# SELF-BODY RELATIONS: ATTACHMENT, MALADAPTIVE SCHEMAS, AND EMOTION REGULATION IN EATING DISTURBANCES

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#### Abstract

Self-concept deficits and difficulties with emotion have long been implicated in the development and maintenance of eating disorders (ED). This thesis aimed to integrate various emotion-regulation, self-regulation, and sociocultural models of ED pathology within an overarching framework of attachment theory. Such an integrated 'self/emotion-regulation model' of ED pathology posits that negative feelings about the self and unexpressed emotions are displaced onto the body and that ED pathology represents the maladaptive function of attempting to manage these painful internal experiences and to enhance self-worth via the body and achievement of idealised standards.

This integrated self/emotion-regulation model was explored in a series of four studies using two distinct heterogeneous samples of women. In accordance with emotion-regulation models of ED pathology, the first study explored the roles of various forms of emotional inhibition (negative beliefs about emotion, ambivalence over emotional expressiveness, silencing-the-self and divided-self schemas), in individually and uniquely explaining variance in ED pathology and examined the possible mediating and moderating role of negative emotionality in the associations between each form of emotional inhibition and ED pathology. The second study then investigated the direct and indirect roles of a number of emotion regulation strategies (reappraisal, suppression, rumination, reflection, and emotional reflection), negative beliefs about emotion, negative emotionality, and depressive symptoms in contributing to variance in ED pathology within path models.

In testing features of the integrated self/emotion-regulation model of ED pathology, the third study explored the role of feelings of low self-worth (internalised shame and low self-esteem), externalised self-perceptions (self-objectification and body surveillance), body shame, and depressive symptoms in directly and indirectly contributing to variance in ED pathology. Finally, the fourth study investigated the structural associations among insecure attachment styles (anxiety and avoidance), defectiveness/shame schemas, emotional inhibition,

unrelenting standards (perfectionism), body shame, depressive symptoms and ED pathology within a path model.

The results need to be interpreted with some caution due to a number of limitations, the most notable of which is the use of cross-sectional designs. Nevertheless, taken together the current findings provide preliminary support for an integrated self/emotion-regulation model of ED pathology grounded in attachment theory. Specifically, in support of emotionregulation models of ED pathology, findings suggest that various forms of emotion inhibition (in particular, negative beliefs about emotion), and other maladaptive emotion-regulation strategies serve to increase the experience of negative emotion, which in turn may trigger ED pathology as a means of alleviating this experience. In support of self-regulation models of ED pathology, findings suggest that negative feelings about the self (low self esteem, internalised shame) are externalised (via increased self-objectification and body surveillance) and displaced onto the body, thereby increasing body shame and giving rise to depressive symptoms and ED pathology. Finally, within a broad self/emotion-regulation model, findings suggest that insecure attachment styles contribute to negative feelings about the self (defectiveness/shame), emotional inhibition, and unrealistically high standards, which are externalised onto the body (in the form of body shame), and thereby contribute to depressive symptoms and ED pathology.

Such evidence highlights the need for therapeutic interventions for young women with EDs to also address underlying difficulties with insecure attachment, feelings of low selfworth, difficulties with emotion regulation and other maladaptive schemas in order to enhance the efficacy of current treatments and improve therapy outcomes.

**Certification by Candidate** 

I certify that the work in this thesis entitled "Self-Body Relations: Attachment,

Maladaptive Schemas, and Emotion Regulation in Eating Disturbances" has not

previously been submitted for a degree nor has it been submitted as part of requirements for a

degree to any other university or institution other than Macquarie University.

I also certify that the thesis is an original piece of research and it has been written by

me. Any help and assistance that I have received in my research work and the preparation of

the thesis itself has been appropriately acknowledged.

In addition I certify that all information sources and literature used are indicated in the

thesis.

The research presented in this thesis was approved by Macquarie University Ethics

Committee (Human Research) on 15 October 2012 (Reference No: 5201200615) and on 13

May 2013 (Reference No: 5201300137).

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# Chapter 1

**Overview of Research** 

#### **OVERVIEW**

Eating disorders (EDs) are serious mental illnesses that are notoriously difficult to treat. EDs include a group of specific disorders characterised by combinations of a range of symptoms. These include disturbances in eating (e.g., dietary restraint and binge eating); extreme weight-control behaviours (e.g., fasting, vomiting, laxative/diuretic abuse, and excessive exercise); disturbances in the experience of one's body in terms of weight, shape, and appearance; pursuit of an idealised body weight/shape; fear of gaining weight or becoming 'fat'; and undue influence of body weight or shape on self-evaluation (herein referred to as 'ED pathology'). While EDs are increasingly relevant to men (Strother, Lemberg, Stanford, & Turberville, 2012), they remain disproportionally experienced by women, with gender constituting one of the most potent and replicated risk factors for EDs (Jacobi & Fittig, 2010). Thus this thesis aimed to consider ED pathology experienced by women specifically, in order to attempt to further elucidate some of the processes contributing to and maintaining EDs in this majority population within whom EDs are overrepresented, whilst controlling for some of the maturational and gender influences on ED pathology.

Current lifetime prevalence estimates of anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED) in women have been found to be in the range of 0.9 to 2.2%, 1.5 to 2.9%, and 1.9 to 3.5%, respectively (Hudson, Hiripi, Pope, & Kessler, 2007; Smink, van Hoeken, & Hoek, 2012). Further, subclinical levels of ED pathology may be experienced by as many as 23% of young Australian women (Wade, Wilksch, & Lee, 2012) and only a minority of women with EDs are thought to seek treatment (Hoek, 2006). AN has also been identified as having one of the highest mortality rates of all psychological illnesses with findings revealing death rates of between 5.9% to 6.5% of participants across various samples (Franko et al., 2013; Sullivan, 1995). Additionally, current estimates of treatment success

reveal that cognitive-behavioural therapy (CBT), the most well supported treatment for EDs, is effective in treating less than 50% of cases (Waller et al., 2014; Zipfel et al., 2014).

Whilst EDs are classified under DSM-5 into categories, the most well-know of which are AN, BN, and BED, such categorical systems have not always been empirically supported (Wonderlich, Joiner, Keel, Williamson, & Crosby, 2007), with significant heterogeneity observed within categories and individuals frequently moving between diagnostic categories across time (Agras, Crow, Mitchell, Halmi, & Bryson, 2009). Further, many individuals with clinically relevant ED pathology fail to meet full-threshold diagnosis of AN, BN, or BED. Such individuals fall within the category of other specified feeding and eating disorder (OSFED) or unspecified feeding and eating disorder (UFED) in DSM-5 (formerly known as EDNOS in DSM-IV). Indeed EDNOS was previously the most prevalent ED diagnosis (J. J. Thomas, Vartanian, & Brownell, 2009). Furthermore, both sub-threshold EDs and even lower subclinical levels of ED pathology have been associated with the experience of significant distress and/or impairment (Rockert, Kaplan, & Olmsted, 2007; Stice, Marti, & Rohde, 2013; Stice, Marti, Shaw, & Jaconis, 2009) and thus worthy of investigation. As such a dimensional approach to measurement of ED pathology has been advocated for as preferential to a categorical approach (Lavender, Crosby, & Wonderlich, 2013) and is the approach that will be taken in the current thesis.

Given the seriousness and complexity of these disorders and the unsatisfactory outcomes of current treatments, considerable research energy has been focused on identifying risk and predisposing factors that may contribute to the development and maintenance of ED pathology. Whilst there is considerable variation across reviews, some of the potential risk factors that have been identified in the literature include female gender, birth-related perinatal/pregnancy complications, sociocultural pressure to be thin, dieting, negative self-evaluation, psychiatric morbidity, the experience of sexual abuse or physical neglect during

childhood, perceived low social support from family, low self-esteem, perfectionism, and an escape-avoidant style of coping with stressful events (Jacobi & Fittig, 2010; Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Stice, 2002). However, numerous difficulties have been identified with the replication of specific risk factors across studies and significant limitations exist within the literature regarding the large proportion of cross-sectional studies versus good quality longitudinal studies truly capable of identifying aetiological risk factors. Nevertheless, despite such limitations, the two strongest and most reproducible risk factors that emerge for EDs are concerns about body weight and shape and negative emotionality (Jacobi & Fittig, 2010; Jacobi et al., 2004).

Whilst a broad range of models of EDs exist in the literature, the most extensively researched and accepted is that of the transdiagnostic maintenance model of EDs (Fairburn, 2008; Fairburn, Cooper, & Shafran, 2003), which is derived from cognitive behaviour theory and lead to the development of an enhanced cognitive-behavioural therapy for eating disorders (CBT-E). This model in its original form proposed a maintenance cycle of the symptoms of BN whereby the over-evaluation of eating, shape and weight and their control (the core ED cognition) is proposed to lead to strict dieting and other weight-control behaviours, to binge eating, and to compensatory vomiting/laxative use, which in turn leads to increased binge eating, then to strict dieting and other weight control behaviours, and increased over-evaluation of eating, shape and weight and their control (Fairburn et al., 2003). This model and CBT-E have since been adapted to suit all EDs (transdiagnostic approach) and its most recent iteration to include the additional maintaining factors of: dietary restraint, mood intolerance, clinical perfectionism, core low self-esteem, and interpersonal problems (Fairburn et al., 2003). The transdiagnostic maintenance model has been highly beneficial in informing the development of CBT-E, the treatment of choice for EDs where there is a minimum body mass index (BMI) of 17.5. However, as mentioned above CBT-E is effective

in treating less than 50% of cases, and even less with regard to EDs where there is a BMI less than 17.5 (Waller et al., 2014; Zipfel et al., 2014). Furthermore, this model is a maintenance model of EDs that does not seek to explain the development of ED pathology (although developmental and maintenance factors may overlap), or the development of predisposing factors that may contribute to the development and maintenance of EDs.

In addition to or underlying these maintaining factors, and given the potent risk factor of negative emotionality, other important contributors to the development and maintenance of ED pathology may be disturbances in development of the sense of self (e.g., Amianto, Northoff, Abbate Daga, Fassino, & Tasca, 2016) and disturbances in emotional development generally, and emotion-regulation capacities specifically (e.g., Haynos & Fruzzetti, 2011). Whilst in recent years there has been a return to recognising the role of emotional difficulties in EDs, this has been a largely overlooked area in ED research over the past several decades (Fox & Goss, 2012); despite the identification by Bruch (1978) in her seminal early writings on AN of the apparent difficulties with emotional processing observed in individuals with AN. It is proposed in this thesis that understanding the importance of emotions and emotional functioning in EDs and addressing underlying difficulties with sense of self and emotion-regulation in ED treatment may provide possible avenues to enhance the efficacy of current treatment outcomes for EDs and promote long-term recovery from ED pathology.

Thus this thesis aims to enhance understanding of the newly recognised maintaining factors within the transdiagnostic maintenance model (namely, mood intolerance, clinical perfectionism, core low self-esteem, and interpersonal problems) by framing these factors within a developmental model grounded in theory and by attempting to explore some of the processes by which these factors maintain ED pathology. As such, the current thesis focused on exploring the role of various forms of emotion-regulation, negative beliefs about emotion, and negative emotionality in accounting for variance in ED pathology. The research further

explored the role of insecure attachment styles, negative feelings about the self and body, externalised self-perceptions, and various maladaptive schemas (perfectionism, emotion inhibition, internal shame), in contributing to ED pathology. Finally, the current thesis sought to combine such self and emotion regulation variables within an integrated overarching model of ED pathology, grounded in attachment theory.

# **Theoretical Background**

Eating disorders have long been linked with difficulties in the processing and regulation of emotion (Bruch, 1973, 1978; Fox & Goss, 2012) and women who suffer from EDs have been found to experience higher levels of negative emotion generally than women without EDs (Kitsantas, Gilligan, & Kamata, 2003). Emotion-regulation models of EDs propose that ED pathology serves the maladaptive emotion-regulation function of 'distracting from', 'blocking', 'inhibiting', or 'escaping from awareness of' the experience and expression of negative emotion (Bekker & Spoor, 2010; Elmore & de Castro, 1990; Heatherton & Baumeister, 1991), and thus constitute avoidant coping strategies (Neckowitz & Morrison, 1991; Troop, Holbrey, Trowler, & Treasure, 1994).

In addition to being viewed as disorders of emotion regulation, EDs have been viewed as disorders of the self more generally (Goodsitt, 1983, 1997; Taylor, Bagby, & Parker, 1997). Early on Bruch (1962, 1978) implicated disturbances in the development of self-definitions and an impoverished sense of self in the development of AN. Similarly BN has been suggested to result from deficits in a clearly defined sense of self, where the body is used to represent inner identity and preoccupation with physical appearance is a concrete solution to the absence of an authentic sense of self (Schupak-Neuberg & Nemeroff, 1993). Research evidence supports the hypothesis that self-concept disturbances underlie both AN and BN (Stein, 1996, 2001; Stein & Corte, 2003, 2007, 2008).

The fundamental principle underlying this view of EDs as disorders of the self is that ED symptoms are maladaptive means of managing the painful internal experiences emanating from deficits in the self (Amianto et al., 2016). Specifically, Bruch (1973, 1978) proposed a displacement theory of emotion in EDs, which argues that unexpressed or undifferentiated emotions and negative feelings about the self are displaced onto the body in the form of 'feeling fat'. Consistent with other emotion-regulation models, displacement theory suggests that ED symptoms serve the function of attempting to avoid or reduce feelings of shame and represent attempts to enhance feelings of self worth and identity via the body.

From a developmental perspective, attachment theory (Bowlby, 1969, 1973, 1980) provides a framework for understanding both deficits in development of self and maladaptive emotion-regulation capacities that predispose women to ED pathology. Attachment theory implicates specific interpersonal processes in the development of intrapersonal characteristics, such as sense of self (Feeney, 2006). The theory specifies that deficiencies in adequate caregiver affective attunement and availability and responsiveness in early attachment relationships, particularly in relation to co-regulation of negative emotion, can result in development of maladaptive emotion-processing systems and the internalisation of negative internal working models (IWM) of the self and/or others. These representations are carried into the future, thereby shaping the individual's feeling about self and others, particularly in the context of close relationships (Bowlby, 1988; Feeney, 2006).

Likewise, difficulties with affective attunement in early infant-caregiver relationships have been implicated in the development of internalised shame (Cook, 1991; Goss & Allan, 2009; Hahn, 2000), which is characteristic of those with negative IWMs of self (Luke, Maio, & Carnelley, 2004). Environments throughout childhood and adolescence that fail to meet the emotional needs of the individual (including attachment needs) are implicated in the development of early maladaptive schemas (EMS) within schema therapy, a therapy based in

part on attachment theory (Young, Klosko, & Weishaar, 2003). Finally, invalidating childhood environments are also implicated in the development of negative beliefs about emotions and difficulties with emotion regulation (Corstorphine, 2006) – all factors implicated in the development and maintenance of ED pathology.

Another theory that implicates deficits in sense of self in the formation and maintenance of ED pathology, also grounded in attachment theory, is self-silencing theory (Jack, 1991). This sociocultural theory provides an account of how women may develop relational-schemas (externalised self-perception, care as self-sacrifice, self-silencing, and divided self) that lead to the inhibition of authentic emotion, thoughts, and behaviour in an attempt to preserve relationships. Women's sense of self is said to be relationally embedded and suppression of parts of the authentic self in the service of relationships is said to be an indicator of attachment insecurity and lead to a loss of voice, loss of self, lowering of self-worth, to further preoccupation with attachment, and to depression (Jack, 1991) and ED pathology (Geller, Srikameswaran, & Cassin, 2010).

#### **Summary**

Exploration of the role of emotion and sense of self in ED pathology are neglected areas when compared with the abundance of ED research that has focused on the treatment and understanding of ED behaviours, thoughts, and their physical consequences. Indeed, it has been suggested that such neglect echoes the emotional avoidance and attachment difficulties characteristic of those who experience ED pathology (Treasure, 2012). Emotion-regulation models of ED pathology propose that ED symptoms function as maladaptive means of regulating, avoiding, or inhibiting emotions and as an escape from the distress of painful self-awareness. Concurrently, self-regulation models, such as displacement theory of EDs, propose that unexpressed emotions and negative feelings about the self are displaced onto the body and control of body weight, shape, and food in the pursuit of an internalised

"thin-ideal" thereby become the focus of identity and self-worth (core ED cognitions) and drive ED behaviours (e.g., dietary restriction/control, binge eating, self-induced vomiting, excessive driven-exercise, and other compensatory behaviours). This thesis argues that emotion-regulation and self-regulation models of EDs may be seen to be congruent and symbiotic, rather than contrary, and that both fit within a broader view of EDs grounded in attachment theory; namely a 'self/emotion-regulation' model of ED pathology (see Figure 1). Given the large number of alternate theoretical positions and models of ED development and maintenance available in the literature it is beneficial to seek to integrate these theories and models and embed them within an overarching theoretical framework, where appropriate, in order to focus future research and inform the development of more effective treatments for EDs.

Thus, the central argument of this thesis is that self-regulation, emotion-regulation, and sociocultural models of EDs are in fact highly congruent, overlapping, and inter-related. Given such overlap, it is challenging to separate discussion of these models and individual constructs within them under unique subheadings, as will be apparent across the background discussion presented in Chapters 2 and 3. However, these models arguably can be seen as essentially presenting a coherent account of several of the processes that may underlie the development and maintenance of ED pathology, and as largely understandable within the overarching framework of attachment theory.

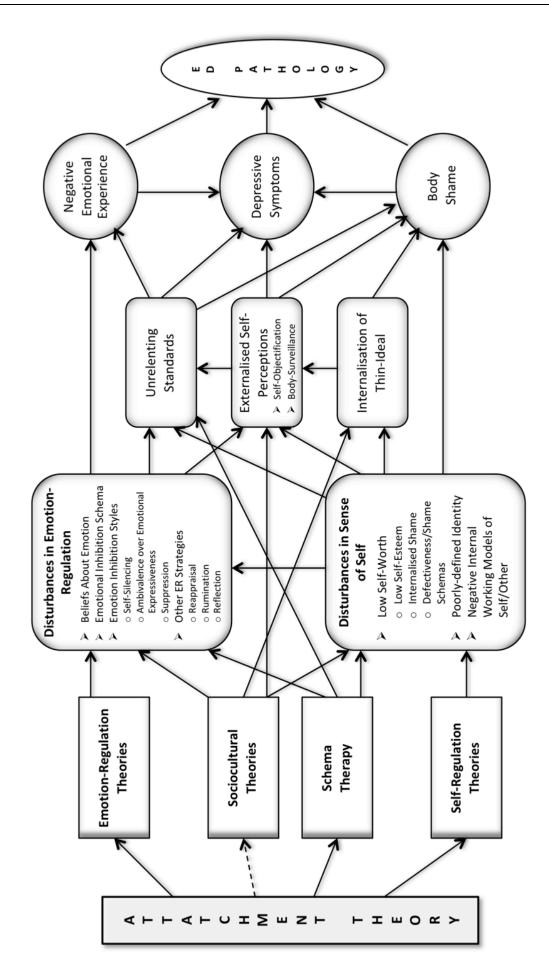


Figure 1. Author's conceptualisation of an integrated self/emotion-regulation model of eating disorders grounded in attachment theory.

#### RATIONALE FOR THE CURRENT RESEARCH

In support of a self/emotion-regulation model of ED pathology grounded in attachment theory, there is a growing body of research exploring the associations between various emotion-related constructs (e.g., awareness, experience, processing, regulation, and interpersonal expression of emotion) and ED pathology. There is also extensive research exploring associations between ED pathology and developmental constructs, such as attachment style, relational and early maladaptive schemas, and also the role of socio-cultural factors such as socialisation and gender-role expectations, cultural influences on idealised views of beauty and femininity, body objectification and internalisation of an observer's perspective of self ("externalised self-perceptions").

This body of research focused initially on exploration of direct associations between single constructs and ED pathology in isolation. Subsequently, studies have attempted to elucidate these associations using multivariable analysis and/or tests of moderation and mediation to determine the relative importance of key constructs in accounting for variance in ED pathology. However, these analyses have typically been confined to consideration of a small number of potentially relevant variables. This thesis seeks to build on this body of research and address some of these limitations by exploring in unison a number of interrelated and potentially overlapping emotion-regulation constructs, self, emotion and relational schemas, and attachment dynamics within an integrated model. The research tests the structural associations among these variables in a series of cross-sectional studies in women, using process mediation analyses and structural equation modelling (SEM) in order to attempt to tease out their individual and collective contributions to ED pathology and gain more insight into the contingent nature of the mechanisms through which such variables may impact the development and maintenance of ED pathology.

#### GOALS OF THE CURRENT RESEARCH

The broad goals of the current research were twofold. First, in accordance with emotion-regulation models of ED pathology, to investigate the associations among various forms of emotional inhibition and several emotion-regulation strategies in predicting ED pathology and to explore the mediating role of negative emotionality in these associations. Second, to explore a combined self/emotion-regulation model of ED pathology grounded in attachment theory, which incorporates emotion-regulation, self-development deficits, displacement theory, and sociocultural conceptualisations of ED pathology.

#### **AIMS**

More specifically, the studies that comprise the current research thesis were designed:

- 1. To explore the individual and unique roles of several forms of emotional inhibition (as one specific maladaptive style of emotion regulation), namely, negative beliefs about emotion, ambivalence over emotional expressiveness, silencing-the-self and divided-self schemas, in explaining variance in ED pathology and to examine any mediating and/or moderating role of negative emotionality in the associations between each form of emotional inhibition and ED pathology (Chapter 5).
- 2. To investigate the direct and indirect roles of various emotion regulation strategies namely, reappraisal, suppression, rumination, reflection, and emotional reflection negative beliefs about emotion, negative emotionality, and depressive symptoms in explaining variance in ED pathology (Chapter 6).
- 3. To explore the role of feelings of low self-worth (internalised shame and low self-esteem), externalised self-perceptions (self-objectification and body surveillance), body shame, and depressive symptoms in directly and indirectly contributing to variance in ED pathology (Chapter 7).

4. To investigate an integrated self/emotion-regulation model of ED pathology grounded in attachment theory by exploring the structural associations among attachment dynamics, core low self-worth (defectiveness/shame schemas), emotional inhibition schemas, unrelenting standards (perfectionism), body shame, depressive symptoms, and ED pathology (Chapter 8).

## RESEARCH DESIGN AND SAMPLES

The abovementioned aims were addressed in a series of several distinct but related studies presented in the form of journal articles submitted or prepared for submission for publication in peer-reviewed journals (Chapters 5 to 8). A summary of the sample, research design and variables considered in each study is presented in Table 1. The research comprised two independent cross-sectional online surveys in two different samples of women, which each yielded data for a number of individual studies. Data from the first survey (N = 403) was used in the analysis of Study 1 (Chapter 5) and Study 3A (Chapter 7). The second survey had two parts. Participants had the option of completing only Part (i) of the survey (N = 724), or also continuing to participate in Part (ii) (N = 548, a subset of the larger sample). Data from Part (i) of the second survey was used in the analysis of Study 2 (Chapter 6) and Study 4 (Chapter 8) and data from Part (ii) was used in the analysis of Study 3B (Chapter 7).

#### THESIS PRESENTATION

This thesis is presented as a thesis by publication. As a result, there is some repetition across chapters. An overview of the theoretical and empirical background relevant to the thesis is presented in Chapters 2 and 3. **Chapter 2** includes a description of emotion-regulation models of EDs and will briefly review relevant research literature related to investigation of the role of emotional experience, emotion inhibition and various forms of emotion-regulation strategies in ED pathology. **Chapter 3** provides an outline of a broad developmental model of EDs as disorders of self-development (i.e., as disorders comprising

significant disturbances to development of sense of self and self-identity), grounded in attachment theory. This chapter provides an overview of attachment theory and how insecure attachment styles have been linked to ED pathology, both directly and indirectly in the context of self-development (via Internal Working Models of self and others and internalised shame), and emotional development (e.g., emotional awareness, expression, and regulation capacities). Finally, the chapter will explore some of the maladaptive core beliefs (i.e., early maladaptive schemas of defectiveness/shame, emotional inhibition, and unrelenting standards) possibly underlying ED pathology. **Chapter 4** describes the aims of the current research and provides an overview of the research approach taken in the four discrete studies. The four empirical studies are then presented in **Chapters 5 through 8**. Finally, **Chapter 9** provides an integrated discussion of the major findings of the research as a whole, the theoretical and clinical implications of this research, its strengths and limitations, and directions for future research.

To avoid repetition, a single comprehensive reference list is provided at the end of the thesis. However, since the individual studies are presented in the form of manuscripts submitted or prepared for submission for publication, each is presented as a standalone work including references within each of Chapters 5 through 8. This thesis has been prepared using Australian English language spelling throughout. However, the manuscripts for the individual studies (Chapters 5 to 8) submitted for publication were prepared using US English language spelling, as these were prepared for journals based in the USA.

Table 1
Summary of Research Design and Key Variables Considered in Each Study

| Survey                                |  | Study                   | Variables/Constructs   |
|---------------------------------------|--|-------------------------|--|
| Survey 1                              | (N = 403)  | Study 1<br>(Chapter 5)  | <ul> <li>ED pathology (EAT-26)</li> <li>Depressive symptoms (DASS-21 depression subscale)</li> <li>Negative emotionality (DES IV-A negative emotionality subscale)</li> <li>Beliefs about emotion (BAES)</li> <li>Ambivalence over emotion expression (AEQ)</li> <li>Silencing-the-self schemas (STSS silencing-the-self subscale)</li> <li>Divided self schemas (STSS divided-self subscale)</li> </ul> |
|                                       |  | Study 3A<br>(Chapter 7) | <ul> <li>ED pathology (EAT-26)</li> <li>Depressive symptoms (DASS-21 depression subscale)</li> <li>Internalised Shame (FASS)</li> <li>Self-esteem (SES)</li> <li>Body shame (OBCS body shame subscale)</li> </ul>  |
| Survey 2 Part (i) Study 2 (Chapter 6) | <ul> <li>ED pathology (EAT-26)</li> <li>ED symptoms (EDDS-SC)</li> <li>Depressive symptoms (DASS-21 depression subscale)</li> <li>Beliefs about emotion (BAES)</li> <li>Cognitive reappraisal (ERQ reappraisal subscale)</li> <li>Expressive suppression (ERQ suppression subscale)</li> <li>Emotional reflection (ROF)</li> <li>Rumination (RRQ rumination subscale)</li> <li>Reflection (RRQ reflection subscale)</li> </ul> |                         |  |
|                                       |  | Study 4<br>(Chapter 8)  | <ul> <li>ED pathology (EAT-26)</li> <li>ED symptoms (EDDS-SC)</li> <li>Depressive symptoms (DASS-21 depression subscale)</li> <li>Attachment-avoidance (ECR-R avoidance subscale)</li> <li>Attachment-anxiety (ECR-R anxiety subscale)</li> </ul>  |

#### Table 1 (continued)

|                        |                         | <ul> <li>Defectiveness/shame schemas (YSQ-S3 defectiveness/shame subscale)</li> <li>Emotional inhibition schemas (YSQ-S3 emotional inhibition subscale)</li> <li>Unrelenting standards schemas (YSQ-S3 unrelenting standards subscale)</li> <li>Body shame (OBCS body shame subscale)</li> </ul>                       |
|------------------------|-------------------------|--|
| Part (ii)<br>(N = 548) | Study 3B<br>(Chapter 7) | <ul> <li>ED pathology (EAT-26)</li> <li>ED symptoms (EDDS-SC)</li> <li>Depressive symptoms (DASS-21 depression subscale)</li> <li>Internalised shame (FASS)</li> <li>Self-objectification (SOQ)</li> <li>Body surveillance (OBCS body surveillance subscale)</li> <li>Body shame (OBCS body shame subscale)</li> </ul> |

EAT-26 = Eating Attitudes Test (Garner, Olmsted, Bohr, & Garfinkel, 1982). DASS-21 = Depression, Anxiety and Stress Scale depression subscale (Lovibond & Lovibond, 1995a). DES IV-A = Differential Emotions Scale IV-A negative emotionality subscale (Izard, 1982). BAES = Beliefs About Emotions Scale (Rimes & Chandler, 2010). AEQ = Ambivalence Over Emotional Expressiveness Questionnaire (King & Emmons, 1990). STSS = Silencing the Self Scale (Jack & Dill, 1992). FASS = Feelings About Self Scale (McIlwain & Warburton, 2004). SES = Rosenberg Self-Esteem Scale (Rosenberg, 1965). OBCS = Objectified Body Consciousness Scale body shame subscale (McKinley & Hyde, 1996). EDDS = Eating Disorder Diagnostic Scale (Stice, Telch, & Rizvi, 2000). ERQ = Emotion Regulation Questionnaire (Gross & John, 2003). ROF = Reflecting on Feelings Scale (McIlwain & Galati, 2005). RRQ = Rumination-Reflection Questionnaire (Trapnell & Campbell, 1999). ECR-R = Experience of Close Relationships-Revised Questionnaire (Fraley, Waller, & Brennan, 2000). YSQ-S3 = Young Schema Questionnaire – Short Form (Young, 2007). SOQ = Self-Objectification Questionnaire (Noll & Fredrickson, 1998).

# Chapter 2

**Background – Emotion-Regulation Models of Eating** 

**Disturbances** 

#### OVERVIEW

As mentioned in Chapter 1, the central argument of this thesis is that self-regulation, emotion-regulation, and sociocultural models of the development and maintenance of eating disorders (EDs) are highly inter-related and best understood as anchored within the overarching conceptual framework of attachment theory. This overlap presents a challenge in separating discussion of such models and the individual constructs and processes implicated within each under unique subheadings within a background literature review. Nevertheless, the approach this thesis adopts is to divide discussion of the emotion-regulation and selfregulation models across this and the next chapter. Following below, this chapter presents emotion-regulation based models of ED pathology and includes a brief review of the relevant empirical evidence linking various emotion-regulation styles and strategies (including selfsilencing schemas as a form of emotion inhibition) to ED pathology. Chapter 3 then presents a broad developmental model of EDs as disorders of self-regulation grounded in attachment theory. An overview of attachment theory, how insecure attachment styles have been linked to ED pathology, both directly and indirectly in the context of self and emotional development and some of the maladaptive core beliefs possibly underlying ED pathology are explored. Finally, sociocultural models of EDs are returned to and explored further at the end of Chapter 3.

#### **EMOTION-REGULATION MODELS OF EATING DISTURBANCES**

Difficulties with emotion have long been conceptualised as a core feature of eating disorders (ED), with Bruch (1962, 1973) early on identifying difficulties in the ability to tolerate, name and express emotions in clients with anorexia nervosa. ED pathology has repeatedly been associated with the experience of negative emotion (Kitsantas et al., 2003; Stice, 2002) and negative emotionality has been found to predict later ED pathology in prospective studies of adolescents (Leon, Fulkerson, Perry, & Cudeck, 1993; Leon, Fulkerson,

Perry, & Early-Zald, 1995; Leon, Fulkerson, Perry, Keel, & Kliump, 1999). Additionally, EDs often present with comorbid depression (e.g., Agras, 2001; Zonnevylle-Bender et al., 2004). A recent review concluded that available evidence suggests that both anorexia nervosa (AN) and bulimia nervosa (BN) are characterised by broad emotion regulation deficits (Lavender et al., 2015). Further, difficulties with emotion regulation have been viewed as a transdiagnostic concern and as characteristic of both depression and EDs (Brockmeyer, Holtforth, et al., 2012; Brockmeyer et al., 2014).

Several emotion-regulation models of ED pathology have been provided. Broadly speaking the symptoms of EDs can be thought of as serving to inhibit emotions (Bekker & Spoor, 2010; Fox & Power, 2009). The 'blocking' model proposes that ED symptoms are maladaptive emotion regulation strategies that provide short-term relief from unacceptable emotion, but which exacerbate distress in the long-term (McManus & Waller, 1995; Root & Fallon, 1989). Similarly the 'escape from awareness model' (Heatherton & Baumeister, 1991) proposes that chronic self-focus may highlight perceived failures to meet idealised standards of self (held by self or others), resulting in painful negative self-evaluation and associated negative emotion, which the individual seeks to divert attention away from by focusing on an external stimulus (food).

Building on these theories, Corstorphine (2006) proposed an affect-regulation model of EDs in which beliefs about the unacceptability of experiencing and expressing negative emotions (BAE) underlie the emotional distress experienced by individuals with EDs. From an etiological perspective, this model implicates early experience of emotionally invalidating environments in the development of beliefs that emotions are 'bad' and that experiencing and expressing negative emotions is dangerous and to be avoided. Such beliefs are thought to then activate secondary negative emotions in response to primary emotions (e.g., shame about feeling loneliness or guilt about feeling sadness or anger), which in turn trigger the use of ED

symptoms (focus on weight/shape and their control, binge eating, compensatory behaviours, and restriction) as a short-term strategy for blocking the experience of such negative emotions (Corstorphine, 2006).

Empirically, a growing body of research has sought to identify specific emotion-regulation styles and strategies that may be linked to ED pathology. Emotion-regulation models of psychopathology suggest that it may not be the experience of emotion-provoking events or negative emotion per se that leads to symptoms, but, rather, the manner in which individuals attend to, process or regulate this experience (either internally or interpersonally) that leads to increased psychopathology (Gratz & Roemer, 2004; Gross, 1998).

