated.

Investigation of mediational pathways showed that lower levels of self-compassion resulted in higher negative affect, which in turn resulted in a greater increase of disordered eating psychopathology. A systematic review by Braun et al. (2016) found support for the role of self-compassion as a protective factor against poor body image and eating pathology among clinical and non-clinical samples. As suggested by Tylka and Kroon Van Diest (2015), self-compassion may protect against eating pathology through various pathways, including reducing eating disorder-related outcomes directly, preventing the development of risk factors and/or eating disorder-related outcomes, and interacting with risk factors to interrupt their deleterious effects. While self-compassion has been considered in the treatment of eating disorders (e.g., compassion-focused therapy; see Goss & Allan, 2014 for overview), it has not yet been considered within the realm of prevention.

These findings give support for variables already considered in the dual-pathway model (i.e., sociocultural influences, body dissatisfaction, and negative affect), as well as

additional variables related to temperament (i.e., difficulties with emotion regulation and self-compassion). In support of this, a systematic review of the existing models for disordered eating by Pennesi and Wade (2016), identified preoccupation with weight and shape (including body dissatisfaction), emotional regulation difficulties, negative affect, thin-ideal internalisation, and external pressure, as common risk factors across models which have informed interventions such as prevention and treatment, suggesting that these factors are fundamental to the development of future effective interventions for disordered eating.

Factors related to temperament should also be considered in the development of future prevention approaches for disordered eating. First, as these measures were able to longitudinally predict the development of disordered eating psychopathology, they could be used to identify women at increased risk for targeted prevention programs. Moreover, the findings suggest that future prevention programs may benefit from incorporating attempts to target malleable temperament factors such as emotion regulation difficulties and self-compassion. For example, programs may teach women more adaptive ways of coping with strong negative emotions, or attempt to bolster body satisfaction through the enhancement of self-compassion.

Despite the strengths of the study, including the longitudinal design, there were several limitations. The most prominent of these was that the data were not missing completely at random; those participants who provided data at one time point only reported significantly higher baseline body dissatisfaction scores. This limits the conclusions that can be drawn from these data. It was for these reasons that a decision was made to reanalyse the data using only cross-sectional (baseline) data with 167 cases, which was presented in the preceding chapter.

The results should also further be interpreted in the context of a number of other limitations. First, disordered eating behaviours were measured using self-report items from

the EDE-Q, a standardised measure of eating disturbances with acceptable reliability and validity; however the use of structured clinical interviews may improve predictive validity. Second, the study relied solely on self-report data. While the use of confidential self-report procedures may encourage honest responding (Lavender & Anderson, 2009), use of multiple-reporter data (e.g., peer or family member reports) and multiple methods of measurement (e.g., interviews, observation, or frequency records) would increase the strength of findings. Third, the present model focuses on the aetiology of disordered eating among females aged between 17 and 25 years, and is unable to inform the development of disordered eating in other groups (e.g., younger/older women and men). Fourth, body dissatisfaction was measured as current weight – ideal weight which may more accurately represent weight dissatisfaction; future studies may consider the use a standardised measure of body dissatisfaction. Fifth, we were unable to investigate disordered eating behaviours as there was no significant variance over time, future research may consider testing the model in a sample at high risk of developing an eating disorder (i.e., where the presence of disordered eating is likely to be higher), such as body-dissatisfied women. Sixth, this is a nonexperimental study and experimental work and prevention trials will be useful to demonstrate hypothesised decreases in the rates of disordered eating psychopathology over time. Lastly, there was a notably high attrition rate with only 34 participants (20.4%) completing all three waves of data collection. Despite addressing this limitation by imputing missing data and using analyses that make maximum use available data, the small number of cases over the three waves ultimately limits the utility of the data to provide reliable effects that remain stable over time. Investigation of a larger sample over time would make the observed effects more convincing.

These findings have several implications for future research. Whereas the present research focused on predicting the full spectrum of eating disturbances, future research might

investigate whether the predictors of diagnostic levels of eating pathology differ. Second, while this study adds to the existing literature examining the dual-pathway model, few independent research teams have replicated the model in its entirety longitudinally, and given the current debate about reproducibility in science (Open Science Collaboration, 2015), it is important that future researchers address this gap in order to inform the development of future effective interventions. Third, researchers should consider testing the model with the inclusion of factors related to temperament (i.e., difficulties with emotion regulation and self-compassion) as well as additional risk factors that may contribute to the development of disordered eating that are not considered within the current model.

**Table 4.1**

*Summary of Descriptive Statistics at Baseline (T1), at 6-Month Follow-Up (T2), and at 12-Month Follow-Up (T3)*

	T1 (N = 167)	T2 (N = 63)	T3 (N = 66)
Variable	Mean (SD)	Mean (SD)	Mean (SD)
<i>Demographic Variables</i>			
Age	19.20 (1.63)	19.50 (1.43)	20.17 (1.43)
BMI	22.04 (2.99)	22.87 (3.29)	22.17 (3.55)
Body dissatisfaction	4.40 (5.00)	5.07 (5.21)	4.74 (5.69)
<i>Independent Variables</i>			
Sociocultural influences	3.37 (.85)	3.29 (.92)	3.24 (.71)
Perfectionism	2.96 (.78)	2.96 (.73)	3.03 (.84)
Self-efficacy	3.34 (.53)	3.38 (.64)	3.34 (.66)
Negative affect	.66 (.57)	.63 (.59)	.67 (.57)
DERS	2.58 (.66)	2.42 (.68)	2.60 (.76)
Self-compassion	2.80 (.57)	3.00 (.77)	2.78 (.70)
<i>Dependent Variables<sup>1</sup></i>			
Objective binge eating	3.51 (5.27)	2.51 (3.79)	1.60 (3.60)
Self-induced vomiting	.22 (2.20)	.14 (.62)	.11 (.75)
Laxative use	.08 (.80)	.89 (4.95)	.00 (.00)
Fasting	1.65 (4.73)	2.25 (4.82)	2.73 (6.97)
Driven exercise	4.58 (6.52)	4.25 (5.84)	3.22 (5.56)
	%, Mean (SD)	%, Mean (SD)	%, Mean (SD)
EDE-Q global <sup>2</sup>	33.5%, 2.19 (1.30)	33.3%, 2.19 (1.31)	21.5%, 2.09 (1.26)
EDE-Q behaviour <sup>3</sup>	80.8%, .03 (.83)	76.2%, .00 (.56)	66.7%, .00 (.62)

*Note.* T1 = time 1; T2 = time 2; T3 = time 3; SD = standard deviation; BMI = body mass index; DERS = difficulties with emotion regulation; EDE-Q = Eating Disorder Examination–Questionnaire.

<sup>1</sup> All eating behaviours are reported as number of episodes over the preceding 28 days, with the exception of fasting which is reported as number of days.

<sup>2</sup> % = proportion of the sample reporting disordered eating psychopathology  $\geq 2.77$  (Norm for young adult women +1SD; Mond et al., 2006).

<sup>3</sup> % = proportion of the sample reporting any disordered eating behaviour.

**Table 4.2**

*Correlations between Baseline (T1) Variables, and between Baseline Variables and Disordered Eating Psychopathology at Baseline (T1) and 12-Month Follow-Up (T3)*

Baseline Variable	1.	2.	3.	4.	5.	6.	7.	8.
1. Sociocultural influences	–							
2. Body dissatisfaction	<b>.307**</b>	–						
3. Perfectionism	<b>.209**</b>	.015	–					
4. Self-efficacy	<b>-.266**</b>	-.137	-.090	–				
5. Negative affect	<b>.316**</b>	.110	.071	<b>-.411**</b>	–			
6. DERS	<b>.357**</b>	<b>.249**</b>	.130	<b>-.579**</b>	<b>.663**</b>	–		
7. Self-compassion	<b>-.481**</b>	<b>-.179*</b>	<b>-.185*</b>	<b>.496**</b>	<b>-.610**</b>	<b>-.726**</b>	–	
8. T1 EDE-Q global	<b>.595**</b>	<b>.545**</b>	.063	<b>-.351**</b>	<b>.375**</b>	<b>.507**</b>	<b>-.470**</b>	–
9. T3 EDE-Q global	<b>.303*</b>	<b>.530**</b>	.131	-.175	.201	<b>.392**</b>	<b>-.316*</b>	<b>.654**</b>

*Note.* T1 = time 1; T3 = time 3; DERS = difficulties with emotion regulation; EDE-Q = Eating Disorder Examination–Questionnaire. Significant values are in **bold**.

\*\* Correlation is significant at the .001 level (2-tailed).

\* Correlation is significant at the .05 level (2-tailed).

**Table 4.3**

*Summary of Baseline (T1) Comparisons between Those Who Provided Data at One Time Point (n=72) and Those Who Provided Data at Multiple Time Points (n=95)*

Baseline Variable	<i>df</i>	<i>t</i>	Sig.	ES (95% CI)
Age (at testing)	165	.790	.431	.12 (-.18 to .43)
BMI	165	.417	.677	.07 (-.24 to .37)
Sociocultural influences	164	1.078	.283	.17 (-.14 to .48)
Body dissatisfaction	165	2.444	<b>.016</b>	.38 (.07 to .69)
Perfectionism	164	-.646	.519	-.10 (-.41 to .21)
Self-efficacy	164	.681	.497	.11 (-.20 to .41)
Negative affect	164	-.762	.447	-.12 (-.43 to .19)
DERS	165	.144	.886	.02 (-.28 to .33)
Self-compassion	164	.560	.576	.09 (-.22 to .40)
EDE-Q global	165	1.655	.100	.26 (-.05 to .57)

*Note.* T1 = time 1; *df* = degrees of freedom; Sig. = *p*-value; ES = effect size (Cohen's *d*); CI = confidence interval; BMI = body mass index; DERS = difficulties with emotion regulation; EDE-Q = Eating Disorder Examination–Questionnaire. Significant values are in **bold**. Cohen's *d* .2 = small, .50 = medium, .80 = large.

**Table 4.4***Information Criteria for EDE-Q Global Growth Mixture Modelling with 1-5 Latent Classes*

Classes	Fit statistics					
	# of free Parameters	AIC	BIC	ABIC	Entropy	LRT <sup>§</sup> <i>p</i>
N <sub>c=1</sub>	8	877.087	902.031	876.701	-	-
N <sub>c=2</sub>	11	853.350	887.648	852.821	.831	<b>.001</b>
N <sub>c=3</sub>	14	849.596	893.248	848.922	.813	.192
N <sub>c=4</sub>	17	845.537	898.543	844.719	.769	.564
N <sub>c=5</sub>	20	843.265	905.625	842.302	.654	.437

*Note.* EDE-Q = Eating Disorder Examination–Questionnaire; AIC = Akaike information criteria; BIC = Bayesian information criteria; ABIC = Adjusted BIC; §LRT = likelihood ratio tests (e.g., Vuong-Lo-Mendell-Rubin likelihood ratio tests; Lo-Mendell-Rubin adjusted LRT test) quantify specific comparisons between the model of interest and a model with one fewer class, C-1; N<sub>c</sub> = Number of latent classes. Significant values are in **bold**.

**Table 4.5**

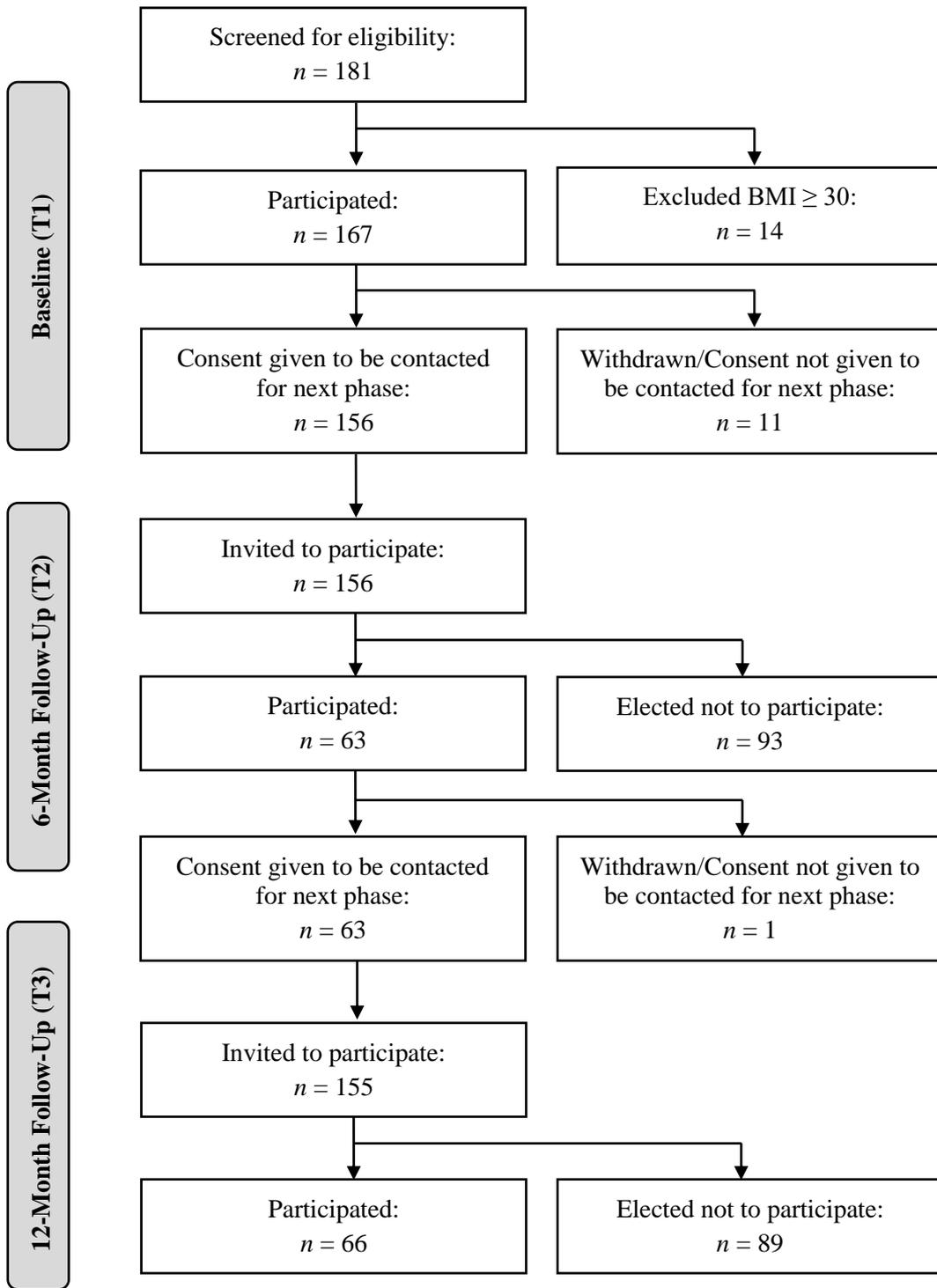
*Testing a Path Analysis Model to Examine whether Negative Affect at T2 Mediates the Relationship between Baseline (T1) Variables and EDE-Q Global at 12-Month Follow-Up (T3) Showing Standardised Parameter Estimates Adjusting for Baseline Negative Affect and EDE-Q Global*

Model: mediational pathway	Independent → negative affect	Independent → EDE-Q global	Negative affect → EDE-Q global
	Standardized Estimate (SE) <i>p</i>	Standardized Estimate (SE) <i>p</i>	Standardized Estimate (SE) <i>p</i>
Body dissatisfaction → negative affect → EDE-Q global	-.001 (.008) .933	-.009 (.027) .749	.841 (.490) .086
Sociocultural influences → negative affect → EDE-Q global	-.032 (.153) .833	.080 (.501) .874	<b>.891 (.359) .013</b>
Perfectionism → negative affect → EDE-Q global	.003 (.043) .949	.139 (.133) .295	<b>.874 (.282) .002</b>
Self-efficacy → negative affect → EDE-Q global	-.001 (.085) .989	.182 (.205) .375	<b>.877 (.377) .020</b>
DERS → negative affect → EDE-Q global	.086 (.063) .167	-.206 (.263) .432	<b>.900 (.376) .008</b>
Self-compassion → negative affect → EDE-Q global <sup>1, 2</sup>	<b>-.182 (.067) .006</b>	.156 (.221) .460	<b>.972 (.373) .009</b>