#### **EMOTION INHIBITION**

Emotion inhibition is a maladaptive form of emotion regulation that includes efforts to both prevent emotion expression and to suppress sensations, thoughts, urges, and feelings associated with the experience of emotion (Krause, Mendelson, & Lynch, 2003). This maladaptive means of coping has both physiological and psychological costs. For instance, individuals who chronically suppress anger have been found to have elevated blood pressure (S. P. Thomas, 1997), while those who attempt to suppress pain experience greater duration of discomfort and increased sensitivity to future discomfort (Cioffi & Holloway, 1993). Suppression of emotion is also associated with impairment in various aspects of psycho-social functioning (Gross & John, 1997, 2003), due in part to the fact that it is actually counterproductive and paradoxically can bring about increases in the very emotions being suppressed (for review see Wenzlaff & Wegner, 2000). Emotional inhibition can be conceptualised as including a number of distinct but related constructs and has been operationalised in different ways. Thus, in the ED literature, emotion inhibition has been viewed, among other things, as conflict or ambivalence over emotional expression (King & Emmons, 1990); self-silencing schemas (Jack & Dill, 1992); expressive suppression (Gross & Emmons, 1990); self-silencing schemas (Jack & Dill, 1992); expressive suppression (Gross &

John, 2003); the early maladaptive schema of emotional inhibition (Young, 1990); and the closely related construct of negative beliefs about emotion expression (Corstorphine, 2006). Evidence bearing on the association of each of these constructs with ED pathology is briefly reviewed in the paragraphs that follow.

#### **Ambivalence over Emotional Expressiveness**

While evidence exists for an association between emotional expression and wellbeing, this association is by no means straightforward (Kennedy-Moore & Watson, 2001). It has been suggested that it is not one's level of emotional expressiveness per se, but rather how conflicted one is over their level of emotional expression or inhibition, that is associated with physical and psychological distress (King & Emmons, 1990). The construct of ambivalence over emotional expressiveness ('ambivalence') relates to an individual's experience of psychological conflict about their level of emotional expression. It is characterised by a desire to express emotion coupled with an inability to do so, as well as emotional expression coupled with regret at having done so (King & Emmons, 1990). Research exploring the association between ambivalence over emotional expressiveness and ED pathology has found ambivalence to be significantly higher in a clinical sample of women with EDs than in controls (Forbush & Watson, 2006). In a female student sample, ambivalence over emotional expressiveness was found to be significantly associated with ED pathology, over and above depressive symptoms and sociotropy (a need for and preoccupation with securing close interpersonal bonds; Krause, Robins, & Lynch, 2000). However, in another student sample ambivalence over emotional expressiveness was not found to be independently associated with ED pathology, over and above alexithymia (difficulty identifying and describing feelings in words) and emotional expression (Quinton & Wagner, 2005). Thus, the nature of the association between ambivalence over emotional expressiveness and ED pathology and

whether this association exists independent of other forms of emotion inhibition (to be discussed below) and depressive symptoms requires further investigation.

#### **Self-Silencing**

Another means through which individuals may inhibit emotion is via self-silencing. Self-silencing theory (Jack, 1991) proposes that women value the maintenance of close positive interpersonal connections and, within a social history of gender inequity, develop a range of cognitive/relational schemas (e.g., putting the needs of others first, self-sacrifice, and self-silencing) as a means of achieving and maintaining intimacy. This theory has since been suggested to also explain EDs (Geller, Cockell, Hewitt, Goldner, & Flett, 2000). Self-silencing theory also embodies concepts of disturbances in sense of self and will be considered further in this regard in Chapter 3. Two of the self-silencing schemas most relevant to emotion inhibition, though, are 'silencing-the-self', which involves attempting to avoid conflict or relationship loss by inhibiting expression of one's thoughts, feelings, or behaviour; and 'divided-self', the experience of developing internal anger and hostility whilst presenting an externally compliant self in order to conform to feminine role constraints (Jack & Dill, 1992).

In addition to evidence of a link between self-silencing schemas and depression (e.g., Cramer, Gallant, & Langlois, 2005; Tan & Carfagnini, 2008), a growing number of studies have investigated the link between self-silencing schemas and ED pathology. For instance, both silencing-the-self and divided-self schemas demonstrated strong positive associations with body dissatisfaction and drive for thinness in a clinical sample of adolescent girls diagnosed with EDs (Buckholz et al., 2007). In a high school sample, female adolescents who engaged in emotional and restrained eating were found to report significantly higher levels of silencing-the-self and anger suppression and lower levels of body-esteem than female adolescents who engaged in healthy eating (Norwood et al., 2011). In a second high school

sample, adolescent girls who scored high on ED symptomatology reported significantly higher silencing-the-self, divided-self, and anger suppression than those who scored low (Zaitsoff, Geller, & Srikameswaran, 2002). In female undergraduates, self-silencing has been found to be positively associated with ED concerns (Shouse & Nilsson, 2011), and both self-silencing schemas and anger suppression were positively associated with ED pathology in community-based samples, independent of their associations with one another (Morrison & Sheahan, 2009; Piran & Cormier, 2005). Further, silencing-the-self specifically has been found to be positively associated with weight and shape concerns, dietary restraint and bulimic behaviours, and divided-self positively associated with weight and shape concerns and uncontrolled eating, in two studies of female undergraduate students (Frank & Thomas, 2003; Ross & Wade, 2004).

With regard to clinical ED pathology, Geller et al. (2000) found that women with AN scored significantly higher than age-matched psychiatric and healthy control women (who did not significantly differ from each other) on silencing-the-self, divided-self, suppressed anger, and depressive symptoms. Across the combined sample silencing-the-self, divided-self, and anger suppression were each found to be significantly positively correlated with body image dissatisfaction. Higher scores on measures of self-silencing schemas and depressive symptoms among women with AN, compared with healthy controls, have also been observed in at least two other clinical samples (Hambrook et al., 2011; Oldershaw et al., 2012). However, in the one study in which group differences in measures of self-silencing were assessed over and above the effects of depression (Hambrook et al., 2011), these differences, and the partial correlations between silencing-the-self and divided-self and ED pathology, were no longer significant when age, depression and anxiety were controlled.

**Summary.** There is consistent preliminary evidence indicating the potential importance of self-silencing schemas in general, and silencing-the-self and divided-self

schemas as specific forms of emotion inhibition, in understanding ED pathology. However, despite the theoretical and empirical link between self-silencing and depression (e.g., Cramer et al., 2005; Jack, 1991), and between depression and EDs (e.g., Presnell, Stice, Seidel, & Madeley, 2009), only one study investigating the link between silencing-the-self and divided-self schemas and EDs has explored whether this association remains significant when controlling for depressive symptoms. Further, research is needed to explore the associations among silencing-the-self and divided-self schemas and ED pathology in unison with other forms of emotion inhibition (e.g., ambivalence over emotional expressiveness and negative beliefs about emotion) in order to determine their independent contributions to variance in ED pathology.

#### **Emotional Inhibition Schemas**

Another way in which emotion inhibition has been conceptualised is as an early maladaptive schema within schema therapy (Young, 1990). The early maladaptive schema of emotional inhibition involves the suppression of spontaneous affect, behaviours, or communication of thoughts/feelings/ideas, in an attempt to avoid feelings of shame, losing control of oneself, or the judgment or disapproval by others (Young, 1990). According to schema therapy, emotional inhibition may develop as a result of defectiveness/shame schemas (to be discussed in Chapter 3) (Young et al., 2003). In relation to ED pathology, emotional inhibition schemas have been associated with an increased drive for thinness (Waller, Dickson, & Ohanian, 2002), have been found to predict the severity of binge eating (Waller, Ohanian, Meyer, & Osman, 2000), and were associated with perceived experience of a chaotic early family environments (Ford, Waller, & Mountford, 2011) in clinical samples of women with EDs.

## **Negative Beliefs About Emotion**

Finally, a construct closely aligned with the early maladaptive schemas of emotional inhibition is that of beliefs about the unacceptability of experiencing or expressing negative emotions. Negative beliefs about emotion have been operationalised using the Beliefs About Emotions Scale (BES; Rimes & Chalder, 2010), the Attitudes Towards Emotional Expression Scale (AEES; Joseph, Williams, Irwing, & Cammock, 1994), and the nonacceptance of emotional responses subscale of the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). Such beliefs are suggested to have implications for emotion processing and regulation (Rimes & Chalder, 2010) and have been implicated theoretically as playing an important role in the formation and maintenance of ED pathology (Corstorphine, 2006).

Research seeking to provide empirical support for the implications of negative beliefs about emotion for ED pathology is in its early stages. However, negative attitudes towards emotional expression have been found to be associated with increased ED symptomology in non-clinical female samples (Haslam, Arcelus, Farrow, & Meyer, 2012; Meyer, Leung, Barry, & De Feo, 2010) and evidence from clinical samples is consistent with such an association. For instance, negative beliefs about emotion were found to be higher in individuals with AN compared with healthy controls (Hambrook et al., 2011; Jansch, Harmer, & Cooper, 2009; Oldershaw et al., 2012) and found to be positively associated with ED pathology, depression, and anxiety in a mixed sample of individuals with current-AN, recovered-AN, and healthy controls (Oldershaw et al., 2012). Negative beliefs about emotion were found to be positively associated with ED pathology, over and above depression and anxiety, in a clinical sample of individuals with AN (Hambrook et al., 2011). Further, non-acceptance of emotional states was positively associated with increased ED symptomology in a clinical sample of individuals with full- and sub-threshold BN and higher among those with full-threshold BN than those with sub-threshold BN (Lavender et al., 2014).

As is the case with silencing-the-self and divided-self schemas, however, it is unclear whether the association between negative beliefs about emotions and ED pathology exists independent of the effects of depressive symptoms. Depressive symptoms were not measured in Lavender et al.'s (2014) study and while depressive symptoms were measured in the studies of Jansch et al. (2009) and Oldershaw et al. (2012), the issue of whether group differences in negative beliefs about emotion between individuals with AN and healthy controls remained after controlling for depressive symptoms was not considered. Nor was it clear whether the positive association between negative beliefs about emotion and ED pathology observed in Oldershaw and colleagues' study remained significant when controlling for symptoms of depression and anxiety. In the study by Hambrook and colleagues (2011), a significant positive association between negative beliefs about emotion and ED pathology among individuals with AN remained after controlling for symptoms of depression and anxiety, whereas differences in levels of negative beliefs about emotion between participants with AN and healthy controls were no longer significant after controlling for age, depressive symptoms, and anxiety. Thus, further research is needed to substantiate the role of negative beliefs about emotion in contributing to ED pathology, over and above depressive symptoms and to consider such a role in unison with other forms of emotion inhibition.

## **Expressive Suppression**

Expressive suppression ('suppression') involves the inhibition of outward signs of emotion expression and is a form of response-focused modulation that occurs late in the emotion generation process (Gross, 2001). Suppression is typically considered in contrast to the more adaptive emotion-regulation strategy of reappraisal (to be discussed below). Whilst suppression decreases the behavioural expression of emotion, it fails to decrease the experience of emotion and actually increases physiological responding to stressors (Gross,

2001). Suppression is a frequently employed method of emotional control but it comes with costs. For instance, it places high cognitive and attentional demands on the individual, taxes memory (Richards & Gross, 2000) and is inversely associated with interpersonal functioning and measures of well-being (Gross & John, 2003). Use of suppression has been associated with the experience of greater negative emotion and less positive emotion (Gross & John, 2003) and can trigger feelings of inauthenticity and low self-esteem (John & Gross, 2004).

In terms of associations with ED pathology, suppression has been found to be associated with bulimic symptoms, over and above BMI, in undergraduate women (Lavender, Jardin, & Anderson, 2009) and to be associated with both ED pathology and depressive symptoms. That is, levels of suppression, depressive symptoms and negative emotion were higher in women with high levels of ED pathology, than those with low levels (McLean, Miller, & Hope, 2007). In clinical samples, suppression was found to be significantly higher among individuals with AN and BN compared with healthy controls (Davies, Swan, Schmidt, & Tchanturia, 2012) and significantly higher in groups of women with AN, BN, and BED compared with healthy controls (Svaldi, Griepenstroh, Tuschen-Caffier, & Ehring, 2012). Additionally, in a meta-analysis of six studies, comprising both clinical and non-clinical samples, suppression was found to have a medium effect on ED pathology, with higher effect sizes for research conducted in clinical versus non-clinical samples (Aldao, Nolen-Hoeksema, & Schweizer, 2010).

### **Summary**

While there is considerable evidence available that emotion inhibition in general is a costly form of emotional coping that may lead to increased ED pathology, the associations among the various forms of emotion inhibition and ED pathology, and whether or not such associations are independent of one another and depressive symptoms, is less clear.

Furthermore, given evidence that use of emotion inhibition to circumvent unwanted emotions

emotion in undergraduate students (Kashdan, Barrios, Forsyth, & Steger, 2006) and the expectations of Corstorphine's (2006) affect-regulation model, exploration of whether negative emotionality mediates and/or moderates the associations among negative beliefs about emotion, ambivalence over emotional expressiveness, silencing-the-self, divided-self, suppression and ED pathology seems warranted and presents a gap in the current literature.

#### **EMOTION REGULATION STRATEGIES**

Emotion-regulation strategies are processes through which individuals attempt either consciously or unconsciously to modulate their response to emotion-eliciting events by changing the size, duration, intensity, and/or type of their emotional experience or expression (Gross & Thompson, 2007). Emotion-regulation strategies can take several forms, in addition to the various forms of emotion inhibition considered above, and can be classified as adaptive or maladaptive (Werner & Gross, 2010). Several additional emotion-regulation strategies that have been linked to ED pathology are cognitive reappraisal, rumination, and reflection.

## **Cognitive Reappraisal**

A more adaptive emotion-regulation strategy that occurs earlier in the emotion generative process and avoids the costs of suppression is cognitive reappraisal ('reappraisal') (Gross, 2001). Reappraisal involves generating alternate interpretations of potentially emotion-provoking events in order to alter the emotions elicited (Gross & John, 2003). Cognitive models of emotional disorders identify maladaptive appraisals as a core mechanism in the development and maintenance of depression and anxiety disorders, and cognitive-behavioural therapies (CBT) for these disorders focus on the development of more adaptive appraisal skills (Beck, 1979; Beck, Emery, & Greenberg, 1985; Beck, Rush, Shaw, & Emery, 1979). Such cognitive models and CBT have also been applied to EDs (e.g., Fairburn, 2008).

Use of reappraisal has been found to be more effective than suppression, as reappraisal

decreases both the experience and expression of emotion, without adverse impacts on memory (Gross, 2001), and is positively associated with interpersonal functioning and well-being (Gross & John, 2003). Reappraisal has also been found to be associated with lower depressive symptoms, controlling for other emotion-regulation strategies (including rumination; to be discussed below), in both female and mixed gender non-clinical and psychiatric samples (Garnefski & Kraaij, 2006; Garnefski, Teerds, Kraaij, Legerstee, & van den Kommer, 2004). Further, high reappraisers were found to report less negative emotion, less physiological arousal, and more positive emotion than low reappraisers in an induced anger laboratory study (Mauss, Cook, Cheng, & Gross, 2007).

In relation to ED pathology, research on reappraisal is limited and largely confined to clinical samples, with results occasionally having been found to vary as a function of ED diagnosis. In one study, reappraisal was found to be high in healthy controls than groups of women with AN, BN, and BED (Svaldi et al., 2012). However, in another study reappraisal was found to be lower among individuals with AN compared to healthy controls, while individuals with BN did not differ from those with AN or controls (Davies et al., 2012). In another clinical sample, reappraisal was found to be higher among women with restrictive AN than those with binge-purge AN or BN. Within the women with restrictive AN, however, there was no association between reappraisal and ED pathology, whereas a moderate negative association (r = -.33) was observed in women with binge-purge AN (Danner, Evers, Stok, van Elburg, & de Ridder, 2012). Finally, a study assessing differences in use of suppression and reappraisal among women with binge-purge AN, restrictive AN, BN, binge eating disorder (BED), and healthy controls, found suppression to be higher in all ED-groups compared to controls, and higher among women in both of the AN groups than women with BN, while women with restrictive AN and BN (but not binge-purge AN or BED) reported lower use of reappraisal then controls (Danner, Sternheim, & Evers, 2014). Also in this study, suppression

was positively associated with depression in all subgroups but with ED pathology only in women with binge-purge AN and BED, while the use of reappraisal was negatively associated with depression only in women with BN and with ED pathology only in women with restrictive AN and BED.

**Summary.** While there is mounting support for the association of suppression with ED pathology (considered above), the role of reappraisal is less clear and there is a need for further research addressing the association between ED pathology and both suppression and reappraisal, controlling for symptoms of depression and other potential covariates. Research addressing these issues in non-clinical samples of women would be particularly helpful.

# Manner of Attending to Emotions: Rumination and Reflection

The manner in which one attends to the self and emotions can serve an important emotion-regulation function, and can also take more or less adaptive forms, such as reflection and rumination respectively (Trapnell & Campbell, 1999; Watkins, 2008).

**Rumination.** Rumination is a maladaptive form of repetitive negative thinking involving the tendency to dwell on the self, upsetting events and symptoms, and their possible causes and consequences, in the absence of problem-solving (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Watkins, 2008). Rumination is suggested to function as a maladaptive means of regulating emotion, best categorised as an avoidant coping strategy (Smith & Alloy, 2009). It is maladaptive in that – like suppression – it has the paradoxical effect of increasing negative emotion and low mood (Mor & Winquist, 2002).

Extensive evidence exists for the role of rumination in both the onset and maintenance of depression. For instance, evidence from numerous prospective longitudinal studies has shown that rumination consistently predicts the future onset of depression, as well as the severity of future depressive symptoms in both non-depressed and clinically-depressed people (Ehring & Watkins, 2008; Nolen-Hoeksema et al., 2008; Watkins, 2008). Further, rumination

has been found to mediate the association between several risk factors for depression and onset of depressive symptoms (Spasojevic & Alloy, 2001). In relation to EDs, rumination has been found to be associated with ED pathology in a student sample (Rawal, Park, & Williams, 2010), while in women with AN rumination has been found to contribute to variance in ED pathology, over and above depression, anxiety and worry (Startup et al., 2013). Rumination has been found to be elevated in women with AN compared with age-matched controls and a reduction in rumination was found to be associated with future reduction in ED pathology (Rawal et al., 2010). In adolescent girls, one longitudinal study found that rumination predicted the onset of both future major depression and binge eating and predicted future increases in depressive and bulimic symptoms, further depressive and bulimic symptoms were also found to predict future increases in rumination (Nolen-Hoeksema, Stice, Wade, & Bohon, 2007).

Finally, response style theory (Nolen-Hoeksema et al., 2008) suggests that binge eating serves the maladaptive function of escaping the aversive self-focus associated with the heightened rumination seen among individuals with BN. Further, ED-specific rumination (e.g., repetitive thinking about calories and control of eating, weight, and shape) is conceptualised as the primary mode of processing in AN, to the exclusion of experiencing emotions and the broader emotional meaning of bodily states (e.g., sensations of hunger and starvation); that in the short-term increases feelings of control, but in the long-term exacerbates dietary restraint (Park, Dunn, & Barnard, 2011, 2012). Such a model is supported by findings that disorder-specific rumination (on eating, weight, and shape concerns) has been uniquely associated with ED pathology, over and above symptoms of depression and anxiety (Cowdrey & Park, 2012).

**Reflection.** Another style of thinking and attending to self, one that may avoid the costs associated with rumination, is reflection. Reflection is defined as a form of "self-

attentiveness motivated by curiosity or epistemic interest in the self' (Trapnell & Campbell, 1999, p. 297). Reflection has been positively associated with openness (Trapnell & Campbell, 1999) and personal growth in undergraduate students (Harrington, Loffredo, & Perz, 2014). Research addressing the association between reflection and ED pathology is currently limited. However, in Rawal et al.'s (2010) study, referred to above, there was no association between reflection and ED pathology among students and levels of reflection did not differ between individuals with AN and controls.

Of relevance to an integrated self/emotion-regulation model of ED pathology grounded in attachment theory, the construct of reflective functioning (or mentalizing capacity) has also been linked with EDs (Tasca & Balfour, 2014a). Within attachment theory to be discussed in Chapter 3), disturbances in early attachments (insecurity of attachment relationships) are believed to interrupt developing capacities for reflective functioning, which in turn impair adaptive emotion-regulation functioning (Fonagy, Gergely, Jurist, & Target, 2002). Fonagy and colleagues further propose that reflective functioning may be central to coherent internal working models of relationships (Fonagy et al., 2002). In relation to EDs, reflective functioning (assessed via the Adult Attachment Interview; Kaplan & Main, 1985) has been found to be lower among individuals with EDs than in both non-clinical and psychiatric comparison groups (Fonagy et al., 1996; Kuipers & Bekker, 2012).

Emotional reflection. Additionally, given the focus on avoidance of negative emotion that is central to both the 'blocking' and 'escape from awareness' theories of EDs, the role of emotional reflection specifically, as opposed to more general reflection, in contributing to variance in ED pathology may warrant greater consideration. Emotional reflection is said to involve a fullness of feeling together with a cognitive and sensory awareness of the emotion and one's response to it (McIlwain, Taylor, & Geeves, 2010). Importantly, it involves awareness of feeling, rather than judgment of feeling (McIlwain et al., 2010). Thus emotional

reflection shares characteristics with awareness and nonjudgmental facets of trait mindfulness, each of which were found to be uniquely associated with ED pathology, over and above symptoms of anxiety and depression in undergraduate women (Lavender, Gratz, & Tull, 2011). Further, another parallel construct, experiential self-focus, but not ruminative self-focus, was found to be associated with lower feelings of change in weight or shape following exposure to imagining eating a large meal among students high in ED pathology and in partially weight-restored women receiving inpatient treatment for AN (Rawal, Williams, & Park, 2011). However, research has yet to explore whether emotional reflection is independently associated with ED pathology over and above general reflection and other emotion-regulation strategies.

# **Negative Emotionality and Depressive Symptoms**

Consideration of the potential mediating roles of negative emotionality and depressive symptoms is a concern that cuts across the associations among the various emotion-regulation strategies, negative beliefs about emotion and ED pathology. As noted above, the experience of negative emotion is strongly associated with ED pathology (Agras & Telch, 1998; Johnson & Larson, 1982). Negative emotion is also a diagnostic feature of major depressive disorder (American Psychiatric Association, 2013) and depression and ED pathology are commonly comorbid (Agras, 2001; Puccio, Fuller-Tyszkiewicz, Ong, & Krug, 2016). Further, dispositional tendencies towards high use of rumination and suppression, and low use of reappraisal have been consistently identified as transdiagnostic concerns across multiple psychopathologies, including depression (Aldao et al., 2010; Nolen-Hoeksema et al., 2007; Nolen-Hoeksema et al., 2008). Hence, when investigating the association between emotion-regulation strategies and ED pathology, it would be important to consider the roles played by both negative emotionality and depressive symptoms.

Despite the conceptual and empirical overlap between negative emotionality and depressive symptoms, it is argued herein that a distinction can be made between these constructs. Whilst negative emotionality, in the form of low mood or sadness, is a core symptom of depression, depressive symptoms include more than just negative emotionality. Thus for the purposes of the current research negative emotionality is defined to include the experience of sadness, disgust, contempt, self-directed hostility, fear, anger, embarrassment, shyness, and guilt (Izard, 1982). Whereas, depressive symptoms are defined to include: a lack of ability to experience positive emotion, lack of motivation, feeling down or blue, feelings of hopelessness, worthlessness, and meaninglessness (Lovibond & Lovibond, 1995a). This is considered to be an important distinction given the possibility that negative emotion may be experienced independent of the presence of other depressive symptoms and that ED pathology may be used as a maladaptive means of regulating negative emotion, independent of the experience of depressive symptoms.

### **Summary**

Few studies have looked at the relative importance of different emotion-regulation strategies in terms of their unique associations with ED pathology. In a correlational study of undergraduate students, use of experiential avoidance, emotion inhibition, suppression, and rumination were found to be associated with higher levels of psychological distress, via increases in the experience of negative affect and negative life events, and lower levels of positive affect, life satisfaction, and positive life events, whereas the use of reappraisal was associated with increases in gratitude, life satisfaction, and curiosity (Kashdan et al., 2006). Also in undergraduate students, a study employing structural equation modelling (SEM), found that suppression, rumination and reappraisal (negatively and weakly) loaded on a single latent emotion-regulation factor, which in turn contributed to variance in ED pathology, depression symptoms, and anxiety symptoms (Aldao & Nolen-Hoeksema, 2010). To date,

however, no research has employed a path model that incorporates maladaptive and adaptive emotion-regulation strategies, negative emotionality, depressive symptoms, and ED pathology, or extended such a model to incorporate potential antecedent constructs, such as negative beliefs about emotion.

# Chapter 3

**Background – Eating Disorders as Disorders of Self** 

#### EATING DISORDERS AS DISORDERS OF SELF

Disruptions in the sense of self have long been identified as a central feature of EDs (Ewell, Smith, Karmel, & Hart, 1996). Indeed adolescence, which is a time of transformation in sense of self (Erikson, 1950, 1968), is well known as a vulnerable period for the onset of ED pathology. The sense of self can be defined as "the sum total of all that [a person] can call his" (W. James, 1890, p. 291). Such a definition implies conscious awareness of the self by an individual and imbues him/her with control over interpreting what does and does not form part of the self; and in this way differs from common definitions of personality (Ewell et al., 1996). Sense of self is suggested to comprise the elements of both objective self-awareness (focusing of attention onto the self) and subjective self-awareness (self-reflection with emotional investment in the contents seen as comprising the self) and also self-understanding, self-representations, and theories of self (Ewell et al., 1996).

Heightened objective self-awareness is a well-recognised feature of EDs (Striegel-Moore, Silberstein, & Rodin, 1993) and forms the basis for Heatherton and Baumeister's (1991) theory of binge eating as serving the function of an escape from self-awareness. With regard to subjective self-awareness, ruptures and splits in subjective self-awareness are frequently identified in those who experience ED pathology and parts of the self or one's life are seen as disparate from the self or disavowed as not "belonging" to the individual (e.g., parts of the body, 'fat' on the body, sensations of hunger, or even parts of the self seen as false or inauthentic) (Ewell et al., 1996). With regard to representations of self, evidence exists regarding distortions in representations of the body in those with EDs, whereby perceived discrepancies among images of the actual self, the ideal self or what the self ought to be, according to the expectations of the self and/or others, are amplified (Mason et al., 2016).

Further, adolescence is characterised by the development of a sense of self, where features of the self are ascribed value in terms of their currency in achieving social acceptance and integration across many and varied contexts in life (e.g., academic success, sports, social activities, etc.) (Ewell et al., 1996). Ewell and colleagues further argue that for many individuals experiencing ED pathology, the only path to achieving such self and/or other acceptance is believed to be via attainment of the idealised body weight or shape. Thus, body weight/shape and their control becomes the central feature of achieving life satisfaction. As such, ED pathology can be seen to be associated with disturbances across all facets of the individual's sense of self and that whilst transitions in sense of self are characteristic of normal adolescent development, for adolescent girls with ED pathology the experience of such transitions are exaggerated and may endure beyond adolescence.

As discussed in Chapter 1, Bruch (1973, 1978) proposed a displacement theory of emotion in ED pathology whereby unexpressed emotions and negative feelings about the self are said to be displaced onto the body. In this context ED symptoms function as maladaptive means of avoiding or reducing feelings of shame and of enhancing feelings of pride or self-worth (Goss & Allan, 2009). That is, the displacement of an underdeveloped sense of self and core feelings of worthlessness or internalised shame involves the redirection of ego-threatening feelings to a less threatening and more concrete target – the body – and to body weight and shape, which can be controlled in a more concrete fashion via disturbed eating behaviours (Schupak-Neuberg & Nemeroff, 1993). Indeed, findings from longitudinal research suggest that past beliefs about the importance of body weight and shape predict future ED behaviours in adolescent girls (Wilksch & Wade, 2010). Furthermore, for individuals with EDs, anorexia nervosa (AN) in particular, schemas for self-evaluation become intricately connected to the achievement of unrealistically high standards and the achievement of these goals then becomes a strategy to overcome or block out feelings of

defectiveness or emptiness. The positive reinforcement provided by achieving such goals (and the associated feelings of pride) then serves to maintain the disorder and becomes part of the identity of the individual (Bruch, 1978).

As discussed in previous chapters, emotion-regulation and self-regulation models of ED pathology share a high amount of overlap, are inter-related, and have been theoretically conceptualised to emanate from common developmental causes (i.e., the quality of early caregiving relationships and environments). As such, these models are arguably best understood as symbiotically anchored within the overarching framework provided by attachment theory.

### **Individual Differences in Adult Attachment**

A developmental understanding of EDs as disorders of the self (Amianto et al., 2016; Goodsitt, 1983, 1997) is grounded within the broader theoretical framework of attachment theory. Attachment theory (Bowlby, 1969, 1973, 1980, 1988), emphasises the importance of the early caregiving environment for later psychological functioning and describes personality development from an interpersonal perspective. According to attachment theory, the capacity to regulate emotion and the development of the self occur as part of a normative emotional developmental process within the context of the primary caregiving relationship. Infants are seen as social animals, possessing an evolutionary drive to form attachments with and to seek proximity to caregivers in order to regulate distress and ensure their own survival (Ainsworth, 1982; Ainsworth, Blehar, Waters, & Wall, 1978; Bowlby, 1988).

Attachment theory proposes that information individuals collect from past experiences in relationships is organised in the form of internal working models (IWMs) of the self and others that operate as relationship schemas, enabling the individual to plan their behaviour and to predict the behaviour of others, particularly in the context of close relationships (Baldwin, 1992; Baldwin, Keelan, Fehr, Enns, & Kohn-Rangarajoo, 1996; Kobak & Sceery,

1988). It is argued that both functional emotion-regulation strategies and positive IWMs of the self and others develop through the child's repeated experiences of emotional attunement with a sensitive and responsive caregiver. As such, disruptions in attunement and insensitive or inconsistent caregiving are believed to result in the development of maladaptive emotion-regulation strategies and the internalisation of negative IWMs of the self and others (Mikulincer & Shaver, 2007; Schore, 1994, 2003; Siegel, 1999, 2006). Internal working models contribute to the formation the implicit memories of the self, self-states, and the self-in-relation to another and are considered to be central elements of personality that guide interactions with others throughout life (Bowlby, 1988; Zimmermann, 1999). Attachment processes, thus, are theorised to be important across the lifespan (Bowlby, 1988). Although amenable to change, IWMs tend to be stable and self-perpetuating as they are unconsciously imposed on future relationships (Ainsworth, 1990; Collins & Read, 1990; Main, Kaplan, & Cassidy, 1985).

Individual differences in adult attachment styles can be measured along the two dimensions of attachment-avoidance, characterised by discomfort with intimacy and independence seeking, and attachment-related anxiety, characterised by fears of rejection and abandonment (Fraley, Waller, & Brennan, 2000). In terms of emotion-regulation, high attachment-avoidance is associated with the defensive inhibition or suppression of threat-related emotional states (e.g., fear, anxiety, anger, shame and sadness) and aims to deactivate the attachment system and minimise interpersonal closeness and interdependence. A potential cost of which is that it often interferes with reappraisal, problem-solving, and support seeking (Mikulincer & Shaver, 2007). Conversely, attachment-anxiety is paradoxically associated with the intensification of expression of negative emotions, the aim of this strategy being to hyper-activate the attachment system in order to attract the attention, care, and protection of unpredictably available attachment figures. This strategy can often be self-defeating and can

lead to feelings of helplessness (Shaver & Mikulincer, 2007).

The dimensional measurement of attachment dynamics (Fraley et al., 2000) is organised within a four-category model of adult attachment (Figure 2) (Bartholomew & Horowitz, 1991). According to these models *secure* attachment is represented by low anxiety and low avoidance and positive models of both the self and other. *Preoccupied* (anxious-ambivalent) attachment is represented by high anxiety and low avoidance and corresponds to a negative model of the self and a positive model of other. *Dismiss*ing (avoidant) attachment is represented by low anxiety and high avoidance with a positive model of the self and a negative model of other. Finally, a *fearful-avoidant* (disorganised) attachment is represented by high anxiety and avoidance and corresponds to negative models of both self and other. Broadly speaking, high attachment-anxiety and/or high attachment-avoidance, either individually or concurrently, are believed to characterise insecure attachment styles.

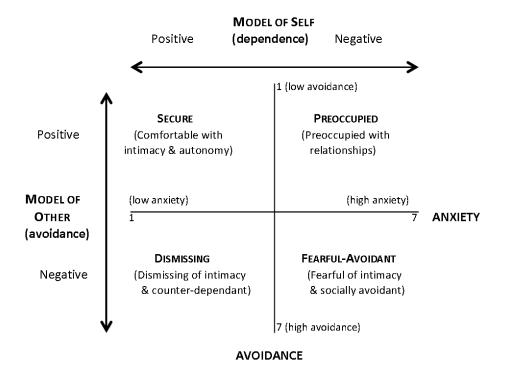


Figure 2. Diagrammatical representation of Bartholomew and Horowitz's (1991) four-category model of adult attachment (included on the left and top sides of the figure) and Fraley, Waller and Brennan's (2000) two-dimensional model (represented in the centre of the figure).

Adult attachment and ED pathology. There is extensive empirical support for a propensity to insecure attachment styles in clinical populations with EDs. For instance, higher rates of insecure adult attachment have been found in clinical samples of women with EDs compared with non-clinical controls (e.g., Latzer, Hochdorf, Bachar, & Canetti, 2002; Troisi, Massaroni, & Cuzzolaro, 2005). Preoccupied attachment style was found to be associated with body dissatisfaction in women with EDs, controlling for body mass index (BMI) and depression (Troisi et al., 2006), while and in one study of 62 women with clinical-EDs all participants were classified as insecurely attached (Ringer & Crittenden, 2007). Further, evidence suggests that attachment dynamics may moderate responses to different treatment modalities. Thus, in a study of individuals with binge eating disorder (BED) receiving specialist treatment, high attachment-anxiety was associated with better outcomes using group psychodynamic interpersonal therapy at 12-month follow-up, whereas low attachment-anxiety was predictive of better outcomes from group CBT at 12-month follow-up (Tasca et al., 2006). Further, attachment-avoidance has been associated with treatment drop-out in women with AN and BED (Tasca et al., 2006; Tasca, Taylor, Ritchie, & Balfour, 2004).

Consistent with the expectations of a self/emotion-regulation model of EDs grounded in attachment theory, emotion dysregulation has been found to mediate the associations between both attachment-anxiety and attachment-avoidance and ED pathology in a community sample of Australian women (Ty & Francis, 2013). Further, the mediating role of up-regulating and down-regulating emotion-regulation strategies on the associations among attachment dimensions, depressive symptoms, and ED pathology was explored using structural equation modelling (SEM) in a cross-sectional study of women with EDs. This study revealed that the association between attachment-anxiety and ED pathology was fully mediated by emotional reactivity and depressive symptoms, whereas the association between attachment-avoidance and ED pathology was only partially mediated by emotional

deactivation and depressive symptoms and the direct association between attachment-avoidance and ED pathology remained significant (Tasca et al., 2009b). Although, these studies employed cross-sectional designs and thus preclude the drawing of conclusions as to whether insecure attachment styles are a cause or effect of ED pathology. However, findings by Sharpe et al. (1998) of an over-representation of insecure attachment styles in teenage girls with increased food and weight concerns (a factor placing them at risk for developing EDs). Taken together with findings by Burge et al. (1997) that insecure attachment styles predicted ED symptoms at 12-month follow-up, suggest that insecure attachment either precedes EDs or contributes to their maintenance via feelings of self-worth and self-development, emotion-regulation, and interpersonal functioning (Tasca & Balfour, 2014b).

Summary. Attachment theory provides an overarching theoretical framework for understanding emotional development and disturbances in emotion-regulation capacities, as well as the development of sense of self and disturbances in self-regulation. Further evidence to support a self/emotion-regulation model of ED pathology grounded in attachment theory, can be found in research investigating the roles of self-esteem, internalised shame, and defectiveness/shame schemas. Research of this kind, along with research exploring other disturbances in self, including the roles of body shame, perfectionistic schemas, and externalised self-perceptions (self-objectification and body surveillance), as these relate to ED pathology, is reviewed in the following sections.

### Low Self-Worth/Shame in Eating Disturbances

Difficulties with feelings of low self-worth, negative self-evaluations, and disturbances in sense of self are thought to play a central role in ED pathology (Basten & Touyz, 2016). Indeed, EDs have been described as 'disorders of shame' (Kaufman, 1996, p. 129). In the ED literature, negative feelings about the self have been conceptualised in a

number of ways, most notably via reference to internalised shame, low self-esteem, and defectiveness/shame schemas.

**Internalised shame.** Within a self/emotion-regulation model of ED pathology the central overwhelming negative emotion that is displaced onto the body is that of internalised shame (also referred to as internal shame). Internalised shame is a self-conscious emotion characterised by an experience of the self as being flawed, worthless and powerless (Tangney, 1995; Tangney, Wagner, & Gramzow, 1992). It is accompanied by a sense of shrinking or feeling inferior and of being exposed (Tangney, 1995) and with intense self-criticism and feelings of self-hatred (Gilbert, 2002). Internalised shame is distinguished from external shame, which relates to negative beliefs about the ways in which one may be perceived and evaluated by others and subsequent fears of rejection (Allan & Goss, 2012). Lewis (1971) further suggested that internalised shame involves a split in self-functioning whereby the self is both agent of observation and object of condemnation, such that the observing self becomes a denigrating audience, vilifying the objective self as worthless and reprehensible (Hahn, 2000; Tangney, 1995). To this end, ED sufferers frequently identify themselves as feeling inherently worthless, deficient, or disgusting (Bruch, 1973; Garfinkel, Garner, & Kennedy, 1985) and often report experiences of feeling trapped in and ashamed of their bodies (Rortveit, Astrom, & Severinsson, 2009).

Given that the experience of internalised shame involves a painful sense of self as undesirable and flawed in some way, it is not surprising that efforts are made to escape or avoid such experience. As noted earlier, displacement theory proposes that such egothreatening feelings (internalised shame and painful sense of self) are redirected to a less threatening and more concrete target – the body – which can be more readily controlled via preoccupation with and pursuit of idealised body weight/shape and through adherence to extreme weight-control behaviours (Schupak-Neuberg & Nemeroff, 1993). That is, a focus on

the control of body weight/shape through ED pathology serves the dual functions of avoiding aversive self-awareness and attempting to enhance self-worth and self-identity through feelings of pride (Button, 1990; Goss & Allan, 2009). Thus, in EDs, the body becomes at once the target of displaced feelings of shame, the object of change, and the vehicle for self-transformation (Basten & Touyz, 2016).

In support of such a theory, associations between internalised shame and ED pathology have been found in both non-clinical (Sanftner, Barlow, Marschall, & Tangney, 1995) and clinical (Troop, Allan, Serpell, & Treasure, 2008) samples, and when controlling for both depressive symptoms (Gee & Troop, 2003; Hayaki, Friedman, & Brownell, 2002b) and negative affect more generally (Gupta, Rosenthal, Mancini, Cheavens, & Lynch, 2008). Further, Grabhorn, Stenner, Stangier, and Kaufhold (2006) found inpatient women with AN and BN reported higher levels of internalised shame than inpatient women with anxiety and mood disorders, suggesting that internalised shame may play a particularly important role in the onset and/or maintenance of ED pathology.

Self-esteem. Another consistent way in which negative feelings about the self (low self-worth) have been associated with ED pathology is via the recurrent identification of low self-esteem in women with EDs. Self-esteem can be defined as confidence in one's overall worth or abilities or as self-respect (Rosenberg, 1965). Low self esteem has been associated with ED pathology in various population-based samples, including college women (Fredrick & Grow, 1996; Mayhew & Edelmann, 1989) and self-esteem has been found to be lower in patients with EDs than in individuals without EDs (Moor, Vartanian, Touyz, & Beumont, 2004). Low self-esteem has also been associated with concerns about fatness in girls as young as 11-12 years (Button, 1990). While in a prospective study of adolescent girls, low self-esteem was found to be associated with EDs at baseline and high self-esteem was a protective factor that significantly reduced the risk of EDs at 18-month follow-up (Cervera et al., 2003;

Gual et al., 2002). In a clinical group of outpatients with EDs, self-esteem was significantly lower compared with controls participants and significantly inversely correlated with body shape concerns in both groups (Beato, Rodriguez Cano, & Belmonte, 2003). Among individuals with BN, low self-esteem has also been associated with poor treatment outcomes and relapse at 12-month follow up (Fairburn, Peveler, Jones, Hope, & Doll, 1993).

Importantly, consistent with a self/emotion-regulation model of EDs grounded in attachment theory, attachment anxiety and avoidance have each been found to demonstrate strong to moderate associations with low self-esteem and secure internal working models of self and others show strong to moderate positive associations with self-esteem (Luke et al., 2004). Luke et al. (2004) found attachment anxiety was strongly associated with negative working models of self and attachment avoidance with negative representations of others in relational contexts. They also reported that positive perceptions of fathers' support and acceptance in childhood contributed to self-esteem indirectly via positive IWMs of self.

**Defectiveness/shame schemas.** A third way in which negative feelings about the self can be conceptualised, closely aligned with internalised shame, is via early maladaptive core beliefs of defectiveness/shame. Defectiveness/shame schemas involve the underlying feeling and belief that one is bad, unwanted, damaged or inferior in important ways or that one would be seen as unlovable if exposed to significant others (Young, 1990). Defectiveness/shame schemas can be paralleled with negative internal working models of the self within attachment theory, as each construct is conceptually symbiotic and seen to have common causes of inconsistent or inadequate care in early caregiving relationships.

Empirically, defectiveness/shame schemas have been found to be significantly higher in women with EDs compared with controls (Leung & Price, 2007; Overton, Selway, Strongman, & Houston, 2005) and were associated with increased ED pathology in a mixed sample of women (Overton et al., 2005). Defectiveness/shame schemas have also been found

to trigger episodes (Hinrichsen, Morrison, Waller, & Schmidt, 2007), and be associated with frequency, of vomiting in women with BN (Waller et al., 2000); and have been found to be higher in women with AN compared with women with anxiety and mood disorders and healthy controls (Deas, Power, Collin, Yellowlees, & Grierson, 2010). These findings suggest that defectiveness/shame may play a particularly important role in the onset and/or maintenance of ED pathology.

Importantly, in support of a self/emotion-regulation model of ED pathology framed in attachment theory, internalised shame and defectiveness/shame schemas are believed to derive in part from compromised early caregiving relationships. Thus, perceptions of low parental care were found to be associated with internalised shame in a clinical sample of women with EDs (L. Keith, Gillanders, & Simpson, 2009). Internalised shame has also been found to fully mediate the association between perceptions of parental overprotection and bulimic symptoms in nonclinical women (Murray, Waller, & Legg, 2000). Further, attachment insecurity has been associated with increased experience of shame in children and adolescents (Muris et al., 2013) and attachment-avoidance and attachment anxiety have both been found to mediate the association between the experience of childhood trauma (e.g., neglect, punishment, or sexual abuse) and ED pathology in adults with EDs (Tasca et al., 2013). Defectiveness/shame schemas have also been associated with anger suppression in women with EDs (Waller et al., 2003). Finally, within attachment theory, having expectations about the unavailability of significant others (associated with attachment-avoidance) may increase emotion inhibition (Dozier & Kobak, 1992) and this down-regulation of emotion, together with depressive symptoms, has been found to mediate the effect of attachmentavoidance on ED pathology in women with EDs (Tasca et al., 2009a).

## **Summary of Internalised Shame and Self-Esteem in Eating Disturbances**

This chapter argues that both self and emotion-regulation models of EDs can be understood within the developmental framework of attachment theory. Whilst there is considerable evidence supporting the links among insecure attachment styles, low self-esteem, internalised shame and defectiveness/shame schemas and ED pathology individually, the distinction between internalised shame and low self-esteem as alternate conceptualisations of low self-worth within ED research is unclear. Self-esteem and internalised shame are highly related but distinct constructs (Kaufman, 1996), although, they are frequently used interchangeably in the ED literature as alternate conceptions of negative feelings about the self. To the author's knowledge, however, research is yet to investigate whether internalised shame is predictive of ED pathology over and above low self-esteem, nor whether such an association exists over and above the impact of depressive symptoms.

Conceivably, a distinction can be drawn between internalised shame and self-esteem. Feelings of low self-worth associated with internalised shame may be seen to relate to feeling that one is fundamentally "not good enough", unlovable, or defective on a whole, as a person. Self-esteem, on the other hand, may be seen to relate to feelings that one is competent, capable or good enough in some ways, and characterised by self-evaluations more with regard to actions, abilities, attributes or parts of the self, or derived from different sources (Goss & Gilbert, 2002). Thus, an individual might believe that he/she is good at something or even talented or special in some unique way, and derive self-esteem from this knowledge, yet still feel she/he is not "good enough" and deep-down fundamentally defective or unlovable (e.g., having thoughts of inauthenticity such as "if they really knew...they would not love me or think I'm good enough").

#### **Body shame**

In addition to the construct of internalised shame, researchers have also explored the

association between ED pathology and shame as it specifically relates to the body (Troop & Redshaw, 2012). Body shame entails experiencing one's physical body as undesirable or unattractive and as a source of the shamed self (Gilbert, 2002). The internalisation of prevailing cultural standards of beauty (i.e., the 'thin-ideal', discussed in more detail later) is said to position women to perceive a disparity between their actual and ideal-body and to experience body shame (McKinley & Hyde, 1996). Further, women who associate achievement of idealised standards of beauty and body weight/shape with their identity, as may be the case in individuals with self-concept deficits who are prone to displace feelings of self-worth onto the body, may be further primed to feel body shame when they perceive their bodies as falling short of these ideals (McKinley & Hyde, 1996). Thus, body shame may increase the risk of psychological distress and ED pathology (Gilbert, 2002).

Specifically, body shame has been found to be higher in female ED patients than non-clinical controls, controlling for depressive symptoms (Swan & Andrews, 2003), and to predict ED pathology, controlling for internalised shame (Burney & Irwin, 2000). Further, whereas both internalised shame and body shame were independently associated with ED pathology in a non-clinical sample of women, only body shame was independently associated with ED pathology in female patients with EDs (Doran & Lewis, 2012). In a longitudinal study of patients with EDs, past body shame was found to independently predict later AN symptoms, controlling for past depressive symptoms and internalised shame, whereas neither internalised shame nor body shame predicted later bulimic symptoms (Troop & Redshaw, 2012).

According to a displacement theory-based self/emotion-regulation model of ED pathology, negative feelings about the self (internalised shame and low self-esteem) are expected to predict both body shame and ED pathology and body shame should mediate the association between negative feelings about the self and ED pathology. Consistent with these

hypotheses, body shame has been found to mediate the association between external shame and ED pathology (Duarte, Pinto-Gouveia, Ferreira, & Batista, 2015) and body shame has been found to mediate the association between low self-esteem and ED pathology in female adolescents (Iannaccone, D'Olimpio, Cella, & Cotrufo, 2016).

# Perfectionism and Unrelenting Standards Schemas

Perfectionism has been consistently identified as a risk factor for ED pathology (Bardone-Cone et al., 2007; Cassin & von Ranson, 2005; Culbert, Racine, & Klump, 2015). Perfectionism can be defined as the propensity to establish and pursue relentlessly high standards, despite negative consequences, and to make stringent self-evaluations (Cassin & von Ranson, 2005). Perfectionism has been found to be associated with body dissatisfaction in a community sample of women (Wade & Tiggemann, 2013) and with ED pathology in clinical-ED samples (Boone, Braet, Vandereycken, & Claes, 2013; La Mela et al., 2015), and when controlling for depressive symptoms (Bernert et al., 2013). It is associated with dietary restraint in women with AN (Lavender et al., 2016) and has been found to be higher in women with AN compared with healthy controls (Bastiani, Rao, Weltzin, & Kaye, 1995) and women with mood disorders, controlling for self-esteem, depressive symptoms and global psychiatric severity (Cockell et al., 2002). Further, perfectionism in early adolescence has been found to predict onset of AN (clinical and subclinical) in young adulthood, controlling for initial ED pathology (Tyrka, Walsron, Graber, & Brooks-Gunn, 2002). Importantly, perfectionism has also be identified as a factor that can interfere with response to treatment within enhanced cognitive-behavioural therapy (CBT) for EDs (CBT-E; Fairburn, Cooper, Shafran, Bohn, & Hawker, 2008).

Perfectionism can also be operationalised as the holding of unrelenting standards schemas within schema therapy, namely, an underlying belief that one must strive to meet very high internalised standards in order to avoid criticism (Young, 1990). Such unrelenting

standards have been found to be highly correlated with other measures of perfectionism in women with clinical EDs (Overton et al., 2005; Waller et al., 2002). Specifically, unrelenting standards schemas have been found to be higher in women with AN compared to women with depression and anxiety (psychiatric controls) and healthy controls (Deas et al., 2010). Unrelenting standards have also been associated with ED pathology in women with AN (Deas et al., 2010), with anger suppression in women with EDs (Waller et al., 2003), and with frequency of binge eating in women with binge eating disorder (BED; Waller, 2003). Further, strong positive associations between perfectionism and emotion inhibition (self-silencing schemas & suppressed anger) have been reported in a mixed sample of AN and healthy control women (Geller et al., 2000), and also with emotion inhibition (self-silencing schemas) and depression in an undergraduate sample (Flett, Besser, Hewitt, & Davis, 2007).

Importantly, in support of a self/emotion-regulation model of ED pathology grounded in attachment theory, cross-sectional research conducted in a large clinical sample of women with EDs found that perfectionism mediated the associations between each of attachment-anxiety and attachment-avoidance and ED pathology, with full mediation in the case of attachment-anxiety and partial mediation in the case of attachment-avoidance (Dakanalis et al., 2014). Further, in two cross-sectional studies of British athletes, one employing structural equation modelling (SEM), found that a path model in which attachment-avoidance predicted self-critical perfectionism, self-esteem, depressive symptoms, and, finally, ED pathology demonstrated good fit for the data (Shanmugam, Jowett, & Meyer, 2011). Further, a second study, employing bootstrapping mediation analysis, found that the association between attachment-anxiety and ED pathology was simultaneously mediated by low self-esteem, self-critical perfectionism, and depressive symptoms (Shanmugam, Jowett, & Meyer, 2012). Finally, as noted above, longitudinal research suggests that perfectionism in young

adolescence predicts the onset of AN in young adulthood, controlling for initial ED pathology (Tyrka et al., 2002).

# **Summary of Early Maladaptive Schemas**

Considered together, schemas relating to defectiveness/shame, emotional inhibition (discussed in the previous chapter), and unrelenting standards have all been found to be significantly higher in women with EDs compared with controls (Leung & Price, 2007; Overton et al., 2005) and associated with increased ED pathology in a mixed sample of women (Overton et al., 2005). These schemas may also play an important role in recovery from EDs and pose a particular challenge for therapy, as such core beliefs are believed to be stable constructs that are self-perpetuating and highly resist to change, thus they may be difficult to challenge via use of conventional CBT (Jones, Leung, & Harris, 2007). For instance, both emotional inhibition and unrelenting standards were found to be higher in women with current-EDs than in those with recovered-EDs and controls (who did not differ from one another), whereas defectiveness/shame schemas were found to be higher in women with current-ED than those with recovered-EDs, and significantly higher in those with recovered-EDs compared with controls (Jones, Harris, & Leung, 2005). Defectiveness/shame and unrelenting standards schemas have also been negatively associated with change in bulimic pathology after 12-weeks of CBT (Leung, Waller, & Thomas, 2000). This suggests that defectiveness/shame beliefs and associated feelings of internalised shame may be particularly resistant to change and may pose an acute challenge for the effectiveness of traditional CBT for EDs (Goss & Gilbert, 2002; Waller, Kennerley, & Ohanian, 2007).

In summary, this chapter has argued that both emotion-regulation and self-regulation models of ED pathology are closely intertwined and best understood as being concurrently anchored in the overarching framework of attachment theory. In the paragraphs that follow, a final set of models also offered to explain ED pathology, namely, sociocultural models, in

particular, self-silencing theory (Jack, 1991) and objectification theory (Fredrickson & Roberts, 1997) will be considered. It will be argued that these models can similarly be seen to fit, to some extent, within an overarching attachment theory view of ED pathology, as insecure attachment styles and disturbances in sense of self (i.e., negative internal models of self) may make some women and girls more likely to internalise certain sociocultural messages.

#### SOCIOCULTURAL MODELS

Sociocultural models have been suggested to explain the increased vulnerability of women and girls, compared with men, to the development of ED pathology (and associated psychological disturbances, such as depression and anxiety). Silencing the self theory (Jack, 1991) builds on Gilligan's (1982) theory of self-development, which proposes that women derive their sense of self-worth from their ability to establish and maintain intimate connections with others (a relational sense of self), whereas men's self-worth is derived from instrumental achievements (an autonomous sense of self). Furthermore, women's sense of self is based on socialised messages stressing the importance of developing feminine traits, such as non-dominance, affiliation, warmth and caring for others, and core self-worth is tied to success in this regard (Jack, 1991). As a result of developing such a relational sense of self, the quality of current relationships, and the wider social context of women in general, are fundamentally important to women's sense of self and their feelings of self-worth (Jack, 1991). Self-silencing theory is grounded in attachment theory and women's self-silencing in the service of relationships has been viewed as an indicator of attachment insecurity that leads to increased loss of self, lower self-worth, and further preoccupation with attachment (Jack, 1991).

A key premise of Jack's theory is that there are costs to self-silencing. As Gilligan (2010) puts it the "stark implication of the self-silencing theory of women's depression is that

the self does not go gently in silence. Whatever the biological substrates or sociological precipitants, depression in women is also a sign or a symptom of a woman's resistance to silencing herself." (p. x). As discussed in the previous chapter, self-silencing has been linked to both depressive symptoms and ED pathology (e.g., Geller et al., 2010; Tan & Carfagnini, 2008). This theory is mentioned again here in the context of how it relates to sense of self, beyond emotion inhibition. Here again the difficulty in differentiating emotion-related and self-related components of such theories is apparent, given the inherent overlap among these constructs. Instead, deficits in sense of self and difficulties with emotion (specifically, emotion inhibition) are thus argued to be better captured within an integrated self/emotion-regulation model of ED pathology grounded in attachment theory.

Sociocultural models further argue that socialisation processes contribute to pressures to conform to feminine ideals of both behaviour and beauty. This focus on the body as a commodity leads to a shift from perceiving oneself as a whole authentic self with unique thoughts, feelings, and characteristics, to an externalised view of the self as an object — what objectification theory (Fredrickson & Roberts, 1997) refers to as 'self-objectification'.

Sociocultural models further suggest that the tendency for women to place importance on body weight and shape and to use the standards of others to inform self-evaluations may predispose them to the development of ED pathology (Frank & Thomas, 2003). Externalised self-perceptions (objectified body consciousness or the extent to which women experience their body as an object and view it from an outside perspective) have been found to be positively associated with ED pathology and negatively related to body-esteem in adolescent girls (Lieberman, Gauvin, Bukowski, & White, 2001). They have also been found to be significantly associated with cognitive aspects of ED pathology, over and above body weight, body dissatisfaction, perceived importance of body shape and self-silencing in undergraduate women (Frank & Thomas, 2003).

According to objectification theory (Fredrickson & Roberts, 1997), self-objectification leads to habitual body monitoring (body surveillance), body shame, appearance anxiety, loss of interoceptive awareness, and, in turn, depressive symptoms and ED pathology. In support of this theory, a number of studies have found evidence of a link between self-objectification and ED pathology (e.g., Peat & Muehlenkamp, 2011), self-objectification and body shame (e.g., Greenleaf & McGreer, 2006) and body surveillance and body shame (Calogero & Thompson, 2009). Thus, evidence from non-clinical samples of women suggests, first, that body shame mediates the association between body surveillance and ED pathology (Moradi, Dirks, & Matteson, 2005; Tylka & Hill, 2004) and between self-objectification and ED pathology (Noll & Fredrickson, 1998); second, that body surveillance and body shame were jointly associated with ED pathology both directly and indirectly via depressive symptoms (Muehlenkamp & Saris-Baglama, 2002); and third, that body surveillance and body shame each mediate the association between self-objectification and both ED pathology (Calogero, 2009; Tiggemann & Slater, 2001) and depressive symptoms (Tiggemann & Kuring, 2004; Tiggemann & Williams, 2012). Further, body shame was found to mediate the association between self-objectification and drive for thinness in a clinical sample of women with EDs (Calogero, Davis, & Thompson, 2005).

While objectification theory identifies sexual objectification as a cultural experience of all women that leads to self-objectification, body surveillance, body shame, and, potentially depressive symptoms and ED pathology, objectification theory does not necessarily account for individual differences in levels of self-objectification among women. Further, explanations of why an almost 'normative discontent' with body weight and shape (Rodin, Silberstein, & Striegel-Moore, 1984) experienced by many women leads to the development of severe body image disturbances and clinical ED pathology in only a minority, rather than all women likewise remains unclear. Although evidence exists that internalisation

of media/cultural messages of the thin ideal leads to increased self-objectification and body dissatisfaction in women (Morry & Staska, 2001). Further, it has been suggested that impairments in identity development and the lack of a clearly defined sense of self may pose a particular vulnerability for women to internalise cultural messages about body weight and shape, and thereby contribute to the development of ED pathology (Stein & Corte, 2003; Vartanian, 2009).

The question of whether internalised shame may predict externalised self-perceptions, in the form of self-objectification and body surveillance, and whether such externalised self-perceptions mediate the associations among internalised shame, body shame, depressive symptoms and ED pathology, are yet to be addressed. Investigation of such associations and processes in women thus represents a gap in the literature.

#### SUMMARY

Various theoretical conceptualisations of the psychosocial processes and constructs that may contribute to the development and maintenance of ED pathology have been offered in the ED literature. From a developmental position, insecure attachment styles, disruptions in self-development, difficulties in emotional-development and emotion-regulation, and sociocultural influences, have all been implicated in the development and maintenance of ED pathology. It is the position of this thesis that, essentially, these theories and models are arguing much the same thing; and when anchored within the theoretical framework of attachment theory, can be seen as largely complimentary. That is, within the context of social pressures particular to women, undue focus is placed on attainment of physical perfection (in the form of the thin ideal) and inhibition of emotion, thoughts and behaviours deemed socially undesirable. However, some women are more vulnerable to such sociocultural pressures than others — perhaps because of insecure attachment styles, unhealthy sense of self, and maladaptive emotion-regulation strategies. Attachment theory suggests that each of these constructs has

their origins in deficiencies in early childhood relationships. Against this backdrop, the current thesis investigated a self/emotion-regulation model of ED pathology grounded in attachment theory, with a view to integrating these inter-related conceptions of EDs and elucidating some of the structural associations among the constructs embedded in them.

# Chapter 4

**The Current Research** 

#### THE CURRENT RESEARCH

In sum, the broad goals of the current research were to investigate both emotion-regulation and self-regulation models of ED pathology, and to integrate elements of each in exploring a combined self/emotion-regulation model of ED pathology grounded in attachment theory.

The specific aims of the current research project (presented in Chapter 1) were addressed in a series of distinct but related studies prepared in the form of journal articles submitted for publication or prepared for submission for publication (Chapters 5 through 8). As mentioned in Chapter 1, two different samples were employed so that initial findings from the first phase of the research project, addressing the first and third aims, could be further investigated in the second phase of the research in a separate sample. Further, in order to maximise recruitment numbers, the studies addressing the first and third aims (Study 1 and Study 3A) were combined in one larger online survey during the first phase of the research (survey 1). In the second phase of the research project, again with the objective of maximising sample numbers for each study, the studies addressing the second, third and fourth aims were combined into a second large survey (survey 2) containing two parts. As stated earlier, Survey 1 (data collected from 23 October 2012 to 23 January 2013) yielded data for Study 1 (Chapter 5) and Study 3A (Chapter 7). In a different sample, Survey 2 (data collected from 14 May 2013 to 11 August 2014) Part (i) collected data for Study 2 (Chapter 6) and Study 4 (Chapter 8) and Survey 2 Part (ii) collected data for Study 3B (Chapter 7).

Given that the research sought to investigate mediation processes, use of a longitudinal design would have been optimal. However, this was deemed to be infeasible in the current research due to the likelihood in an adult population of insufficient change in the outcome variable expected over the timeframe for a doctoral thesis, a lack of empirical evidence to support a rationale for the timing of waves in order to capture meaningful changes

in the relationships among the study variables, and difficulties in obtaining sufficiently high completion rates across waves to ensure valid analyses. The recruitment of large crosssectional samples and the use of relatively sophisticated analytic methods (PROCESS mediation analyses and structural equation modelling with bootstrapping procedures) was deemed the best possible compromise, hence this approach was chosen for the current research. Further, the recruitment of heterogeneous samples of women was seen to be important in terms of ensuring variability in the level of key study variables, to maximise the generalisability of the findings, and to ensure sufficiently large samples to allow for subgroups of participants with probable-ED pathology and no-ED pathology to be identified in the second phase of the research. Accordingly, women were recruited from a number of sources including psychology undergraduate students, members of the broader community, and visitors to support websites related to eating disorders. The samples comprised adult women, with adolescents and men excluded in order to control for potential gender and maturational influences on the associations between ED pathology and other study variables. Whilst it is recognised that men and adolescent girls are certainly populations of interest in understanding ED pathology, the current research sought to extend research previously conducted primarily with adult females (the population for which the largest body of evidence currently exists).

The four broad aims of the current research were addressed in four separate studies (the third of which comprised two separate studies from surveys 1 and 2) as follows:

Paper 1 (Chapter 5): Emotion Inhibition, Negative Emotional Experience, and Eating Disorder Pathology in Women (manuscript submitted to the *Journal of Abnormal Psychology*, March 2017). Whilst there is growing evidence linking different forms of emotion inhibition to ED pathology, few studies have examined their relative importance in accounting for variance in ED pathology, nor the potential role of negative emotionality in

moderating or mediating these associations, and no study has examined these different possible pathways whilst controlling for the occurrence of depressive symptoms. This study (Study 1) aimed to address this gap in the literature. First, the study sought to examine the associations between different forms of emotion inhibition, namely negative beliefs about emotion, ambivalence over emotional expressiveness, silencing-the-self and divided self schemas, in order to explore the unique and shared effects of these variables in explaining variance in ED pathology, while controlling for depressive symptoms. Second, the study sought to examine whether and how negative emotionality might influence the associations between each form of emotional inhibition and ED pathology. In view of preliminary evidence suggesting that both moderating and mediating effects may be worthy of investigation, both of these possibilities were considered, again controlling for depressive symptoms in each case.

Paper 2 (Chapter 6): Beliefs About Emotion and Eating Pathology in Women:
The Role of Emotion Regulation, Negative Emotionality, and Depressive Symptoms
(manuscript submitted to Clinical Psychology & Psychotherapy, March 2017). In accordance with affect-regulation models of ED pathology (Corstorphine, 2006), this study investigated emotional experience in EDs and considered the role played by negative beliefs about emotion and various emotion-regulation strategies. Specifically, Study 2 first aimed to establish whether the various emotion-regulation strategies (rumination, reflection, emotional reflection, reappraisal, and suppression) and negative beliefs about emotion were each individually associated with ED concerns; second, whether emotional reflection would explain unique variance in ED pathology over and above general reflective capacity; and third, given the similarity between the various emotion-regulation strategies and negative beliefs about emotion, to determine if they overlap or were independently significant in contributing to variance in ED pathology. Fourth, this study tested a path model to determine whether the

association between negative beliefs about emotion and ED pathology was mediated by the various emotion-regulation strategies, negative emotionality, and depressive symptoms. Finally, the study tested whether such a model would demonstrate good fit within subgroups of women with probable-ED and no ED diagnoses separately.

Paper 3 (Chapter 7): Feelings about the Self and Body in Eating Disturbances:

The Role of Internalised Shame, Self-Esteem, Externalised Self-Perceptions, and Body
Shame (manuscript submitted to Self and Identity, March 2017). Study 3 explored in two
independent samples of women a displacement theory-based model of ED pathology,
whereby negative feelings about the self are displaced onto the body (in the form of increased
body shame), thereby promoting ED pathology. Study 3A examined the distinction between
low self-esteem and internalised shame as two distinct, but closely related, conceptualisations
of low self-worth and assessed their independent and unique contributions in explaining
variance in ED pathology, over and above depressive symptoms and one another. The
possibility that body shame mediates the association of each with ED pathology was also
tested. Study 3B aimed to explore, using SEM, a path model that would extend the
displacement theory of ED pathology by incorporating elements of objectification theory,
specifically by including self-objectification and body surveillance as additional mediators of
the association between internalised shame and body shame, depressive symptoms and ED
pathology.

Paper 4 (Chapter 8): The Role of Attachment Dynamics, Maladaptive Schemas, and Body Shame in Contributing to Eating Pathology in Women (manuscript submitted to the *Journal of Personality and Social Psychology*, March 2017). Study 4 explored a self/emotion-regulation model of ED pathology grounded in attachment theory, wherein insecure attachment styles (avoidance and anxiety) are thought to give rise to increased experience of shame schemas, and the development of emotional inhibition and unrelenting

standards schemas in response. Negative feelings about the self (defectiveness/shame), unexpressed emotions, and perfectionistic standards are then displaced onto the body, leading to increases in body shame and depressive symptoms, and, in turn, ED pathology (the pursuit of an idealised body weight/shape, dietary restriction, binge eating, and compensatory behaviours) employed as a means of regulating such painful emotions and internal experiences and endeavouring to enhance feelings of self-worth (Goss & Gilbert, 2002). Much of the empirical support for such a model to date, however, is derived from drawing together disparate and overlapping sources of evidence for portions of the model. To evaluate this conception, path models incorporating these developmental features of EDs were explored and model fit tested both in the overall sample and within subgroups of women with probable-EDs and no EDs separately.

# Chapter 5

# **Emotion Inhibition, Negative Emotional Experience, and Eating**

# **Disorder Pathology in Women**

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# **Author Contributions**

First author Kim Woodward was responsible for conceptualisation, design, and conduct of the current study, managed data collection, undertook all statistical analyses, conducted the literature searches, and wrote the first draft of the manuscript. Author Doris McIlwain contributed to the conceptualisation and design of the study at a supervisory level. Author Jonathan Mond contributed to revisions of the manuscript.

#### Abstract

**Objective:** To investigate the role of various forms of emotion inhibition and negative emotional experience in predicting eating disorder (ED) pathology. Methods: Four hundred and three women completed measures of beliefs about unacceptability of negative emotions (BAE), ambivalence over emotional expression (AEE), silencing-the-self schemas, dividedself schemas, negative emotionality, depressive symptoms, and ED pathology. **Results:** Individually, BAE, AEE, silencing-the-self, divided-self, and negative emotionality were each significantly independently associated with ED pathology, controlling for depressive symptoms. When considered together BAE and depressive symptoms were each independently associated with ED pathology, over and above the effects of the other emotion inhibition constructs and negative emotionality. Further, negative emotionality significantly mediated each of the associations between BAE, AEE, silencing-the-self, and divided-self, and ED pathology, controlling for depressive symptoms. Conclusions: BAE appears to be an important predictor of ED pathology, above other emotion inhibition constructs, negative emotionality and symptoms of depression. Further, one of the possible mechanisms by which various forms of emotion inhibition are associated with ED pathology appears to be via increases in the experience of negative emotion. Results support an emotion-regulation model of ED pathology and the implications for therapy are discussed.