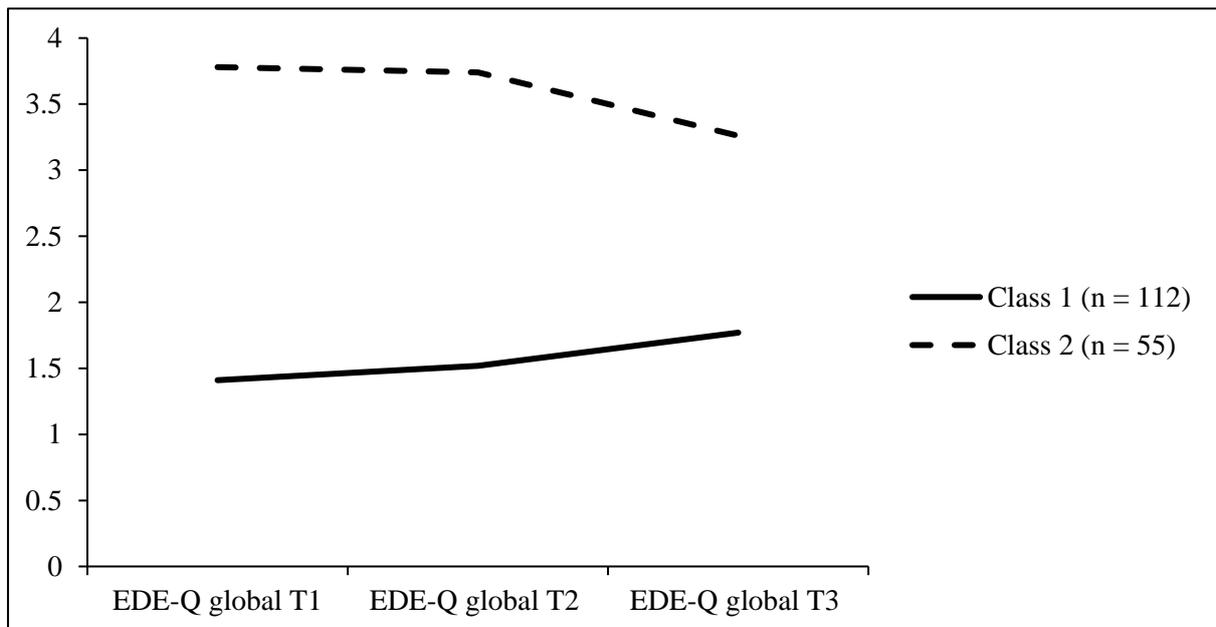
*Note.* T1 = time 1; T3 = time 3; EDE-Q = Eating Disorder Examination–Questionnaire; SE = standard error; DERS = difficulties with emotion regulation. Significant pathways are in **bold**.

<sup>1</sup> analyses repeated using 56.9% of the data (those with 2 or more observations) gave similar results.

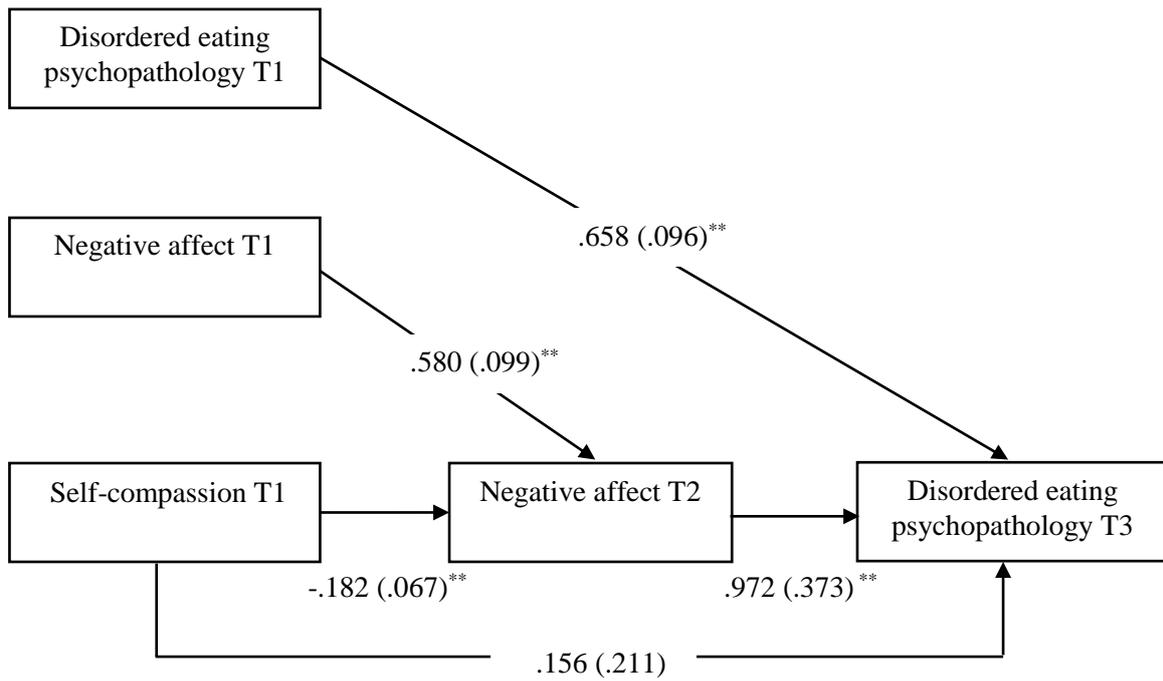
<sup>2</sup> across the set of 10 imputed datasets the estimates for the indirect pathway ranged from -.373 to -.111, with *p* values <0.05 for 9 of 10 datasets (*p* values ranged from <.001 to .05).



**Figure 4.1** PRISMA flow diagram for participation. Figure adapted from Liberati et al. (2009) and Moher, Liberati, Tetzlaff, and Altman (2009). T1 = time 1; T2 = time 2; T3 = time 3; BMI = body mass index.



**Figure 4.2** Trajectories of two latent classes using mean EDE-Q global identified by Growth Mixture Modelling (GMM) across baseline (T1), 6-month follow-up (T2), and 12-month follow-up (T3). T1 = time 1; T2 = time 2; T3 = time 3; EDE-Q = Eating Disorder Examination–Questionnaire; Class 1 = *Low increasing psychopathology*; Class 2 = *High decreasing psychopathology*.



**Figure 4.3** The path model for the mediational model. Showing the mediational pathway between baseline (T1) self-compassion, negative affect at T2, and disordered eating psychopathology at T3 (12-month follow-up), and the direct pathway between baseline self-compassion and disordered eating psychopathology at T3. Path coefficients are standardized estimates (with standard errors). Baseline negative affect and disordered eating psychopathology are included as covariates. T1 = time 1; T2 = time 2; T3 = time 3. \* $p < .05$ ; \*\* $p < .01$ .

## **Chapter 5**

### **Imagery Rescripting and Cognitive Dissonance: A Randomised Controlled Trial of Two Brief Interventions to Reduce Body Dissatisfaction for Women**

## 5.1 Abstract

The present study compared two approaches to reducing body dissatisfaction, imagery rescripting and cognitive dissonance, to a control condition within an experimental session. We also examined the degree to which these approaches modified factors in addition to body dissatisfaction, namely negative affect and self-compassion. Female university students ( $N = 201$ , 17 to 28 years of age) were randomized to one of the three conditions (i.e., imagery rescripting, cognitive dissonance, control), and then completed a baseline assessment, body dissatisfaction induction, and brief intervention. Findings provide support for both imagery rescripting and cognitive dissonance interventions, with participants in these conditions reporting higher self-compassion compared to those in the control condition. Further, both approaches showed promise in reducing negative affect. There was no significant impact of either approach on body dissatisfaction. These findings suggest that both imagery rescripting and cognitive dissonance may be useful in increasing self-compassion, at least in the short term. Further these findings suggest that imagery-based techniques (e.g., imagery rescripting) may have utility in future prevention approaches, and are worthy of further research.

## 5.2 Introduction

The main aim of the current study was to examine two different approaches to reducing body dissatisfaction (i.e., a robust risk factor for the development of disordered eating pathology; Dakanalis et al., 2017; Jacobi & Fittig, 2010; Stice, 2016) in order to identify which approach appears most promising for further development, namely imagery rescripting and cognitive dissonance. Cognitive dissonance-based interventions (DBI) target thin-ideal internalisation through use of cognitive-attitudinal activities such as asking participants to critique the thin ideal of beauty (Stice & Presnell, 2007). While cognitive dissonance has been established as an effective intervention for reducing body dissatisfaction (Becker & Stice, 2017; Shaw & Stice, 2016), little is known about the efficacy of imagery rescripting as an intervention approach for reducing body dissatisfaction.

Imagery rescripting is a technique developed to modify the negative meanings linked to early traumatic or distressing experiences and the implications that they continue to hold in the present (Tatham, 2011). While imagery rescripting has been traditionally used in the treatment of social phobia and PTSD (Arntz, Tiesema, & Kindt, 2007; Wild & Clark, 2011), and there is preliminary support for its use in the treatment of bulimia nervosa (Cooper et al., 2007; Ohanian, 2002), imagery techniques are yet to been investigated in the prevention of eating disorders. Somerville and Cooper (2007) argue that approaches which rely on cognitive restructuring and rational argument may be less effective at tackling negative core beliefs of relevance to eating disorders (such as self-criticism), which have stronger emotional than rational bases, compared to emotion-focused techniques such as compassion-focused imagery (Gilbert & Irons, 2005) which is used in imagery rescripting. This is consistent with the findings in the cross-sectional study in **Chapter 3**, which showed difficulties with emotion regulation to be an important addition to the model that has informed the use of cognitive dissonance with body dissatisfaction.

In the current study, conducted with female university students, we examined the efficacy of imagery rescripting and cognitive dissonance in reducing body dissatisfaction compared to controls, within an experimental session following a body dissatisfaction induction. We also examined the degree to which each approach modified other theoretically-supported factors in addition to body dissatisfaction, namely negative affect and self-compassion, in order to identify variables that appear to be most effective for change. The knowledge gained from this research will help to inform the content of future effective prevention approaches for eating disorders and disordered eating.

### **5.3 Method**

#### **5.3.1 Participants.**

Females were recruited from a first year university student population and wider university sample. Given that disordered eating occurs predominantly among young women (Hudson et al., 2007; Liechty & Lee, 2013) and that early adulthood increases risk for the onset of new eating pathology (Allen et al., 2013; Favaro et al., 2009; Kessler et al., 2013; Stice, Marti, et al., 2013), this sample was considered to be at greater risk for disordered eating. After the full baseline assessment, we excluded those women considered to be likely to have an eating disorder that required immediate attention ( $n = 8$ ). This involved a score of  $\geq 4$  on the over-evaluation of weight or shape items on the self-report Eating Disorder Examination–Questionnaire (EDE-Q; Fairburn & Beglin, 1994) and, either (a) a body mass index (BMI) of  $< 17.5$  (i.e., the criteria for low weight; World Health Organization [WHO], 2006), or (b) using self-induced vomiting or laxatives as a means of controlling weight or shape on more than half the days over the past 28 days. These women were provided with information about appropriate treatment options. Given the prevalence of self-reported disordered eating, including binge eating, purging, fasting, and driven exercise, in community samples of young women (Hay et al., 2008; LePage, Crowther, Harrington, & Engler, 2008;

Liechty & Lee, 2013; Wade et al., 2012), the presence of these behaviours were not considered as means for exclusion. The analytic sample for this study included 201 females ranging in age from 17 and 28 years ( $M_{age} = 20.18$ ,  $SD = 1.98$ ) with a mean BMI in the normal range (i.e., BMI 18.5–24.99; WHO, 2006;  $M = 23.75$ ,  $SD = 5.06$ ). The majority of participants (72.1%) self-reported as Caucasian, with the next largest groups being Asian (18.9%), African (2.5%), Indian (2.0%), and Middle Eastern (2.0%).

### 5.3.2 Procedures.

The study was presented as an investigation of different strategies to improve body image and eating in women, and involved a single experimental session; the procedure is depicted in **Figure 5.1**. Before commencing, height and weight (current and ideal) were measured in order to derive BMI (measured as  $\text{weight} / \text{height}^2$ ) and a rating of body dissatisfaction (measured as  $\text{current weight} - \text{ideal weight}$ ). Participants were randomized, using the randomization function in *Qualtrics*, to one of the three conditions: cognitive dissonance, imagery rescripting, or control. Participants completed self-report state measures (Time 1; T1) in a research laboratory with the principal researcher available behind a screen to answer any questions. For all measures, if an item was left unanswered, the participant was asked if they wanted to “answer the question”, or “continue without answering”. Hence there was no missing data for any of the dependent variables. For the next five minutes all participants were asked to close their eyes and imagine (or visualise), and then describe in the first person (as if it were happening to them “right now”), the “first or earliest memory of a personal unpleasant body experience from the past where you may have felt ashamed or embarrassed of your body or how your body looked”. Examples were provided, including: trying on clothes in a change-room, getting ready with friends to go to a party, walking past a group of people who were looking at you, being teased about your body, looking at yourself

in the mirror. This was used to equalise groups given that the first stage of imagery rescripting typically requires recall and visualisation of past traumatic memories (Wild & Clark, 2011), and was also intended to serve as a body dissatisfaction induction procedure to enhance effect sizes for any subsequent changes in the state measures. After the recall, participants completed a second assessment of the state measures (T2), before receiving the baseline intervention (i.e., imagery rescripting, cognitive dissonance, control), then completed the state measures for the final time (T3). Data were collected between October 2016 and April 2017. A choice of course credits, cash or gift voucher were offered for research participation. The Flinders University Social and Behavioural Research Ethics Committee approved this study.

### **5.3.3 Techniques.**

Video instructions were used for all conditions in order to enhance understanding and compliance (Kingston, Gray, & Williams, 2010), and written instructions were subsequently provided on the computer screen as indicated below.

*Imagery rescripting.* This procedure was designed for this study based on methods used previously with traumatic memories in social phobia (Wild & Clark, 2011); but with an emphasis on the latter active stages of treatment. Participants were presented with the following instructions, presented in two parts and make up a single intervention exercise:

(Part 1) Please think about your first or earliest memory of a personal unpleasant body experience from the past where you may have felt ashamed or embarrassed of your body or how your body looked. This time, when you close your eyes and imagine (or visualize) this event, imagine it from an observers' perspective. Imagine that your adult self is in the room observing what's happening right now, watching the events unfold. Imagine that you are observing your younger self reliving the event. Please only continue once you have this event in mind. For the next 2.5 minutes, please describe what you see from an observers' perspective as if it were happening to your younger self

right now. Please write in the third person (e.g., “I see Sarah in the change room, she is trying on a pair of blue jeans...”). Try to be descriptive and include as many details as you can, for example, where is Sarah, what is Sarah doing, who is Sarah with, how might Sarah be feeling (emotions), and what might Sarah be telling herself (thoughts).

(Part 2) Please again think about your first or earliest memory of a personal unpleasant body experience from the past where you may have felt ashamed or embarrassed of your body or how your body looked. This time, when you close your eyes and imagine (or visualize) this event, imagine it as if it is happening to you right now, but this time your wiser and more compassionate adult self is with you in the room and she can intervene if you want her to. She can offer you compassion or provide new updated information based on what you know now as an adult. She can talk to you (or others), or do anything else that feels helpful or right in the situation. Please only continue once you have this event in mind. For the next 2.5 minutes, please describe what you see as if it is happening to you right now but this time your adult self is with you and she can intervene if you want her to. Please write in the first person (e.g., “I’m in the change room, I’m trying on a pair of blue jeans...”) unless you are referring to the adult you (e.g., “Adult Sarah said...”, or “older Sarah said...”). Try to be descriptive and include as many details as you can, including where you are, what you are doing, who you are with, how you are feeling (emotions), and what you are telling yourself (thoughts). Please also describe if your adult self intervenes or does anything in the situation.

***Cognitive dissonance.*** This procedure was adapted from techniques described by Stice et al. (2006), which have subsequently been used by Wade, George, and Atkinson (2009) as a brief intervention for body dissatisfaction. Participants were presented with the following instructions, accompanied by images of the thin-ideal stereotype for women:

Please read the definition of the thin-ideal stereotype for women below and view the accompanying images which show what this ideal looks like. Please only continue after reading the definition and viewing the images. *“The thin-ideal stereotype for women, as portrayed by the media, is that of an ultra-slender, feminine physique with a small waist, long legs and little body fat.”* For the next 5 minutes, please brainstorm about the following: 1) the costs and consequences

of pursuing the thin-ideal (thin-ideal stereotype for women as portrayed in the media), and 2) the positive attributes about yourself (these can be physical, emotional, or behavioural). Try to spend an even amount of time on each topic.