**Key words/phrases:** Eating disorder pathology, emotion inhibition, negative emotionality, emotion-regulation

#### Introduction

Eating disorders (EDs) have long been linked with difficulties in the processing and regulation of emotion (Bruch, 1973, 1978; Fox & Goss, 2012). From an emotion regulation perspective, the symptoms of eating disorders (EDs) can be thought of as serving a function in the inhibition of emotions (Bekker & Spoor, 2010; Fox & Power, 2009). That is, ED symptoms are seen as serving a maladaptive emotion regulatory function in the form of a 'distraction from', 'blocking', or 'escape from awareness' of experiencing and expressing negative emotion (Elmore & de Castro, 1990; Heatherton & Baumeister, 1991; Root & Fallon, 1989), and thus constitute a form of avoidant coping (Neckowitz & Morrison, 1991; Troop et al., 1994). Emotion inhibition is taken to include both efforts to suppress sensations, urges, thoughts, and feelings associated with the experience of emotion and efforts to prevent emotion expression (Krause et al., 2003). It is a maladaptive means of coping that can be conceptualised as including a number of distinct but related constructs and operationalised in a variety of different ways. Thus, in the ED literature, emotion inhibition has been viewed, among other things, as: emotional suppression (Gross & John, 2003); conflict or ambivalence over emotional expression (King & Emmons, 1990); self-silencing (Jack & Dill, 1992); low emotional expressiveness (King & Emmons, 1990); the early maladaptive schema of emotional inhibition (Young, 1990); and avoidance of affect (Corstorphine, Mountford, Tomlinson, Waller, & Meyer, 2007).

Regardless of how it is operationalised, it is apparent that the inhibition of emotion has both physiological and psychological costs. For instance, individuals who chronically suppress anger have been found to have elevated blood pressure (S. P. Thomas, 1997). Suppression of emotion is also associated with impairment in various aspects of psycho-social functioning (Gross & John, 1997, 2003), due in part to the fact that it can actually be counterproductive and paradoxically bring about the very emotions and thoughts being

suppressed (for review see Wenzlaff & Wegner, 2000). In terms of associations with ED pathology, emotional inhibition has been found to be associated with greater body dissatisfaction in female undergraduates, even after controlling for depressive symptoms (Hayaki, Friedman, & Brownell, 2002a), and with greater body dissatisfaction, drive for thinness and bulimic symptoms in a clinical sample of women with EDs (Ioannou & Fox, 2009). Evidence for an association between ED pathology and several forms of emotional inhibition in particular is mounting, namely, ambivalence over emotional expression and self-silencing schemas, and the closely related construct of beliefs about the unacceptability of experiencing and expressing emotions.

# **Ambivalence Over Emotional Expressiveness**

It has been suggested that it is how conflicted one is over their level of emotional expression or inhibition, rather than one's actual level of emotional expressiveness, that is associated with physical and psychological distress (Kennedy-Moore & Watson, 2001; King & Emmons, 1990). The construct of ambivalence over emotional expression (AEE) relates to an individual's experience of psychological conflict over their level of emotional expression, where there is a desire to do so, or the presence of emotional expression, together with regret at having done so (King & Emmons, 1990). AEE has been found to be associated with alexithymia (the difficulty identifying and describing feelings in words) and various measures of psychological distress in non-clinical samples (King & Emmons, 1990, 1991; King, Emmons, & Woodley, 1992), and with depression, hopelessness and suicidal ideation in clinical psychiatric samples (Lynch, Robins, Morse, & Krause, 2001).

Research exploring the relationship between AEE and ED pathology has found AEE to be higher among women with EDs than non-clinical controls (Forbush & Watson, 2006) and to be associated with ED pathology in a non-clinical female undergraduate sample, over

and above the impact of depressive symptoms and sociotropy (a need for and preoccupation with securing close interpersonal bonds; Krause et al., 2000). In a different non-clinical sample, however, AEE was not independently associated with ED pathology when controlling for alexithymia and emotional expression (Quinton & Wagner, 2005). Hence the nature of the association between AEE and ED pathology and the extent to which this association exists independent of other emotion inhibition and emotional style constructs and depressive symptoms requires further investigation.

# **Self-Silencing in the Service of Relationships**

Another means by which individuals may inhibit emotion is via self-silencing. Silencing the self theory (Jack, 1991) proposes that women value the maintenance of close positive interpersonal connections and, within a social history of gender inequity, develop a range of cognitive/relational schemas (e.g., putting the needs of others first, self-sacrifice, and self-silencing) as a means of achieving and maintaining intimacy. Women's sense of self is said to be relationally embedded and by silencing their authentic feelings, thoughts, and behaviours in the service of relationship maintenance (e.g., to avoid conflict or fit the expectations of others), women run the risk of experiencing loss of self, loss of voice, low self-esteem, and depression (Jack, 1991; Jack & Dill, 1992). This theory of silencing the self ('self-silencing') has since been applied to ED pathology (Geller et al., 2000), the over-representation of which among women is well-known (American Psychiatric Association, 2000).

Jack and Dill (1992) developed The Silencing the Self Scale (STSS) to measure self-silencing among women and to capture the specific schemas of externalised self-perceptions (evaluating the self based on the standards of others/society/norms), care as self-sacrifice (placing the needs of others ahead of the self in order to secure attachments), silencing-the-self (attempting to avoid conflict or relationship loss by inhibiting expression of one's

thoughts, feelings, or behaviour), and divided-self (the experience of developing internal anger and hostility whilst presenting an externally compliant self). Of specific relevance to emotion inhibition are the schemas of silencing-the-self and divided-self.

Empirical evidence supports an association between self-silencing and depression (e.g., Cramer et al., 2005; Tan & Carfagnini, 2008) and a growing number of studies have examined the link between self-silencing and various forms of ED pathology. Specifically, both silencing-the-self and divided-self schemas were found to be strongly positively associated with measures of body dissatisfaction and drive for thinness in a clinical sample of 149 girls diagnosed with EDs (Buckholz et al., 2007) and silencing-the-self and anger suppression were significantly higher in girls who scored high in ED pathology compared with those who scored low, in two school-based samples of female adolescents (Norwood et al., 2011; Zaitsoff et al., 2002). In female undergraduates, self-silencing was found to be positively associated with body dissatisfaction and various forms of ED pathology (Shouse & Nilsson, 2011) and both self-silencing and anger suppression have been found to associated with ED pathology in community-based samples, independent of their associations with each other (Morrison & Sheahan, 2009; Piran & Cormier, 2005). Further, silencing-the-self was found to be positively associated with weight and shape concerns, dietary restraint and bulimic behaviours, and divided-self positively associated with weight and shape concerns and uncontrolled eating, in two female undergraduate student samples (Frank & Thomas, 2003; Ross & Wade, 2004).

In a clinical sample, women with anorexia nervosa (AN) scored significantly higher than age-matched psychiatric and healthy control women on silencing-the-self, divided-self, anger suppression and depressive symptoms and across the combined sample silencing-the-self, divided-self and anger suppression were positively associated with body dissatisfaction (Geller et al., 2000). Higher scores on measures of self-silencing, and depression, among

women with AN, compared with healthy controls, have also been observed in at least two other clinical samples (Hambrook et al., 2011; Oldershaw et al., 2012). However, in the one study in which group differences in measures of self-silencing were assessed over and above the effects of depressive symptoms (Hambrook et al., 2011), these differences, and the partial correlations between silencing-the-self and divided-self and ED pathology, were no longer significant when age, depressive symptoms and anxiety were controlled.

Thus, while there is good preliminary evidence to suggest a role for silencing-the-self and divided-self schemas in accounting for variance in ED pathology, further research is needed to determine whether and to what extent associations between silencing-the-self, divided-self and ED pathology remain when controlling for depressive symptoms. Further research is also needed to examine the association of silencing-the-self and divided-self and ED pathology taking into account associations between each and other forms of emotion inhibition, namely, ambivalence over emotional expression (as outlined above) and beliefs about the unacceptability of negative emotions (as outlined below), in order to determine the unique contributions of these constructs to variance in ED pathology.

# **Beliefs About the Unacceptability of Negative Emotions**

Another construct that has been identified as co-occurring with emotional inhibition in individuals with EDs (Corstorphine, 2006), and which aligns with various operational definitions of emotional inhibition (particularly the early maladaptive schema of emotional inhibition; Young, 1990), is that of beliefs about the unacceptability of experiencing or expressing negative emotions (BAE). Such beliefs are thought to have implications for emotion processing and regulation (Rimes & Chalder, 2010) and, more specifically, to be implicated in the development and maintenance of ED pathology (Corstorphine, 2006). According to this theory, individuals who grow up in environments that are emotionally invalidating may develop beliefs that emotions are 'bad' and that experiencing and expressing

emotions is dangerous and to be avoided (Corstorphine, 2006). Such beliefs may then activate secondary negative emotions (e.g., shame about feeling loneliness or guilt about feeling sadness or anger), which may, in turn, give rise to ED symptoms (focus on weight & shape and their control, binge eating, compensatory behaviours, and restriction), as a short-term strategy to alleviate the experience of these emotions (Corstorphine, 2006). Based on this affect-regulation model, Corstorphine (2006) developed cognitive-emotional-behaviour-therapy for EDs (CEBT-ED), which aims to reduce the experience of secondary negative emotions and, in turn, the need for ED symptoms, by challenging underlying beliefs about the unacceptability of experiencing and expressing emotions.

Empirical support for the role of BAE in ED pathology is currently limited and the effectiveness of CEBT-ED is unclear. However, negative attitudes towards emotional expression have been found to be associated with ED pathology in non-clinical samples of women (Haslam et al., 2012; Meyer et al., 2010) and there is preliminary support for this association from clinical samples. For instance, BAE was found to be significantly higher among individuals with AN compared with healthy controls (Hambrook et al., 2011; Jansch et al., 2009; Oldershaw et al., 2012) and higher among individuals with full-threshold bulimia nervosa (BN) than those with sub-threshold BN (Lavender et al., 2014). Further, BAE was found to be positively associated with ED pathology, depressive symptoms and anxiety in a mixed sample of individuals with AN, individuals recovered from AN, and healthy controls (Oldershaw et al., 2012), and with ED pathology after controlling for depressive symptoms and anxiety in a clinical sample of individuals with AN (Hambrook et al., 2011). In Lavender et al.'s (2014) study, referred to above, non-acceptance of emotional states was positively associated with ED pathology in a clinical sample of 72 women and 8 men with full- and sub-threshold BN.

However, as is the case with silencing-the-self and divided-self schemas, it is unclear whether the association between BAE and ED pathology exists independent of the effects of depressive symptoms. Depressive symptoms were not controlled for in the Lavender et al. (2014) study and while depressive symptoms were measured in the studies of Jansch et al. (2009) and Oldershaw et al. (2012), the issue of whether group differences in BAE between individuals with AN and healthy controls remained after controlling for depressive symptoms was not considered. Nor was it clear whether the positive association between BAE and ED pathology observed in Oldershaw et al.'s (2012) study remained significant when controlling for depressive symptoms and anxiety. In Hambrook and colleagues' (2011) study, a significant positive association between BAE and ED pathology among individuals with AN remained after controlling for symptoms of depression and anxiety, whereas differences in BAE levels between participants with AN and healthy controls were no longer significant after controlling for age, depressive symptoms, and anxiety. Thus, while there is preliminary evidence to suggest that BAE is associated with ED pathology, the nature of this association, and its existence when controlling for depressive symptoms and other forms of emotion inhibition, requires further investigation.

## The Role of Negative Emotionality

An additional consideration, which cuts across that of the associations between these different forms of emotional inhibition and ED pathology, concerns the potential moderating and/or mediating role of negative emotionality per se. ED pathology is strongly associated with the experience of negative emotions (Agras & Telch, 1998; Johnson & Larson, 1982), negative emotion is a diagnostic feature of major depressive disorder (American Psychiatric Association, 2013), depression is a common comorbidity of ED pathology (e.g., Agras, 2001; Zonnevylle-Bender et al., 2004), and difficulties with emotion regulation have been viewed as a transdiagnostic concern and as characteristic of both depression and EDs (Brockmeyer,

Bents, et al., 2012). Thus, when investigating the association between emotion inhibition (and the associated schema and beliefs that may underlie its various forms) and ED pathology, it seems important to consider the potential roles played by both negative emotionality and depressive symptoms.

While there is little direct evidence bearing on whether and how negative emotionality and emotion inhibition might interact in giving rise to ED pathology, the potential for an effect of this kind is suggested by research in cardiac patients in which the tendency to inhibit emotional expression has been found to moderate the association between negative emotional experience and poor long-term health outcomes (including death); that is, only individuals who score high on both negative emotion and emotion inhibition have been found to be at increased risk of negative health outcomes (Denollet et al., 2006; Denollet et al., 1996). One study failed to confirm the hypothesis, however, that negative emotion moderates the association between either of two possible coping styles (avoidant versus emotion-oriented) and emotional eating (Spoor, Bekker, Van Strien, & van Heck, 2007).

Alternatively, or in addition, emotion inhibition may give rise to ED pathology via an increase in the experience of negative emotions. A mediating influence of this kind accords with Corstorphine (2006)'s affect-regulation model and is supported by evidence that while expressive suppression may inhibit the behavioural response associated with negative emotion, it does not reduce the subjective experience of this emotion and, indeed, paradoxically exacerbates this experience (John & Gross, 2004). Further, evidence suggests that the use of emotion inhibition, suppression, and experiential avoidance to circumvent unwanted emotions is conducive to psychological distress and impairment in psycho-social functioning more generally among undergraduate students at least (Kashdan et al., 2006).

#### **The Present Study**

Despite, growing evidence linking emotion inhibition to ED pathology, few studies have examined the relative importance of different various forms of emotion inhibition in accounting for variance in ED pathology, nor the potential role of negative emotionality in moderating or mediating these associations, and no study has examined these different possible pathways while controlling for the occurrence of depressive symptoms.

The goal of the current study was to address this gap in the literature. First, we sought to examine the associations between several different forms of emotion inhibition which have been linked with ED pathology in previous research, namely BAE, AEE, silencing-the-self and divided-self, in order to explore the unique and shared contributions of these variables in explaining variance in ED pathology, while controlling for depressive symptoms. Second, we sought to examine whether and how negative emotionality might influence the associations between each form of emotional inhibition and ED pathology. In view of preliminary evidence suggesting that both moderating and mediating effects may be worthy of investigation, both of these possibilities were considered in the current study, again controlling for depressive symptoms in each case.

The following specific hypotheses were formulated: (1) BAE, AEE, silencing-the-self, divided-self, and negative emotionality will be individually associated with higher levels of ED pathology; (2) these associations will remain significant when controlling for symptoms of depression; (3) when fitted together, the variance in ED pathology explained by BAE, AEE, silencing-the-self, and divided-self will be overlapping, such that the independent effects of each will be reduced; (4) negative emotionality will moderate the associations between one of more forms of emotion inhibition – BAE, AEE, silencing-the-self or divided-self – and ED pathology; and (5) the effects of BAE, AEE, silencing-the-self and divided-self on ED pathology will each be either partially or fully mediated by negative emotionality.

#### Method

# **Participants**

Participants were 403 women aged 18 years or older ( $M_{age} = 23.9$ , SD = 6.9, age range: 18-62 years; the "full sample"). Of these, 102 were first year psychology students who participated in return for course credit (the "student sample"), 175 were recruited via advertisements displayed on eating disorder support websites (the "web sample"), and 126 were recruited via flyers displayed around the Macquarie University campus and via email invitation to participate forwarded to prospective participants by family and friends of the researchers (the "community sample"). Inclusion criteria were being female and aged 18 years or older. Males were not included because of the need for unduly large sample sizes to examine associations with ED pathology in this demographic in community-based samples. Only adult females were included because of the complex nature of the constructs under investigation and in order to control for maturational influences relating to the manifestation of these constructs. From a total of 448 respondents, data for two respondents who were aged less than 18 years and 45 respondents who identified as males were excluded.

#### Measures

Eating Attitudes Test (EAT-26). ED pathology was measured dimensionally via the EAT-26 (Garner et al., 1982). EAT-26 a 26-item multiple-choice scale that measures attitudes towards eating and includes items assessing *dieting*, *bulimia* and *food* preoccupation, and *oral control*. Items are rated on a 6-point scale and scored such that never, rarely, and sometimes = 0, often = 1, usually = 2 and always = 3, with the one negatively worded item reverse scored. EAT-26 scores can be utilised in a number of ways, including to obtain subscale scores or to create cut-off categories for individuals at risk of EDs (with scores of 20 and above indicating ED risk). In the current study EAT-26 items were summed to yield a total score from 0 to 78. The EAT-26 has been found to have high internal reliability (Garner

& Garfinkel, 1979; Garner et al., 1982), and correlate positively with a number of validated measures of ED pathology, including clinical classification (Garner et al., 1982; Lee, Kwok, Liau, & Leung, 2002; Mintz & O'Halloran, 2000). Further, EAT-26 is one of the most widely used and validated screening tools for the assessment of ED risk (Garner et al., 1982).

It is acknowledged that EAT-26 is not a diagnostic tool for the purposes of identifying DSM-5 EDs and that EAT-26 scores above 20 may capture individuals without full-syndrome AN or BN and thus overestimate actual cases of EDs. However, a dimensional approach to measuring ED pathology via EAT-26 total scores was seen as beneficial in the present study in order to capture sub-threshold and even lower levels of ED pathology, which may be associated with significant distress and/or impairment and worthy of investigation (Rockert et al., 2007; Stice et al., 2013; Stice et al., 2009).

**Depression, Anxiety and Stress Scale (DASS-21).** Depressive symptoms were measured using the 7-item depression subscale of the DASS-21 (Lovibond & Lovibond, 1995a). Items are rated on a 4-point scale (from 0 = did not apply to me at all to 3 = applied to me very much, or most of the time) and summed and then multiplied by two to yield a total score from 0 to 42. The DASS-21 depression subscale has been found to have adequate internal consistency (Cronbach's  $\alpha = .81$ ) and good construct validity in correlating highly with other validated measures of depression (Lovibond & Lovibond, 1995b).

**Differential Emotions Scale IV-A (DES IV-A).** Negative emotion was assessed using the negative emotionality subscale of the DES IV-A (Izard, 1982), a 36-item trait measure of the experience of discrete emotions, namely, interest, joy, surprise, sadness, anger, disgust, contempt, inner-directed self-hostility, fear, shame, shyness, and guilt. The DES-IV asks participants to rate how often in their daily life they feel each emotion statement on a 5-point scale (where  $I = rarely \ or \ never$ ,  $2 = hardly \ ever$ , 3 = sometimes, 4 = often, and  $5 = very \ often$ ). The three items tapping the emotions of sadness, anger, disgust, contempt, inner-

directed self-hostility, fear, shame, shyness, and guilt were summed to obtain a score for negative emotionality. The DES-IV has been found to have moderate to strong internal consistency for individual subscales (α's in the range of .60 to .85), demonstrating stability over a 3-years; and the negative emotionality subscale has good construct validity and has been found to be highly positively correlated with measures of depression, anxiety and neuroticism (Blumberg & Izard, 1985, 1986; Izard, Libero, Putnam, & Haynes, 1993).

Beliefs About Emotions Scale (BAES). The BAES (Rimes & Chalder, 2010) is a 12item scale that assesses beliefs about the unacceptability of experiencing and expressing negative emotions. The BAES contains items such as "It would be a sign of weakness to show my emotions in public", each of which is rated on a 7-point Likert scales (where  $\theta = totally$ disagree,  $\theta = totally$  agree). The BAES has demonstrated unidimensionality, high internal consistency ( $\theta = totally$  agree) and construct validity, correlating highly with measures of perfectionism, dysfunctional attitudes, self-sacrifice, depression, and anxiety (Rimes & Chalder, 2010). Furthermore, the BAES has been found to demonstrate sufficient sensitivity to detect meaningful reduction in BAE following completion of CBT in individuals suffering from chronic fatigue syndrome (Rimes & Chalder, 2010).

Ambivalence Over Emotional Expressiveness Questionnaire (AEQ). The 28-item AEQ (King & Emmons, 1990) was used to assess AEE. The AEQ includes items such as: "I often can't bring myself to express what I am feeling" and "I feel guilty after I have expressed anger to someone", agreement with which is rated on a scale from 1 to 5 (I = never feel that way, 5 = f frequently feel that way). A total score (ranging from 28 to 140) is obtained as the sum of the 28 items, with higher scores indicated higher levels of AEE. The AEQ has been found to have good internal consistency (Cronbach's  $\alpha = .89$ ), test-retest reliability (at a 6-week interval, r = .78), and construct validity, having been found to be negatively correlated with both self-reported and peer-rated emotional expressiveness and positively correlated with

several indices of psychological distress (King & Emmons, 1990).

Silencing the Self Scale (STSS). The STSS was developed by Jack and Dill (1992) to measure self-silencing among women and contains subscales designed to capture the schemas of externalised self-perceptions, care as self-sacrifice, silencing the self and divided self. In the current study, the aspects of self-silencing specifically related to emotion inhibition were assessed via the silencing-the-self and divided-self subscales. The silencing-the-self and divided-self subscales are designed to assess, respectively, inhibition of self-expression in the service of relationship maintenance and the experience of internal anger and hostility whilst presenting an externally compliant self. The silencing-the-self and divided-self subscales contain 10-items and 6 items, respectively; each rated on a 5-point Likert scale from 1 (*strongly disagree*) to 5 (*strongly agree*). Negatively worded items are reverse scored and items summed to obtain total scores from 10 to 50 (silencing-the-self) or 6 to 30 (divided-self). Both subscales have been found to have good internal consistency (silencing-the-self:  $\alpha = .83$  & divided-self:  $\alpha = .78$ ), test-retest reliability, and significantly correlated with a well-validated measure of depression (Jack & Dill, 1992).

#### Procedure

Data were collected as part of a 30-minute online survey using Qualtrics. Participants were provided a brief description of the research and invited to indicate their informed consent to participate before commencing the survey. Participants were asked their age (to the nearest year), height (to nearest cm), weight (to nearest kg), and gender. The survey then presented the study measures in randomised order (presentation of items within scales was also randomised) so as to minimise order effects. Participants in the student sample received course credit for their participation, while those in the community and web samples were offered entry in a prize draw for one of five AUD\$50 iTunes gift vouchers. The study methods were approved by the Macquarie University Human Research Ethics Committee.

#### **Statistical Analysis**

All analyses were conducted using the IBM Statistical Package for Social Sciences (SPSS) version 22. Preliminary analysis comparing samples across recruitment methods on each of the study variables was conducted using General Linear Model Analysis of Variance (GLM ANOVA) with simple contrast tests (using a Bonferroni adjusted p-value of < .017 for significance) and GLM ANOVA was used to test for interaction effects between recruitment groups and study variables. Pearson correlations were used to evaluate the strength and direction of the bivariate associations between study variables. Multiple linear regression analyses were used to determine the extent to which the IVs were independently and collectively associated with ED pathology, including the use of hierarchical regression analysis to test for moderation effects. Given the number of analyses, a conservative alpha level of .01 was employed for this analysis. Missing Value Analysis was performed using SPSS and missing data found to be Missing Completely at Random (MCAR) according to Little's MCAR test ( $\chi^2_{(73)} = 61.309$ ; p = .834) (Little, 1988), thus missing data were excluded pairwise (Garson, 2015). Mediation analyses were conducted using the PROCESS tool (A. F. Hayes, 2013), which entailed using 10,000 bootstrap samples to estimate the size of the total, direct, and indirect effects and to yield bias-corrected confidence intervals (CIs) around each effect in order to test for statistical significance at the 99% confidence level. Given that age and body mass index (BMI) have been identified in past research as inversely associated with ED pathology, age and BMI were included as covariates in all primary analyses.

## Results

## **Preliminary Analysis**

The GLM ANOVA contrast tests revealed the web sample scored significantly higher on each of the study variables than the community and student samples (see Appendix A).

The community and student samples differed only on divided-self subscale, where the

community sample reported significantly higher scores than the student sample. These differences were expected given the more clinical presentation of participants recruited via ED self-help websites. Additional analysis indicated that there were no significant interactions between recruitment method and any of the independent variables in predicting ED pathology, hence pooling of the data across recruitment methods was deemed appropriate (see Appendix B).

EAT-26 data was found to be positively skewed (as would be expected in a primarily general population sample) and potential violations of the assumptions of regression analysis (i.e., normality, linearity, and homoscedacity) were apparent. A square-root transformation of EAT-26 scores (EAT-26-Sqrt) yielded an approximately normal distribution of the data and no violations of the regression assumptions were apparent when the analysis was repeated, as such EAT-26-Sqrt was used as the DV in the primary analyses. Given the strong correlations among the IVs multicollinearity was assessed but no major concerns were identified, with collinearity tolerance statistics falling in the range of .298 to .926 (all > .1) for predictors in the multiple regression model.

Descriptive statistics and inter-correlations for all study variables are shown in Table 1 and 2 respectively. Approximately half (46.3%) of the total sample were classified as being "at risk" of an ED according to the accepted EAT-26 cut-point of 20 or greater.

# **Primary Analysis**

To test the hypothesis that BAE, AEE, silencing-the-self, divided-self, and negative emotionality were each individually associated higher levels of ED pathology, a series of hierarchical multiple regression analyses was performed (with the covariates of age and BMI entered in Block 1 and each predictor variable in Block 2), results of which are shown in Table 3. To test the hypothesis that BAE, AEE, silencing-the-self, divided-self, and negative emotionality were each individually associated with increased ED pathology when controlling

Table 1

Descriptive Statistics for Study Variables

|                       |     |       |       |     |      | Skewn | ess  |
|-----------------------|-----|-------|-------|-----|------|-------|------|
|                       | n   | M     | SD    | Min | Max  | Stat  | SE   |
| Age                   | 403 | 23.90 | 6.88  | 18  | 62   | 2.20  | .122 |
| BMI                   | 400 | 22.28 | 4.99  | 13  | 53   | 2.19  | .122 |
| EAT-26                | 373 | 22.91 | 19.76 | 0   | 75   | .696  | .126 |
| EAT-26-Sqrt           | 373 | 4.240 | 2.224 | 0   | 8.66 | .070  | .126 |
| Negative emotionality | 378 | 74.99 | 22.08 | 27  | 128  | .010  | .125 |
| Depressive symptoms   | 380 | 16.44 | 12.94 | 0   | 42   | .456  | .125 |
| BAE                   | 375 | 42.60 | 16.71 | 0   | 72   | 272   | .126 |
| AEE                   | 378 | 90.72 | 24.35 | 28  | 140  | 374   | .125 |
| Silencing-the-self    | 374 | 28.25 | 7.33  | 11  | 49   | 086   | .126 |
| Divided-self          | 374 | 16.30 | 5.25  | 6   | 30   | .023  | .126 |

BMI = Body Mass Index. EAT-26 = Eating Attitudes Test (ED pathology). EAT-26-Sqrt = Square root transformation of EAT-26 scores. BAE = Beliefs about the unacceptability of negative emotion. AEE = Ambivalence over emotional expression.

for depressive symptoms, a further series of hierarchical regression analyses were performed (with age, BMI, and depressive symptoms entered in Block 1 and each predictor variable in Block 2). As can be seen in Table 3, the contribution of each of the predictor variables to variance in ED pathology remained significant when controlling for depressive symptoms. Another set of hierarchical multiple regression analyses were used to test the hypothesis that when fitted together the variance in ED pathology explained by BAE, AEE, silencing-the-self, and divided-self would be overlapping. Results confirmed that the unique variance in ED pathology explained by each of the predictor variables was reduced when these variables were entered simultaneously (over and above the impact of age and BMI), such that only BAE and divided-self were independently significantly associated with ED pathology (Table 4). When also controlling for depression, only BAE (and depression) were independently significantly associated ED pathology. However, when only BAE and divided-self were entered in the regression model, along with depressive symptoms, the contributions of BAE (t = 5.379, p < 0.001), divided-self (t = 2.995, p = 0.003) and depressive symptoms (t = 5.122, t < 0.001) each

remained significant and the total variance in ED pathology explained ( $R^2 = .352$ ) was not significantly reduced ( $R^2$  change = .002,  $F_{(2.353)} = .509$ , p = .602).

**Moderation effects.** To test the hypothesis that negative emotionality would moderate the association between emotion inhibition and ED pathology, hierarchical multiple regression analysis was performed, with age, BMI, depressive symptoms, BAE, AEE, silencing-the-self, and divided-self fitted together in Block 1 and the interactions of BAE, AEE, silencing-the-self, and divided-self with negative emotion fitted together in Block 2. Results of this analysis indicated that the addition of the interaction terms in Block 2 was not statistically significant ( $R^2$  change = .012,  $F_{(4,348)}$  = 1.634, p = .165) (see Table 5). That is, there was no evidence to suggest that negative emotionality moderated the associations between any of the emotion inhibition variables and ED pathology.

Mediation effects. A series of PROCESS analyses was conducted to test the hypothesis that the effects of BAE, AEE, silencing-the-self, and divided-self on ED pathology would be mediated by negative emotionality, controlling for depressive symptoms. Results of this analysis indicated that AEE had a significant indirect effect on ED pathology via negative emotionality and that the direct effect was no longer significant (mediation), whereas BAE, silencing-the-self, and divided-self each had significant indirect effects on ED pathology via negative emotionality and that the direct effects remained significant in each case (mediation; Table 6).

Table 2

Correlations Among Study Variables

|    |                            | -    | 6     | ۲,   | 4    | 6    | 9    | 7     | ×     | 0     | 10    |
|----|----------------------------|------|-------|------|------|------|------|-------|-------|-------|-------|
|    |                            | ,    | 1     | ,    | ٠    | ,    |      | ,     | 0     | ,     | 7.0   |
| -  | Age                        | ı    |       |      |      |      |      |       |       |       |       |
| 2  | BMI                        | .262 | I     |      |      |      |      |       |       |       |       |
| 3  | ED Pathology (EAT-26)      | .040 | 157   | .96° |      |      |      |       |       |       |       |
| 4  | ED Pathology (EAT-26-Sqrt) | .053 | -1.04 | .972 | I    |      |      |       |       |       |       |
| 2  | Negative emotionality      | .033 | 059   | .549 | .504 | .96ª |      |       |       |       |       |
| 9  | Depressive symptoms        | 028  | 960:- | .520 | .546 | .746 | .94ª |       |       |       |       |
| 7  | BAE                        | .003 | 035   | .505 | .526 | .663 | .535 | .93 a |       |       |       |
| 8  | AEE                        | .011 | 063   | .418 | .427 | .661 | .522 | .692  | .95 a |       |       |
| 6  | Silencing-the-self         | .047 | 007   | .365 | .365 | .470 | .342 | .477  | .569  | .85 a |       |
| 10 | Divided-self               | 090  | 066   | .427 | .438 | .601 | .468 | .529  | .556  | .705  | .83 a |

\*. Correlation significant at p < .001 (2-tailed). \*\* Cronbach's  $\alpha$  in the present study. BMI = Body Mass Index. EAT-26 = Eating Disorder Test (ED pathology). EAT-26-Sqrt =

Square root transformation of EAT-26 scores. BAE = Beliefs about the unacceptability of negative emotion. AEE = Ambivalence over emotional expression.

Comparison of Standardised Regression Coefficients and Unique Variance in ED Pathology Explained by Each Emotion Inhibition Variable

Table 3

When Fitted Individually, Controlling for Depressive Symptoms, and Controlling for both Negative Emotion and Depressive Symptoms

|                                    | Emotio | Emotion Inhibition IVs, Neg<br>Emotion and Depressive Sym<br>fitted independently <sup>a</sup> | on IVs, No<br>essive Sy<br>endently | egative<br>mptoms | Emotion Inhibition IVs and Negative emotion fitted independently, controlling for depressive symptoms <sup>a</sup> | n Inhibitio<br>ed indepen<br>epressive | Emotion Inhibition IVs and Negative ion fitted independently, controlling for depressive symptoms a | Negative<br>atrolling    | Emoti<br>independer<br>emotion | Emotion Inhibition IVs fitted ndependently controlling for negative emotion & depressive symptoms a | on IVs fitte<br>lling for ne<br>ive sympto | ed<br>gative<br>ms <sup>a</sup> |
|------------------------------------|--------|--|-------------------------------------|-------------------|--|--|---|--------------------------|--------------------------------|---|--|---------------------------------|
| Dependent Variable:<br>EAT-26-Sqrt | β      | ı  | d                                   | Unique $R^2$      | β  | 1                                      | d   | Unique<br>R <sup>2</sup> | β                              | t   | d  | Unique<br>R <sup>2</sup>        |
| Depressive symptoms                | .496   | 10.935   | < .001                              | .244              | I  |  | 1   | ı                        | ı                              | 1   |  | I                               |
| Negative emotion                   | .536   | 12.168   | < .001                              | .285              | .382   | 5.849                                  | < .001  | .065                     | I                              | I   | I  | ı                               |
| BAE                                | .520   | 11.734   | < .001                              | .270              | .359   | 7.150                                  | < .001  | .092                     | .278                           | 4.900   | < .001                                     | .043                            |
| AEE                                | .417   | 8.814  | < .001                              | .173              | .219   | 4.221                                  | < .001  | .035                     | .100                           | 1.741   | .083                                       | 900.                            |
| Silencing-the-self                 | .363   | 7.452  | < .001                              | .132              | .225   | 4.773                                  | < .001  | .045                     | .147                           | 2.990   | .003                                       | .017                            |
| Divided-self                       | .427   | 8.982  | < .001                              | .181              | .264   | 5.309                                  | < .001  | .054                     | .184                           | 2.773   | 900.                                       | .019                            |

 $^{a}$ . = Controlling for age and Body Mass Index (BMI).  $\beta$  = Standardized Regression Coefficients. EAT-26-Sqrt = Square root transformation of Eating Disorder Test (EAT-26) scores (ED pathology). BAE = Beliefs about the unacceptability of experiencing and expressing negative emotion. AEE = Ambivalence aver emotional expression.

Pahle 1

Comparison of Standardised Regression Coefficients and Unique Variance in ED Pathology Explained by Each Inhibition Variable Fitted Simultaneously, Simultaneously Controlling for Depressive Symptoms, and Simultaneously With Negative Emotion and Controlling for

Depressive Symptoms

|                                    | Emo  | tion Inhib | Emotion Inhibition IVs fitted | itted        | Em          | Emotion Inhibition I | ition IVs fitted | tted                     | Full Model: Emotion | Emotion In  | hibition IV    | 's fitted    |
|------------------------------------|------|------------|-------------------------------|--------------|-------------|----------------------|------------------|--------------------------|---------------------|-------------|----------------|--------------|
|                                    |      | simultan   | simultaneously ab             |              |             | Simultaneous         | ously,           |                          | simultaneously v    | vit         | n negative emo | tion &       |
|                                    |      |            |                               |              | controlling | ing for depress      | sive sympt       | oms ac                   | controlling f       | for depress | ive sympto     | oms ad       |
| Dependent Variable:<br>EAT-26-Sqrt | β    | t          | d                             | Unique $R^2$ | β           | t.                   | d                | Unique<br>R <sup>2</sup> | β                   | ı           | d              | Unique $R^2$ |
| Negative emotion                   | 1    | ı          | ı                             | ı            |             | 1                    | ı                | ı                        | 181.                | 2.339       | .020           | .010         |
| Depressive symptoms                | I    | I          | I                             | I            | .274        | 5.0156               | < .001           | .048                     | .190                | 2.976       | .003           | .016         |
| BAE                                | .384 | 6.1111     | < .001                        | .072         | .300        | 4.784                | < .001           | .041                     | .264                | 4.110       | <.001          | .030         |
| AEE                                | .033 | .496       | .620                          | <.001        | 039         | 600                  | .549             | .001                     | 072                 | -1.089      | .277           | .002         |
| Silencing-the-self                 | .026 | 397        | .692                          | <.001        | .061        | .944                 | .346             | .002                     | .063                | 166:        | .323           | .002         |
| Divided-self                       | .184 | 2.773      | 900.                          | .015         | .126        | 1.913                | .057             | .007                     | .092                | 1.380       | .168           | .003         |

 $^{a}$  = Controlling for age and Body Mass Index (BMI).  $^{b}$  Model:  $R^{2}$  = .313.  $^{c}$  Model:  $R^{2}$  = .362.  $^{d}$  Full Model:  $R^{2}$  = .372.  $\beta$  = Standardized Regression Coefficients. EAT-26-

Sqrt = Square root transformation of Eating Disorder Test (EAT-26) scores (ED pathology). BAE = Beliefs about the unacceptability of experiencing and expressing negative emotion. AEE = Ambivalence aver emotional expression.

Table 5

Results of Moderation Analysis with Study Variables and Covariates included in Model 1 of the Hierarchical Multiple Regression and the Inclusion of the Interaction of Each Study Variable with Negative Emotion Fitted in Model 2

|                                     |      | Model 2 |        |
|-------------------------------------|------|---------|--------|
|                                     | β    | t       | p      |
| Age                                 | .051 | 1.144   | .254   |
| BMI                                 | 075  | -1.701  | .090   |
| Depressive symptoms                 | .184 | 2.885   | .004   |
| Negative emotion                    | .171 | 2.209   | .028   |
| BAE                                 | .279 | 4.347   | < .001 |
| AEE                                 | 070  | -1.040  | .299   |
| Silencing-the-self                  | .047 | .734    | .463   |
| Divided-self                        | .108 | 1.614   | .107   |
| Negative Emotion*BAE                | .076 | 1.151   | .251   |
| Negative Emotion*AEE                | 010  | 139     | .889   |
| Negative Emotion*Silencing-the-self | .129 | 1.906   | .057   |
| Negative Emotion*Divided-self       | 114  | -1.674  | .095   |

NB: Dependent Variable = EAT-26-Ssrt (Square root transformation of Eating Disorder Test total scores).  $\beta$  = Standardised Regression Coefficient. Model 1:  $R^2$  = .372. Model 2:  $R^2$  = .383.  $R^2$  change = .012,  $F_{(4,348)}$  = 1.634, p = 165. BAE = Beliefs about the unacceptability of experiencing and expressing negative emotion. AEE = Ambivalence aver emotional expression.

able 6

Summary of Mediation Analyses Testing the Indirect Effects of Each Emotion Inhibition Variable on ED Pathology via Negative Emotion, Controlling for

Depressive Symptoms

|                           | Total effect      |       | Direct effect     |       |                     |       | Indirect effects  |      |                   |         |
|---------------------------|-------------------|-------|-------------------|-------|---------------------|-------|-------------------|------|-------------------|---------|
| ,                         |                   |       |                   |       | Path a              |       | Path b            |      | Total indirect e  | effect  |
| Hypothesis                | B (99% CI)        | d     | B (99% CI)        | d     | B (99% CI)          | d     | B (99% CI)        | d    | B (99% CI)        | $p_{p}$ |
| BAE→NegEm→ED <sup>3</sup> | .048 (.030, .065) | <.001 |                   | <.001 | .498 (.373, .624)   | <.001 |                   |      | .011 (.002, .022) | .004    |
| AEE→NegEm→ED <sup>a</sup> | .022 (.008, .032) | <.001 | .009 (004, .023)  | .083  | .333 (.249, .418)   | <.001 | .032 (.013, .051) |      | .011 (.004, .019) | <.001   |
| STS→NegEm→ED <sup>3</sup> | .067 (.031, .104) | <.001 | .044 (.006, .083) | .003  | .760 (.487,1.033)   | <.001 | .044 (.006, .083) | .003 | .023 (.009, .042) | <.001   |
| DS→NegaEm→ED <sup>a</sup> | .112 (.058, .167) | <.001 |                   | .001  | 1.369 (.973, 1.766) | <.001 |                   |      | .038 (.014, .072) | <.001   |

<sup>a</sup>. Controlling for depressive symptoms, age, and Body Mass Index (BMI). <sup>b</sup> Normal Theory Test p-value. Path a = Path from predictor variable to the mediator variable. Path b = Path from mediator variable to the dependent variable. B = Unstandardized Effect Coefficient. 99% CI = Bootstrapped 99% Confidence Intervals around Unstandardized Effect. p = Twotailed significance. BAE = Beliefs about the unacceptability of experiencing and expressing negative emotions. NegEm = Negative emotion ED = Eating disorder pathology (EAT-26-

Sqrt: Square root transformation of Eating Attitude Test [EAT-26] scores). AEE = ambivalence over emotional expression. STS = silencing-the-self. DS = divided-self.

#### Discussion

The aims of the current study were, first, to examine the associations between various forms of emotion inhibition, namely BAE, AEE, silencing-the-self, divided-self, and negative emotionality and ED pathology, in a diverse sample of women; and second, to explore the potential role of negative emotionality in moderating and/or mediating these associations. It was hypothesised, first, that BAE, AEE, silencing-the-self, divided-self, and negative emotionality would each individually predict increased ED pathology; and second, that each would do so over and above the contribution of depressive symptoms. These hypotheses were supported, confirming previous findings concerning the associations between ED pathology and negative emotional experience (Spoor et al., 2007), BAE (Hambrook et al., 2011), and AEE (Krause et al., 2000). With respect to silencing-the-self and divided-self, the current findings extend earlier research (Hambrook et al., 2011; Piran & Cormier, 2005) in suggesting independent effects of each of silencing-the-self and divided-self schemas, in predicting ED pathology, controlling for depressive symptoms.

Third, it was hypothesised that, given the theoretical overlap between the different inhibition constructs, the variance in ED pathology explained by these constructs, taken together, would be reduced. This hypothesis was also supported, the results indicating that only BAE and divided-self schemas were independently associated with ED pathology. Further, when controlling for depressive symptoms, only BAE (and depressive symptoms) were independently associated with ED pathology. When AEE and silencing-the-self were removed from the model, however, the independent contributions to ED pathology of both BAE and divided-self remained significant, controlling for depressive symptoms, and the total variance in ED pathology explained was not reduced. These findings accord with Quinton and Wagner (2005) finding that AEE was not independently associated with ED pathology when controlling for emotional expression and alexithymia and suggest that while the variance in

ED pathology explained by AEE and silencing-the-self overlaps with that explained by BAE and divided-self, both BAE and divided-self explain unique aspects of and are of independent importance in understanding ED pathology.

Fourth, it was hypothesised that negative emotionality would moderate the associations among BAE, AEE, and divided-self and ED pathology. However, this hypothesis was not supported in that there was no evidence that the strength of the associations between the respective emotion inhibition constructs and ED pathology varied as a function of participants' levels of negative emotionality. This finding would appear to be at odds with the finding of an interaction between emotion inhibition and negative emotion in predicting poor outcomes in cardiac patients (Denollet et al., 1996), but is consistent with the failure to find evidence of an interaction effect between either avoidant or emotion-oriented copying styles and negative emotion in accounting for emotional eating reported by Spoor et al. (2007). The current findings, taken with those of Spoor and colleagues, suggest that lower negative emotionality may not be protective in mitigating the association between emotion inhibition and ED pathology. This interpretation accords with Corstorphine's (2006) affect-regulation model of ED pathology and with King and Emmons' (1990) conceptualisation of AEE. In both of these models, it is not the individual's level of emotion per se that is important in giving rise to distress and use of maladaptive coping strategies but, rather, his or her interpretation of their emotional experience and expression (or lack thereof).

Finally, it was hypothesised that the effects of emotion inhibition on ED pathology would be mediated, partially or fully, by negative emotionality. The results supported this hypothesis and provided evidence that negative emotionality partially accounts for the associations between each of BAE, silencing-the-self and divided-self schemas and ED pathology, and fully accounts for the association between AEE and ED pathology, controlling for depressive symptoms. These findings converge with evidence that emotion inhibition in

the form of expressive suppression (Gross & John, 2003) and AEE (King & Emmons, 1990) is associated with increased experience of negative emotion, while further suggesting a role for emotion inhibition in indirectly predicting ED pathology. As such, the current findings provide important new information concerning one of the processes by which different forms of emotion inhibition may influence ED pathology via negative emotionality and provide preliminary support for one part of Corstorphine's (2006) affect-regulation model of ED pathology, namely, the indirect effect of BAE on ED pathology via increased negative emotional experience.

# **Study Implications**

The current findings contribute to the body of evidence suggesting that emotional inhibition is a maladaptive and costly means of emotion regulation that may have adverse flow-on effects, one of which may be increases in ED pathology. By inhibiting the experience and expression of emotion, women may lose the opportunity to learn from and understand their emotional experience (Denollet, Nyklicek, & Vingerhoets, 2010; Kennedy-Moore & Watson, 2001). Insight of this kind is important in helping individuals to use the information gained from their emotional experience not only to guide their thoughts and behaviours but also to process, regulate, and cope more adaptively with negative emotions (Kennedy-Moore & Watson, 1999; Salovey, Hsee, & Mayer, 1993; Watson & Greenberg, 1996). Further, inhibition of emotion expression in the interpersonal context may impede not only communication and intimacy but also the ability to gain validation from others, to access social support and to resolve interpersonal difficulties associated with or triggering the experience of negative emotions (Kennedy-Moore & Watson, 2001).

The findings of the current study provide support for an emotion regulation model of ED pathology, in which ED symptoms serve the maladaptive function of avoiding, distracting, or escaping awareness of negative emotion (Fox, Federici, & Power, 2012; Heatherton &

Baumeister, 1991). According to this model, attempts by women to avoid or manage emotional experience or expression through one or more forms of emotion inhibition, and in particular BAE, are counterproductive, and rather are conducive to increases in the experience of negative emotion and, in turn, the ED pathology used to manage this experience. The implication is that if more adaptive means of emotion-regulation were to be employed – in place of emotion inhibition – then the occurrence of ED pathology should be reduced. To this end, several ED therapies have been developed with the specific goal of addressing maladaptive emotion inhibition or avoidance. These include CEBT-ED (Corstorphine, 2006), emotion-focused therapy for EDs (EFT; Dolhanty & Greenberg, 2007), schema-focused CBT for EDs (SFCBT; Waller, Cordery, et al., 2007), integrative cognitive-affective therapy for BN (Wonderlich et al., 2010) and DBT for BN and binge eating disorder (Safer, Telch, & Chen, 2009). Indeed, the role of ED symptoms functioning as a means of escaping from or reducing negative emotionality is now emphasised in several contemporary CBT models of ED pathology (Cooper, 2005; Cooper, Wells, & Todd, 2004; Fairburn et al., 2003) and mood intolerance has been incorporated into CBT-E (Fairburn, 2008).

## Study Strengths, Limitations and Directions for Future Research

A notable strength of the current study lies in its efforts to tease out the unique and shared contributions of several different emotion inhibition constructs in accounting for variance in ED pathology. In this regard the research fills a significant gap in the literature as previous research was largely confined to consideration of these constructs in isolation. Further, the study provides preliminary insights into one of the processes by which emotion inhibition contributes to increased ED symptoms, namely, via increases in negative emotionality. The research contributes new evidence supporting the theory that maladaptive attempts at emotion regulation (via inhibition) are likely to be counterproductive in reducing the experience of negative emotion, and rather lead to further negative emotion that may then

be regulated via ED pathology. A further strength of the current research is the recruitment of participants from a number of sources – ED information websites, general community, and undergraduate students – thereby ensuring a broad range of values on each of the study variables and improving the generalizability of the findings.

There were also significant limitations of the current research, the most notable of these being the use of a cross-sectional study design. Mediational processes, such as those referred to in Corstorphine's (2006) model, imply causality and direction of effect, which cannot be tested using a cross-sectional design. However, A. F. Hayes (2013) argues that where a theoretical foundation exists to support the directionality of effects it is appropriate to use PROCESS analyses to test for mediation effects using cross-sectional data. Further, the forms of emotion inhibition considered in the current study are conceived of as originating from deficits in early childhood environments and from deficits in development of emotion regulation capacities; and are thus thought to precede current emotional experience (Corstorphine, 2006; Krause et al., 2003). Nevertheless, there are likely to be complex, bidirectional pathways underpinning the associations among the different forms of emotion inhibition, negative emotionality and ED pathology and clearly longitudinal research will be needed to elucidate these pathways. The inclusion of potentially relevant variables not assessed in the current research, such as those measuring some of the etiological factors theorised to contribute to emotion inhibition and other maladaptive emotional styles (e.g., attachment dynamics); and any specific emotion regulation strategies (e.g., rumination, reflection, reappraisal, and suppression) that may further mediate the relationship between BAE, negative emotionality, and ED symptoms will also be important in future research.

A second important limitation of the current research is the reliance on self-report measures of all study variables. While online surveys are an efficient means of data collection that allow for high levels of participant privacy and anonymity, potential problems with the

use of self-report measures of ED symptoms in particular are well-known (House, Eisler, Simic, & Micali, 2008). This is of particular relevance with regard to self-report of current body weight and height used to derive BMI in the current study, as women have been found to demonstrate significant inaccuracies in self-reporting of weight and height, in particular (Barnes, White, Masheb, & Grilo, 2010; Bowring et al., 2012). However, as BMI was not found to be significantly correlated with ED pathology in the current study, this limitation is unlikely to impact interpretation of result here. Nevertheless, the inclusion of interview data and/or data obtained from multiple sources, and independent assessment of weight and height, would clearly be of interest in future research. Ideally, the validity of self-report measures of key study variables would be confirmed in a sub-group of participants before administration of these measures to the complete study sample.

It should also be noted that while the recruitment of participants from various sources may be acknowledged as a strength of the current research, this method leaves unanswered the question of whether similar results might be observed in general population and/or clinical samples considered separately. On the one hand, the current findings are likely to have clinical relevance, given that approximately half of the current sample would be classified as being "at risk" for an ED based on their EAT-26 scores. Replication of the current findings in a clinical sample of individuals with EDs would have more direct implications for treatment practice, however, and would permit exploration of potential differences in the observed associations across ED diagnoses (e.g., Frank & Thomas, 2003; Ross & Wade, 2004).

Replication of the current findings in a general population sample, on the other hand, may have implications for ED prevention programs.

Also concerning the recruitment methods employed in the current study, it should be noted that these methods are susceptible to selection bias and that this may detract from the generalizability of the findings. For example, it is possible that emotionally avoidant

individuals are less likely to volunteer to participate in research aimed at assessing emotional experience and emotion styles. The relatively high proportion of participants with high levels of ED pathology is another example of selection bias, albeit an asset in the current context. Finally, the fact that the current research was confined to adult females needs to be acknowledged as a potential limitation of the research. Although this was reasonable for a study designed to extend research previously conducted in this demographic, the inclusion of younger women and of males in future research would be of interest.

## Conclusion

Within the limits of a cross-sectional study design, the current findings support the view that various forms of emotion inhibition, BAE in particular, may serve to increase the experience of negative emotion, which in turn may trigger ED pathology as a means of alleviating this experience. The findings are consistent with an emotion-regulation model of ED pathology and support the role of therapeutic interventions for ED pathology designed to challenge beliefs about the unacceptability of experiencing and expressing negative emotions and related cognitions.

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Appendix A

Descriptive Statistics for Study Variables in Each Recruitment Sample and Contrast Tests for Group Differences Across Samples

|                       | Studer | Student Sample (SS) | (SS)  | Commun   | ity Sample (CS) | (CS)  | Butterfly | <b>3 autterfly Sample (BS)</b> | (BS)  | Contrasts <sup>a</sup> |
|-----------------------|--------|---------------------|-------|--|-----------------|-------|-----------|--------------------------------|-------|------------------------|
|                       | и      | M                   | SD    | и  | M               | QS    | и         | M                              | SD    |                        |
| Age                   | 102    | 21.27               | 5.44  | 126  | 23.33           | 5.68  | 175       | 25.85                          | 7.80  | SS = CS < BS           |
| BMI                   | 102    | 22.06               | 4.43  | 124  | 22.93           | 4.80  | 174       | 21.94                          | 5.40  | SS = CS = BS           |
| EAT-26                | 101    | 13.48               | 15.22 | 125  | 16.33           | 14.04 | 147       | 34.99                          | 20.56 | SS = CS < BS           |
| Negative emotionality | 102    | 66.51               | 20.70 | 125  | 70.80           | 20.58 | 151       | 84.19                          | 20.93 | SS = CS < BS           |
| Depressive symptoms   | 101    | 13.50               | 11.49 | 125  | 12.42           | 11.64 | 154       | 21.64                          | 13.14 | SS = CS < BS           |
| BAE                   | 102    | 36.13               | 15.91 | 125  | 40.54           | 15.41 | 148       | 48.80                          | 16.26 | SS = CS < BS           |
| AEE                   | 101    | 82.58               | 24.70 | 125  | 80.98           | 23.08 | 152       | 99.95                          | 22.17 | SS = CS < BS           |
| Silencing-the-self    | 102    | 26.01               | 08.9  | 125  | 26.83           | 6.72  | 147       | 31.01                          | 7.35  | SS = CS < BS           |
| Divided-self          | 102    | 14.01               | 4.84  | 125  | 15.88           | 5.23  | 147       | 18.26                          | 4.81  | SS < CS < BS           |
| a                     |        | 24 1 4 23 14 1      | ı     | the state of the s |                 |       |           |                                | 0     |                        |

<sup>&</sup>lt;sup>a</sup>. Bonferroni adjusted simple contrast test. BMI = Body Mass Index. EAT-26 = Eating Attitudes Test. BAE = Beliefs about the unacceptability of negative emotion

experience and expression. AEE = Ambivalence over emotional expression.

Appendix B

Results of General Linear Model Analysis of Variance to Test for Possible Interaction Effects

Between Recruitment Group and Any Study Independent Variables in Predicting ED

Concerns

|                           | $oldsymbol{\eta}^2_{partial}$ | df       | F      | p     |
|---------------------------|-------------------------------|----------|--------|-------|
| Group                     | .003                          | (2, 343) | .554   | .575  |
| Age                       | .002                          | (1, 343) | .633   | .427  |
| BMI                       | <.001                         | (1, 343) | .145   | .703  |
| Negative emotion          | .017                          | (1, 343) | 5.995  | .015  |
| Depressive symptoms       | .007                          | (1, 343) | 2.463  | .117  |
| BAE                       | .040                          | (1, 343) | 14.249 | <.001 |
| AEE                       | .002                          | (1, 343) | .595   | .441  |
| Silencing-the-self        | <.001                         | (1, 343) | .004   | .952  |
| Divided-self              | .004                          | (1, 343) | 1.310  | .253  |
| Group*Negative emotion    | .003                          | (2, 343) | .494   | .610  |
| Group*Depressive Symptoms | .008                          | (2, 343) | 1.312  | .271  |
| Group*BAE                 | .005                          | (2, 343) | .788   | .455  |
| Group*AEE                 | .011                          | (2, 343) | 1.829  | .162  |
| Group*Silencing-the-self  | <.001                         | (2, 343) | .009   | .991  |
| Group*Divided-self        | .010                          | (2, 343) | 1.722  | .180  |

NB: Dependent Variable = EAT-26-Sqrt (Square root transformation of Eating Disorder Test total scores).

 $<sup>\</sup>eta^2_{partial}$  = Partial Eta Squared. BAE = Beliefs about the unacceptability of experiencing and expressing negative emotion. AEE = Ambivalence aver emotional expression.

# Chapter 6

Beliefs About Emotion and Eating Pathology in Women: The Roles of Emotion Regulation, Negative Emotionality, and Depressive Symptoms

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# **Author Contributions**

First author Kim Woodward was responsible for conceptualisation, design, and conduct of the current study, managed data collection, undertook all statistical analyses, conducted the literature searches, and wrote the first draft of the manuscript. Author Doris McIlwain contributed to the conceptualisation and design of the study at a supervisory level. Author Jonathan Mond contributed to revisions of the manuscript.

#### Abstract

**Objective:** To investigate the direct and indirect roles of various emotion regulation (ER) strategies, negative beliefs about emotions (BAE), negative emotionality, and depressive symptoms in predicting eating disorder (ED) pathology. **Methods:** Participants were 724 women (197 recruited via ED support websites, 208 from the community, and 319 undergraduate students; 385 of whom were classified as having no probable ED and 339 as having a probable DSM-5 ED) who completed measures of BAE, cognitive reappraisal, expressive suppression, rumination, general reflection, emotional reflection, negative emotionality, depressive symptoms, and ED pathology. **Results:** BAE, each ER strategy, negative emotion and depressive symptoms individually predicted ED pathology. Path analyses assessing the indirect relationship of ER strategies with ED pathology, excluding BAE, revealed the relationships between reappraisal, suppression, and rumination and ED pathology are each mediated by negative emotion and depressive symptoms, while reflection had no independent effect. A path model including BAE, suppression, rumination, negative emotionality, depressive symptoms, and ED pathology demonstrated very good fit for the data (in the full sample and in the non-ED and probable-ED subgroups separately) and revealed BAE has significant direct and indirect effects on ED pathology. Conclusions: BAE appears to predict ED pathology both directly and also indirectly via increased use of the maladaptive ER strategies of suppression and rumination and, in turn, increases in negative emotionality and depressive symptoms. Findings are consistent with an ER model of ED pathology. Implications for clinical practice are discussed.

**Keywords:** Eating disorder pathology; beliefs about emotions; emotion regulation; negative emotionality; suppression; rumination

#### Introduction

The symptoms of eating disorders (EDs) have long been thought of as serving a function in the psychic and emotional economy of the individual (Bruch, 1973). It is well known that many individuals with EDs are highly emotionally vulnerable, that these individuals are prone to experience difficulties in regulating emotions, and that the experience of emotion is often a trigger for ED symptoms (Corstorphine, 2006). One of the theories suggested to explain the link between negative emotion and ED pathology is the 'blocking' model, which proposes that ED symptoms, such as binge eating and extreme weight-control behaviours, are maladaptive emotion regulation strategies that provide short-term relief from unacceptable emotion but which in the long-term exacerbate distress (McManus & Waller, 1995; Root & Fallon, 1989). Closely aligned with this theory is the 'escape from awareness model' (Heatherton & Baumeister, 1991), which suggests that ED pathology serves the function of diverting attention away from painful self-awareness. As such, ED symptoms are conceptualised as a form of avoidant coping (Neckowitz & Morrison, 1991; Troop et al., 1994). A growing body of research has sought to identify specific ER strategies that may be linked to EDs, as ER models of psychopathology suggest that it may not be the experience of emotion-provoking events or negative emotion per se that leads to symptoms, but, rather, the manner in which individuals attend to, process or regulate this experience (either internally or interpersonally) that leads to increased psychopathology (Gratz & Roemer, 2004; Gross, 1998).

# **Emotion Regulation Strategies**

Emotion regulation (ER) strategies are processes through which individuals attempt either consciously or unconsciously to modulate their response to emotion-eliciting events by changing the size, duration, intensity, and/or type of their emotional experience or expression (Gross & Thompson, 2007). Emotion-regulation strategies can take on several forms and can

be classified as adaptive or maladaptive (Werner & Gross, 2010). Several ER strategies that have been linked to psychopathology, including ED pathology, are expressive suppression ('suppression'), cognitive reappraisal ('reappraisal), rumination and reflection.

**Suppression and reappraisal.** One influential model of ER (Gross, 1998) explains emotion generation and expression to be a temporal process and posits that ER strategies can occur at any of five points in the process. The timing of use of the ER strategy is believed to impact its outcome, with strategies occurring earlier in the process being more adaptive than those occurring later (Gross, 2001). Two strategies that have received extensive research attention in this regard are expressive suppression and cognitive reappraisal.

Suppression is a form of response-focused modulation that occurs late in the emotion generation process and involves the inhibition of outward signs of emotion expression (Gross, 2001). Whilst suppression serves to decrease the behavioural expression of emotion, it fails to decrease the experience of emotion and is associated with various costs including increased physiological response to stressors, cognitive overload, negative emotionality, and impairment in psycho-social functioning more broadly (Gross, 2001; Gross & John, 2003; John & Gross, 2004; Richards & Gross, 2000). Thus, suppression is actually counterproductive and may paradoxically give rise to the very emotions from which relief is sought (for review see Wenzlaff & Wegner, 2000).

In terms of associations with ED pathology, suppression was associated with both ED pathology and depressive symptoms in undergraduate women and levels of suppression, depressive symptoms, and negative emotion were all elevated among women with elevated levels of ED pathology (McLean et al., 2007). In a clinical sample, suppression was found to be significantly higher among individuals with anorexia nervosa (AN) and bulimia nervosa (BN) than among healthy controls (HC), whereas AN and BN groups did not differ with respect to levels of suppression (Davies et al., 2012). In a meta-analysis of six studies,

comprising both clinical and non-clinical samples, suppression was found to have a medium effect on ED pathology, with higher effect sizes for research conducted in clinical versus non-clinical samples (Aldao et al., 2010). Additionally, there is also a growing literature linking various other forms of emotion inhibition to ED pathology, such as self-silencing schemas and anger suppression (Geller et al., 2000; Hambrook et al., 2011), ambivalence over emotion expression (Krause et al., 2000), and emotional inhibition schemas (Waller et al., 2000).

A more adaptive ER strategy that occurs earlier in the emotion generation process (an antecedent-focused strategy) and avoids the costs of suppression is reappraisal (Gross, 2001). Reappraisal involves generating alternate interpretations of potentially emotion-provoking events in order to alter the emotions elicited (Gross & John, 2003). Cognitive theories identify maladaptive appraisals as a core mechanism in the development and maintenance of common mental health problems, such as depression and anxiety, and cognitive-behavioural therapy (CBT) for these problems is focused on the development of more adaptive appraisal skills (Beck, 1979). This framework has since been applied to the maintenance and treatment of ED pathology (e.g., Fairburn, 2008).

Unlike suppression, reappraisal decreases the experience and expression of emotion without any adverse impact on memory (Gross, 2001) and is associated with better psychosocial functioning (Gross & John, 2003). Reappraisal and has been found to predict lower depressive symptoms, controlling for other ER strategies, in both female and mixed gender and clinical and non-clinical samples (Garnefski & Kraaij, 2006; Garnefski et al., 2004), and high reappraisers report less negative emotion, less physiological arousal, and more positive emotion than low reappraisers (Mauss et al., 2007). In terms of associations with ED pathology, the evidence is limited and confined to clinical samples. In one study, reappraisal was found to be lower among individuals with AN compared to HCs, whereas individuals with BN did not differ from those with AN or from the HC group (Davies et al., 2012). In a

second study, reappraisal was higher among women with restrictive AN (ANR) than those with binge-purge AN (ANBP) or BN. Within the ANR group, however, there was no association between reappraisal and ED pathology, whereas a moderate negative association (r = -.33) was observed in the ANBP group (Danner et al., 2012). A third study, in which both reappraisal and suppression were assessed, found higher levels of suppression among ED patients than HCs and among AN patients than BN patients, while ANR and BN patients reported lower use of reappraisal than HCs (Danner et al., 2014). Suppression was positively associated with depressive symptoms in all subgroups, but with ED pathology in only the ANBP and binge eating disorder (BED) groups, whereas reappraisal was negatively associated with depression in the BN group only and with ED pathology in the ANR and BED groups only.

Such findings suggest that whilst there is mounting support for the association of suppression with ED pathology, the role of reappraisal in ED pathology is less clear and there is a need for further research addressing the association between ED pathology and both suppression and reappraisal, while controlling for symptoms of depression and other potential covariates. Research of this kind in one or more non-clinical samples would be particularly welcome.

Rumination and reflection. Rumination is a form of repetitive negative thinking involving the tendency to dwell on self, upsetting events, and symptoms and their possible causes and consequences, in the absence of problem-solving (Nolen-Hoeksema et al., 2008; Watkins, 2008). It is deemed to be a maladaptive means of regulating emotion, best categorised as an avoidant coping strategy, that (like suppression) has the paradoxical effect of increasing negative affect and low mood (Mor & Winquist, 2002; Smith & Alloy, 2009). There is extensive evidence for the role of rumination in both the onset and maintenance of depression, including from numerous prospective longitudinal studies (Ehring & Watkins,

2008; Nolen-Hoeksema et al., 2008; Watkins, 2008).

In relation to EDs, rumination has been found to be significantly associated with ED pathology in a student sample (Rawal et al., 2010). In women with AN rumination has been found to predict ED pathology, over and above depression, anxiety, and worry (Startup et al., 2013), has been found to be elevated in women with AN compared with age-matched controls, and reduction in rumination at 10-month follow-up was associated with reduction in ED pathology (Rawal et al., 2010). In adolescent girls, one longitudinal study found that rumination predicted the onset and maintenance of both depressive and bulimic symptoms and vice-versa (Nolen-Hoeksema et al., 2007). Finally, an experimental study comparing the use of rumination versus distraction in response to induced feelings of sadness found that rumination lead to a significant increase in the desire to abstain from eating among women with AN and a significant increase in the desire to binge in women with BN, but had no impact on eating behaviour in non-ED control participants (distraction had no effect in ED participants but significantly reduced the desire to binge in the control group). In all groups, rumination resulted in significant increases in subjective feelings of sadness, whereas distraction did not (Naumann, Tuschen-Caffier, Voderholzer, Caffier, & Svaldi, 2015).

According to response style theory (Nolen-Hoeksema et al., 2008), binge eating serves the maladaptive function of escaping the aversive self-focus associated with the elevated rumination characteristic of individuals with BN. Further, ED-specific rumination (e.g., repetitive thinking about eating, weight or shape) is conceptualised as the primary mode of processing in AN, to the exclusion of experiencing and recognising emotions and bodily states (e.g., sensations of hunger and starvation). Rumination of this kind may serve to increase feelings of control in the short-term but exacerbate dietary restraint in the long-term (Park et al., 2011, 2012). Such rumination has been found to be associated with ED pathology over and above its association with symptoms of depression and anxiety (Cowdrey & Park,

2012) and over and above depression, anxiety, stress, BMI and disorder-specific reflection (Dondzilo, Rieger, Palermo, Byrne, & Bell, 2016).

Another style of repetitive thinking, one that may avoid the costs associated with rumination and may even be beneficial in reducing negative emotion and psychopathology, is reflection. Reflection is defined as a form of "self-attentiveness motivated by curiosity or epistemic interest in the self" (Trapnell & Campbell, 1999, p. 297). Reflection has been found to be positively correlated with the personality trait of openness (Trapnell & Campbell, 1999) and has been positively associated with personal growth in undergraduate students (Harrington et al., 2014). There has been little research to date regarding the association of reflection to ED pathology, however, in one study that found rumination was associated with ED pathology (Rawal et al., 2010).

Given the focus on avoidance of emotion central to blocking and escape from awareness theories of EDs, emotion reflection specifically, rather than general reflection, may be of particular relevance to ED pathology. Emotion reflection is said to involve a fullness of feeling together with a cognitive and sensory awareness of the emotion and one's response to it (McIlwain et al., 2010). Importantly, emotion reflection involves awareness of feeling, rather than judgment of feeling (McIlwain et al., 2010). As such, it can be thought of as akin to awareness and nonjudgmental facets of trait mindfulness, each of which were found to uniquely predict ED pathology over and above symptoms of anxiety and depression in undergraduate women (Lavender et al., 2011). Further, experiential self-focus (conceptually akin to reflection), when compared with ruminative self-focus, has been found to be associated with lower feelings of change in weight or shape following exposure to imagining eating a large meal in both students high in ED pathology and women with AN (Rawal et al., 2011). To date, however, no research has examined whether emotional reflection is associated

with ED pathology and whether emotional reflection predicts ED pathology when controlling for general reflection and other potential covariates.