**Control.** The control procedure was adapted from May and colleagues (2010).

Participants did not receive any training but were asked to let their mind wander, and were presented with the following instructions:

For the next 5 minutes, just sit and let your mind wander wherever it would like to, let your attention drift. There is no need to control your thoughts in any way, just let them wander. You may find that your mind wanders to thoughts about many different things or thoughts of nothing at all. You may find that your mind wanders to thoughts about the personal unpleasant body experience you described earlier. Wherever your mind wanders it's OK. Just think about whatever you like. Thoughts are not right or wrong. Just let your attention drift.

#### **5.3.4 Outcome measures.**

**State body dissatisfaction.** Three visual analogue scales (VAS) were used to measure state body dissatisfaction. Following methods used previously by Heinberg and Thompson (1995) which were subsequently adapted by Wade et al. (2009), participants were asked to indicate their response to questions, including: "How satisfied do you feel about your weight right now?", "How satisfied do you feel about your appearance right now?", and "How distressed are you by your feelings about your body right now?". Responses were indicated by dragging a slider along a 100-pixel horizontal line representing a scale from *not at all* to *very much*, with responses rated 0 (*extreme satisfaction*) to 100 (*extreme dissatisfaction*). VAS have shown to be valid and reliable measures of fluctuations in body dissatisfaction, can be completed quickly, and provide improved sensitivity to small changes (Heinberg & Thompson, 1995; Tiggemann & McGill, 2004; Wade et al., 2009).

**State negative affect.** The 10-item Negative Affect scale of the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) was used to measure state negative affect, which reflects a general dimension of subjective distress. The PANAS contains a list of adjectives describing various mood states, and participants were asked to indicate the extent to which they feel this way “right now (that is, at the present moment)”. Items were scored on 4-point scales, ranging from *very slightly or not at all* to *extremely*. The momentary version of the PANAS has been shown to be a reliable measure of state negative affect in previous daily diary studies ( $\alpha = .88$ ; Merz & Roesch, 2011) and has demonstrated moderate convergent validity with general measures of distress and psychopathology (Watson et al., 1988). In the current study internal consistency was .90.

**State self-compassion.** Different aspects of state self-compassion were measured using three VAS designed for this study, using items adapted from 12-item Self-Compassion Scale (Raes et al., 2011), the Fears of Compassion Scales (Gilbert, McEwan, Matos, & Rivis, 2011), and the Forms of Self-Criticising/Attacking and Self-Reassuring Scale (Gilbert, Clarke, Hempel, Miles, & Irons, 2004). All of these scales have acceptable reliability and validity ratings (Gilbert et al., 2004; Gilbert et al., 2011; Raes et al., 2011). Participants were asked to indicate the extent to which they believe particular statements “right now (that is, at the present moment)”, including: “I am very disapproving about my own flaws and inadequacies“, “I don’t deserve to be kind and forgiving to myself“, and “There is a part of me that feels I am not good enough”. Items were negatively-worded and therefore responses were reverse-scored, with higher scores indicating higher self-compassion. Responses were indicated by dragging a slider along a 100-pixel horizontal line representing a scale from *not at all* to *very much*, with responses rated 0 (*absent*) to 100 (*extreme*).

### **5.3.5 Statistical analyses.**

Of the 209 participants who entered the study, 8 (3.8%) participants were excluded and referred for appropriate treatment, leaving 201 participants in the analytic sample. Following Cumming and Calin-Jageman's (2016) open science approach to interpreting significance, all between- and within-group comparisons were examined using an effect size (ES, Cohen's  $d$ , where .2 = small, .5 = medium, .8 = large; Cohen, 1988) with 95% confidence interval (CI). If the 95% CI does not include the null hypothesised value (i.e., zero), the null hypothesis is rejected and the difference between or within groups is considered to be statistically significant (Hayes, 2009). Baseline (T1) values across the three conditions were compared using one-way analyses of variance (ANOVAs).

**Manipulation check.** A manipulation check was performed using a 3 (condition: imagery rescripting, cognitive dissonance, control) by 2 (time: baseline, post-induction) repeated measures ANOVA on state body dissatisfaction, negative affect, and self-compassion, respectively. One-way ANOVAs were used to compare the three conditions at T1 and T2.

**Change in outcome.** Repeated measures ANOVAs were used to compare the effectiveness of the three conditions with respect to the outcome variables. The dependent variable was change at T3, and T1 was included as a covariate.

## 5.4 Results

### 5.4.1 Descriptives.

Means and standard deviations are shown in **Table 5.1**. Skewness for each variable was within the accepted range of  $Z = +/-2.575$  ( $\alpha = .01$ ) with the exception of state negative affect, which was positively skewed. The (reflected) inverse of state negative affect was used in all analyses (Tabachnick & Fidell, 2013). The majority of participants (63.7%) fell within the normal BMI range (i.e., BMI 18.5–24.99; WHO, 2006), 7.0% were classified as

underweight (i.e., BMI <18.5), and 29.4% were overweight or obese (i.e., BMI  $\geq$ 25). The majority of participants (89.6%) wanted to reduce their current body weight, and 8.5% wanted a higher body weight. Participants did not differ across the three conditions on any of the variables. Correlations are presented in **Table 5.2**, all in the expected direction. While our measure of body dissatisfaction was highly correlated with BMI ( $r = .847$ ), these variables were only used to describe the sample and not included in the analyses.

#### **5.4.2 Manipulation check.**

Across all groups, the induction successfully resulted in increasing negative affect, which was associated with a significant main effect of time, (ES = -.25, 95% CI = -.44, -.05). There was no accompanying significant increase in body dissatisfaction (ES = -.18, 95% CI = -.37, .02) or decrease in self-compassion (ES = .06, 95% CI = -.13, .26). There were no significant main effects of group or interactions between time and group for any of the state variables. After the induction procedure, state body dissatisfaction, negative affect and self-compassion were not significantly different across the three conditions (body dissatisfaction, ES = -.06, 95% CI = -.39, .28; negative affect, ES = -.06, 95% CI = -.28, .40; self-compassion, ES = -.05, 95% CI = -.29, .39).

#### **5.4.3 Change in outcome.**

**Table 5.3** shows the estimated means and standard errors for the outcome measures at T2 and T3, and the within-group effect sizes for change at T3, adjusting for T1 variables. Across all conditions, participants reported significantly lower state body dissatisfaction and negative affect and higher state self-compassion at T3. The between-group effect sizes for change at T3 are reported in **Table 5.4**. Participants in both the imagery rescripting and the cognitive dissonance condition reported significantly higher state self-compassion than those

in the control condition at T3. There were no accompanying significant differences between conditions for state body dissatisfaction and negative affect.

## 5.5 Discussion

The aim of the current research was to examine the relative effectiveness of two different approaches to reducing body dissatisfaction compared to a control condition, within an experimental session. These approaches included imagery rescripting and cognitive dissonance. While cognitive dissonance has been found to be a robustly effective prevention approach for this purpose (Becker & Stice, 2017; Shaw & Stice, 2016), very little was known about the potential efficacy of imagery rescripting. We were also interested in the degree to which these approaches modified factors in addition to body dissatisfaction, namely negative affect and self-compassion. Findings provide support for both imagery rescripting and cognitive dissonance interventions, with participants in these conditions reporting higher state self-compassion compared to those in the control condition. Of note, imagery rescripting was superior, showing the largest effect ( $d = 1.01$ ) compared to cognitive dissonance ( $d = 0.76$ ) for within-group change in self-compassion.

Unexpectedly imagery rescripting and cognitive dissonance were not significantly different to the control condition with respect to changes in state body dissatisfaction and negative affect over the experimental session. However both approaches were promising, where imagery rescripting and cognitive dissonance showed very large effect sizes ( $d = 1.23$  and  $d = 1.10$ , respectively) compared to the control condition ( $d = 0.63$ ) for within-group change in negative affect. While research has shown that body dissatisfaction and negative affect are robust risk factors for disordered eating (Dakanalis et al., 2017; Jacobi & Fittig, 2010; Stice, 2016), an emerging body of research suggests that self-compassion may be a protective factor for disordered eating pathology (Braun et al., 2016; Goss & Allan, 2014;

Kelly & Carter, 2015; Kelly, Wisniewski, Martin-Wagar, & Hoffman, 2016; Tylka & Kroon Van Diest, 2015).

The brief interventions examined in the current study can only indicate strategies that are worthy of further research in terms of developing future effective prevention approaches. A major contribution of this research is to provide a direct comparison of strategies that may show promise in reducing body dissatisfaction and negative affect, and increasing self-compassion, thus reducing risk of future disordered eating. In this regard, we have identified that both imagery rescripting and cognitive dissonance may be useful in increasing self-compassion, in the short term. A further major contribution is that this study represents the first evaluation of imagery techniques in reducing body dissatisfaction in the prevention of disordered eating. To date, only two studies have investigated the use of imagery-based techniques in relation to eating disorders, in both cases bulimia nervosa (Cooper et al., 2007; Ohanian, 2002). These findings suggest that imagery rescripting may have utility in future prevention approaches.

These results should be interpreted in the context of several limitations. First, in this research we only examined brief interventions over the experimental session (i.e., over a 5 minute period); further research is required to examine the longer term impact of using such techniques before conclusions can be drawn about their clinical usefulness. It would also be valuable to investigate whether increased exposure to these procedures, including lengthier training and practice, may produce larger effects. Second, while the present study compared two distinct intervention approaches, there was no control over participant responses which may have muddied the waters. For example, participants in the imagery rescripting condition may have engaged in some form of cognitive dissonance during the rescripting process. Future research may wish to investigate this further. Third, participants were young female university students; it is therefore unclear whether these findings would generalize to other

populations, including males or the wider community. Relatedly, while the population examined in this study is considered to be at increased risk for disordered eating, future research would benefit from examining the use of these intervention approaches in high-risk populations including body-dissatisfied women.

In summary, the results of the current study suggest that brief interventions related to imagery rescripting and cognitive dissonance were able to significantly increase self-compassion during an experimental session in a female university sample compared to the control condition. Further, both imagery rescripting and cognitive dissonance showed promise in reducing negative affect, at least in the short term. Further research is necessary to investigate the longer term efficacy of these approaches. Second, while this study provides preliminary support for the use of imagery techniques in the prevention of disordered eating, further exploration is recommended. It would be of interest to investigate the use of more traditional forms of imagery rescripting, including face-to-face or therapist-led protocols (e.g., Wild & Clark, 2011). Third, self-compassion was highlighted as an important factor for change in this study; future research should examine this further in the context of prevention interventions.

**Table 5.1***Descriptive Statistics at Baseline (T1)*

Variable	Whole sample	Imagery rescripting	Cognitive dissonance	Control	ES (95% CI)
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	
<i>Demographic Variables</i>					
Age (at testing)	20.18 (1.98)	20.25 (2.20)	20.05 (2.02)	20.24 (1.70)	.10 (-.24 to .44)
BMI	23.75 (5.06)	23.64 (4.95)	23.33 (4.54)	24.29 (5.66)	-.06 (-.28 to .40)
Body dissatisfaction	6.70 (7.95)	6.75 (7.16)	6.01 (6.85)	7.33 (9.65)	-.09 (-.24 to .43)
<i>Dependent Variables</i>					
State body dissatisfaction	50.84 (22.72)	52.13 (22.36)	51.20 (22.34)	49.15 (23.69)	.04 (-.30 to .38)
State negative affect	1.71 (.69)	1.68 (.70)	1.63 (.55)	1.82 (.80)	.07 (-.26 to .41)
State self-compassion	53.35 (26.08)	54.89 (23.61)	52.89 (25.49)	52.21 (29.26)	.08 (-.26 to .41)

*Note.* T1 = time 1; SD = standard deviation; ES = between groups effect size (Cohen's *d*); CI = confidence interval; BMI = body mass index. Whole sample (*N* = 201); imagery rescripting (*n* = 69); cognitive dissonance (*n* = 66); control (*n* = 66). Significant ES are in **bold**. Cohen's *d* .2 = small, .50 = medium, .80 = large (Cohen, 1988).

**Table 5.2***Correlations between Baseline (T1) Variables*

Baseline Variable	1.	2.	3.	4.
1. BMI	–			
2. Body dissatisfaction	<b>.847**</b>	–		
3. State body dissatisfaction	<b>.321**</b>	<b>.381**</b>	–	
4. State negative affect	.085	.092	<b>.572**</b>	–
5. State self-compassion	-.084	-.117	<b>-.543**</b>	<b>-.574**</b>

*Note.* T1 = time 1; BMI = body mass index. Significant values are in **bold**.

\*\* Correlation is significant at the .001 level (2-tailed).

\* Correlation is significant at the .05 level (2-tailed).

**Table 5.3**

*Estimated Means (and Standard Errors) at Post-Induction (T2) and Post-Intervention (T3), with Within-Group Effect Sizes (Cohen's *d* and 95% Confidence Intervals), Controlling for Baseline (T1)*

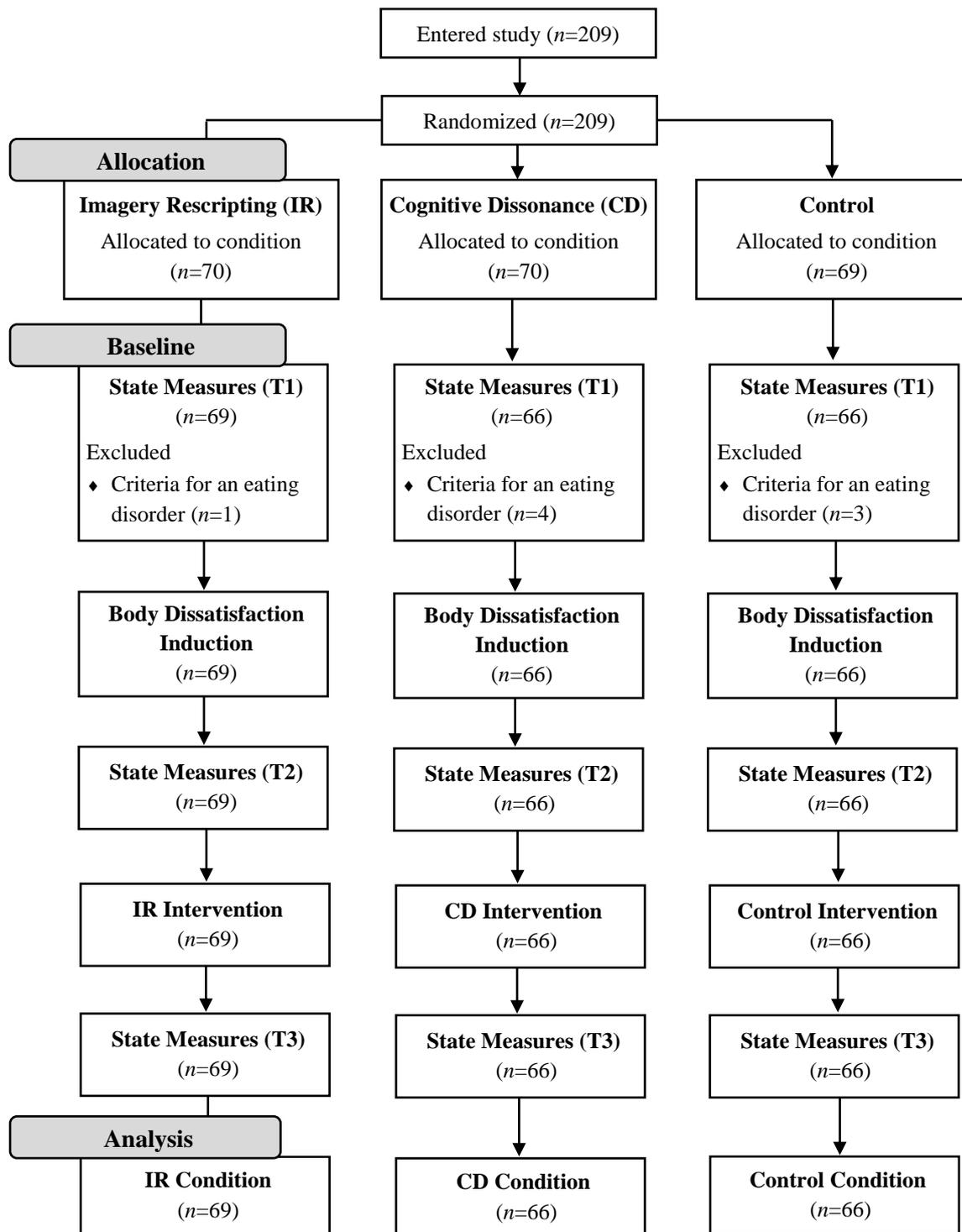
Variable	T2	T3	T2-T3 Change
	Estimated Mean (SE)	Estimated Mean (SE)	ES (95% CI)
State body dissatisfaction (T1 covariate mean = 50.84)			
Imagery rescripting	53.61 (1.59)	45.80 (1.62)	<b>.59 (.25 to .93)</b>
Cognitive dissonance	55.65 (1.63)	48.79 (1.66)	<b>.52 (.17 to .86)</b>
Control	55.38 (1.63)	49.03 (1.66)	<b>.48 (.13 to .82)</b>
State negative affect (T1 covariate mean = .33)			
Imagery rescripting	.42 (.01)	.26 (.02)	<b>1.23 (.86 to 1.59)</b>
Cognitive dissonance	.40 (.01)	.26 (.02)	<b>1.10 (.73 to 1.46)</b>
Control	.38 (.01)	.30 (.02)	<b>.63 (.28 to .98)</b>
State self-compassion (T1 covariate mean = 53.35)			
Imagery rescripting	52.04 (1.31)	63.46 (1.43)	<b>-1.01 (-1.36 to -.66)</b>
Cognitive dissonance	52.52 (1.34)	61.07 (1.46)	<b>-.76 (-1.11 to -.40)</b>
Control	50.58 (1.34)	56.70 (1.46)	<b>-.54 (-.89 to -.19)</b>

*Note.* T1 = time 1; T2 = time 2; T3 = time 3; SE = standard error; ES = within-group effect size (Cohen's *d*). Imagery rescripting ( $n = 69$ ); cognitive dissonance ( $n = 66$ ); control ( $n = 66$ ). Significant ES are in **bold**. Cohen's *d* .2 = small, .50 = medium, .80 = large (Cohen, 1988). LSD adjustments.