More generally, few studies have looked at the relative importance of different ER strategies in terms of their unique associations with ED pathology. In one correlational study of undergraduate students, use of experiential avoidance, emotion inhibition, suppression, and rumination was found to be associated with higher levels of psychological distress, via increases in the experience of negative affect and negative life events, and deceases in positive affect, life satisfaction, and positive life events; whereas use of reappraisal was associated with increases in gratitude, life satisfaction, and curiosity (Kashdan et al., 2006). Another study of undergraduate students, using structural equation modelling (SEM), found that suppression, rumination and reappraisal (negatively and weakly) loaded on a single latent ER factor, which in turn predicted ED and comorbid psychopathology (Aldao & Nolen-Hoeksema, 2010). To date, however, no research has employed a path model that incorporates maladaptive and adaptive ER strategies, negative emotionality, depressive symptoms, and ED pathology, or extended such a model to incorporate potential antecedent constructs, such as negative beliefs about the experience and expression of emotions.

# **Beliefs About the Unacceptability of Negative Emotions**

Building on the blocking and escape from awareness models of ED pathology, it has been suggested that beliefs about the unacceptability of experiencing and expressing negative emotions (BAE) underlie the distress experienced by individuals with EDs (Corstorphine, 2006). According to this affect regulation model of EDs, the experience of emotionally invalidating childhood environments is said to engender beliefs that emotions are 'bad' and the experience or expression of negative emotions is unacceptable (Corstorphine, 2006). Such beliefs are suggested to activate the experience of secondary negative emotions in response to emotional experience (e.g., guilt about feeling anger or shame about feeling fear or sadness),

and trigger the use of ED behaviour as a maladaptive short-term means of blocking such negative affective experience (Corstorphine, 2006). BAE is thought to have implications for emotion processing and regulation and has been found to be positively associated with elevated levels of general depression, anxiety, and psychological distress in individuals suffering from multiple sclerosis and chronic fatigue syndrome (Dennison, Moss-Morris, Silber, Galea, & Chalder, 2010; Rimes & Chalder, 2010) and, together with rumination, low mindfulness, and low self-compassion, to predict increased psychological distress (depression, stress and anxiety) in undergraduate students (K. James, Verplanken, & Rimes, 2015).

BAE has also been found to be associated with ED pathology in various study populations. For instance, various measures of BAE have been found to be positively associated with ED pathology in non-clinical women (Haslam et al., 2012; Meyer et al., 2010), in individuals with BN (Lavender et al., 2014), and with ED pathology, depression, and anxiety in a mixed sample of individuals with AN, recovered from AN, and healthy controls (Oldershaw et al., 2012). Also in individuals with AN, BAE has also been associated with ED pathology, controlling for depression and anxiety (Hambrook et al., 2011). In research conducted by the authors (Woodward, McIlwain, & Mond, 2016), in a mixed student/community sample of women, BAE was found to be associated with ED concerns independent of its association with depressive symptoms, negative emotional experience, and various other styles of emotion inhibition. Further, the association between BAE and ED concerns was partially mediated by negative emotionality, controlling for depressive symptoms and emotion inhibition. These findings are consistent with the affect regulation model of ED pathology proposed by Corstorphine (2006), whereby one of the mechanisms by which BAE impacts ED symptomology is indirectly via increases in negative emotional experience. However, these findings need to be replicated in a different sample and the roles

of other processes potentially linking BAE with ED pathology, such as the use of specific maladaptive ER strategies need to be explored.

# The Role of Negative Emotionality and Depressive symptoms

Cutting across the associations between ER strategies, BAE and ED pathology are the potential mediating roles of negative emotionality and depressive symptoms. As noted above, negative emotionality is a risk factor for ED pathology (Culbert et al., 2015). Negative affect is also a diagnostic feature of major depressive disorder (American Psychiatric Association, 2013) and depressive symptoms and ED pathology are commonly comorbid (Puccio et al., 2016). Further, difficulties with ER have been identified as features of both depression and EDs (Brockmeyer, Bents, et al., 2012) and high dispositional use of rumination and suppression, and low use of reappraisal have consistently been identified as being characteristic of multiple forms of psychopathology (including depression, various forms of anxiety, EDs, and substance-abuse disorders; Aldao et al., 2010; Nolen-Hoeksema et al., 2007; Nolen-Hoeksema et al., 2008). Hence, when investigating the association between BAE, ER strategies and ED pathology, it is important to consider the role played by both negative emotionality and depressive symptoms.

The central thesis of the current study is that BAE and maladaptive ER strategies may give rise to ED pathology both directly and indirectly via increases in the experience of negative emotion and depressive symptoms. A mediating influence of this kind is suggested by Corstorphine (2006)'s affect regulation model and is supported by considerable longitudinal and experimental evidence linking rumination to increases in the experience of negative affect and depressive symptoms (Mor & Winquist, 2002; Watkins, 2008); and by evidence that while suppression may inhibit the behavioural response associated with negative emotion, it does not reduce, and in fact increases, the subjective experience of this emotion (Gross & John, 2003). In view of this evidence, it is not surprising that the use of

emotion suppression and experiential avoidance to bypass unwanted emotions has been found to give rise to higher levels of psychological distress (Kashdan et al., 2006). There is also some evidence that distress mediates the association between the processing of emotion-related information and ED pathology among women with AN and BN (Gilboa-Schechtman, Avnon, Zubery, & Jeczmien, 2006). Finally, both BAE and suppression have been linked to negative emotionality in individuals with mood disorders (Campbell-Sills, Barlow, Brown, & Hofmann, 2006) and results of a recent meta-analysis examining the direction of the association between depression and ED pathology are consistent with an affect-regulation model, whereby ED pathology may develop as a means of regulating low mood among individuals with depressive symptoms (Puccio et al., 2016).

Why, then, might BAE be conducive to increased use of maladaptive ER strategies? Suppression serves the function of inhibiting the expression of emotion, while rumination may serve the function of narrowing attention and thereby avoiding or numbing aversive emotional awareness (Startup et al., 2013), albeit both maladaptively. Women with high BAE, thus, may employ various maladaptive ER strategies (and fail to use various adaptive ER strategies) as a means of reducing awareness and expression of aversive emotions. However, given that both rumination and suppression may actually lead to increases in negative emotion and depressive symptoms, ED pathology may then be called on as an additional maladaptive means of escaping aversive emotions, the latter now exacerbated by the use of ineffective ER strategies. To date, however, no research has explored a path model that includes mediation of the effect of BAE on ED pathology via increased use of maladaptive ER strategies (reappraisal, reflection, and emotional reflection), negative emotionality, and depressive symptoms.

#### **The Present Study**

With these considerations in mind, the goal of the current research was to examine the

role played by BAE and the ER strategies of rumination, reflection, suppression and reappraisal, in predicting ED pathology, both directly and indirectly via increases in negative emotionality and symptoms of depression. Specifically, the study aims, firstly, to determine whether and to what extent the different ER strategies and BAE are uniquely associated with ED pathology; whether emotional reflection is associated with ED concerns over-and-above general reflection; and whether the various ER strategies, BAE, negative emotionality, and depressive symptoms overlap or are independently significant in predicting ED pathology. Secondly, the study aims to determine whether the associations between the ER strategies and ED pathology are mediated by negative emotion and depressive symptoms. Thirdly, the study aims to investigate a path model that allows for both direct associations between BAE and ED pathology and associations between these variables mediated by the use of the various ER strategies, negative emotionality, and depressive symptoms in both the full sample and subgroups of probable-ED and non-ED participants separately.

In regard to the first aim it is hypothesised that: (1a) BAE, rumination, suppression, and depressive symptoms will each predict increased ED pathology, while reflection, emotional reflection and reappraisal will each predict reduced ED pathology; (1b) emotional reflection will predict reduced ED pathology over-and-above the influence of general reflection; (1c) the variance in ED pathology explained by the various ER strategies, when fitted simultaneously, will be overlapping, such that the independent effect of each will be reduced; and (1d) when fitted simultaneously with BAE, negative emotion, and depressive symptoms, the independent effect of the ER strategies will be further reduced. With regard to the second aim of the study it is hypothesised that negative emotion and depressive symptoms will mediate the associations between each of the ER strategies and ED pathology. Finally, in relation to the third study aim, it is hypothesised that (3a) the various ER strategies will mediate the association between BAE and ED pathology, such that a path model that includes

ER strategies, negative emotionality, and depressive symptoms will be a good fit for the data in the full sample; and (3b) that this final path model will demonstrate a good fit for the data in both probable-ED and non-ED subgroups.

#### Method

# **Participants**

Participants were 724 women aged between 18 to 62 years ( $M_{age} = 22.62$ , SD = 6.52;  $M_{BMI} = 22.28$ , SD = 4.71; the "full sample"). Of these, 197 were recruited via advertisements displayed on eating disorder support websites ("web sample"), 208 were recruited via email invitation to participate forwarded by family and friends of the researchers to prospective participants and via flyers displayed on Macquarie University campus ("community sample"), and 319 were first year psychology students ("student sample"). Inclusion criteria were being female and aged 18 years or older. These methods were designed to control for potential gender and maturational influences on eating pathology while ensuring the recruitment of a sufficiently large subgroup of participants with high levels of ED pathology. The student sample participated in return for course credit, whilst those in the community and web samples were able to enter a prize draw for one of ten AUD\$100 ColesMyer Gift Cards.

#### Measures

Eating Attitudes Test (EAT-26). In the current study ED pathology was assessed using two measures of ED concerns and symptomology, the first of which was the EAT-26 (Garner et al., 1982), a 26-item multiple-choice measure of attitudes towards eating. EAT-26 includes items that assess *dieting*, *bulimia* and *food* preoccupation, and oral control, rated on a 6-point scale from never to always (scored such that never, rarely, sometimes = 0, often = 1, usually = 2 and always = 3). One negatively worded item is reverse scored and items summed to obtain a total score from 0 to 78, where scores of 20 or above indicate eating disorder risk (Garner et al., 1982). The EAT-26 is widely used as a screening measures for ED symptoms

and concerns and has been found to demonstrate high reliability (Garner & Garfinkel, 1979; Garner et al., 1982) and validity in various study populations (Garner et al., 1982; Lee et al., 2002; Mintz & O'Halloran, 2000). Cronbach's alpha for each measure in the current study full sample is presented in Table 2.

Eating Disorder Diagnostic Scale (EDDS). The second measure of ED pathology used was the EDDS (Stice et al., 2000), a 22-item self-report measure of ED pathology designed to assess the diagnostic criteria for, and thereby generate, specific (DSM-IV) ED diagnoses. It contains 4 items assessing the attitudinal symptoms of AN and BN during the past 3 months, rated on a 7-point Likert scale (from  $0 = not \ at \ all \ to \ 6 = extremely$ ); 4 items assessing the frequency of binging behaviour in number of times per week (BN) and number of days per week (BED); 6 items assessing the experiential elements of binge episodes; 4 items assessing frequency of purging behaviours (vomiting, use of diuretics or laxatives, fasting, or excessive exercise); and 4 items measuring height, weight, number of missed menstrual periods, and whether contraceptive pill used in past 3 months. These responses are used to generate DSM-IV diagnoses of AN, BN, and BED. Additionally, relevant items may be summed to obtain an ED symptom composite score (EDDS-SC), which can be used as a continuous, global measure of ED pathology. EDDS has been shown to be reliable and valid in non-clinical and clinical samples of adult women; demonstrating good criterion validity with interview-based diagnoses, convergent validity with validated measures of eating pathology, and internal consistency (Krabbenborg et al., 2012; Stice, Fisher, & Martinez, 2004; Stice et al., 2000). The EDDS-SC has also shown test-retest reliability (r = .87), internal consistency ( $\alpha = .89$ ), and convergent validity with other measures of eating pathology (Stice et al., 2000).

For the current study, the algorithm used to generate DSM-IV ED diagnoses was adapted to align with DSM-5 (American Psychiatric Association, 2013) criteria for AN, BN,

BED, and other specified feeding or eating disorders (OSFED; namely, purging disorder and restrained eating disorder) (SPSS syntax for the scoring algorithm is available from the first author upon request). On this basis, participants were classified into "non-ED" (no probable ED diagnosis) and "probable-ED" (probable diagnosis of any DSM-5 ED). In the current study, to test hypotheses related to the first study aim, EAT-26 total score was used as a continuous, global measure of ED pathology. In testing hypotheses related to the second and third study aims, both EAT-26 total score and EDDES-SC score were used as indicators of an ED pathology latent variable in the SEM models, in order to reduce measurement error.

Beliefs about Emotions Scale (BAES). The BAES (Rimes & Chalder, 2010) is a 12item scale that assesses beliefs about the unacceptability of experiencing and expressing negative emotions. The BAES contains items such as "To be acceptable to others, I must keep any difficulties or negative feelings to myself" rated on a 7-point Likert scales (where  $\theta$  = totally disagree,  $\theta$  = neutral, and  $\theta$  = totally agree), which are summed to obtain a total score ranging from 0 to 72. The BES has demonstrated high internal consistency ( $\alpha$  = .91), construct validity, and unidimensionality (Rimes & Chalder, 2010).

Emotion Regulation Questionnaire (ERQ). The ERQ (Gross & John, 2003) is a 10item self-report scale measuring the two ER strategies of cognitive reappraisal and expressive
suppression. The reappraisal subscale contains 6-itmes (e.g., "When I want to feel more
positive emotion I change the way that I am thinking about the situation") and the
suppression subscale 4 items (e.g., "I control my emotions by not expressing them"), each
rated on a 7-point scale (1 = strongly disagree to 7 = strongly agree). Items are averaged to
obtain a score from 1 to 7 on each subscale. The ERQ suppression and reappraisal subscales
have demonstrated good convergent and discriminant validity with measures of inauthenticity,
coping, mood, rumination, and big-five personality dimensions (Gross & John, 2003; John &
Gross, 2004), the two-factor structure has been supported across a number of samples (Gross

& John, 2003; Melka, Lancaster, Bryant, & Rodriguez, 2011), with subscales demonstrating adequate internal consistency ( $\alpha$  = .75 to .82 and .68 to .76 for reappraisal and suppression respectively; Gross & John, 2003).

Rumination-Reflection Questionnaire (RRQ). The RRQ (Trapnell & Campbell, 1999) is a 24-item measure of the distinction between ruminative and reflective dispositional self-attentiveness. The 12-item rumination subscale assesses the disposition to engage in repetitive thinking about the past (e.g., "I often find myself reevaluating something I've done") and the 12-item reflection subscale assesses the disposition to engage in reflective self-exploration (e.g., "My attitudes and feelings about things fascinate me"). Each item is rated on a 5-point scale (1= strongly disagree to 5 = strongly agree), negatively worded items are reverse scored, and items averaged to obtain a mean score for each subscale (ranging from 1 to 5). The two unique factors of the RRQ have been supported by factor analysis, with each subscale demonstrating high internal consistency (rumination:  $\alpha$  = .90; and reflection:  $\alpha$  = .91), and good convergent and discriminant validity with measures of five-factor personality and private self-consciousness (Trapnell & Campbell, 1999).

Reflecting on Feelings Scale (ROF). The ROF (McIlwain & Galati, 2005) is a 10item measure of emotional reflection. It contains items such as "I can change the way I feel by the way I pay attention to that feeling" answered on a 5-point Likert scale (where I =never, 2 = hardly ever, 3 = sometimes, 4 = often, and 5 = all the time). Items were summed to obtain a total score ranging from 10 to 50. Initial studies have found the ROF to demonstrate acceptable internal consistency ( $\alpha = .76$ ; McIlwain & Galati, 2005), however, further research is required to establish construct validity.

**Differential Emotions Scale IV-A (DES IV-A) negative emotionality subscale.** The DES IV-A (Izard, 1982) is a 36-item trait measure of the experience of discrete emotions of interest, joy, surprise, sadness, anger, disgust, contempt, inner-directed self-hostility, fear,

shame, shyness, and guilt. The DES-IV asks participants to rate how often in their daily life they feel each emotion statement on a 5-point scale ( $I = rarely \ or \ never$  to  $S = very \ often$ ). The three items tapping each of the emotions of sadness, anger, disgust, contempt, inner-directed self-hostility, fear, shame, shyness, and guilt were summed to obtain a total score for negative emotionality, ranging from 27 to 135. The DES-IV has shown moderate to strong internal consistency for each emotion subscale ( $\alpha$ 's in the range of .60 to .85) and evidence of good construct validity (Blumberg & Izard, 1985, 1986; Izard et al., 1993).

**Depression, Anxiety and Stress Scale (DASS-21) depression subscale.** Depression was measured using the depression subscale of the DASS-21 (Lovibond & Lovibond, 1995a), a 7-item measure of depression over the period of the past week. Items are rated on a 4-point scale ( $0 = did \ not \ apply \ to \ me \ at \ all \ to \ 3 = applied \ to \ me \ very \ much, \ or \ most \ of \ the \ time)$  and summed and then multiplied by two to obtain a total score ranging from 0 to 42. The depression subscale has demonstrated adequate internal consistency (Cronbach's  $\alpha = .81$ ) and good construct validity (Lovibond & Lovibond, 1995b).

# **Procedure**

Data were collected via an online survey using Qualtrics software. Participants were given a brief description of the research and indicated their informed consent to participate prior to commencement of the survey. They were then asked to provide basic demographic information, including age, height and weight, before commencing the survey proper (the order of study measures, and of items within measures, were randomised to minimise order effects). Following completion of the survey participants were directed to a separate webpage to enter the prize draw or apply for course credit. All aspects of the study methods were approved by Macquarie University Human Research Ethics Committee.

# **Statistical Analysis**

Unless otherwise indicated, analyses were conducted using IBM Statistical Package

for Social Sciences (SPSS) version 24 with alpha set to 0.05. Missing values were present in data obtained by less than 1 per cent of participants (0.55%) and SPSS Missing Value Analysis revealed such data to missing at random (MAR), as such it was deemed appropriate to exclude missing data listwise (Cheema, 2014; Garson, 2015). Samples were first compared across each of the study variables using General Linear Model Analysis of Variance (GLM ANOVA) with simple, Bonferroni-adjusted contrast tests, and GLM ANOVA used to test for interaction effects between recruitment group and each of the independent variables in predicting ED concerns and ED symptoms, in order to confirm the appropriateness of pooling across recruitment methods. The strength and direction of the associations between study variables was assessed by Pearson bivariate correlations. Differences in subgroup mean scores on study variables were compared using independent samples t-tests. Multiple linear regression analysis was then employed to assess the extent to which study variables individually and jointly predicted ED pathology. Consistent with previous research, age and BMI were included as covariates in all Regression Analyses. However, such inclusion did not change the statistical significance of any of the results. Given this, and consideration of the number of variables to be included and complexity of the models tested in the path analyses, BMI and age were not included as covariates in SEM analyses in AMOS.

Mediation effects and path model fit were tested via SEM using the maximum likelihood method (ML) in IBM SPSS AMOS (version 22) and using 10,000 bootstrap samples to estimate the size of the total, direct, and indirect effect and to provide 95% confidence intervals (CIs) around each effect to test for statistical significance (Byrne, 2010). Bootstrapping is a resampling method that estimates standard errors and creates confidence intervals to allow for the testing of mediation effects and, importantly, has the benefit of being free from the requirement of multivariate normality that underlies SEM (Byrne, 2010). SEM in AMOS used full information maximum likelihood method to estimate missing data.

The overall goodness-of-fit of structural models was assessed based on the Tucker-Lewis Index (TLI), Comparative Fit Index (CFI), root mean square error of approximation (RMSEA) and RMSEA closeness of fit (PCLOSE) fit indices. According to commonly accepted conventions (Byrne, 2010; Schreiber, Stage, King, Nora, & Barlow, 2006), a priori standards of acceptable goodness-of-fit chosen were TLI greater than .95, CFI greater than .95, RMSEA less than .08, and PCLOSE greater than .05. Chi-square difference ( $\Delta \chi^2$ ) tests were used to compare nested models, with a Bonferroni adjusted alpha level for the number of comparisons (adjusted *p*-value of < .05/3 = .017 for significance).

#### Results

# **Preliminary Analysis**

The GLM ANOVA contrast tests indicated that the web sample scored significantly higher than both the community and student samples (which did not significantly differ from one another) on each of the study variables, with the exception of reflection and BMI scores, which did not vary across any groups. These differences were as expected given the bias towards the more clinical presentation of participants recruited via ED self-help websites. However, there were no significant interactions between recruitment method and any independent variables (IVs) in predicting ED concerns, therefore, pooling of data across the recruitment methods was deemed appropriate.

EAT-26 data were positively skewed and potential violations of the assumptions of regression analysis were apparent. A square-root transformation of EAT-26 scores (EAT-26-Sqrt) yielded an approximately normal distribution of the data and no violations of the regression assumptions were apparent when the analysis was repeated using EAT-26-Sqrt. Since there were no changes to the statistical significance of any of the results when repeating the analysis, and for ease of interpretation, results obtained with the original EAT-26 scores are presented. Given the strong correlations among the independent and mediator variables

multicollinearity among all predictor variables was assessed but no major concerns were identified, with collinearity tolerance statistics falling in the range of .339 to .950 (> .2; Field, 2009) for predictors in the multiple regression model. Finally, given that age and body mass index (BMI) have been identified in past research as inversely associated with ED pathology, age and BMI were included as covariates in all regression analyses; although the statistical significance of results did not vary with inclusion of age and BMI as covariates.

Descriptive statistics and correlations between study variables in the full sample are given in Tables 1 and 2. According to the EDDS, 385 participants were classified as having no probable DSM-5 ED diagnosis (53.2%; "non-ED") and the remaining 339 participants met operational criteria for a probable DSM-5 ED diagnosis (46.8%; "probable-ED"); of these, 67 were classified as probable AN, 130 probable BN, 30 probable BED, 56 purging disorder, and 56 restrained eating disorder. Descriptive statistics for each subgroup and tests of group differences on study variables are presented in Table 1. As can be seen, the probable-ED subgroup scored significantly higher than the non-ED subgroup on measures of ED pathology, depressive symptoms, negative emotionality, suppression, and rumination, and lower on measures of reappraisal, reflection, and emotional reflection. There were no differences between the subgroups in age and BMI.

# **Regression Analysis**

Hierarchical multiple regression analysis, results of which are shown in Table 3, indicated that suppression, rumination, BAE, negative emotionality, and depressive symptoms each individually significantly predicted increased ED concerns, whereas reappraisal, emotional reflection, and general reflection each significantly predicted a decrease in ED concerns. When emotional reflection and general reflection were simultaneously regressed on ED concerns, emotional reflection ( $\beta = .268$ , t = -6.934, p < .001), but not general reflection ( $\beta = .000$ , t = .011, t = .991), was found to be independently associated with ED concerns.

Table 1

Descriptive Statistics for Study Variables in the Full Sample and Comparison of Non-ED and Probable-ED Subgroups

|             |     |       | Fu    | Full Sample |       |          |      | Non-ED   | ED    | Probable-ED | le-ED | t-tests | S,    | C      | Cohen's d      |
|-------------|-----|-------|-------|-------------|-------|----------|------|----------|-------|-------------|-------|---------|-------|--------|----------------|
|             |     |       |       | •           |       | Skewness | ssən | subgroup | dno   | subgroup    | dno   |         |       |        |                |
|             | u   | M     | QS    | Min         | Max   | Stat     | SE   | M        | QS    | M           | SD    | t       | d     | p      | 95% CI         |
| Age         | 724 | 22.62 | 6.58  | 18          | 62    | 2.35     | .091 | 22.53    | 6.67  | 22.72       | 6.35  | 380     | .704  | 028    | .118,174       |
| BMI         | 724 | 22.28 | 4.71  | 12.88       | 51.02 | 1.88     | .091 | 22.19    | 4.65  | 22.39       | 4.78  | 560     | .575  | 042    | .104,188       |
| ED Sx       | 724 | 29.80 | 19.07 | 0           | 111   | 1.04     | .091 | 18.26    | 12.11 | 42.91       | 16.94 | -22.254 | <.001 | -1.658 | -1.488, -1.823 |
| (EDDS-SC)   |     |       |       |             |       |          |      |          |       |             |       |         |       |        |                |
| ED concerns | 724 | 20.07 | 18.19 | 0           | 75    | .91      | .091 | 10.07    | 10.75 | 31.43       | 18.26 | -18.852 | <.001 | -1.404 | -1.241, -1.567 |
| (EAT-26)    |     |       |       |             |       |          |      |          |       |             |       |         |       |        |                |
| Depressive  | 724 | 15.30 | 12.41 | 0           | 42    | .59      | .091 | 10.20    | 9.78  | 21.10       | 12.54 | -12.912 | <.001 | 962    | 088, -1.116    |
| symptoms    |     |       |       |             |       |          |      |          |       |             |       |         |       |        |                |
| Negative    | 723 | 73.40 | 22.05 | 27          | 135   | .16      | .091 | 63.35    | 19.18 | 84.85       | 19.38 | -14.963 | <.001 | -1.115 | 958, -1.272    |
| emotion     |     |       |       |             |       |          |      |          |       |             |       |         |       |        |                |
| BAE         | 724 | 41.62 | 16.56 | 0           | 72    | 204      | .091 | 34.95    | 15.23 | 49.19       | 14.65 | -12.778 | <.001 | 952    | 797, -1.105    |
| Reappraisal | 723 | 4.35  | 1.30  | -           | 7     | 354      | .091 | 4.55     | 1.25  | 4.11        | 1.31  | 4.642   | <.001 | 356    | .199, .493     |
| Suppression | 723 | 3.75  | 1.43  | -           | 7     | 80:      | .091 | 3.42     | 1.39  | 4.13        | 1.39  | -6.966  | <.001 | 519    | 371,668        |
| ROF         | 724 | 30.59 | 6.63  | 10          | 20    | 15       | .091 | 31.77    | 6.04  | 29.24       | 7.00  | 5.176   | <.001 | 386    | .238, .533     |
| Rumination  | 721 | 3.85  | 9/.   | -           | 2     | 41       | .091 | 3.59     | .74   | 4.14        | 99:   | -10.522 | <.001 | 786    | 634,937        |
| Reflection  | 721 | 3.30  | .71   | -           | 2     | .05      | .091 | 3.36     | .71   | 3.23        | .71   | 2.471   | .014  | .185   | .038, .331     |
|             |     |       |       |             |       |          |      |          |       |             |       |         |       |        |                |

BMI = Body Mass Index. ED Sx = Eating disorder symptoms. EDDS-SC = Eating Disorder Diagnostic Scale ED symptom composite score. EAT-26 = Eating Attitudes Test. BAE = Beliefs about the unacceptability of negative emotion experience & expression. ROF = Reflecting on Feelings Scale (emotional reflection). Cohen's d = between

group effect size. 95% CI = 95% confidence intervals around Cohen's d.

Table 2

Correlations Among Study Variables in the Full Sample

|    |                     | 1     | 2    | 3    | 4                | 5                | 9     | 7                | ~    | 6                | 10        | 11        | 12        |
|----|---------------------|-------|------|------|------------------|------------------|-------|------------------|------|------------------|-----------|-----------|-----------|
| -  | Age                 | I     |      |      |                  |                  |       |                  |      |                  |           |           |           |
| 7  | BMI                 | .195  | I    |      |                  |                  |       |                  |      |                  |           |           |           |
| 3  | ED symptoms         | .078  | .080 | I    |                  |                  |       |                  |      |                  |           |           |           |
| 4  | ED concerns         | .062  | 103  | .738 | .95 <sup>a</sup> |                  |       |                  |      |                  |           |           |           |
| 5  | Depressive symptoms | .037  | 019  | .518 | .577             | .93 <sup>a</sup> |       |                  |      |                  |           |           |           |
| 9  | Negative emotion    | .053  | 013  | .569 | .616             | .740             | .96ª  |                  |      |                  |           |           |           |
| 7  | BAE                 | .065  | 016  | .486 | .539             | .560             | .625  | .93 <sup>a</sup> |      |                  |           |           |           |
| ~  | Reappraisal         | 039   | .072 | 192  | 225              | 341              | 312   | 197              | .90ª |                  |           |           |           |
| 6  | Suppression         | 013   | 024  | .332 | .398             | .426             | .447° | .588             | 002  | .83 <sup>a</sup> |           |           |           |
| 10 | ROF                 | 035   | .029 | 227  | 267**            | 340**            | 325   | 282              | .558 | 177              | $.86^{a}$ |           |           |
| 11 | Rumination          | .064  | .004 | .355 | .435             | .498             | .611  | .534             | 275  | £399°.           | 309       | $.92^{a}$ |           |
| 12 | Reflection          | £200. | .047 | 097  | 094              | 102              | 072   | 098              | .254 | 159              | .367**    | .088      | $.87^{a}$ |

\*. Correlation significant at p < .05 (2-tailed). \*\*\* Correlation significant at p < .01 (2-tailed). \*\* Cronbach's  $\alpha$  in the present study. BAE = Beliefs about the unacceptability of

negative emotion experience & expression. BMI = Body Mass Index. ROF = Reflecting on Feelings Scale (emotional reflection).

Summary of Results of Regression Analysis in the Full Sample

|                  | Each var | Each variable fitted individually predicting ED concerns <sup>a</sup> | d individ<br>concerr | lually in<br>ns <sup>a</sup> | Emotion regulation variables fitted simultaneously ab | regulation simultaneou | variables<br>usly <sup>a b</sup> | fitted                   | Emotion regulat together with BAE, and Depressive | Emotion regulation<br>ther with BAE, Ne<br>and Depressive syr | on variables fitted egative emotion mptoms a c * | s fitted                 |
|------------------|----------|---|----------------------|------------------------------|---|------------------------|----------------------------------|--------------------------|---|---|--|--------------------------|
| DV: ED concerns  | β        | t   | р                    | Unique $R^2$                 | β   | t                      | d                                | Unique<br>R <sup>2</sup> | β   | t   | d  | Unique<br>R <sup>2</sup> |
| Depressive Sx    | .573     | 18.845  | <.001                | .327                         | I   | ı                      | ı                                | ı                        | .206  | 4.745   | <.001  | .017                     |
| Negative emotion | .613     | 20.916  | <.001                | .375                         | I   | Ι                      | I                                | I                        | .298  | 6.226   | <.001  | .030                     |
| BAE              | .533     | 16.939  | <.001                | .283                         | I   | I                      | I                                | I                        | .174  | 4.180   | <.001  | .014                     |
| Reappraisal      | 218      | -5.978  | <.001                | .047                         | 099   | -2.546                 | .011                             | 900:                     | .004  | .124  | .902   | <.001                    |
| Suppression      | .397     | 11.664  | <.001                | .158                         | .297  | 8.858                  | <.001                            | .077                     | 090   | 1.656   | 860.   | .002                     |
| ROF              | 268      | -7.470  | <.001                | .071                         | 068   | -1.733                 | .084                             | .003                     | 031   | 897   | .370   | <.001                    |
| Rumination       | .431     | 12.847  | <.001                | .185                         | .293  | 8.407                  | <.001                            | .070                     | .028  | .749  | .454   | <.001                    |
| Reflection       | 099      | -2.676  | 800.                 | .010                         | I   | I                      | I                                | I                        | I   | I   | I  | I                        |

unacceptability of negative emotion experience & expression. Depressive Sx = Depressive symptoms. ROF = Reflecting on Feelings Scale (Emotional Reflection). <sup>b</sup>. Model  $^{a}$  = Controlling for age and Body Mass Index (BMI).  $\beta$  = Standardised Regression Coefficients. ED Concerns = Eating Attitudes Test (EAT-26). BAE = Beliefs about the

1:  $R^2 = .343$ . Evall Model:  $R^2 = .442$ . R<sup>2</sup> Change (Model 1 to Full Model) = .10,  $F_{(2.707)} = 64.07$ , p < .001.

General reflection was therefore excluded from all further analyses. Additional analysis indicated that when the ER variables were fitted simultaneously; suppression, rumination and reappraisal strategies remained independently associated with ED concerns, whereas the contribution of emotional reflection was no longer independently significant (Table 3). When BAE, negative emotionality and depressive symptoms were included in the regression models, only these three variables remained independently associated with ED concerns (Table 3).

# **SEM Analysis of Model Fit and Mediation Effects**

Hypothesis 2 – Model fit. To test the hypothesis that both negative emotionality and depressive symptoms would mediate the relationships between ER strategies and ED pathology the hypothesised path model (Model A1) was fitted first (see Figure 1). Results revealed that Model A1 demonstrated very good fit for the data (Table 4). Since a number of non-significant paths were apparent, however, Model A2, which constrained these paths to zero was fitted. This model had equivalent overall model fit (no significant  $\Delta \chi^2$ ) and very good fit of the data and was therefore deemed to be more parsimonious.

*Mediation.* The indirect effect of ER strategies on ED concerns via negative emotionality and depressive symptoms are shown in Table 5. As can be seen, the indirect effect of each of reappraisal, suppression, and rumination on ED pathology via negative emotion and depressive symptoms were significant and the direct effect of reappraisal and rumination on ED concerns was no longer significant, indicating evidence of mediation via negative emotionality and depressive symptoms in the case of reappraisal, rumination and suppression. In the presence of the other predictors, emotional reflection did not have any (direct or indirect) independent effect on ED pathology.

**Hypothesis 3(a)** – **Model fit.** To test the hypothesis that a path model including ER strategies, negative emotionality, and depressive symptoms would mediate the association between BAE and ED pathology, three alternate models were assessed. The hypothesised

model (Model 1) was tested first (Figure 2). Model 2 constrained to zero all of the non-significant paths from Model 1, while Model 3 removed reappraisal (Figure 3). As can be seen in Table 4, Model 1 had poor fit for the data, there being a number of non-significant paths. Model 2 had significantly improved fit for the data (according to the  $\chi^2\Delta$  test), relatively poor fit according to several goodness-of-fit indices and adequate fit according to others. In Model 3, reappraisal, which had the weakest path coefficients and which was negatively associated with ED pathology, was removed. Since this led to significantly improved overall fit and very good fit of the data, Model 3 was considered the final mediation model. Testing of the indirect effect of BAE on ED pathology via ER strategies, negative emotionality, and depressive symptoms in Model 3 (the final model) showed that BAE has a significant indirect effect on ED pathology via suppression, rumination, negative emotionality, and depressive symptoms and that the direct effect of BAE on ED pathology remained significant, indicating evidence of mediation (Table 5). That is, BAE explained some unique variance in ED pathology that was not also explained by suppression, rumination, negative emotionality, and depressive symptoms.

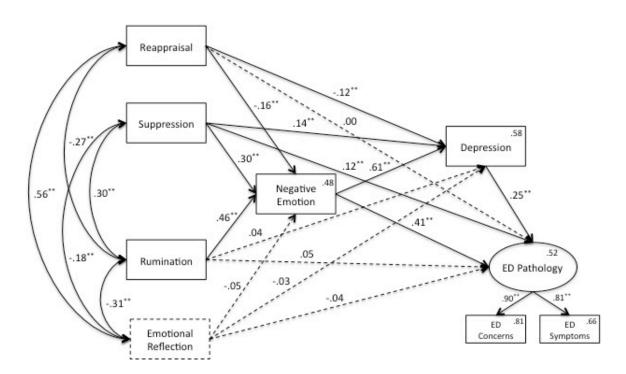


Figure 1. Path Model A1 – Mediation of relationship between emotion regulation strategies and ED pathology by negative emotionality and depressive symptoms. Model fit:  $\chi^2 = 7.549$ , df = 6, p = .273, CMIN/df = 1.258, CFI = .999, TLI = .997, RMSEA = .019 (90%CI: .000, .055, PCLOSE = .917). \*\* = p < .001. Dashed path lines are not significant (p > .05).

*Note*. Reappraisal and suppression are conceptualised as orthogonal constructs and indeed they were uncorrelated in the current study and thus the covariation path between these variables was removed from the model.

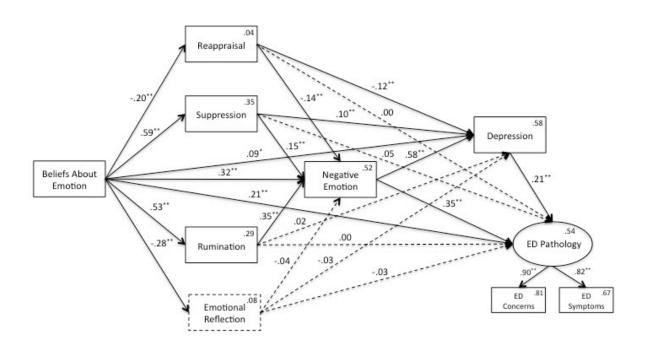


Figure 2. The Hypothesised Full Mediation Model (Model 1). Model explains 54% of the variance in ED Pathology. Model fit:  $\chi^2 = 312.970$ , df = 12, p < .001, CMIN/df = 26.081, CFI = .903, TLI = .710, RMSEA = .187 (90%CI: .169, .205, PCLOSE < .001). \*\* = p < .001. Dashed path lines are not significant (p > .05).

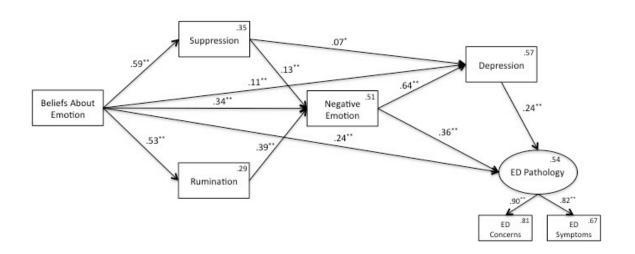


Figure 3. The Final Mediation Model (Model 3). All possible indirect effects statistically significant (p < .001). The Final Model explains 54% of the variance in ED pathology. Model fit:  $\chi^2 = 11.552$ , df = 8, p = .172, CMIN/df = 1.444, CFI = .999, TLI = .997, RMSEA = .025 (90%CI: .000, .054, PCLOSE = .916). \*\* = p < .001.

Table 4

Summary of Results of Path Analyses Comparing Models A1 and A2 and Models 1 (Hypothesised Model), Model 2 (removing non-significant paths/variables), and Model 3 (further removing Reappraisal)

|                                 | ×       | df  | d      | CMIN/df | CFI  | TLI  | TLI RMSEA | (90% CI) PCLOSE | PCLOSE | $\Delta \chi^2(df)$ | р     |
|---------------------------------|---------|-----|--------|---------|------|------|-----------|-----------------|--------|---------------------|-------|
| Hypothesis 2(a) Model A1        | 7.549   | 9 0 | .273   | 1.258   | 666. | 766. | .019      | (.000, .055)    | .917   | (6) 203 1           | 30    |
| Model A2 Hypothesis 3(a)        | 12.133  | 0   | . I 45 | /15.1   | 866. | 666. | .027      | (.000, .033)    | 9006.  | 4.386 (2)           | .v.   |
| Model 1                         | 312.970 | 12  | <.001  | 26.081  | .903 | .710 | .187      | (.169, .205)    | <.001  |                     |       |
| Model 2                         | 56.580  | 12  | <.001  | 4.715   | .984 | .963 | .072      | (.054, .091)    | .025   |                     | <.001 |
| Model 3                         | 11.552  | ∞   | .172   | 1.444   | 666. | 766. | .025      | (.000, .055)    | .916   | 44.028 (4)          | <.001 |
| Hypothesis 3(b)<br>Non-clinical |         |     |        |         |      |      |           |                 |        |                     |       |
| Model 3<br>ED-clinical          | 17.924  | ∞   | .022   | 2.241   | 686  | .971 | .057      | (.021, .093)    | .328   |                     |       |
| Model 3                         | 20.221  | ∞   | .010   | 2.528   | 786. | 965  | 890.      | (.031, .105)    | .186   |                     |       |

 $\chi^2 = \text{Chi-squared}$ . df = Degrees of Freedom. p = Chi-squared significance test. CMIN/df = Chi-squared/degrees of freedom ratio. CFI = Comparative Fit Index. TFI = Tucker-(BAE), ER strategies, negative emotion, depression, and ED pathology. Model 2 = Constrained to zero the non-significant paths in Model 1. Model 3 = The Final Mediation Lewis Index. RMSEA = Root Mean Square Error of Approximation. 90% CI = 90% Confident Interval around RMSEA. PCLOSE = RMSEA closeness of fit.  $\Delta \chi^2 = \text{Chi-}$ depression. Model A2 = Constrained all non-significant pathways in Model A1 to zero. Model 1 = Hypothesised Full Mediation Model including beliefs about emotion square difference. Model A1 = Hypothesised path model testing mediation of the relationships between ER strategies and ED pathology by negative emotion and then Model (further removed reappraisal from Model 2).

NB: Model A1 and A2 represented nested models. Models 1, 2 and 3 represent nested models.

Table 5

Summary of Results of Mediation Analyses in the Full Sample

|  | Total effect      |       | Direct effect     |       | Indirect          |       | Result    |
|--|-------------------|-------|-------------------|-------|-------------------|-------|-----------|
| -  | $\beta$ (95% CI)  | d     | $\beta$ (95% CI)  | d     | $\beta$ (95% CI)  | Ь     |           |
| Hypothesis 2   |                   |       |                   |       |                   |       |           |
| Model A1   |                   |       |                   |       |                   |       |           |
| Reapp $\rightarrow$ NegEm $\rightarrow$ Dep $\rightarrow$ ED | 117 (199,034)     | .005  | 001 (074, .074)   | 866.  | 117 (139,076)     | <.001 | Mediation |
| Supp $\rightarrow$ NegEm $\rightarrow$ Dep $\rightarrow$ ED  | .324 (.255, .395) | <.001 | .124 (.059, .189) | <.001 | .200 (.162, .242) | <.001 | Mediation |
| Rum $\rightarrow$ NegEm $\rightarrow$ Dep $\rightarrow$ ED   | .314 (.244, .381) | <.001 | .046 (031, .121)  | .241  | .268 (.220, .321) | <.001 | Mediation |
| $ROF \rightarrow NegEm \rightarrow Dep \rightarrow ED$       | 074 (167, .021)   | .129  | 039 (113, .038)   | 309   | 036 (084, .011)   | .140  | ns        |
| Model A2 <sup>a</sup>  |                   |       |                   |       |                   |       |           |
| Reapp → NegEm → Dep → ED                                     | 146 (189,108)     | <.001 | I                 | I     | 146 (189,108)     | <.001 | Mediation |
| Supp $\rightarrow$ NegEm $\rightarrow$ Dep $\rightarrow$ ED  | .347 (.281, .412) | <.001 | .125 (.061, .190) | <.001 | .222 (.183, .262) | <.001 | Mediation |
| Rum → NegEm → Dep → ED                                       | .283 (.239, .327) | <.001 | I                 | Ι     | .239 (.220, .327) | <.001 | Mediation |
| Hypothesis 3(a)  |                   |       |                   |       |                   |       |           |
| Model 3 (Final Mediation Model)                              |                   |       |                   |       |                   |       |           |
| BAE→Suppr/Rum→NegEm→Dep→ED                                   | .596 (.542, .646) | <.001 | .236 (.157, .309) | <.001 | .360 (.308, .416) | <.001 | Mediation |
| Supp $\rightarrow$ NegEm $\rightarrow$ Dep $\rightarrow$ ED  | .084 (.048, .121) | <.001 |                   |       | .084 (.048, .121) | <.001 | Mediation |
| Rum $\rightarrow$ NegEm $\rightarrow$ Dep $\rightarrow$ ED   | .201 (.161, .242) | <.001 | I                 |       | .201 (.161, .242) | <.001 | Mediation |
| $NegEm \rightarrow Dep \rightarrow ED$                       | .516 (.446, .584) | <.001 | .364 (.269, .453) | <.001 | .152 (.093, .219) | <.001 | Mediation |
|  |                   |       |                   |       |                   |       |           |

Rumination. ROF = Reflecting on Feelings (emotional reflection). ns = no statistically significant effect. <sup>a</sup>. Chi-squared difference between Model A1 and Model A2 = 4.528, strategies and ED pathology by negative emotion and then depression. Model A2 = Constrained all non-significant pathways in Model A1 to zero. Model 1 = Hypothesised degrees of freedom = 2, p > .05 (no significant difference between fit of models). Model A1 = Hypothesised path model testing mediation of the relationships between ER Full Mediation Model. Model 2 = Constrained to zero the non-significant paths in Model 1. Model 3 = The Final Mediation Model testing mediation of the relationships  $\beta$  = Standardised Effect Coefficient. 95% CI = Bootstrapped 95% Confidence Intervals around Standardised Effect. p = Two-tailed significance. Reapp = Reappraisal. NegEm = Negative emotion. Dep = Depression. ED = Eating disorder psychopathology latent variable (ED concerns & ED symptoms). Supp = Suppression. Rum = among beliefs about emotion (BAE), ER strategies, negative emotion, depression, and ED pathology (removing all non-significant paths and reappraisal).

**Hypothesis 3(b)** – **Model fit.** To test the hypothesis that the final path model would have a good fit for the data in both the non-ED and probable-ED subgroups, the fit of Model 3 was assessed in these groups separately. As can be seen from the fit indices reported in Table 4, the model demonstrated very good fit in each subgroup.

### **Discussion**

The aims of the current study were to determine, first, if various ER strategies – suppression, reappraisal, rumination, reflection, and emotional reflection – and BAE each individually and uniquely predict ED pathology; second, if the effect of ER strategies on ED pathology is mediated by negative emotionality and depressive symptoms; and third if the association between BAE and ED pathology is mediated by use of the certain ER strategies, negative emotionality, and depressive symptoms. In addressing the first aim, it was hypothesised that BAE and each of the ER strategies would individually predict ED pathology and that emotional reflection would do so over-and-above general reflection. These hypotheses were supported, confirming previous findings concerning the associations between ED pathology and BAE (Hambrook et al., 2011), suppression (McLean et al., 2007), and rumination (Rawal et al., 2010), and extending previous findings with regard to reappraisal, reflection, and emotional reflection each being inversely associated with ED pathology (e.g., Danner et al., 2012; Rawal et al., 2010; Rawal et al., 2011). With regard to reflection specifically, emotional reflection appears to be particularly relevant to ED pathology, over-and-above general reflection. It was further hypothesised that the variance in ED pathology explained by the various ER strategies would be reduced when these strategies were fitted together and that the independent effect of each ER strategy would be further reduced with the inclusion of BAE and the proposed mediating variables of negative emotionality and depressive symptoms in the model. These hypotheses were supported in that when the ER strategies were fitted together the unique variance in ED pathology explained by each was reduced. In this model, the contributions of suppression, rumination, and reappraisal to variance in ED pathology remained independently significant, whereas that of emotion reflection did not. When the ER strategies were fitted with BAE, negative emotionality, and depressive symptoms, none of the ER strategies were independently significant in predicting ED pathology.

In addressing the second study aim, it was hypothesised that negative emotionality and depressive symptoms would mediate the associations between each of the ER strategies and ED pathology. Results supported this hypothesis with regard to reappraisal, rumination, and suppression, there being evidence of full mediation in the case of reappraisal and rumination and partial mediation in the case of suppression, whereas emotional reflection did not have any independent direct or indirect effect on ED pathology. These findings converge with evidence that suppression (Gross & John, 2003), rumination (Mor & Winquist, 2002; Watkins, 2008), and reappraisal (inversely) (Garnefski & Kraaij, 2006; Garnefski et al., 2004; Gross & John, 2003) are associated with increased negative emotionality and depressive symptoms, while further suggesting a role of these ER strategies in indirectly predicting ED pathology. As such, the current findings provide important new information concerning one of the processes by which ER strategies may influence ED pathology, namely, via increase in the experience of negative emotionality and depressive symptoms.

Finally, in addressing the third study aim it was hypothesised that the various ER strategies would mediate the relationship between BAE and ED concerns, such that a path model that included ER strategies, negative emotionality, and depressive symptoms would be a good fit for the data and that this would be the case among both non-ED and probable-ED subgroups. Results provided support for these hypotheses in that a reduced path model in which BAE predicted ED pathology both directly and indirectly via suppression, rumination, negative emotion, and depressive symptoms (Model 3) demonstrated excellent fit for the data

in the total sample and good fit when tested in the non-ED and probable-ED subgroups separately. These findings are consistent with evidence that BAE (K. James et al., 2015; Oldershaw et al., 2012), suppression (Gross & John, 2003), and rumination (Mor & Winquist, 2002; Watkins, 2008), are each associated with increased experience of negative emotionality and depressive symptoms, while further suggesting a role for BAE in predicting ED pathology both directly and indirectly. The current findings provide important new information concerning the possible processes by which BAE may influence ED pathology via the maladaptive ER strategies of expressive suppression and rumination, negative emotionality, and depressive symptoms.

While the adaptive ER strategy of reappraisal was found to predict ED pathology over-and-above the maladaptive ER strategies of suppression and rumination, the hypothesised path model from BAE to ED pathology via the ER strategies, negative emotionality, and depressive symptoms, demonstrated good fit only after the removal of reappraisal. These results suggest that the use of maladaptive ER strategies (suppression and rumination) may be particularly problematic in exacerbating the experience of negative emotionality and depressive symptoms and, in turn, ED pathology, whereas the more adaptive ER strategy of reappraisal may be less important in this regard. This is interesting given the emphasis on cognitive reappraisal skills training in CBT for ED (e.g., Fairburn et al., 2003), and suggests a possible explanation for why CBT is ineffective in a substantial proportion of cases (Waller et al., 2014; Zipfel et al., 2014).

# **Study Implications**

The findings of the current study provide support for an ER model of ED pathology, in which ED symptoms serve the maladaptive function of blocking, avoiding, or escaping awareness of negative emotion (Fox et al., 2012; Heatherton & Baumeister, 1991). According to such a model, attempts to avoid or manage emotional experience and expression in

response to BAE through the use of suppression and rumination are counterproductive in that they are likely to exacerbate the experience of negative emotion and depressive symptoms and, in turn, ED pathology.

According to the ER model, the experience of secondary negative emotion and depressive symptoms in response to BAE not only exacerbate distress, but also hinder the ability to attend to primary emotions and resolve the problems signalled by such emotions (Corstorphine, 2006). Further, individuals who ruminate and suppress emotional expression may fail to respond appropriately to others in and/or avoid social situations (John & Gross, 2004). Indeed, the use of suppression has also been linked to lower levels of social support, closeness with others, and social satisfaction (Srivastava, Tamir, McGonigal, John, & Gross, 2009). More broadly, the use of maladaptive ER strategies in an attempt to avoid negative emotional experience and expression, and the additional negative emotion and depressive symptoms that this engenders, may serve to interrupt the signal function of affect in alerting the individual to what is important, both intrapersonally and interpersonally, and in guiding their thoughts and actions (Corstorphine, 2006; Kennedy-Moore & Watson, 2001).

The direct experience of emotion has been found to be more psychologically adaptive than the avoidance of it via suppression or the repetitive mulling over of causes and consequences of symptoms characteristic of rumination (Watkins, 2004, 2008). As such mindfulness and mindfulness-based therapies may be helpful in the treatment of EDs. Evidence suggests that mindfulness training serves to reduce rumination and improve negative affect in people with mood disorders (Broderick, 2005; Ramel, Goldon, Carmona, & McQuaid, 2004). Mindfulness-based cognitive therapy (MBCT) has been found to reduce body image concerns, food cravings, dichotomous thinking, and emotional eating among women with disordered eating (Alberts, Thewissen, & Raes, 2012), and both mindfulness-based eating awareness training (MB-EAT) and mindfulness-action based cognitive

behavioural therapy (MACBT) have been found to reduce binge eating episodes, associated ED pathology, and depressive symptoms among individuals with BED (Courbasson, Nishikawa, & Shapira, 2011; Kristeller, Wolever, & Sheets, 2013). Further, teaching experiential processing-based self-awareness has been associated with increased self and body-acceptance among women with AN (Rawal, Enayati, Williams, & Park, 2009). Indeed, it has been suggested that mindfulness can restore connection with the signal function of affects and sensations and aid in adaptive ER (Linehan, 2015). Additionally, several other therapies have been developed with the goal of addressing emotional avoidance and suppression as these occur among individuals with ED. These include: cognitive-emotional-behaviour-therapy (CEBT-ED; Corstorphine, 2006), emotion-focused therapy for EDs (EFT; Dolhanty & Greenberg, 2007), schema-focused CBT for EDs (FSCBT; Waller, Cordery, et al., 2007), integrative cognitive-affective therapy for BN (Wonderlich et al., 2010), acceptance and commitment therapy for EDs (Sandoz, Wilson, & Dufrene, 2010), and DBT for BN and BED (Safer et al., 2009).

# Study Strengths, Limitations and Directions for Future Research

A key strength of the current study lies in its effort to bring together and explore in unison the role of several different ER strategies in accounting for variance in ED pathology, both directly and indirectly via negative emotionality and depressive symptoms. This is notable because previous exploration of ER strategies has been largely confined to the direct associations between ER strategies and negative emotion, depressive symptoms, and ED pathology conceived of as independent outcomes. The current research further extends previous research by exploring BAE potentially underlying the use of maladaptive ER strategies, thereby providing preliminary insights into the processes by which BAE contributes to increased ED pathology, namely, via use of maladaptive ER strategies and increases in negative emotionality and depressive symptoms. The research contributes new

evidence supporting the theory that maladaptive attempts at ER are likely to be counterproductive in that they may give rise to further negative emotion and depressive symptoms and, in turn, ED behaviour. Additional strengths of the current research were the recruitment of a relatively large sample of participants from a number of sources, thereby ensuring a broad range of values on each of the study variables and permitting the identification of a subgroup of probable ED cases, and the use of relatively sophisticated statistical methods (SEM and Bootstrapping procedures) and inclusion of multiple outcome measures in accounting for ED pathology. The fact that close to half of the sample was identified with probable-ED diagnoses suggests that the findings may have clinical relevance. Nevertheless, replication of the current findings in a treatment-seeking sample of individuals with EDs would have more direct implications for clinical practice and would permit exploration of potential differences in the observed associations across ED diagnoses (e.g., Danner et al., 2014; Davies et al., 2012; Naumann et al., 2015).

The most notable limitation of the current research was the use of a cross-sectional study design. Process analysis using SEM implies causality and direction of effects, which strictly speaking cannot be tested with cross-sectional data. Where there is a theoretical foundation to support the assumptions of direction of effect made, however, it may be appropriate to test mediation analyses with cross-sectional data (A. F Hayes, 2013). In support of the direction of effects in the model tested in the current study Corstorophine's (2006) affect-regulation theory provides that the relationship between BAE and ED pathology is mediated by negative emotion and response style theory (Nolen-Hoeksema et al., 2008) provides that rumination leads to ED pathology. Additionally, there is strong empirical evidence to support the direction of effects in the SEM tested in the current study from rumination to negative emotionality and depressive symptoms, and that rumination mediates the relationship between BAE and depression (Ehring & Watkins, 2008; Spasojevic & Alloy,

2001; Watkins, 2008). Further, a recent systematic review and meta-analysis of longitudinal studies on direction of effect between depression and ED pathology (Puccio et al., 2016) found support for an ER model of ED pathology, whereby ED pathology may develop as a maladaptive means of reducing negative mood, while also being a risk factor for the future development of depressive symptoms.

Nevertheless, complex bidirectional relationships likely exist between most variables in the final model tested in this research. Indeed, the occurrence of such bidirectional associations is anticipated in Corstorphine's (2006) model, which incorporates a feedback loop between ED pathology and negative emotion. There is also evidence for bidirectional associations between rumination and negative emotion (Mor & Winquist, 2002) and for mediations of the association between ED pathology and depressive symptoms by rumination (Harrell & Jackson, 2008). Thus, clearly longitudinal research is needed to elucidate these pathways and future research employing cross-panel time-lagged longitudinal designs that allow for the testing of non-recessive relationships to determine the temporal nature of bidirectionality among such variables would be particularly useful.

A second notable limitation of the current research is the reliance on self-report measures of study variables. While the use of online surveys has many advantages, potential problems with the use of self-report measures of ED symptoms in particular are well-known (House et al., 2008). Interview assessment of key study constructs would clearly be desirable in future research, for example, the Eating Disorder Examination (EDE-17.0D; Fairburn, Cooper, & O'Connor, 2014) for ED pathology and the Emotion Regulation Interview (Werner, Goldin, Ball, Heimberg, & Gross, 2011) for ER strategies.

Concerning the recruitment methods employed in the current study, these methods are susceptible to selection bias and that this may detract from the generalisability of the findings. For example, it is possible that emotionally avoidant individuals are less likely to volunteer to

participate in research aimed at assessing emotional experience. The relatively high proportion of participants with high levels of ED pathology is an example of such bias, albeit an asset in the current context. The fact that the current research was confined to adult women also limits the generalisability of the findings. Although this was reasonable for a study designed to extend research previously conducted in this demographic, the inclusion of adolescent girls and of males in future research clearly would be of interest. Finally, it should be noted that classification of participants into non-ED and probable-ED subgroups relied on a measure of ED pathology (the EDDS) designed to generate operational diagnoses according to DSM-IV criteria and validated for this purpose. The adapted algorithm employed in the current study, which permitted the generation of probable DSM-5 ED diagnoses, has not yet been validated. However, comparing model fit across non-ED and probable-ED subgroups was not the primary aim of the current research.

### Conclusion

The findings of the current study are consistent with an ER model of ED pathology and suggest that women who possess negative beliefs about the experience and expression of emotion may benefit from therapies that target maladaptive emotion schemas and ER difficulties and aim to enhance adaptive emotion awareness and regulation.

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Appendix A

Results of General Linear Model Analysis of Variance to Test for Possible Interaction Effects

Between Recruitment Group and Any Study Independent Variables in Predicting ED

Concerns and ED Symptoms

|                            | $\eta^2_{\it partial}$ | df       | F      | p     |
|----------------------------|------------------------|----------|--------|-------|
| DV = EAT-26                |                        |          |        |       |
| Group                      | .002                   | (2,689)  | .774   | .461  |
| Age                        | .005                   | (1,689)  | 3.684  | .055  |
| BMI                        | .009                   | (1,689)  | 6.042  | .014  |
| Depressive symptoms        | .020                   | (1,689)  | 14.222 | <.001 |
| Negative emotion           | .041                   | (1,689)  | 29.719 | <.001 |
| BAE                        | .020                   | (1,689)  | 13.840 | <.001 |
| Reappraisal                | .001                   | (1,689)  | .687   | .407  |
| Suppression                | .001                   | (1,689)  | .631   | .427  |
| Emotional Reflection       | <.001                  | (1, 343) | .174   | .677  |
| Rumination                 | <.001                  | (1, 343) | .093   | .761  |
| Reflection                 | .002                   | (1, 343) | 1.383  | .240  |
| Group*Negative emotion     | .004                   | (2,689)  | 1.518  | .220  |
| Group*Depressive Symptoms  | <.001                  | (2,689)  | .003   | .997  |
| Group*BAE                  | .001                   | (2,689)  | .419   | .658  |
| Group*Reappraisal          | .001                   | (2,689)  | .484   | .616  |
| Group*Suppression          | .007                   | (2,689)  | 2.566  | .078  |
| Group*Emotional Reflection | .003                   | (2,689)  | 1.046  | .352  |
| Group*Rumination           | <.001                  | (2,689)  | .127   | .881  |
| Group*Reflection           | .001                   | (2, 689) | .326   | .722  |
| DV = EDDS-SC               |                        |          |        |       |
| Group                      | .001                   | (2,689)  | .491   | .612  |
| Age                        | .002                   | (1,689)  | 1.209  | .272  |
| BMI                        | .020                   | (1,689)  | 13.756 | <.001 |
| Depressive symptoms        | .016                   | (1,689)  | 11.086 | .001  |
| Negative emotion           | .048                   | (1,689)  | 34.999 | <.001 |
| BAE                        | .022                   | (1,689)  | 15.380 | <.001 |
| Reappraisal                | <.001                  | (1,689)  | .236   | .627  |
| Suppression                | <.001                  | (1,689)  | .274   | .601  |
| Emotional Reflection       | <.001                  | (1, 343) | .090   | .764  |
| Rumination                 | .002                   | (1, 343) | 1.561  | .212  |
| Reflection                 | .003                   | (1, 343) | 1.780  | .183  |
| Group*Negative emotion     | .002                   | (2, 689) | .823   | .440  |
| Group*Depressive Symptoms  | <.001                  | (2, 689) | .022   | .978  |
| Group*BAE                  | <.001                  | (2,689)  | .006   | .994  |
| Group*Reappraisal          | .001                   | (2, 689) | .279   | .757  |
| Group*Suppression          | <.001                  | (2, 689) | .168   | .846  |
| Group*Emotional Reflection | .001                   | (2, 689) | .256   | .774  |
| Group*Rumination           | <.001                  | (2, 689) | .024   | .976  |
| Group*Reflection           | .002                   | (2,689)  | .834   | .435  |

DV = Dependent Variable. EAT-26 = Eating Disorder Test total scores). EDDS-SC = Eating Disorder Diagnostic Scale ED symptom composite score.  $\eta^2_{partial}$  = Partial Eta Squared. BAE = Beliefs about the unacceptability of experiencing and expressing negative emotion. AEE = Ambivalence aver emotional expression.

# Chapter 7

Feelings about the Self and Body in Eating Disturbances: The Role of Internalised Shame, Self-Esteem, Externalised Self-Perceptions and Body Shame

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# **Author Contributions**

First author Kim Woodward was responsible for conceptualisation, design, and conduct of the current study, managed data collection, undertook all statistical analyses, conducted the literature searches, and wrote the first draft of all manuscript. Author Doris McIlwain contributed to the conceptualisation and design of the study at a supervisory level. Author Jonathan Mond contributed to revisions of the manuscript.

#### Abstract

**Objective:** To investigate the roles of feelings of low self-worth (internalised shame and low self-esteem), externalised self-perceptions (self-objectification and body surveillance), body shame, and depressive symptoms in directly and indirectly explaining variance in eating disorder (ED) pathology across two studies. **Study 1:** In 403 women, internalised shame and self-esteem were found to each be independently associated with ED pathology, over and above one another, depressive symptoms, age and BMI. Further, body shame fully mediated the relationships between ED pathology and internalised shame and self-esteem, controlling for one another, depressive symptoms, age and BMI. Study 2: A different sample of 548 women, Structural Equation Modelling (SEM) revealed that a path model in which internalised shame predicts ED pathology both directly and indirectly via self-objectification, body surveillance, body shame, and depressive symptoms demonstrated very good fit for the data and explained 68% of the variance in ED pathology. **General conclusions:** Results support an understanding of EDs as disorders of self, in which negative feelings about the self (internalised shame and low self-esteem) are displaced onto the body (in the form of externalised self-perceptions and body shame) and are associated with increases in depressive symptoms and ED pathology, both directly and indirectly.

### Introduction

Self-concept deficits and difficulties with emotions have long been implicated in the development and maintenance of eating disorders (EDs) (Bruch, 1962, 1973); and low self-esteem is a well-accepted feature of EDs. From a developmental perspective framed in attachment theory, EDs are suggested to result from and be maintained by disturbances in self-development (Amianto et al., 2016). The fundamental principle underlying such a view of EDs as disorders of the self, is that ED symptoms are used as maladaptive means of managing the painful internal experiences emanating from deficits in the self (Amianto et al., 2016). Such a self-deficit understanding of EDs accords with emotion-regulation theories of ED pathology, such as the escape model (Heatherton & Baumeister, 1991), whereby binge eating is said to serve the function of an escape from painful self-awareness via a narrowing of attention to an unreflective focus on immediate sensations and stimuli.

Closely aligned with self-development deficit and emotion-regulation conceptualisations of EDs, Bruch (1973, 1978) proposed a displacement theory of emotion in EDs, which argues that unexpressed or undifferentiated emotions and negative feelings about the self are displaced onto the body in the form of 'feeling fat'. Consistent with emotion-regulation models, within displacement theory, ED symptoms (e.g., the pursuit of an idealised body weight/shape via extreme weight-control behaviours) are seen to function as maladaptive attempts to avoid or reduce feelings of shame and also further to enhance feelings of pride (Goss & Allan, 2009). As such, the displacement of core feelings of worthlessness or an underdeveloped self involves the redirection of ego threatening feelings to a less threatening and more concrete target – the body – and to body weight and shape, things that can ostensibly be controlled in a more concrete fashion via disturbed eating behaviours (Schupak-Neuberg & Nemeroff, 1993).

Evidence to support a combined self-deficit, emotion-regulation, and displacement theory model ('self/emotion-regulation model') of ED pathology can be found in research investigating the roles of self-esteem, internalised shame, body shame, and externalised self-perceptions (self-objectification and body surveillance) as these relate to ED pathology.

### **Self-Esteem**

One consistent way that negative feelings about the self (low self-worth) have been associated ED pathology is via the recurrent identification of low self-esteem in women with EDs. Self-esteem can be defined as confidence in one's overall worth or abilities or as self-respect (Rosenberg, 1965). Self-esteem has been found to be inversely associated with ED pathology in various population-based samples, including college students (Fredrick & Grow, 1996; Mayhew & Edelmann, 1989), lower among individuals with EDs receiving outpatient treatment than those in control group and inversely associated with body shape concerns in both groups (Beato et al., 2003). Further, differences of this kind have been found to remain after controlling for depressive symptoms (Jacobi, Paul, de Zwaan, Nutzinger, & Dahme, 2004). Low self-esteem has been associated with concerns about fatness in girls as young as 11-12 years (Button, 1990) and in a prospective study of adolescent girls, low self-esteem was associated with ED pathology at baseline, while high self-esteem was a protective factor that significantly reduced risk of onset of ED pathology at 18-month follow-up (Cervera et al., 2003; Gual et al., 2002). Thus, from preadolescence to adulthood, low self-esteem appears to be a risk factor for both preclinical and clinical ED pathology.

Additionally, improvements in self-esteem have been suggested to be integral for full recovery from EDs, with pre-treatment low self-esteem found to predict poor treatment outcomes and relapse at 12-month follow-up in individuals with bulimia nervosa (Fairburn et al., 1993). While individuals with both active and partially recovered EDs (physical and behavioural but not psychological recovery) have been found to have lower self-esteem than

fully recovered individuals and controls, the latter groups not differing with respect to levels of self-esteem (Bardone-Cone et al., 2010).