**Table 5.4***Between-Group Effect Sizes (Cohen's  $d$  and 95% Confidence Intervals) at Post-Intervention (T3), Controlling for Baseline (T1)*

Comparison	State body dissatisfaction	State negative affect	State self-compassion
Imagery rescripting vs cognitive dissonance	-.22 (-.56 to .11)	.00 (-.34 to .34)	.20 (-.14 to .54)
Imagery rescripting vs control	-.24 (-.58 to .10)	-.27 (-.61 to .07)	<b>.57 (.23 to .92)</b>
Cognitive dissonance vs control	-.02 (-.36 to .32)	-.27 (-.62 to .07)	<b>.37 (.03 to .72)</b>

*Note.* T1 = time 1; T3 = time 3. Imagery rescripting ( $n = 69$ ); cognitive dissonance ( $n = 66$ ); control ( $n = 66$ ). Significant values are in **bold**. Cohen's  $d$  .2 = small, .50 = medium, .80 = large (Cohen, 1988). LSD adjustments.



**Figure 5.1** CONSORT flow diagram for study. T1 = time 1 (baseline); T2 = time 2 (post-induction); T3 = time 3 (post-intervention).

## Chapter 6

### **Imagery Rescripting and Cognitive Dissonance: A Randomised Controlled Trial of Two Brief Interventions for Women at Risk of Developing an Eating Disorder**

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#### **Author Contributions:**

JP led the study design, recruitment, data collection, statistical analyses, results and interpretation, and manuscript preparation. TW contributed to the study design, statistical analyses, results and interpretation, and manuscript preparation.

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## 6.1 Abstract

The present study compared two brief interventions, imagery rescripting and cognitive dissonance, to a control condition in a sample of body dissatisfied young women at risk of developing an eating disorder. We examined the degree to which each intervention reduced disordered eating and modified risk and protective factors for eating disorders. Female university students ( $N = 107$ , 17 to 28 years of age) completed a screening questionnaire, followed by random allocation to one of the three conditions, baseline assessment, a body dissatisfaction induction, brief intervention, home practice, and one-week follow-up. Findings provide support for the imagery rescripting intervention, with participants reporting higher body image acceptance (Cohen's  $d = 0.49$ ) and self-compassion ( $d = 0.59$ ), and lower levels of disordered eating ( $d = 0.59$ ), at follow-up. There was no significant impact of cognitive dissonance on any factors. Change in body image acceptance and self-compassion mediated the relationship between allocated condition and change in disordered eating. These findings provide support for the use of imagery-based techniques (e.g., imagery rescripting) to reduce risk for the development of an eating disorder by strengthening protective factors. Further exploration of the use of imagery strategies in the prevention of disordered eating is required, including prospective tests of the mechanisms of action.

## 6.2 Introduction

Disordered eating and eating disorders are prevalent among young women (Goldschmidt et al., 2016; Hay et al., 2008; Wade et al., 2012), and have the potential for serious consequences including increased mortality rates, emotional distress, physical morbidity, psychosocial impairment, and decreased quality of life (Allen et al., 2013; Fichter & Quadflieg, 2016; Winkler et al., 2014). Body dissatisfaction and negative affect are two of the most prominent risk factors associated with the development of disordered eating (Dakanalis et al., 2017; Jacobi & Fittig, 2010; Stice, 2016), and are the most common risk factors to be included across the eating disorder models that have been used to inform interventions (Pennesi & Wade, 2016), including prevention approaches.

In a prospective test of the dual-pathway model of bulimic pathology, Stice (2001) found that perceived pressure to be thin promotes internalisation of the thin-ideal and body dissatisfaction, which leads to dieting and negative affect, which in turn leads to bulimic pathology. This model resulted in the development of a cognitive dissonance-based intervention (DBI), targeting the thin-ideal of beauty through cognitive-attitudinal activities such as asking participants to argue against these ideals (Stice & Presnell, 2007). DBIs have been shown to be robustly effective prevention approaches, reducing thin-ideal internalisation, body dissatisfaction, negative affect, psychosocial functioning and eating disorder symptoms, and risk for eating disorder onset (Becker & Stice, 2017; Stice, Butryn, et al., 2013; Stice, Rohde, et al., 2009). Effects have been maintained for up to 3 years (Stice, Rohde, Butryn, Shaw, & Marti, 2015; Stice, Rohde, et al., 2011).

A growing body of research has also suggested self-compassion may act as a protective factor against poor body image and eating pathology (Braun et al., 2016; Goss & Allan, 2014; Kelly & Carter, 2015; Kelly et al., 2016; Steindl, Buchanan, Goss, & Allan, 2017; Tylka & Kroon Van Diest, 2015). Self-compassion (a form of general compassion)

involves having empathy and compassion for oneself, especially during failures or in time of difficulty (Gilbert, 2014; Neff, 2011). In a cross-sectional study of adult women, Tylka, Russell, and Neal (2015) found that self-compassion was protective against the effects of mediating risk factors (e.g., thin-ideal internalisation) for disordered eating. Somerville and Cooper (2007) argue that using cognitive approaches to tackle negative core beliefs of relevance to eating disorders (such as self-criticism and shame) will be less effective than approaches that focus on emotional mechanisms, such as compassion-focused imagery (Gilbert & Irons, 2005). Recent experimental work has shown that imagery has a greater impact on emotions than verbalizing (Holmes & Mathews, 2005) and has led to an interest in the use of imagery rescripting which changes the negative meanings linked to autobiographical memories of early traumatic or distressing experiences by asking a patient to imagine or visualize a specific memory in the present and then to intervene (e.g., say or do something) so as to change its meaning (Stopa, 2011). Across a variety of psychological disorders there has been growing use and evaluation of imagery rescripting as an adjunct to standard cognitive-behavioural treatments, with preliminary research showing that it can improve treatment outcomes, and can also be effective as a stand-alone treatment in some disorders, including when previous treatments have failed (Arntz et al., 2007; Grunert, Weis, Smucker, & Christianson, 2007; Wheatley et al., 2007; Wild, Hackmann, & Clark, 2007).

To date, only two studies have investigated the use of imagery techniques to target core beliefs related to eating disorders (Tatham, 2011), in both cases bulimia nervosa. Ohanian (2002) presented a single case report investigation of the use of imagery rescripting in conjunction with cognitive behaviour therapy (CBT) for bulimia nervosa (Fairburn, 1997), and found that while eight sessions of conventional CBT reduced symptom behaviours by 50%, one session of imagery rescripting led to an almost complete cessation of initial binge-purge behaviours. Cooper et al. (2007) compared a single session imagery intervention

(imagery modification; i.e., restructuring of core beliefs through imagery) to a control (i.e., verbal restructuring with no imagery), and found that imagery was more effective in reducing emotionally held belief ratings, negative mood, and urges to binge. Other research (Cesa et al., 2013; Riva, 2011) has suggested the role of imagery rescripting methods based on virtual reality in treating body image disturbance in obesity and binge eating disorder. Imagery rescripting has not previously been investigated for reducing body image disturbance in young women at risk of developing an eating disorder.

Thus, the aim of the present study was to compare two brief interventions, imagery rescripting and cognitive dissonance, to a control condition in a sample of body-dissatisfied women considered to be at risk of developing an eating disorder, in terms of reducing disordered eating. We also investigated the extent to which each intervention modified established risk and protective factors for eating disorders. The information gained from this research will help to inform the development of future prevention approaches for eating disorders and related problems with body, weight and shape.

## **6.3 Method**

### **6.3.1 Participants.**

Females were recruited from a first year university student population and wider university sample. Inclusion criterion for the study included those women at risk of developing an eating disorder, as indicated by a score of  $\geq 47$  on the Weight Concerns Scale (WCS; Killen et al., 1996; Killen et al., 1994), used in previous studies to screen for high-risk status (e.g., Jacobi et al., 2011). After a full baseline assessment, we excluded women considered to be likely to have an eating disorder that required urgent attention ( $n = 8$ ). This included self-reporting  $\geq 4$  on the over-evaluation of weight or shape items in the Eating Disorder Examination–Questionnaire (EDE-Q; Fairburn & Beglin, 1994) and, either a body

mass index (BMI) of <17.5 (i.e., the criteria for low weight; World Health Organization [WHO], 2006), or using vomiting or laxatives as a means of controlling shape or weight for more than half the days over the past 28 days. We provided these women with information about appropriate treatment options. Given that a moderate degree of disordered eating (including binge eating, fasting, and driven exercise) is considered normative among young women (LePage et al., 2008; Liechty & Lee, 2013; Wade et al., 2012), the presence of these behaviours were not considered as exclusion criteria. The analytic sample for this study included 107 females ranging in age from 17 to 28 years ( $M_{age} = 20.27$ ,  $SD = 2.02$ ) with a mean BMI in the normal range (i.e., BMI 18.5–24.99; WHO, 2006;  $M = 24.57$ ,  $SD = 5.08$ ). The majority of participants (70.1%) self-reported as Caucasian, with the next largest groups being Asian (21.5%), African (2.8%), Indian (2.8%), and Middle Eastern (1.9%).

### **6.3.2 Procedures.**

The study was presented as an investigation of different strategies to improve body image and eating in women, and involved two phases of data collection over a period of one week, depicted in **Figure 6.1**. Before commencing, participants completed a brief screening questionnaire (i.e., WCS; Killen et al., 1996; Killen et al., 1994) to determine eligibility for the study, and height and weight were measured in order to derive BMI. Following screening, participants were randomized, using the randomization function in *Qualtrics*, to one of the three conditions: imagery rescripting, cognitive dissonance, or control.

In phase 1 (baseline) participants completed self-report trait measures (Time 1; T1) followed by state measures (pre-induction) in a research laboratory with the principal researcher available behind a screen to answer any questions. For five minutes all participants were asked to imagine (or visualise), and then describe in the first person (as if it were happening to them “right now”), the “first or earliest memory of a personal unpleasant body experience from the past where you may have felt ashamed or embarrassed of your body or

how your body looked”. Examples were provided e.g., trying on clothes in a change-room, getting ready with friends to go to a party, walking past a group of people who were looking at you, being teased about your body, looking at yourself in the mirror. This was used to equalise all groups given that the first stage of imagery rescripting requires recalling and visualising traumatic memories (Wild & Clark, 2011), and was also intended to serve as a body dissatisfaction induction exercise to enhance effect sizes for the state measures used in this phase. After the recall, participants completed a second rating of the state measures (post-induction), before receiving the baseline intervention (i.e., imagery rescripting, cognitive dissonance, or control).

Phase 2 (follow-up) was completed online. Participants in the two experimental conditions completed home practice (i.e., a condensed form of the baseline intervention) over five consecutive days, and then completed the trait measures again (T2). Data were collected between October 2016 and April 2017. The mean time between assessments was 7.6 days ( $SD = 0.9$  days). A choice of course credits, cash or gift voucher were offered for research participation. The Flinders University Social and Behavioural Research Ethics Committee approved this study.

### **6.3.3 Techniques.**

Video instructions were used for all conditions at baseline in order to enhance understanding and compliance (Kingston et al., 2010), however written instructions were also provided on the computer screen as indicated below, and were also used for home practice.

***Imagery rescripting.*** This procedure was designed for this study based on methods used previously with traumatic memories in social phobia (Wild & Clark, 2011); however with an emphasis on the latter active stages of treatment. Participants were presented with the following instructions, presented in two parts and make up a single intervention exercise:

(Part 1) Please think about your first or earliest memory of a personal unpleasant body experience from the past where you may have felt ashamed or embarrassed of your body or how your body looked. This time, when you close your eyes and imagine (or visualize) this event, imagine it from an observers' perspective. Imagine that your adult self is in the room observing what's happening right now, watching the events unfold. Imagine that you are observing your younger self reliving the event. Please only continue once you have this event in mind. For the next 2.5 minutes, please describe what you see from an observers' perspective as if it were happening to your younger self right now. Please write in the third person (e.g., "I see Sarah in the change room, she is trying on a pair of blue jeans..."). Try to be descriptive and include as many details as you can, for example, where is Sarah, what is Sarah doing, who is Sarah with, how might Sarah be feeling (emotions), and what might Sarah be telling herself (thoughts).

(Part 2) Please again think about your first or earliest memory of a personal unpleasant body experience from the past where you may have felt ashamed or embarrassed of your body or how your body looked. This time, when you close your eyes and imagine (or visualize) this event, imagine it as if it is happening to you right now, but this time your wiser and more compassionate adult self is with you in the room and she can intervene if you want her to. She can offer you compassion or provide new updated information based on what you know now as an adult. She can talk to you (or others), or do anything else that feels helpful or right in the situation. Please only continue once you have this event in mind. For the next 2.5 minutes, please describe what you see as if it is happening to you right now but this time your adult self is with you and she can intervene if you want her to. Please write in the first person (e.g., "I'm in the change room, I'm trying on a pair of blue jeans...") unless you are referring to the adult you (e.g., "Adult Sarah said...", or "older Sarah said..."). Try to be descriptive and include as many details as you can, including where you are, what you are doing, who you are with, how you are feeling (emotions), and what you are telling yourself (thoughts). Please also describe if your adult self intervenes or does anything in the situation.

The part 2 instructions were used for the imagery rescripting home practice; however participants were not given a time limit in which to respond.

**Cognitive dissonance.** This procedure was adapted from techniques described by Stice et al. (2006), which have subsequently been used by Wade et al. (2009) as a brief intervention for body dissatisfaction. Participants were presented with the following instructions, accompanied by images of the thin-ideal stereotype for women:

Please read the definition of the thin-ideal stereotype for women below and view the accompanying images which show what this ideal looks like. Please only continue after reading the definition and viewing the images. *“The thin-ideal stereotype for women, as portrayed by the media, is that of an ultra-slender, feminine physique with a small waist, long legs and little body fat.”* For the next 5 minutes, please brainstorm about the following: 1) the costs and consequences of pursuing the thin-ideal (thin-ideal stereotype for women as portrayed in the media), and 2) the positive attributes about yourself (these can be physical, emotional, or behavioural). Try to spend an even amount of time on each topic.

The same instructions were used for the cognitive dissonance home practice; however participants were not presented with the definition of the thin-ideal or the accompanying images, and were not given a time limit in which to respond.

**Control.** The control procedure was adapted from May et al. (2010). Participants did not receive any training but were asked to let their mind wander, and were presented with the following instructions:

For the next 5 minutes, just sit and let your mind wander wherever it would like to, let your attention drift. There is no need to control your thoughts in any way, just let them wander. You may find that your mind wanders to thoughts about many different things or thoughts of nothing at all. You may find that your mind wanders to thoughts about the personal unpleasant body experience you described earlier. Wherever your mind wanders it’s OK. Just think about whatever you like. Thoughts are not right or wrong. Just let your attention drift.

#### **6.3.4 Fidelity.**

A quality rating scheme was developed to assess intervention fidelity at home practice, based on that previously described by Allen et al. (2016); the complete scale is provided in **Appendix A**. Individual mean item ratings were summed to give a total score, with higher scores indicating greater fidelity. Possible scores ranged from 0 to 12 for the imagery rescripting intervention, and from 0 to 6 for the cognitive dissonance intervention; scores for the cognitive dissonance intervention were multiplied by two in order to directly compare scores across interventions. The number of intervention exercises completed during the follow-up period was also recorded. The maximum number of intervention exercises offered to participants in each condition was five.

The first author and a blind rater co-rated responses independently with subsequent discussion of ratings until an agreement was met; a third rater was consulted where the first author and blind rater were unable to come to an agreement. Once rating agreement was consistently reached, raters independently scored a random selection of responses from each of the intervention groups including: 9 responses from the imagery rescripting condition (5.3%); and 9 responses from the cognitive dissonance condition (5.5%). Inter-rater reliability was high:  $r = .94$  and  $r = .84$ , respectively. All subsequent responses were rated by the blind rater. Across all participants, the quality rating scheme showed high internal consistency for both the imagery rescripting ( $\alpha = .90$ ) and the cognitive dissonance intervention ( $\alpha = .91$ ).

### **6.3.5 Screening measure.**

**Weight concern.** The WCS (Killen et al., 1996; Killen et al., 1994) was adapted for an Australian audience (i.e., 3lb was replaced with 1.36kg). It consists of 5 items that assess worry about weight and shape, fear of gaining weight, last time on a diet, importance of weight, and feelings of fatness. A score of  $\geq 47$  on the WCS has good predictive validity for eating disorder cases (Jacobi, Abascal, & Taylor, 2004; Killen et al., 1996; Killen et al.,

1994), and the WCS has been used in previous studies to screen for high-risk (e.g., Jacobi et al., 2011) and has good test-retest reliability (7-month interval,  $r = .71$ ; Killen et al., 1994).

### **6.3.6 Outcome measures.**

***Disordered eating.*** This was assessed using the 22-item global score of the EDE-Q (Fairburn & Beglin, 1994), a self-report measure adapted from the interview-based Eating Disorder Examination (EDE; Fairburn & Cooper, 1993). The global score from the past 28 days is used as an indicator of outcome (i.e., recovery) in treatment studies (Bardone-Cone et al., 2010; Fairburn et al., 2009). Items were scored on 7-point scales, with higher scores indicating higher pathology. The scale at T2 was adapted to reflect the past 7 days, with scores ranging from *no days* to *6-7 days* in order to account for the one-week follow-up. The EDE-Q global score has demonstrated strong internal consistency ( $\alpha = .95$ ; Kelly et al., 2013) and high convergent validity with the EDE global score ( $r = .84$ ; Mond et al., 2004). In the current study internal consistency ranged from .90 to .92, and the intra-class correlation coefficient was .74. The diagnostic items from the EDE-Q were used to exclude the eight people described above.

***Body image acceptance.*** This was measured using the 12-item Body Image-Acceptance and Action Questionnaire (BI-AAQ; Sandoz et al., 2013), which measures the capacity to experience the ongoing perceptions, sensations, feelings, thoughts, and beliefs associated to one's body fully and intentionally while pursuing chosen values. Items were scored on 7-point scales, with higher scores indicating higher acceptance. The BI-AAQ has demonstrated high internal consistency ( $\alpha = .92$ ) and good concurrent-related, criterion-related, and incremental validity (Sandoz et al., 2013). In the current study internal consistency ranged from .91 to .92, and the intra-class correlation coefficient was .84.

**Negative affect.** This was measured using the 21-item Depression Anxiety Stress Scale-21 (DASS-21; Henry & Crawford, 2005). Items were scored on 4-point scales, with higher scores indicating higher psychopathology. The DASS-21 has demonstrated high internal consistency ( $\alpha = .93$ ) and the DASS-21 subscales have demonstrated high convergent validity with independent measures of anxiety and depression (Henry & Crawford, 2005). In the current study internal consistency ranged from .94 to .95, and the intra-class correlation coefficient was .91.

**Self-compassion.** This was measured using the 12-item version of the Self-Compassion Scale (SCS; Raes et al., 2011). Items were scored on 5-point scales, with higher scores indicating higher self-compassion. The SCS has demonstrated high internal consistency ( $\alpha = .92$ ) and test-retest reliability (3-week interval,  $\rho_1 = .93$ ; Neff, 2003a). In the current study internal consistency was .78 at each time point, and the intra-class correlation coefficient was .84.

**Quality of life.** The 16-item Clinical Impairment Assessment–Questionnaire (CIA; Bohn et al., 2008; Bohn & Fairburn, 2008) was used to assess psychosocial impairment caused by eating problems. The CIA measures the severity of psychosocial impairment as a result of eating disorder features over the past 28 days, including: mood and self-perception; cognitive functioning; interpersonal functioning; and work performance. The scale at T2 was adapted to reflect the past 7 days. Items were scored on 4-point scales, with higher scores indicating higher clinical impairment (lower quality of life). The CIA has demonstrated high internal consistency ( $\alpha = .97$ ) and test-retest reliability (3-day interval; Bohn & Fairburn, 2008). In the current study internal consistency ranged from .93 to .95, and the intra-class correlation coefficient was .83.

### **6.3.7 State measures.**

These were assessed before (pre-induction) and after (post-induction) the body dissatisfaction induction. Three visual analogue scales (VAS) were used to measure state body dissatisfaction. Following Heinberg and Thompson (1995), participants were asked to indicate their response to the questions “How satisfied do you feel about your weight right now?”, “How satisfied do you feel about your appearance right now?”, and “How distressed are you by your feelings about your body right now?”. Responses were recorded on a 100-pixel horizontal sliding scale from *not at all* to *very much*, ranging from 0 (*extreme satisfaction*) to 100 (*extreme dissatisfaction*). VAS are shown to be reliable measures of fluctuations in body dissatisfaction, are quick and easy to administer, and are sensitive to small changes (Tiggemann & McGill, 2004; Wade et al., 2009).

The 10-item Negative Affect scale of the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988) was used to measure state negative affect. The PANAS consists of a number of words that describe different feelings and emotions, and participants were asked to indicate the extent to which they feel this way “right now (that is, at the present moment)”. Items were scored on 4-point scales, ranging from *very slightly or not at all* to *extremely*, with higher scores indicating higher negative affect. The momentary version of the PANAS has been shown to be a reliable measure of state positive and negative affect in previous daily diary studies ( $\alpha = .88-.92$ ; Merz & Roesch, 2011) and has demonstrated moderate convergent validity with independent measures of distress and psychopathology (Watson et al., 1988).

### **6.3.8 Statistical analyses.**

Of the 115 eligible participants, 8 (7.0%) participants were excluded so that they could be referred to appropriate treatment, leaving 107 participants in the analytic sample; 1 (0.9%) participant declined to participate at phase 2 and a further 6 (5.6%) participants did not complete the follow-up (T2) questionnaire. Comparisons between those participants who

provided data at T1 only and those who provided data at T1 and at T2 were investigated using Little's (1988) Missing Completely At Random (MCAR) test: chi square=14.073 ( $df = 9$ ;  $p = .120$ ) with a chi-square/df ratio of 1.56, indicating that the data were missing completely at random. Given the small amount of missing data, we utilised an intent-to-treat analysis using the expectation maximization function in the *Statistical Package for the Social Sciences (SPSS)* version 23. Therefore data from all participants ( $n = 107$ ) were analysed. Consistent with an open science approach to interpreting significance (Cumming & Calin-Jageman, 2016), all comparisons were investigated using a between- or within-group effect size (ES) with 95% confidence intervals (CIs). If the CI does not contain zero, the null hypothesis is rejected and the difference between groups is considered to be statistically significant (Hayes, 2009). ES was calculated using Cohen's  $d$ , where .2 = small, .5 = medium, and .8 = large (Cohen, 1988), unless otherwise stated. Baseline values across the three conditions were compared using one-way analyses of variance (ANOVAs).

***Manipulation check.*** A manipulation check was performed using a 3 (condition: imagery rescripting, cognitive dissonance, control) by 2 (time: pre-induction, post-induction) repeated measures ANOVA on state body dissatisfaction and negative affect, respectively.

***Change in outcome over time.*** One-way analyses of covariance (ANCOVAs) were used to compare the effectiveness of the three conditions with respect to the outcome variables. The dependent variable was change at T2, and T1 was included as a covariate.

***Ratings of quality.*** Hierarchical multiple regressions were used to investigate whether fidelity or the number of intervention exercises completed predicted change in outcome at T2. T1 was included as a covariate in all analyses. Independent samples  $t$ -tests were used to compare between group differences.

**Mediational pathways.** Mediation was tested using PROCESS macro (Hayes, 2013), which allows for path analysis-based mediation analysis and generates direct and indirect effects and bootstrap CIs in mediation models with single or multiple mediators operating in parallel or serial (Hayes, 2013; Hayes & Preacher, 2013; Preacher & Hayes, 2004). In particular, we were interested in investigating whether any variables mediated the relationship between condition and change in disordered eating. The term ‘total effect’ refers to the relationship between the predictor and the outcome variable, ‘indirect effect’ refers to the mediating pathway between the predictor and the mediator(s), and between the mediator(s) and the outcome, and ‘direct effect’ reflects the relationship between the predictor and the outcome while controlling for the indirect effects (Preacher & Hayes, 2004, 2008). As recommended by Hayes (2013) and Rucker et al. (2011), we did not test preconditions or interpret the present findings in terms of *complete* or *partial* mediation. Indirect effects were tested through corrected bootstrapping, which is recommended in mediation analysis with small to moderate sample sizes (Hayes, 2009; MacKinnon et al., 2004; Shrout & Bolger, 2002). Bootstrap analysis is less sensitive to violations of the statistical assumptions of normality and linearity, and has strongest statistical power in detecting indirect effects (Hayes, 2009; MacKinnon et al., 2004). It also has the best Type-I error control and allows for improved accuracy of CIs. Bootstrapping with 10000 resamples was used to generate 95% CIs to determine the significance of the indirect (or mediating) effect.

## 6.4 Results

### 6.4.1 Descriptives.

Means and standard deviations are shown in **Table 6.1**, skewness for each variable was within the accepted range of  $Z = +/-2.575$  ( $\alpha = .01$ ). Participants did not differ across the three conditions on any of the variables. The majority of participants (61.7%) were within the

normal BMI range (i.e., BMI 18.5–24.99; WHO, 2006); 2.8% were classified as underweight (i.e., BMI <18.5), and 35.5% were overweight or obese (i.e., BMI  $\geq$ 25). The majority of participants (95.3%) reported engaging in some form of disordered eating behaviour, specifically: driven exercise/fasting ( $n = 91$ , 85.0%); bingeing ( $n = 83$ , 77.6%); and self-induced vomiting/laxative misuse ( $n = 21$ , 19.6%). Over two thirds of participants (71.0%) reported eating disorder psychopathology (i.e., EDE-Q global) above the clinical cut-off (i.e.,  $\geq$ 2.77, norm for young adult women +1SD; Mond et al., 2006). Mean WCS for this sample was well above the cut-off for high risk of developing an eating disorder (i.e.,  $\geq$ 47; Killen et al., 1996; Killen et al., 1994;  $M = 62.96$ ,  $SD = 11.17$ ).

#### **6.4.2 Manipulation check.**

Across all groups, the induction successfully resulted in increasing negative affect, which was associated with a significant main effect of time, (ES = -.28, 95% CI = -.55, -.02). There was no accompanying significant increase in body dissatisfaction (ES = -.17, 95% CI = -.44, .10). There were no significant main effects of group or interactions between time and group for either of the state variables. After the induction procedure, both state body dissatisfaction and negative affect were not significantly different across the three conditions (body dissatisfaction, ES = -.03, 95% CI = -.50, .43; negative affect, ES = -.05, 95% CI = -.51, .41).

#### **6.4.3 Change in outcome over time.**

**Table 6.2** shows the estimated means and standard errors for the outcome measures at T2, adjusting for T1 variables, and between group effect sizes are reported in **Table 6.3**. Participants in the imagery rescripting condition reported significantly higher body image acceptance than those in the cognitive dissonance condition, as well as significantly higher

self-compassion and lower levels of disordered eating than those in the control condition at T2.

#### **6.4.4 Fidelity and outcome.**

There was no significant difference between groups for fidelity (imagery rescripting,  $M = 9.08$ ,  $SD = 2.10$ ; cognitive dissonance,  $M = 8.32$ ,  $SD = 2.19$ ;  $ES = .35$ , 95%  $CI = -.11, .82$ ) or for the number of intervention exercises completed (imagery rescripting,  $M = 4.54$ ,  $SD = 1.12$ ; cognitive dissonance,  $M = 4.66$ ,  $SD = .64$ ;  $ES = -.13$ , 95%  $CI = -.59, .33$ ). Fidelity and the number of intervention exercises completed did not predict change in the outcome measures in either condition.

#### **6.4.5 Mediation pathways.**

Given the findings showing imagery rescripting to be associated with significant change in body image acceptance, self-compassion, and disordered eating, a dichotomous variable was computed for condition, where participants were classified as belonging either to the imagery rescripting condition or to the cognitive dissonance/control conditions. Change in body image acceptance and self-compassion were investigated as mediators of the relationship between condition and changes in disordered eating, adjusting for T1 variables. Mediators were investigated separately (Model 1 & Model 2) and simultaneously (Model 3). **Table 6.4** presents the model summary and indirect effect summaries. The indirect effect (mediational pathway) was statistically significant for all models. Variance explained and effect size indicated that the model containing both body image acceptance and self-compassion (Model 3) was superior to the models containing body image acceptance (Model 1) or self-compassion (Model 2) alone, and accounted for 59% of the variance in disordered eating (see **Figure 6.2**).

### **6.5 Discussion**

The aim of the present research was to investigate the effectiveness of two brief interventions, imagery rescripting and cognitive dissonance, to a control condition in a sample of young women at risk of developing an eating disorder, with respect to reducing disordered eating. Findings provide some qualified support for the imagery rescripting techniques over the DBI techniques and control conditions. Imagery rescripting was associated with significant improvements in body image acceptance compared to the cognitive dissonance condition but not compared to the control condition. In addition, imagery rescripting was associated significant improvements in self-compassion and levels of disordered eating compared to the control condition but not compared to the cognitive dissonance condition.

This somewhat confused pattern of results somewhat limited the strength of conclusions that could be made about the usefulness of imagery rescripting, which was addressed somewhat by further analysis indicating that change in body image acceptance and self-compassion mediated the relationship between condition and change in disordered eating, where the imagery rescripting condition was associated with higher body image acceptance and higher self-compassion, which was associated with lower levels of disordered eating, compared to the cognitive dissonance/control conditions. Model testing indicated that the model containing both body image acceptance and self-compassion was superior to the models containing either body image acceptance or self-compassion alone, suggesting that each of these variables play a role in influencing change in disordered eating.

The present research adds to the early literature suggesting that imagery-based techniques (e.g., imagery rescripting) may be useful adjuncts in interventions for eating disorders (Tatham, 2011). While imagery rescripting strategies have been shown to be effective in the treatment of bulimia nervosa (Cooper et al., 2007; Ohanian, 2002), the present findings are the first to provide preliminary evidence for the use of imagery rescripting in the

context of reducing risk factors and increasing protective factors for disordered eating. While there is considerable support for the effectiveness of DBIs for reducing body dissatisfaction and body image problems in young women (Stice, Rohde, et al., 2009; Stice, Shaw, et al., 2007), the current findings indicate that imagery-based approaches may be more effective (e.g., increasing body image acceptance).

We highlight a number of reasons why the DBI techniques in the current study were not as effective as previous research suggests. First, we utilised an individual online format, whereas DBIs are typically delivered, and have been most widely evaluated, in a group-based format. A recent evaluation of clinician-led, peer-led and internet-delivered DBIs by Stice et al. (2017) found that the internet-based intervention was less effective. Second, while the length of the intervention was the same for imagery rescripting, the intervention was much briefer than usual (Stice et al., 2009). Third, participants were exposed to the thin-ideal stereotype during the baseline intervention (but not during home practice), which may have diluted the impact of the exercise. Fourth, we only used one aspect of DBIs repeatedly, and not the full intervention; repetition may be better suited to imagery rescripting than cognitive dissonance. Finally, we included the use of a body dissatisfaction induction prior to delivering the intervention, however this procedure was used in all conditions. Future prevention research would benefit from further exploration of the use of imagery in the prevention of eating disorders, especially as an adjunct to evidence-based interventions.

These findings contribute to the emerging research highlighting the potential advantages of using imagery-based approaches over more verbal-based approaches. Researchers have suggested that, compared to verbal representation, imagery has a stronger association with emotion and therefore can provide greater impact on psychological disorders (Holmes & Matthews, 2005). For example, Holmes, Matthews, Mackintosh, and Dalglish (2008) demonstrated that use of a mental image compared to use of a verbal sentence

consistently elicited greater emotional responses, despite the use of identical cues. Imagery-based strategies may be better able to evoke—and therefore modify—emotionally held core beliefs, compared to verbal-based strategies that are utilised in CBT. In line with this, research has demonstrated that core (self) beliefs are associated with greater emotional than rational bases (Somerville & Cooper, 2007), which may explain why core beliefs are more difficult to modify using cognitive therapies. Early work has indicated that rescripting of early memories (i.e., imagery rescripting) may help to modify core beliefs in eating disorders (Cooper, 2011; Hinrichsen, Morrison, Waller, & Schmidt, 2007; Somerville & Cooper, 2007). While the present findings provide support the use of imagery rescripting in the context of reducing disordered eating, yet to be explored are the mechanisms of action and relationship to maladaptive core beliefs. Our research suggests that imagery rescripting reduces risk for the development of an eating disorder by strengthening protective factors, but further exploration of the use of imagery strategies in the prevention of disordered eating is required, including prospective tests of the mechanisms of action.

These findings are also in line with a greater emphasis on resilience, and concord with the emerging literature on protective factors (Gillen, 2015; Tylka, 2011a), which suggests that appreciation for, acceptance of, and connection with, one's physical body, is associated with important health-related indicators including eating behaviour (e.g., Hahn Oh et al., 2012). Further, higher self-compassion was associated with lower disordered eating in our sample, consistent with cross-sectional research suggesting that self-compassion may help to protect against eating pathology (Tylka et al., 2015). These findings provide preliminary support for the further testing of approaches targeting compassion to improve body- and disordered eating-related outcomes (Adams & Leary, 2007; Gale, Gilbert, Read, & Goss, 2014; Kelly & Carter, 2015; Neff & Germer, 2013; Steindl et al., 2017).

The results of the study should be considered in the context of several limitations. First, disordered eating was a self-report assessment, using the EDE-Q, a standardised measure of eating pathology with acceptable reliability and validity (Berg et al., 2011; Luce & Crowther, 1999) able to distinguish between eating disorder and non-eating disorder cases (Mond et al., 2004); however the use of structured clinical interviews may further improve predictive validity. While the use of anonymous self-report measures likely induces more candid responding, particularly for sensitive behaviours e.g., self-induced vomiting, laxative/diuretic use, eating furtively (Lavender & Anderson, 2009), the use of interview data may increase the strength of findings. Second, participants were undergraduate females aged between 17 and 28 years and primarily Caucasian (70.1%); it is therefore unclear whether these findings would generalize to other populations, including younger/older women, men, ethnic minorities, or the wider community. Third, the study included a mind wandering control condition, a procedure that has been used previously by May and colleagues (2010) as a control; however the procedure may have contained elements of mindfulness i.e., noticing thoughts in the moment with non-judgement (Kabat-Zinn, 2003). Future research may consider the use of a true control condition (e.g., Wade et al., 2009). Fourth, in this study we only included a one-week follow-up; further research is required to examine the longer term impact of the intervention.

In sum, the present findings have several implications for future research. While this study provides preliminary support for the use of imagery rescripting in the prevention of disordered eating among a sample of women at risk of developing an eating disorder, further exploration is recommended, including longer follow-up. Second, while the present study investigated an online imagery rescripting intervention, it would be of interest to compare this to more traditional face-to-face or therapist-led imagery protocols (e.g., Wild & Clark, 2011). Third, it would be of interest to investigate a fuller version of an imagery rescripting

intervention to the gold standard 4-session DBIs (Stice, Rohde, et al., 2009; Stice, Shaw, et al., 2007) in order to examine if these interventions differ. Further, combined approaches may enhance intervention effectiveness, enabling development of more effective future prevention approaches for disordered eating.

**Table 6.1***Descriptive Statistics at Baseline (T1)*

Variable	Whole Sample	Imagery rescripting	Cognitive dissonance	Control	ES (95% CI)
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	
<i>Baseline Variables only</i>					
Weight concern	62.96 (11.71)	64.19 (12.19)	61.76 (11.19)	62.86 (11.89)	-.21 (-.67 to .26)
Age	20.27 (2.02)	20.55 (2.43)	19.90 (1.84)	20.36 (1.68)	-.32 (-.79 to .14)
BMI	24.57 (5.08)	24.28 (4.80)	25.21 (4.62)	24.24 (5.83)	.18 (-.28 to .65)
<i>Dependent Variables</i>					
Body image acceptance	3.61 (1.12)	3.64 (1.04)	3.59 (1.04)	3.61 (1.29)	-.04 (-.51 to .42)
Negative affect	1.09 (.61)	.99 (.62)	1.10 (.60)	1.19 (.61)	.18 (-.28 to .65)
Self-compassion	2.56 (.49)	2.65 (.47)	2.54 (.44)	2.47 (.56)	-.23 (-.69 to .24)
Quality of life	2.28 (.60)	2.36 (.61)	2.24 (.56)	2.23 (.64)	-.20 (-.66 to .27)
Disordered eating	3.34 (.94)	3.55 (.84)	3.30 (.83)	3.15 (1.11)	-.27 (-.73 to .19)
<i>State Variables</i>					
Body dissatisfaction	62.14 (19.03)	63.48 (18.81)	61.19 (19.08)	61.67 (19.69)	-.12 (-.58 to .34)
Negative affect	1.98 (.75)	1.88 (.78)	1.88 (.55)	2.17 (.87)	.00 (-.46 to .46)

*Note.* T1 = time 1; SD = standard deviation; ES = between groups effect size (Cohen's *d*); CI = confidence interval; BMI = body mass index. Sample (*N* = 107); imagery rescripting (*n* = 37); cognitive dissonance (*n* = 35); control (*n* = 35). Significant values are in **bold**. Cohen's *d* .2 = small, .50 = medium, .80 = large (Cohen, 1988).

**Table 6.2***Estimated Means (and Standard Errors) at 1-Week Follow-Up (T2), Controlling for Baseline (T1) Variables*

Variable	Imagery rescripting	Cognitive dissonance	Control
Body image acceptance (T1 covariate mean = 3.61)	4.19 (.14)	3.78 (.14)	3.82 (.14)
Negative affect (T1 covariate mean = 1.09)	.85 (.06)	.95 (.06)	.86 (.06)
Self-compassion (T1 covariate mean = 2.56)	2.70 (.06)	2.64 (.06)	2.49 (.06)
Quality of life (T1 covariate mean = 2.30)	1.93 (.08)	2.11 (.08)	2.06 (.08)
Disordered eating (T1 covariate mean = 3.34)	2.68 (.15)	3.02 (.15)	3.20 (.15)

*Note.* T1 = time 1; T2 = time 2. Imagery rescripting ( $n = 37$ ); cognitive dissonance ( $n = 35$ ); control ( $n = 35$ ). LSD adjustments.

**Table 6.3**

*Between-Group Effect Sizes (Cohen's  $d$  and 95% Confidence Intervals) at 1-Week Follow-Up (T2), Controlling for Baseline (T1) Variables*

Comparison	Body image acceptance	Negative affect	Self-compassion	Quality of life	Disordered eating
Imagery rescripting vs cognitive dissonance	<b>.49 (.03 to .96)</b>	-.28 (-.75 to .18)	.17 (-.29 to .63)	-.38 (-.85 to .09)	-.38 (-.85 to .08)
Imagery rescripting vs control	.45 (-.02 to .91)	-.02 (-.49 to .43)	<b>.59 (.12 to 1.06)</b>	-.27 (-.74 to .19)	<b>-.59 (-1.06 to -.11)</b>
Cognitive dissonance vs control	-.05 (-.52 to .42)	.26 (-.21 to .73)	.43 (-.05 to .90)	.11 (-.36 to .58)	-.21 (-.68 to .26)

*Note.* T1 = time 1; T2 = time 2. Imagery rescripting ( $n = 37$ ); cognitive dissonance ( $n = 35$ ); control ( $n = 35$ ). Significant values are in **bold**. Cohen's  $d$  .2 = small, .50 = medium, .80 = large (Cohen, 1988). LSD adjustments.

**Table 6.4***Testing Mediation Pathways at 1-Week Follow-Up (T2), Controlling for Baseline (T1) Variables*

Model: Mediation pathway for disordered eating as DV; condition (IR or CD/control) as IV	Model Summary					Indirect Effect		
	R square	<i>F</i>	df1	df2	<i>p</i>	Effect	Boot SE	Boot 95% CI
<i>Model 1 MV</i>								
T2 body image acceptance	.560	32.46	4	102	<.001	<b>.014</b>	<b>.006</b>	<b>.004, .027</b>
<i>Model 2 MV</i>								
T2 self-compassion	.489	24.38	4	102	<.001	<b>.009</b>	<b>.004</b>	<b>.002, .018</b>
<i>Model 3 MV (in parallel)</i>								
T2 body image acceptance & T2 self-compassion	.590	23.99	6	100	<.001	<b>.016</b>	<b>.006</b>	<b>.006, .030</b>

*Note.* T1 = time 1; T2 = time 2; DV = dependent variable; IV = independent variable; MV = mediator variable; df = degrees of freedom; Boot SE = bootstrap standard error; Boot 95% CI = bootstrap 95% confidence interval; IR = imagery rescripting; CD = cognitive dissonance. Imagery rescripting (*n* = 37); cognitive dissonance/control (*n* = 70). Significant indirect effects are in **bold**.

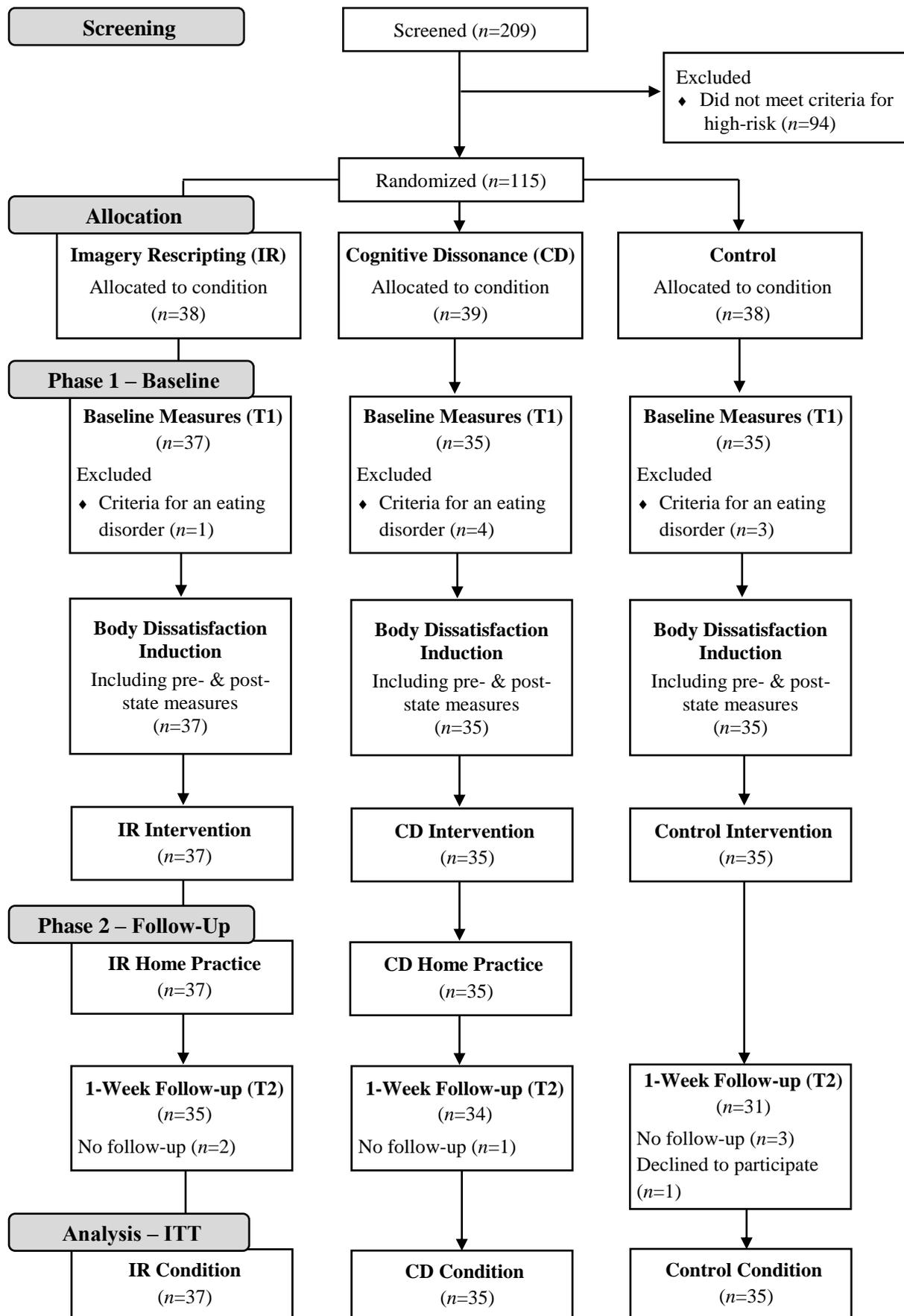
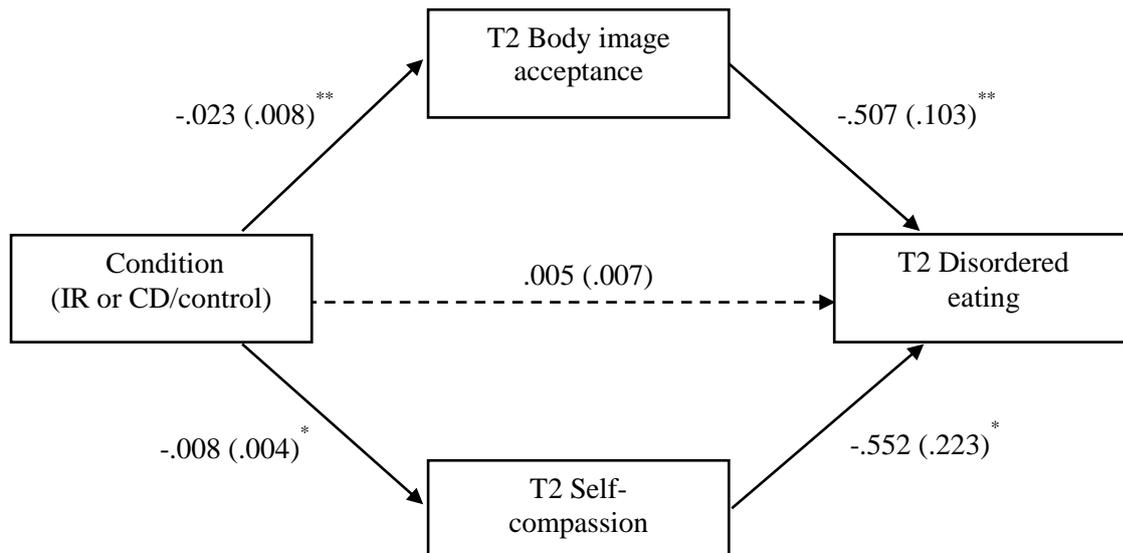


Figure 6.1 CONSORT flow diagram for study. T1 = time 1; T2 = time 2; ITT = intent-to-treat.



**Figure 6.2** Multiple mediation analysis with unstandardized path coefficients (and standard errors) and the estimates of the direct effect of condition (IR or CD/control) on disordered eating. Baseline (T1) variables were included as covariates. IR = imagery rescripting; CD = cognitive dissonance; T1 = time 1. Imagery rescripting ( $n = 37$ ); cognitive dissonance/control ( $n = 70$ ). Significant pathways are indicated with a solid line.  $*p < .05$ ;  $**p < .01$ .

## **Chapter 7**

### **General Discussion**

## **7.1 Overview**

The purpose of the following discussion is to integrate the findings from the three studies conducted in this thesis, thereby presenting a summary of its overall contribution to this field of research. Methodological limitations of the research are considered, theoretical and clinical implications are discussed, and directions for future research are suggested.

## **7.2 Summary of the Present Research**

In line with the current Medical Research Council (MRC; Craig et al., 2008) guidelines, it is vital that we work towards developing more effective approaches for eating disorders that integrate theory development, risk factor testing, and intervention evaluation in order to significantly reduce the impact of eating disorders. With this overall aim in mind, this research aimed to identify variables implicated in the development and/or maintenance of disordered eating, investigate theoretical models to identify key variables to include in future interventions, and investigate interventions for disordered eating to identify those variables that appear to be most effective for change within prevention research. Such insights are essential to enhance the effectiveness of extant prevention programs, and inform the development of future effective interventions, and may help to advance public health approaches to eating disorder prevention.

A systematic review of the existing eating disorder literature identified that while an extensive range of theoretical models describing the development and/or maintenance of disordered eating exist, very few models led to the development of effective interventions. Most notably, the vast majority of theories that have been developed have not progressed beyond the initial stages (i.e., identifying the relevant risk factors, developing the theory or model, and modelling associations between these factors and the outcome as delineated in the theory). The recommendations provided in the systematic review for bridging the gap between the initiation of theories for disordered eating and the development of interventions,

with the aim of informing the development of more effective interventions, subsequently guided the development of the remaining empirical studies for this thesis. Accordingly, the present research not only contributes insights into the specific variables considered to be important in the aetiology of disordered eating pathology, including those that appear to be most promising for change, it also contributes to the development of future more effective approaches to eating disorder prevention in order to achieve significant impact.

### **7.3 Integration of Key Findings with Theoretical and Clinical Implications**

#### **7.3.1 Identifying variables implicated in the development and/or maintenance of disordered eating.**

A review of the literature identified ten robust eating disorder models that have been used to inform interventions such as prevention and treatment (Augustus-Horvath & Tylka, 2011; Cooper et al., 2009; Fairburn, 2008a; Lyon et al., 1997; Neumark-Sztainer et al., 2003; Stice, 2001; Treasure & Schmidt, 2013; Wildes et al., 2010; Wilfley et al., 2000; Yamamiya et al., 2008). A number of variables emerged as common risk factors across these models for the development of disordered eating, namely preoccupation with weight and shape, emotion regulation difficulties, self-esteem deficits, interpersonal issues, and negative affect; suggesting that these variables are considered important to the development and maintenance of disordered eating, and should be considered in any future model development. Consistent with previous risk factor research (Dakanalis et al., 2017; Jacobi & Fittig, 2010; Stice, 2016), preoccupation with weight and shape including body dissatisfaction, and negative affect are two of the most reliable risk factors and contributors to the development of disordered eating pathology. Further, longitudinal researchers have identified body dissatisfaction as a precursor for negative affect (i.e., shame, anxiety, and sadness), given the importance of appearance in western culture, which further increases the risk of disordered eating (Jacobi, Hayward, et al., 2004; Stice, 2001, 2016). In support, prospective research of the dual-

pathway model found that perceived sociocultural pressure to be thin promotes both thin-ideal internalisation and body dissatisfaction, which promotes dieting and negative affect, which in turn results in bulimic pathology (Stice, 2001).

A body of literature now supports the relationship between difficulties with emotion regulation and eating pathology (Gianini et al., 2013; Svaldi et al., 2012). It is suggested that individuals with eating disorders display deficits in emotion regulation and lack the skills required to adaptively and effectively cope with adverse emotional states, and therefore may rely more on dysfunctional coping methods such as binge eating and/or purging behaviour, as a way of coping with, or managing, difficult emotions (Stice, 1994).

Research has also suggested that self-esteem is important in the aetiology and treatment of bulimia nervosa (Gordon et al., 2005). It is thought that low self-esteem makes women more vulnerable to external social pressures and more likely to try to achieve the thin body ideal in order to feel more effective (Fairburn, Wilson, et al., 1993). Further, it is suggested that women with low self-esteem may be more susceptible to self-sabotaging bulimic behaviour (i.e., binge eating) because of their negative expectations for themselves (Vohs et al., 1999). Self-esteem has also been investigated as a prognostic indicator in the treatment of bulimia nervosa (Fairburn et al., 1987); it is suggested that low self-esteem is associated with less positive treatment outcomes. Recently, it has been suggested that self-efficacy (i.e., a dimension of global self-esteem) is a more relevant component of the construct of interest (i.e., doubt about one's ability to achieve; Gordon et al., 2005), and thus self-efficacy is also considered important in this context.

There is also research to support the role of interpersonal factors in the development of binge eating (Wilfley et al., 2000). It is proposed that difficulties with sociocultural functioning contributes to low self-esteem and negative affect, which in turn causes binge eating in an attempt to cope with strong negative emotions (Wilfley et al., 2000). Further, the

role of family or relationships with close others has been investigated as a maintaining factor in the treatment of anorexia nervosa (e.g., the Maudsley model of anorexia nervosa treatment for adults [MANTRA]; Schmidt et al., 2014).

Other important variables have also been identified, namely thin-ideal internalisation, external pressure (i.e., pressure to be thin, pressure to diet, and sociocultural influences), perfectionism, cognitive factors (i.e., negative self-belief, negative automatic thoughts, and pro-anorectic beliefs), and dietary restraint. While these variables were referred to less frequently across the existing models in the current review, a moderate degree of evidence also exists to support these risk factors for the development and/or maintenance of disordered eating (see Culbert, Racine, & Klump, 2015; Dakanalis et al., 2017; Stice, 2002, 2016 for reviews).

Together, the present findings suggest the range of variables identified by the review are likely to be highly influential risk factors, and their inclusion within comprehensive aetiological models for disordered eating may be fundamental in informing the development of future effective interventions for disordered eating.

### **7.3.2 Emotional regulation: A key variable that ought to be considered in future interventions.**

The findings of the current research highlight the role of difficulties with emotion regulation, which is most often ignored in the development of prevention strategies for disordered eating. Emotion regulation difficulties are characterised by lack of emotional awareness, alexithymia, emotional avoidance, and heightened emotional vulnerability and reactivity (Bydlowski et al., 2005; Fruzzetti et al., 2008; Harrison, Sullivan, Tchanturia, & Treasure, 2009; Haynos & Fruzzetti, 2011). Several studies examining problems with emotion regulation have found that individuals with anorexia nervosa and bulimia nervosa

report more difficulties with emotion regulation compared to healthy controls (Gilboa-Schechtman, Avnon, Zubery, & Jeczmierni, 2006; Harrison et al., 2009; Harrison, Sullivan, Tchanturia, & Treasure, 2010; Svaldi et al., 2012).

It is suggested that women who lack adaptive emotion regulation strategies, including the ability to identify and appropriately cope with adverse emotional states, are more vulnerable to urges to binge (i.e., triggered by negative affect and/or restrictive eating behaviours), and are more likely to engage in dysfunctional or problematic coping methods, such as binge eating, as a way of regulating emotions (Gianini et al., 2013; Sim & Zeman, 2006; Whiteside et al., 2007). In this way, negative emotionality increases risk of disordered eating by interacting with, and amplifying the effects of, other risk factors i.e., negative affect (Stice, 2002). These findings are in line with what one would expect in a negative affect regulation model of disordered eating (see Stice, Agras, et al., 2001; Whiteside et al., 2007 for overviews), where binge eating is used as a coping mechanism (i.e., albeit dysfunctional) for short-term relief or escape from painful or distressing emotions (Heatherton & Baumeister, 1991; Polivy & Herman, 1993). While negative affect is already considered within the dual-pathway model, the specific contribution of difficulties with emotion regulation is yet to be investigated. In the complete model, Stice and Agras (1998) posit that personality traits such as self-esteem and inadequate coping contribute to the onset of bulimic pathology, however these variables have yet to be examined empirically within the model. In the present research, negative affect did not explain unique variance disordered eating after accounting for emotion regulation difficulties. This supports existing research indicating that difficulties with emotion regulation explains unique variance in binge eating and general eating pathology beyond the contribution of other factors included in the model such as negative affect (Gianini et al., 2013; Whiteside et al., 2007). This insight offers an important clarification of the aspects of negative affect that need to be targeted in future interventions,

and further suggests that emotion regulation difficulties may help to improve the models predictive ability.

While the dual-pathway model has resulted in the development of the gold standard of prevention approaches for eating disorders among at risk young women (e.g., cognitive dissonance-based intervention [DBI]; Stice, Rohde, et al., 2009), the present findings suggest that the role of temperament factors, in particular emotion regulation strategies or emotion-focused coping skills, should also be considered in the development of future interventions. For example, programs could teach women more adaptive ways of dealing with strong negative emotions, including strategies aimed at reducing or tolerating emotional arousal such as exposure and response prevention, behavioural activation, acceptance, self-validation, or other self-management strategies. Such emotion regulation strategies are shown to be more effective than cognitive strategies in the context of extreme emotional arousal (Fruzzetti et al., 2008). There is considerable evidence supporting the use of such emotion regulation strategies in the treatment of eating disorders, in particular dialectical behaviour therapy (DBT; Linehan, 1993), where the central focus is emotion regulation. While typically applied to individuals with borderline personality disorder or other severely dysfunctional behaviours such as suicidality (see Feigenbaum, 2007; Robins & Chapman, 2004 for reviews), DBT has also shown to be effective in the treatment of bulimia nervosa and binge eating disorder (Chen, Matthews, Allen, Kuo, & Linehan, 2008; Safer et al., 2001; Telch et al., 2001). Recently, a focus on emotion regulation has been added to cognitive behaviour therapy (CBT) for eating disorders (e.g., Fairburn, 2008a), where dysfunctional responses to adverse events or negative moods, such as binge eating, are posited as a maintaining factor; in line with affect regulation models (Heatherton & Baumeister, 1991; Polivy & Herman, 1993). Further, research in the prevention field has suggested that emotional acceptance-based approaches such as mindfulness may be useful in reducing disordered eating risk (e.g.,

Atkinson & Wade, 2012, 2015). For example, Atkinson and Wade (2012) found that mindfulness-based acceptance training reduced negative affect and increased weight and appearance satisfaction in individuals at risk of emotion regulation difficulties and disordered eating. Future prevention efforts would benefit from further exploration of the use of emotion regulation strategies to reduce the risk of disordered eating.

In addition, the longitudinal study in this thesis found that negative affect and self-compassion were also implicated in the development of disordered eating psychopathology over time. A mediational pathway showed that lower levels of self-compassion contributed to higher negative affect, which in turn contributed to a greater increase in disordered eating. While negative affect has been established as a risk factor for the onset of disordered eating (Dakanalis et al., 2017; Jacobi & Fittig, 2010; Stice, 2016; Stice, Gau, Rohde, & Shaw, 2017), this finding suggests that self-compassion may also be involved. However, it should be noted that the longitudinal study had significantly high attrition, with only 34 participants (20.4%) completing all three waves of data collection, and further the data were not missing at random, which limits the conclusions that can be drawn. While these findings suggest a promising line of enquiry for the role of self-compassion, further research is needed to better understand these effects over time.

Nonetheless, the present results did find some support for variables considered in the dual-pathway model, namely sociocultural and body dissatisfaction, as well as additional variables related to temperament, namely difficulties with emotion regulation. Together, these findings suggest that each of these variables play a key role in the development of disordered eating pathology and are therefore fundamental to the development of future effective interventions for disordered eating. Furthermore, these findings suggest that factors related to difficulties with emotion regulation should also be considered in future testing of the dual-pathway model. Further, while this research adds to the existing literature examining

the dual-pathway model, few independent research teams have replicated the model in its entirety longitudinally (e.g., Engler et al., 2006), and given the recent discussion about the importance of reproducibility and replication in psychological science (Open Science Collaboration, 2015), it is essential that future researchers address this gap in order to inform the development of future effective interventions for disordered eating.

The focus on emotion regulation subsequently resulted in the final study, a direct comparison between a more cognitive approach to reducing disordered eating (DBI) and a more emotionally-based approach, imagery rescripting. Self-compassion and body image acceptance, and also to a lesser extent negative affect, were identified as important factors for change. Change in state variables were examined over the experimental session and both imagery rescripting and cognitive dissonance were associated with increases in self-compassion compared to the control condition. This picture changed somewhat with the investigation of trait variables over the one-week follow-up. In this study, imagery rescripting was associated with increases in body image acceptance compared to the cognitive dissonance condition, as well as increases in self-compassion and decreases in disordered eating compared to the control condition, at follow-up. There was no significant impact of cognitive dissonance on any trait variables in this study.

These findings add to the growing research suggesting that self-compassion may be a protective factor against poor body image and eating disorder pathology (Braun et al., 2016; Goss & Allan, 2014; Kelly & Carter, 2015; Kelly et al., 2016; Steindl et al., 2017; Tylka & Kroon Van Diest, 2015). This follows increasing interest in the prevention field toward identifying protective factors that have the potential to prevent or ameliorate eating disorder risk factors (Levine & Smolak, 2016; Tylka & Kroon Van Diest, 2015). It is thought that protective factors can interrupt the processes by which risk factors contribute to the development of disordered eating (e.g., Levine & Smolak, 2016). In a recent meta analysis,

Braun et al. (2016) suggested that self-compassion may protect against eating pathology through multiple pathways, including reducing eating disorder-related outcomes directly, preventing the initial development of risk factors, interacting with risk factors to interrupt (i.e., moderate) their deleterious effects, and disrupting the mediational chain through which risk factors operate (Tylka & Kroon Van Diest, 2015). In support, Tylka et al. (2015) found that self-compassion buffered the relationships between perceived pressure to be thin and eating disorder-related outcomes i.e., thin-ideal internalisation and disordered eating. Further, in treatment studies of individuals with eating disorders, self-compassion has been associated with better patient outcomes; Kelly, Carter, and Borairi (2014) identified that greater increases in self-compassion early in treatment resulted in faster decreases in eating disorder symptoms such as shame over 12 weeks.

In addition, this research also supports the role of body image acceptance (or body image flexibility) as a protective factor for disordered eating. This is in line with a greater emphasis on psychological flexibility and resilience to promote healthy psychological functioning (e.g., Sandoz et al., 2013), and also agrees with the emerging literature on protective factors (e.g., Gillen, 2015; Piran, 2015; Tylka, 2011a), which posits that appreciation for, protection and acceptance of, and connection with, one's physical body, is associated with important mental and physical health-related indicators including eating behaviour (Hahn Oh et al., 2012). A number of acceptance and mindfulness-based approaches to eating disorders include a focus on this kind of flexibility i.e., building awareness and openness to experience while engaging in more intentional, constructive behaviours, in particular acceptance and commitment therapy (ACT; Sandoz, Wilson, & DuFrene, 2011). Preliminary research has shown ACT to be effective in reducing disordered eating (Berman, Boutelle, & Crow, 2009; Juarascio, Forman, & Herbert, 2010), and improving treatment outcomes such as quality of life and psychological distress (Lillis,

Hayes, Bunting, & Masuda, 2009). Further, prevention research has suggested that mindfulness-based acceptance approaches may be useful in reducing eating disorder symptoms and risk factors for eating disorders including weight and shape concern, dietary restraint, thin-ideal internalisation, and negative affect (e.g., Atkinson & Wade, 2012, 2015). While this research suggests that self-compassion and body image acceptance may play a role in influencing change in disordered eating as potential protective factors, future research would benefit from further investigation of these factors in the prevention of disordered eating. Further, this research suggests that future prevention approaches should consider the use of acceptance and mindfulness strategies to reduce the risk of disordered eating.

A major contribution of this research was to provide a direct comparison of strategies that may be useful in modifying risk and protective factors for eating disorders, namely body dissatisfaction, negative affect, self-compassion, and body image acceptance, with the aim of informing the development of future prevention approaches. The present research provides some support for both imagery rescripting and cognitive dissonance interventions in increasing self-compassion, and further for imagery rescripting in increasing body image acceptance, in the short term. While support was also found for the imagery rescripting intervention in reducing disordered eating, this was not significantly different to the cognitive dissonance condition. It should be noted that the present study only examined brief interventions, which can only indicate approaches that are worthy of further research in terms of developing future effective prevention approaches. Future research is required to examine the longer term impact of using such techniques before absolute conclusions can be made about their clinical usefulness. Nonetheless, these findings provide preliminary support for the use of these strategies and suggest that further research is warranted. It would also be valuable to examine whether increased exposure to these techniques, including lengthier training and practice, may produce larger, or different, effects.

These findings add to the early literature suggesting that imagery-based techniques such as imagery rescripting may be useful adjuncts in interventions for eating disorders, in particular CBT (Tatham, 2011). While studies have shown that imagery techniques are effective in the treatment of bulimia nervosa (Cooper et al., 2007; Ohanian, 2002) and may play a role in treating body image disturbance in obesity and binge eating disorder (Cesa et al., 2013; Riva, 2011), the present findings are the first to provide preliminary evidence to support the use of imagery rescripting in the context of reducing risk factors and increasing protective factors for disordered eating. While there is a considerable support for the effectiveness of DBIs such as cognitive dissonance for reducing body dissatisfaction and body image problems in young women (e.g., Stice, Rohde, et al., 2009; Stice, Shaw, et al., 2007), the present findings suggest that imagery-based approaches may be more effective in some instances i.e., for increasing body image acceptance. However, it should be noted that only one aspect of DBIs was used in the present research, not the full intervention. It would be important for future researchers to compare a fuller version of an imagery rescripting intervention to the gold standard 4-session DBIs (e.g., Stice, Rohde, et al., 2009; Stice, Shaw, et al., 2007). Nonetheless, the present research suggests that future prevention research would benefit from further exploration of the use of imagery in the prevention of disordered eating, particularly as an adjunct to standard evidence-based interventions.

The present research adds to the research suggesting that imagery-based approaches have advantages over more verbal-based approaches that are utilised in CBT (Cooper, 2011; Somerville & Cooper, 2007; Tatham, 2011). It has been proposed that relative to verbal representation (i.e., use of thoughts and words), imagery has an especially strong relationship with emotion and therefore can provide greater impact on emotional states in psychological disorders (Holmes & Mathews, 2005). For example, research has demonstrated that use of a mental image compared to use of a verbal sentence produced more powerful emotional

responses, despite using the same material (Holmes et al., 2008). To further this, it has been suggested that compared to cognitive approaches, imagery techniques may be better able to evoke, and therefore modify, more emotionally-held core beliefs (Somerville & Cooper, 2007). For example, Somerville and Cooper (2007) demonstrated that negative core beliefs have stronger emotional compared to rational bases, which may help to explain why core beliefs tend to be more resistant to change using traditional CBT methods. Several researchers have indicated that imagery rescripting of early negative or traumatic memories may help to modify negative core beliefs of relevance to eating disorders such as self-criticism and shame (Cooper, 2011; Hinrichsen et al., 2007; Mountford & Waller, 2006; Somerville & Cooper, 2007; Somerville, Cooper, & Hackmann, 2007). For example, in a single-case report investigation of imagery rescripting in conjunction with CBT (Ohanian, 2002), one session of imagery rescripting resulted in further reductions in binge-purge behaviours when conventional CBT had been unsuccessful. Further, in an experimental study by Cooper et al. (2007), a single session imagery intervention was more effective in reducing emotionally-held core beliefs and associated mood and eating disorder symptoms compared to a matched control group. While the present research provides preliminary support for the use of imagery in the context of reducing disordered eating, future research would benefit from exploring the mechanisms of action and relationship to negative core beliefs. Further, while these findings suggest that imagery rescripting reduces risk for disordered eating by strengthening protective factors such as self-compassion and body image acceptance, future exploration of the use of such imagery-based strategies in the prevention of eating disorders is necessary in order to inform the development of future interventions.

#### **7.4 Methodological Considerations**

An important limitation of this research was the nature of the sample population investigated. This research was conducted exclusively with female undergraduates aged

between 17 and 28 years, it is therefore unclear whether the present findings would generalize to other populations, including males, younger or older women, or the wider community. This population was selected for two reasons. First, there is a wealth of evidence to suggest that eating disorders, including threshold and subthreshold disordered eating, predominantly affect women (Hudson et al., 2007; Liechty & Lee, 2013). Second, research has shown that early adulthood increases risk for the emergence of new eating pathology (Allen et al., 2013; Favaro et al., 2009; Kessler et al., 2013; Stice, Marti, et al., 2013). While this population was considered to be at greater risk of developing disordered eating, it is not necessarily representative of the range of people who may also be at risk of eating disturbances, including males. Recently, there has been increasing awareness of disordered eating among men (see Dakanalis & Riva, 2013; Murray et al., 2017; Tylka, 2011b for reviews), with estimates suggesting that males account for approximately one third of adults reporting disordered eating (Mitchison et al., 2013). In addition, several studies of specialist eating disorder clinics (e.g., Madden, Morris, Zurynski, Kohn, & Elliot, 2009; Nicholls, Lynn, & Viner, 2011) have revealed that males represent between 25% and 33% of preadolescent eating disorders in Australia and the United Kingdom. On a related note, eating pathology can occur across the age spectrum (e.g., Hay et al., 2008). Estimates suggest that eating disorders typically emerge in mid-late adolescence (Doyle, Smyth, & le Grange, 2012; Stice, Marti, Shaw, & Jaconis, 2009), but can occur earlier (Madden, 2012; Madden et al., 2009); with one study (Madden et al., 2009) identifying early-onset eating disorders in children as young as 5 years of age. The prevalence of disordered eating has also been noted in middle age and older (Ackard, Richter, Frisch, Mangham, & Cronemeyer, 2013; Hay et al., 2008; Reas & Stedal, 2015), with up to 11% of women (Fairweather-Schmidt et al., 2015) and 7% of men (Mangweth-Matzek, Kummer, & Pope, 2016) aged 40 years and over self-reporting disordered eating. Thus, it is vital that future researchers also examine risk factors

for disordered eating among males, younger/older women, and community samples in order to further improve the development of approaches to future interventions for these groups.

This research relied solely on the use of self-report data, which should be noted as a potential limitation. In addition, disordered eating was measured using self-report items from the Eating Disorder Examination–Questionnaire (EDE-Q; Fairburn & Beglin, 1994) While the EDE-Q is a standardised measure of eating disturbances with acceptable reliability and validity (Berg et al., 2011, 2012; Luce & Crowther, 1999), and has the ability to distinguish between eating disorder and non-eating disorder cases (Mond et al., 2004), the use of structured clinical interviews such as the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993) may further improve predictive validity. While the use of confidential self-report procedures may encourage honest responding, particularly for sensitive or potentially shameful behaviours such as self-induced vomiting, laxative or diuretic use, or eating furtively (Lavender & Anderson, 2009), the use of interview data may increase the strength of findings. Studies have found consistently higher rates of eating pathology reported via self-report questionnaires versus interview formats (e.g., Keel, Crow, Davis, & Mitchell, 2002; Perry et al., 2002); with several theories proposed to explain this inconsistency e.g., complex or ill-defined symptoms, social desirability, or shame (Anderson, Simmons, Milnes, & Earleywine, 2007; Fairburn & Beglin, 1994). Further, the use of multiple-reporter data such as peer or family member reports, and multiple methods of measurement such as interviews, observation, or frequency records, may also be useful in this context.

While this research targets a number of theoretically indicated variables for the development and/or maintenance of disordered eating, there are a number of variables that are not considered within the present research for which there is some empirical support, including interpersonal functioning, self-surveillance, self-objectification, social comparison, genetics, and environmental and developmental factors (see Culbert et al., 2015;

Fitzsimmons-Craft, 2011; Pennesi & Wade, 2016; Stice, Gau, et al., 2017; Trace, Baker, Peñas-Lledó, & Bulik, 2013; Wang et al., 2013 for reviews). While it was beyond the scope of the current research to investigate every known eating disorder or disordered eating risk factor, future researchers should consider investigating these additional risk factors within the development or revision of eating disorder models and/or interventions for disordered eating.

## **7.5 Summary and Conclusions**

This thesis presents an important framework linking model development, testing of putative risk factors, and evaluation of interventions that can inform the development of more effective interventions for eating disorders and disordered eating. In line with the current best practice guidelines (MRC; Craig et al., 2008), the present research identified a number of key variables of pertinence to the development and/or maintenance of disordered eating that ought to be considered in the development or revision of models that can inform the development of future interventions, including those which appear to be most effective for change within prevention research. In particular, factors that can lead to more effective emotion regulation may be key in seeking improved outcomes.

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## **APPENDIX A**

### **Quality Rating Scheme**

## Quality Rating Scheme

### Adherence to Cognitive Dissonance Intervention

Note. The following applies to the cognitive dissonance intervention instructions presented to participants during the baseline intervention exercise and at each home practice exercise.

**1a) Cost/Consequences.** The response details the *costs and/or consequences of pursuing the thin-ideal* (i.e., the thin-ideal stereotype for women as portrayed in the media). Where this is done well, the response includes details of several costs and/or consequences of pursuing the thin-ideal. An example of an excellent response (a rating of 3) is: *“Body issues. Forcing yourself to throw up to lose weight. Constantly feeling like you need to go to the gym. Constantly feeling like you need to diet. Constantly feeling like you aren’t pretty enough. Constantly feeling like there is someone who is always better than you. Feeling generally ugly about your size and weight. Feeling like you would be easily rejected due to your appearance.”*

0 = no mention of costs and/or consequences

1 = mentioned 1-2 costs and/or consequences but did not describe in detail

2 = mentioned 3 or more costs and/or consequences but did not describe in detail

3 = described 3 or more costs and/or consequences in detail

**1b) Positive Attributes.** The response details the *positive attributes about themselves* (i.e., positive attributes about the participant). Where this is done well, the response includes details of several positive attributes (e.g., physical, emotional, behavioural). An example of an excellent response (a rating of 3) is: *“I am welcoming and warm. Smart and good at writing. I’m getting better at understanding myself and trying to know what I need for my body and mind. Friendly. Creative. I can go outside of my comfort zone and try new things. Persistent. Dedicated. Resilient.”*

0 = no mention of positive attributes about self

1 = mentioned 1-2 positive attributes about self (i.e., not including those referring to appearance) but did not describe in detail

2 = mentioned 3 or more positive attributes about self (i.e., not including those referring to appearance) but did not describe in detail

3 = described 3 or more positive attributes about self (i.e., not including those referring to appearance) in detail

## Adherence to Imagery Rescripting Intervention

Note. The following applies to the imagery rescripting intervention (part 1) instructions presented to participants during the baseline intervention exercise only.

**2a) Personal Unpleasant Body Experience.** The response describes a personal *unpleasant body experience* from the past where the participant felt ashamed or embarrassed of their body or how their body looked. Where this is done well, the response is descriptive and includes details and emotions (e.g., where or when is the event taking place, what is taking place, who is present, how they might be the feeling [emotions], what they might be telling themselves [thoughts]). An example of an excellent response (a rating of 3) is: “*Steph is standing at the beach with a group of friends. Steph looks worried and anxious but her friends look like they are having fun. She hides behind that oversized t-shirt and is wrapped in an extra towel, she seems concerned about her body. It appears the thoughts she is having are making her overwhelmed and distressed and that this is the last place she would be comfortable.*”

0 = no mention of unpleasant body experience

1 = mentioned unpleasant body experience but didn't describe in detail

2 = described unpleasant body experience in detail but didn't mention emotions or thoughts

3 = described unpleasant body experience in detail and described emotions and/or thoughts

**2b) Observer's perspective.** The response describes a personal unpleasant body experience *from an observer's perspective* (i.e., as if the event were happening to their younger self right now). Where this response is done well, the response is written in the third person as an observer, as if their adult self is in the room observing what is happening to their younger self, watching the event's unfold and their adult self is telling the story. The response is descriptive and includes details about their younger self (e.g., where are they, what are they doing, who are they with, what might they be feeling [emotions], what might they be telling themselves [thoughts]). An example of an excellent response (a rating of 3) is: “*I see younger Paige in her bedroom staring at her own reflection in the mirror. She looks displeased with the way she looks and is trying to cover up her collarbones. She thinks it's wrong and they shouldn't be there.*”

0 = did not describe event from an observers' perspective (i.e., in the third person)

1 = described the event from an observer's perspective but did not mention themselves by name or describe details about their younger self

2 = described the event from an observer's perspective and mentioned themselves by name but did not describe details about their younger self

3 = described the event from an observer's perspective and mentioned themselves by name and described details about their younger self

Note. The following applies to the imagery rescripting intervention (part 2) instructions presented to participants during the baseline intervention exercise and at each home practice exercise.

**2c) Personal Unpleasant Body Experience.** See scoring criteria above for 2a. Note. This criterion is used when rating the imagery rescripting home practice exercise ONLY (this criterion is NOT used when rating the imagery rescripting part 2 baseline intervention exercise).

**2d) First/Third Person Language.** The response describes a personal unpleasant body and *both first and third person language is used*. Where this is done well, the response is written in the first person (e.g., “I am in the change room, I’m trying on a pair of blue jeans...”) unless they are referring to their adult self, in which case this is written in the third person (e.g., “*adult Ellie said*”, “*older Ellie approached me*”). An example of an excellent response (a rating of 3) is: “*I am getting ready to go out for one of my best friend’s birthdays. I am wearing a short black dress that belongs to my mum. I don’t like it because I feel fat in it and you can see the outline of my stomach in the dress. I feel out of my comfort zone and wish I could change. Older Ellie steps in while I am turning around in the mirror trying to see if my stomach disappears from another angle, and she tells me that there is nothing there and that you can’t see a thing, and tells me that it is only in my head and that I look beautiful.*”

0 = not written in the first person *or* the third person

1 = written in the first person, but not in the third person (i.e., did not refer to their adult self)

2 = written in the third person, but not in the first person (i.e., did not refer to “I”)

3 = written in the first person (i.e., when referring to the present) and written in the third person (i.e., when referring to their adult self)

**2e) Adult Self Present & Engaged.** The response describes a personal unpleasant body experience and *their adult (or older) self is with them and intervenes or does something in the situation that is right or helpful*. Where this is done well, the response is descriptive and makes explicit reference to their adult self in the room as the events unfold (e.g., reference to “*adult Sarah*”, “*older Sarah*”, “*compassionate Sarah*”) and describes details of their adult self intervening or doing something in the situation that is right or helpful (e.g., what is adult Sarah doing, what did adult Sarah say). An example of an excellent response (a rating of 3) is: “*I’m in the playground playing Cops and Robbers with my friends and classmates. One of the girls stops me and says she no longer wants me on her team because I am slower than the others and larger. I feel crushed. Older Sarah comes up to me. She says not to worry about what others think of me because I have no idea what the future holds. She tells me I’m going to be successful and I’m going to work hard and achieve my dreams.*”

0 = no mention of adult (or older) self present

1 = mentioned adult (or older) self present

2 = mentioned adult (or older) self present and described their adult self intervening or doing something in the situation

3 = mentioned adult (or older) self present and described their adult self intervening or doing something in the situation that is right or helpful (i.e., that may positively influence the situation)

**2f) Self-Compassion/Compassionate Language.** The response describes a personal unpleasant body experience and their *wiser and more compassionate adult self is with them and offers them compassion or provides them with new updated information based on what they know now as an adult*. Where this is done well, the response is descriptive and includes an array of examples of compassionate intervening statements and/or gestures, and the response overall has a strong sense of compassion/self-compassion in the language used. An example of an excellent response (a rating of 3) is: *“I was at home trying on some old clothes. I was with older Amy. As I was trying to out on some old shorts, I realised they were too small for me. I start to become upset and stressed. Older Amy said to me ‘getting bigger is simply a part of growing older, you are not becoming fatter but becoming more like a woman, you look great just the way you are now’.”*

0 = made no reference to intervening statements and/or gestures

1 = made reference to intervening statements and/or gestures, but these were not compassionate

1 = made reference to intervening statements and/or gestures that were compassionate, but the compassion/self-compassion is discounted and overall sense of the response is to minimize compassion/self-compassion

2 = made reference to intervening statements and/or gestures that were compassionate

3 = made reference to intervening statements and/or gestures that were compassionate, and there was a strong sense of compassion/self-compassion in the language used