# **Internalised Shame**

An alternative way to conceptualise negative self-evaluations and their association with ED pathology is via reference to the construct of internalised shame (Cook, 1991). Within a self/emotion regulation model of ED pathology, one of the key overwhelming emotions displaced onto the body is that of shame. Shame is a self-conscious emotion characterised by an experience of the self as being flawed, worthless and powerless, and accompanied by a sense of shrinking or feeling inferior and of being exposed (Tangney, 1995; Tangney et al., 1992). Lewis (1971) further suggested that shame involves a split in selffunctioning in which the self is both agent of observation and object of condemnation, whereby the observing self becomes a denigrating audience, vilifying the object self as worthless and reprehensible (Hahn, 2000; Tangney, 1995). To this end, ED sufferers frequently identify themselves as feeling inherently worthless, deficient, or disgusting (Bruch, 1973; Garfinkel et al., 1985) and often report experiences of feeling trapped in and ashamed of their bodies (Rortveit et al., 2009). Indeed, EDs have been described as 'disorders of shame' (Kaufman, 1996, p. 129) and shame aversion has been found to moderate the association between feelings of shame and ED pathology, over and above depressive symptoms, negative emotion, and experiential avoidance in undergraduate women (Manjrekar, Schoenleber, & Mu, 2013).

Given that the experience of internalised shame involves a painful sense of self as undesirable and flawed in some way, it is not surprising that efforts are made to escape or avoid such experience. Displacement theory proposes that such ego-threatening feelings (shame and painful sense of self) may be redirected to a less threatening target, namely, the body, the latter being something that can more readily be controlled, for instance, via

preoccupation with and pursuit of idealised body weight/shape through adherence to extreme weight-control behaviours (Schupak-Neuberg & Nemeroff, 1993). That is, a focus on the control of body weight/shape through ED behaviour serves the dual functions of avoiding aversive self-awareness and attempting to enhance feelings of pride and self-worth (Button, 1990; Goss & Allan, 2009).

In support of such a theory, associations between internalised shame and ED pathology have been found in both non-clinical (Sanftner et al., 1995) and clinical (Troop et al., 2008) samples, and when controlling for depressive symptoms (Gee & Troop, 2003; Hayaki et al., 2002b) and negative emotion (Gupta et al., 2008). Internalised shame was found to predict depressive symptoms in a longitudinal study of college students (Andrews, Qian, & Valentine, 2002). Further, Grabhorn et al. (2006) found inpatient women with anorexia nervosa (AN) and bulimia nervosa (BN) (two of the most well known EDs) reported higher levels of internalised shame than inpatient women with anxiety and mood disorders, suggesting that internalised shame may play a particularly important role in the onset and/or maintenance of ED pathology.

Whilst there is considerable evidence supporting the link between both low selfesteem and internalised shame and ED pathology individually, the relative merits of these
constructs as alternate conceptualisations of low self-worth within ED research is unclear.

Thus, to our knowledge, research is yet to investigate whether internalised shame is predictive
of ED pathology over and above low self-esteem, nor whether such a relationship exists over
and above the influence of depressive symptoms. Conceivably, a distinction can be drawn
between shame and self-esteem. Feelings of low self-worth associated with internalised
shame may be seen to relate to feeling that one is fundamentally "not good enough",
unlovable, or defective as a person. Self-esteem, by contrast, may be seen to relate to feelings
of competency or capability in specific ways, for instance, self-evaluation as this relates to

actions, abilities, attributes, or specific parts of the self (Goss & Gilbert, 2002). Thus, an individual might believe that they are good at something or even talented or special in some unique way, and derive self-esteem from this knowledge, yet still feel that they are not "good enough" and deep-down fundamentally defective or unlovable.

# **Body Shame**

Researchers have also explored the association between ED pathology and shame specifically related to the body, as opposed to the more global construct of internalised shame (Troop & Redshaw, 2012). Body shame entails experiencing one's physical body as undesirable or unattractive and as a source of the shamed self (Gilbert, 2002). Additionally, internalisation of prevailing cultural standards of beauty (i.e., the 'thin-ideal'), position women to perceive a disparity between their actual and ideal-body and to experience body shame (McKinley & Hyde, 1996). Further, women who associate achievement of idealised standards of beauty and body weight/shape with their identity, as may be the case in individuals with self-concept deficits who are prone to displace feelings of self-worth onto the body, may be further primed to feel body shame when they perceive their bodies as falling short of these ideals (McKinley & Hyde, 1996). As such, body shame may increase the risk of both general psychological distress and ED pathology (Gilbert, 2002).

When considering the associations between ED pathology and specific types of shame, Burney and Irwin (2000) found that body shame and shame about eating behaviour specifically, rather than internalised shame, were associated with ED pathology in an Australian female community sample. However, Swan and Andrews (2003) found that female ED patients scored higher than non-clinical control participants on all four measures of shame considered – internalised shame, behavioural shame, bodily shame, and shame around eating – controlling for depressive symptoms. Further, whereas both internalised shame and body shame were independently associated with ED pathology in a non-clinical sample of women,

only body shame was independently associated with ED pathology in female ED patients (Doran & Lewis, 2012). In a longitudinal study of ED patients, past body shame was found to independently predict later anorexic symptoms, controlling for past depressive symptoms and internalised shame, whereas neither internalised shame nor body shame predicted later bulimic symptoms (Troop & Redshaw, 2012). Finally, body shame was found to mediate the association between low self-esteem and ED pathology in a school-based study of female adolescents (Iannaccone et al., 2016).

According to a displacement theory model of ED pathology, negative feelings about the self (internalised shame and low self-esteem) should be associated with both ED symptoms and body shame. Body shame should also be associated with ED pathology and should mediate the association between negative feelings about the self and ED pathology. Consistent with these hypotheses, body shame has been found to mediate the association between external shame and ED pathology (Duarte, Pinto-Gouveia, Ferreira, et al., 2015) and body image cognitive fusion has been found to mediate the association between external shame and binge eating, among individuals with binge eating disorder (BED), controlling for BMI and depressive symptoms (Duarte, Pinto-Gouveia, & Ferreira, 2015). Whilst dissatisfaction with body weight and shape is so common as to be characterised as a 'normative discontent' (Rodin et al., 1984), and while only a minority of women who report preoccupation with weight or shape and/or disturbed eating behaviours develop clinically-relevant eating disturbances, possessing shame-based negative evaluations of the self may be one factor distinguishing those who develop clinically significant ED pathology from those who do not (Goss & Gilbert, 2002).

# **Externalised Self-Perceptions**

Another factor that has been linked to women's experiences of feelings of worthlessness, body shame, and ED pathology is that of externalised or objectified self-

perceptions (Fredrickson, Roberts, Noll, Quinn, & Twenge, 1998). The theory that negative feelings about the self may be externalised and displaced onto the body accords with various sociocultural models that highlight externalised self-perceptions (objectified body consciousness or the extent to which women experience their body as an object and view it from an outside perspective) as a risk factor for ED pathology, for instance silencing the self theory (Jack, 1991), objectified body consciousness (McKinley & Hyde, 1996), and objectification theory (Fredrickson & Roberts, 1997). Objectification theory suggests that day-to-day life exposes women to sexually objectifying experiences which lead women to internalise an observer's perspective of themselves, labelled self-objectification (Calogero, Tantleff-Dunn, & Thompson, 2011). Objectification entails being treated – and evaluated – as an object rather than as a subject. According to objectification theory, self-objectification leads to habitual body monitoring (body surveillance), body shame (and also appearance anxiety and loss of interoceptive awareness), and, in turn, depressive symptoms and ED pathology (Fredrickson & Roberts, 1997).

In support of this theory, a number of studies have found evidence for a link between self-objectification and ED pathology (e.g., Peat & Muehlenkamp, 2011), self-objectification and body shame (e.g., Greenleaf & McGreer, 2006) and body surveillance and body shame (Calogero & Thompson, 2009). Thus, evidence from non-clinical samples of women suggests that body shame mediates the relationship between body surveillance and ED pathology (Moradi et al., 2005; Tylka & Hill, 2004) and between self-objectification and ED pathology (Noll & Fredrickson, 1998); that body surveillance and body shame jointly are associated with ED pathology both directly and indirectly via depressive symptoms (Muehlenkamp & Saris-Baglama, 2002); and that body surveillance and body shame each mediate the association between self-objectification and both ED pathology (Calogero, 2009; Tiggemann & Slater, 2001) and depressive symptoms (Tiggemann & Kuring, 2004; Tiggemann &

Williams, 2012). Further, body shame was found to mediate the association between self-objectification and drive for thinness in a clinical sample of women with EDs (Calogero et al., 2005).

Whilst objectification theory identifies sexual objectification as a cultural experience of all women that leads to self-objectification, body surveillance, body shame, and, in due course, depressive symptoms and ED pathology, objectification theory does not necessarily account for individual differences in levels of self-objectification among women. One possible factor that may increase self-objectification is internalised shame and the predisposition to displace negative feelings about the self onto the body. Consistent with this suggestion, research in college women found that low self-esteem is associated with both body surveillance and body shame (Mercurio & Landry, 2008) and that externalised self-perceptions predict ED pathology over and above the impact of shape and weight-based self-esteem (Frank & Thomas, 2003). Also in college women, in a study employing structural equation modelling (SEM), a path model that included sexual objectification (via appearance feedback), low self-esteem, body surveillance, body comparison, and body shame, was found to account for 67.9% of the variance in ED pathology (Tylka & Sabik, 2010).

## **The Present Research**

The current research seeks to explore in two mixed undergraduate/community samples of women a displacement theory-based self/emotion-regulation model of ED pathology, whereby negative feelings about the self are displaced onto the body (in the form of increased body shame), thereby promoting ED pathology. Study 1 seeks to explore the distinction between low self-esteem and internalised shame as two closely related but distinct conceptualisations of low self-worth and to assess their independent significance in explaining variance in ED pathology, over and above depressive symptoms; and investigate whether body shame mediates the relationship of each with ED pathology. Study 2 then aims

to explore, using SEM, a path model that seeks to extend displacement theory of ED pathology by incorporating elements of objectification theory, specifically by including self-objectification and body surveillance as additional mediators of the associations among internalised shame, body shame, depressive symptoms and ED pathology.

### STUDY 1

Whilst evidence of an association between internalised shame and ED pathology over and above the impact of low mood is mounting, the precise role played by shame – whether it is an etiological factor, a consequence, or part of the phenomenology of EDs – is less clear. Whether the role of internalised shame is independent of the effects of self-esteem and body shame is similarly unclear. A displacement theory of ED pathology, whereby negative feelings about the self are externalised and displaced onto the body, implies an etiological role of internalised shame and predicts that body shame will mediate the relationship between internalised shame and ED pathology.

With these considerations in mind, the aims of the first study reported here were, first, to investigate the individual relationships among ED pathology and internalised shame, low self-esteem, and body shame, and whether each is associated with ED pathology over and above depressive symptoms; second, given the conceptual similarity between the constructs of internalised shame and self-esteem, to determine whether internalised shame and low self-esteem overlap or are independent in explaining variance in ED pathology, when controlling for depressive symptoms; and third, to determine whether internalised shame is externalised and focused on the body, such that the association between shame and ED pathology is mediated by body shame, when controlling for depressive symptoms and low self-esteem. Specifically, it was hypothesised that, first, internalised shame, low self-esteem, and body shame will each individually be associated with ED pathology, controlling for depressive symptoms; second, the variance in ED pathology explained by low internalised shame and

low self-esteem, when fitted together, will be overlapping, such that the independent effects of each in explaining variance in ED pathology will be reduced, but that internalised shame will remain independently significantly associated with ED pathology, controlling for depressive symptoms; and third, body shame will mediate the associations between internalised shame and/or self-esteem and ED pathology, controlling for depressive symptoms and one another.

#### Method

# **Participants**

Participants were 403 women aged 18 years or older ( $M_{age} = 23.9$ , SD = 6.9, age range: 18-62 years; the "full sample"). Of these, 102 were first year psychology students who participated in return for course credit (the "student sample"), 126 were recruited via flyers displayed around Macquarie University campus and via email invitation to participate forwarded to prospective participants by family and friends of the researchers (the "community sample"), and 175 were recruited via advertisements displayed on eating disorder support websites (the "web sample"). Inclusion criteria were being female and aged 18 years or older, in order to control for potential gender and maturational influences. Of the 405 respondents, two were excluded on the basis of being under the age of 18 years.

### Measures

Eating Attitudes Test (EAT-26). ED pathology was assessed using the EAT-26 (Garner et al., 1982), a 26-item scale that includes items assessing *dieting*, *bulimia* and *food* preoccupation, and oral control. Participants are instructed to select a response from never to always for each items, responses of never, rarely and sometimes being scored = 0, often = 1, usually = 2 and always = 3. One negatively worded item is reverse scored and items summed to yield a total score ranging from 0 to 78, with higher scores indicating higher levels of ED pathology and scores of 20 or above indicating eating disorder risk (Garner et al., 1982). The

EAT-26 is widely used as a measure of ED pathology in population-based samples of women and has been found to have high reliability (Garner & Garfinkel, 1979; Garner et al., 1982) and validity (Garner et al., 1982; Lee et al., 2002; Mintz & O'Halloran, 2000). Cronbach's alpha in the current study for this and other study measures are presented in Table 2.

Depression, Anxiety and Stress Scale (DASS-21) depression subscale. Depressive symptoms were measured using the 7-item depression subscale of the DASS-21 (Lovibond & Lovibond, 1995a). Each item is answered on a 4-point scale (from 0 = did not apply to me at all to 3 = applied to me very much, or most of the time) and items summed and then multiplied by two to yield a total score ranging from 0 to 42, with higher scores indicating higher symptoms levels. The depression subscale has been found to have adequate internal consistency (Cronbach's  $\alpha = .81$ ) and good construct validity (Lovibond & Lovibond, 1995b).

The Feelings about Self Scale (FASS). The FASS (McIlwain & Warburton, 2004) was used to measure participants' feelings of internalised (trait) shame. The FASS is a 20-item self-report measure of feelings of shame, containing items such as: "Sometimes I just want to hide", "Sometimes I feel that I am in bits and pieces", "It's only a matter of time before people discover that I am a fake", and "If I let people know what I'm really like, they would reject me". Participants are instructed to rate each item of a 6-point Likert scale (where I=completely untrue of me and 6=describes me perfectly). Negatively worded items are reverse scored and items summed to yield a total score ranging from 20 to 120, with higher scores indicating higher levels of internalised shame. High internal consistency and construct validity have been reported in undergraduate samples ( $\alpha = .91$  to .93; Warburton, Edwards, Hossieny, Pieper, & Yip, 2008; Warburton & McIlwain, 2005).

Rosenberg Self-Esteem Scale (SES). Self-esteem was measured using the SES (Rosenberg, 1965), a 10-item self-report measure of global self-esteem. It contains items such as "On a whole, I am satisfied with myself", and "I feel that I have a number of good

qualities" answered on a 4-point scale (from I = strongly disagree to 4 = strongly agree). Negatively worded items are reverse scored and items summed to provide a total score ranging from 10 to 40, with higher scores indicating higher self-esteem. The SES is a widely used measure of global self-esteem and has excellent psychometric properties in a broad range of study populations including young adult women (Rosenberg, 1965).

Body Shame subscale of the Objectified Body Consciousness Scale (OBCS). The OBCS (McKinley & Hyde, 1996) is a 24-item scale designed to measure the three dimensions of body surveillance, body shame, and control beliefs. The Body Shame subscale contains 8-items (e.g., "When I'm not the size I think I should be, I feel ashamed") and assesses feelings of shame associated with one's body not conforming to cultural standards rated on a 6-point scale (from  $I = strongly \ disagree$  to  $6 = strongly \ agree$ ). Negatively worded items are reverse scored and items averaged to provide mean score ranging from 1 to 6, with higher scores indicating greater body shame. The body shame subscale has been found to have acceptable test-retest reliability, internal consistency ( $\alpha = .84 \ \& .70$ , for undergraduate and middle-aged women, respectively), and construct validity in non-clinical sample of women, having been found to correlate negatively with body esteem and positively was ED pathology (McKinley & Hyde, 1996).

## Procedure

Data were collected as part of a 30-minute online survey constructed using Qualtrics software. Participants were provided a brief description of the research and invited to indicate their informed consent to participate prior to commencement of the survey. Participants were asked their age (to the nearest year), height (to nearest cm), weight (to nearest kg), and gender and were then presented the study measures in randomised order, with the order of presentation of items within scales also randomised (so as to minimise order effects). Participants in the student sample received course credit in return for participation, whilst

those in the community and web samples were eligible to enter a prize draw for one of five AUD\$50 iTunes gift vouchers. The Macquarie University Human Research Ethics Committee approved all aspects of the study design and methods.

# **Statistical Analyses**

All analyses were undertaken using IBM Statistical Package for Social Sciences (SPSS) version 22. Missing Value Analysis were performed using SPSS and missing data found to be Missing Completely at Random (MCAR) according to Little's MCAR test ( $\chi^2_{(73)}$ = 61.309; p = .834) (Little, 1988), thus missing data were excluded pairwise (Garson, 2015). Preliminary analysis comparing samples across the various recruitment methods was performed using General Linear Models Analysis of Variance (GLM ANOVA) with simple contrast tests (using a Bonferroni adjusted p-value of < .017 for significance). Pearson bivariate correlations were used to determine the strength and direction of the associations between study variables. Multiple linear regression analysis was used to determine the relative importance of the independent variables (IVs), namely, self-esteem, internalised shame, and body shame in accounting for variance in ED pathology. Consistent with previous research, age and BMI were included as covariates in all multivariable analysis. Mediation analysis were conducted using the PROCESS tool in SPSS, with 10,000 bootstrap samples to estimate the size of the total, direct, and indirect effects and to provide 99% confidence intervals (CIs) around each effect. Given the number of analyses, a conservative alpha level of .01 was employed for significance testing.

## **Results**

# **Preliminary Analysis**

GLM ANOVA indicated that the web sample scored significantly higher than both the community and student samples (which did not differ from one another) on each of the study variables, with the exception of Body Mass Index (BMI), which did not vary across any

groups), as would be expected given recruitment of the web sample from ED self-help websites. There were, however, no significant interactions between recruitment method and any IVs in predicting ED pathology. Hence it was deemed appropriate to pool data across recruitment methods.

As would also be expected, in a population-based sample, EAT-26 data were found to be significantly positively skewed. A square-root transformation (EAT-26-Sqrt) yielded an approximately normal distribution of the dependent variable (DV), with no violation of the relevant analytic assumptions. Hence the transformed variable was used in subsequent analysis. Assessment of multicollinearity among predictor variables indicated tolerance statistics in the range of .236 to .936 (> .2) and variance inflation factor (VIF) statistics in the range of 1.126 to 4.230 (< 7) for all predictors (T. Z. Keith, 2006). Finally, given that age and BMI have been identified in past research as inversely associated with ED pathology, age and BMI were included as covariates in all analyses.

Descriptive statistics and correlations among study variables are given in Table 1. Approximately half (46.3%) of participants in the total sample were classified as being "at risk" of an ED (EAT-26 score  $\geq$  20).

## **Regression Analysis**

To test the hypothesis that internalised shame, self-esteem, and body shame, each individually be associated with increased ED pathology over and above depressive symptoms, a series of hierarchical regression analyses were conducted with age, BMI, and depressive symptoms entered in Block 1 and each predictor variable in Block 2. As can be seen in Table 2, each predictor was significantly associated with ED pathology, controlling for age, BMI, and depressive symptoms. Hierarchical multiple regression analysis was similarly employed to test the hypotheses that the variance in ED pathology explained by internalised shame and

Table 1

Study 1: Descriptive Statistics and Correlations Among Study Variables

|   |               |        |       |            |        |        |       |       |      |     |       |       |      |      | Skewness | ess  |
|---|---------------|--------|-------|------------|--------|--------|-------|-------|------|-----|-------|-------|------|------|----------|------|
|   |               | 1      | 2     | 3          | 4      | 5      | 9     | 7     | 8    | u   | M     | SD    | Min  | Max  | Stat     | SE   |
| _ | Age           | 1      |       |            |        |        |       |       |      | 403 | 23.90 | 88.9  | 18   | 62   | 2.197    | .122 |
| 7 | BMI           | .262** | I     |            |        |        |       |       |      | 400 | 22.28 | 4.99  | 13.3 | 52.7 | 2.191    | .122 |
| 3 | ED pathology  | .040   | 157*  | .96ª       |        |        |       |       |      | 373 | 22.91 | 19.76 | 0    | 75   | 969.     | .126 |
|   | (EAT-26)      |        |       |            |        |        |       |       |      |     |       |       |      |      |          |      |
| 4 | ED pathology  | .053   | 104   | .972**     | I      |        |       |       |      |     |       |       |      |      |          |      |
|   | (EAT-26-Srqt) |        |       |            |        |        |       |       |      |     |       |       |      |      |          |      |
| 2 | Depressive    | 028    | 960'- | 096 .520** | .504** | .94 a  |       |       |      | 378 | 16.44 | 12.94 | 0    | 42   | .456     | .125 |
|   | symptoms      |        |       |            |        |        |       |       |      |     |       |       |      |      |          |      |
| 9 | Internalised  | 880.   | 084   | .564**     | .560** | .731** | .93 a |       |      | 378 | 98.69 | 22.37 | 24   | 118  | 272      | .126 |
|   | shame         |        |       |            |        |        |       |       |      |     |       |       |      |      |          |      |
| 7 | Self-esteem   | .054   | .103  |            | 560**  | 723**  |       | .93 a |      | 379 | 15.62 | 6.99  | 0    | 30   | 374      | .125 |
| 8 | Body Shame    | .039   | 990.  | .717**     | .730** | .546** | .693  | 705** | .87ª | 379 | 3.72  | 1.19  | -    | 9    | 086      | .126 |

\*. Correlation significant at p < .01 (2-tailed). \*\* Correlation significant at p < .001 (2-tailed). \*. Cronbach's  $\alpha$  in the present study. BMI = Body Mass Index. EAT-26 =

Eating Attitudes Test (ED pathology). EAT-26-Sqrt = Square root transformation of EAT-26 scores.

Table 2

Study 1: Comparison of Standardised Regression Coefficients and Unique Variance in ED Concerns Explained by Each Variable when Fitted Individually and Fitted Individually Controlling for Depressive Symptoms

|                     | I<br>and b | nternalised<br>ody shame<br>ontrolling fa | Internalised shame, self-esteem<br>and body shame fitted individually,<br>controlling for depression <sup>a</sup> | esteem,<br>dually,<br>n | Internalis | Internalised shame & self-esteem fitted simultaneously, controlling for depression | z self-estee<br>lling for de | m fitted<br>pression <sup>a</sup> | Interr<br>body<br>con | Internalised shame, self-esteem and body shame fit simultaneously, controlling for depression <sup>a</sup> | ne, self-esi<br>imultaneou<br>depressior | teem and isly, |
|---------------------|------------|---|---|-------------------------|------------|--|------------------------------|-----------------------------------|-----------------------|--|--|----------------|
| DV: EAT-26-Sqrt     | β          | 1   | d   | Unique $R^2$            | β          | t  | р                            | Unique $R^2$                      | β                     | t  | р  | Unique $R^2$   |
| Depressive symptoms | .496°      | $10.935^{b}$                              | < .001 <sup>b</sup>   | .244 <sup>b</sup>       | .144       | 2.181  | .030                         | 800.                              | .163                  | 3.036  | .003                                     | .011           |
| Internalised shame  | .401       | 6.239                                     | < .001  | .072                    | .222       | 2.626  | 600                          | .017                              | 033                   | 461  | .645                                     | < .001         |
| Self-Esteem         | 407        | -6.536                                    | < .001  | 620.                    | 265        | -3.228   | .001                         | .018                              | .036                  | .502   | .616                                     | < .001         |
| Body shame          | 999.       | 16.045                                    | < .001  | 308                     | I          | I  | I                            | I                                 | 695                   | 13.353   | < .001                                   | .217           |

 $<sup>^{</sup>a}$  = Controlling for age and Body Mass Index (BMI).  $^{b}$  = Depressive symptoms fitted alone controlling for age and BMI.  $\beta$  = Standardised Regression Coefficients. EAT-26-

Sqrt = Square root transformation of Eating Disorder Test (EAT-26) scores (ED pathology).

Table 3

Study 1: Summary of Mediation Analyses Testing the Indirect Effect of Internalised Shame and Self-esteem on ED Pathology via Body Shame, Controlling for Depressive Symptoms

|                  | sct                   | $p^{b}$    | <.001                                |                       | <.001   |                       |
|------------------|-----------------------|------------|--------------------------------------|-----------------------|---|-----------------------|
|                  | Total indirect effect | B (99% CI) | .025 (.012, .041)                    |                       | .095 (146,050)                                      |                       |
| cts              |                       | d          | <.001                                |                       | <.001   |                       |
| Indirect effects | Path b                | B (99% CI) | 1.311 (1.057,                        | 1.566)                | 1.311 (1.057,                                       | 1.566)                |
|                  |                       | d          | <.001                                |                       | <.001   |                       |
|                  | Path a                | B (99% CI) | .019 (.010, .029)                    |                       | .073 (103,043)                                      |                       |
|                  | '                     | d          | .645                                 |                       | . 919.  |                       |
| Direct effect    |                       | B (99% CI) | 022 (.000, .044) .009003 (022, .041) |                       | .011 (047, .070)                                    |                       |
|                  |                       | d          | 600.                                 |                       | .001  |                       |
| Total effect     |                       | B (99% CI) | .022 (.000, .044)                    |                       | Self-esteem→Body084 (152,017) .001 .011 (047, .070) |                       |
|                  |                       | Hypothesis | Shame→Body                           | shame→ED <sup>a</sup> | Self-esteem > Body                                  | shame→ED <sup>b</sup> |

Bootstrapped 99% Confidence Intervals around Unstandardised Effect. p = Two-tailed significance. Shame = Internalised shame. ED = Eating disorder pathology (EAT-26-Sqrt: Square root transformation of Eating Attitude Test [EAT-26] scores).

<sup>a</sup>. Controlling for depressive symptoms, age, Body Mass Index (BMI), and self-esteem. <sup>b</sup>. Controlling for depressive symptoms, age, BMI, and internalised shame. <sup>c</sup>. Normal Theory Test

p-value. Path a = Path from predictor variable to the mediator variable. Path from mediator variable to the dependent variable. B = Unstandardised Effect Coefficient. 99% CI =

self-esteem, when fitted together, would be overlapping. Results revealed that both internalised shame and self-esteem were independently associated with ED pathology, over and above one another, age, BMI, and depressive symptoms (Table 2).

# **Mediation Analysis**

Results of the PROCESS (mediation) analyses, testing the hypothesis that the effects of internalised shame and/or self-esteem on ED pathology would be mediated by body shame are summarised Table 3. As can be seen, evidence of mediation was observed in analysis of both the mediating role of body shame on the association between internalised shame and ED pathology (controlling for age, BMI, depression, and self-esteem) and the mediating role of body shame on the association between self-esteem and ED pathology (controlling for age, BMI, depression, and internalised shame).

#### Discussion

In a diverse sample of young adult women, Study 1 aimed to investigate the individual and independent associations between ED pathology and low self-esteem, internalised shame, and body shame, controlling for depressive symptoms and one another; and to determine whether the association between shame and/or low self-esteem and ED pathology is mediated by body shame, controlling for depressive symptoms. It was hypothesised, first, that low self-esteem, internalised shame, and body shame would each be individually associated with ED pathology over and above the contribution of depressive symptoms. This hypothesis was supported, confirming previous findings concerning the associations between ED pathology and internalised shame (e.g., Troop et al., 2008), low self-esteem (e.g., Jacobi, Paul, et al., 2004), and body shame (e.g., Tiggemann & Kuring, 2004), over and above depressive symptoms.

It was further hypothesised that the variance in ED pathology explained by low selfesteem and internalised shame, when fitted together, would be reduced but that internalised shame would remain independently associated with ED pathology, controlling for depressive symptoms. This hypothesis was also supported. While the individual variance explained in the ED pathology was reduced when fitting internalised shame and self-esteem together, both variables remained independently associated with ED pathology, also controlling for depressive symptoms. This provides important new evidence that while internalised shame and low self-esteem may be highly correlated; they each contribute uniquely to variance in ED pathology even after controlling for depressive symptoms.

Finally, it was hypothesised that the effects of low self-esteem and/or internalised shame on ED pathology would be mediated by body shame, over and above the contribution of depressive symptoms. The results supported this hypothesis in that body shame fully accounted for the associations between both internalised shame and low self-esteem and ED pathology, controlling for depressive symptoms and one another. These findings converge with those of a recent study suggesting that body shame mediates the association between self-esteem and ED pathology (Iannaccone et al., 2016), while extending this evidence to the association between ED pathology and internalised shame. These findings provide important evidence concerning one of the processes by which negative feelings about the self (in the forms of both internalised shame and low self-esteem) may influence ED pathology, namely, via increases in body shame, and provide preliminary support for the view that negative feelings about the self that are displaced onto the body contribute to ED pathology.

## STUDY 2

Building on the results of study 1, study 2 aims to extend objectification theory by considering externalised self-perceptions (self-objectification and body surveillance) within a displacement theory of ED pathology and to test a path model exploring the direct and indirect effects of internalised shame, self-objectification, body surveillance, body shame, and depressive symptoms on ED pathology. It is hypothesised, first, that the association between

internalised shame and ED pathology will be mediated by self-objectification, body surveillance, body shame, and depressive symptoms, such that a path model including these variables will demonstrate good fit for the data; second, that body shame will mediate the association between internalised shame and both depressive symptoms and ED pathology; third, that internalised shame will have a direct effect on both self-objectification and body surveillance; fourth, that, in accordance with objectification theory, the association between each of self-objectification and body surveillance and ED pathology will be fully mediated by body shame; and fifth, that depressive symptoms will partially mediate the association between body shame and ED pathology, in addition to a direct effect of body shame on ED pathology.

#### Method

## **Participants**

Participants were 548 women aged between 18 to 62 years ( $M_{age} = 22.94$ , SD = 6.85; the "full sample"). Of these, 230 were first year psychology students ("student sample"), 161 were recruited via email invitation to participate forwarded by family and friends of the researchers to prospective participants and via flyers displayed on Macquarie University campus ("community sample"), and 157 were recruited via advertisements displayed on eating disorder support websites ("web sample"). As in Study 1, and for the same reasons, inclusion criteria were being female and aged 18 years or older.

## Measures

Study 2 used several measures described in Study 1, namely, EAT-26, FASS, OBCS body shame subscale, and the DASS-21 depression subscale (see above for descriptions and see Table 4 for Cronbach's  $\alpha$  for each in Study 2). Additional measures used in Study 2 were:

**Eating Disorder Diagnostic Scale (EDDS).** In the current study ED pathology was assessed using two measures, namely the EAT-26 and the EDDS (Stice et al., 2000). The

EDDS is a 22-item self-report measure that can be used to generate probable DSM-IV (American Psychiatric Association, 1994) ED diagnoses. It contains items assessing the attitudinal symptoms of AN and BN (answered on a 7-point Likert scale), frequency of key eating disorder behaviours, namely, binging behaviour, experiential elements of binge episodes, frequency of purging behaviours (vomiting, use of diuretics or laxatives, fasting, or excessive exercise), height, weight, number of missed menstrual periods and contraceptive pill use. Items can be standardised and summed to obtain a composite score (EDDS-SC) that can be used as a continuous measure of global ED pathology. The EDDS (and EDDS-SC, when used as a global measure of ED pathology) has been found to have good psychometric properties in non-clinical and clinical samples of adolescent girls and women, including good criterion validity with interview-based diagnoses, convergent validity with other measures of ED pathology, test-retest reliability and internal consistency (Krabbenborg et al., 2012; Stice et al., 2004; Stice et al., 2000). In the current study EAT-26 total score and EDDES-SC score were each used as indicators of an ED pathology latent variable in the SEM models, in order to reduce measurement error.

Self-Objectification Questionnaire (SOQ). The SOQ (Noll & Fredrickson, 1998) asks participants to rank order (from *greatest* to *least*) 10 body attributes in terms of their impact on physical self-concept. Five of these attributes are appearance-based (*weight*, *sex appeal*, *physical attractiveness*, *firm/sculpted muscles*, and *measurements*), the remaining five competence-based (*physical coordination*, *health*, *strength*, *energy level*, and *physical fitness level*). The SOQ does not assess participants' level of satisfaction with their body attributes, but rather measures concern with appearance independent of an evaluative component. Scores are calculated by summing the ranking for the appearance and competence attributes separately and then computing a difference score (ranging from -36 to 36). The SOQ has been shown to have adequate construct validity, having been found to be positively correlated with

measures of body shame and ED pathology (Noll & Fredrickson, 1998).

Body Surveillance subscale of the Objectified Body Consciousness Scale (OBCS). The Body Surveillance subscale of the OBCS (McKinley & Hyde, 1996; see description above) contains 8-items (e.g., "*I rarely think about how I look*" [R]) designed to measure the extent to which the body is viewed by oneself as an outside observer, rated on a 6-point scale (from I = strongly disagree to 6 = strongly agree). Negatively worded items are reverse scored and items averaged to provide a mean score ranging from 1 to 6, with higher scores indicating greater body surveillance. It has been shown to have good test-retest reliability, acceptable internal consistency (surveillance scale,  $\alpha = .79$  & .76 for undergraduate and middle-aged women, respectively), and construct validity, having been found to be negatively correlated with body esteem and positively related to ED pathology (McKinley & Hyde, 1996).

#### **Procedure**

Data were obtained via an online survey using Qualtrics software. A brief description of the research was presented to participants prior to commencement and informed consent to participate was obtained. The survey first asked participants demographic questions (age, height, and weight) before presenting the study measures (and items within measures) in randomised order (so as to minimise order effects). Participants in the student sample participated in return for course credit, while those in the web and community samples could enter a prize draw for one of ten AUD\$100 ColesMyer Gift Cards and one of five AUD\$30 iTunes gift vouchers. The Macquarie University Human Research Ethics Committee approved all aspects of the study design and methods.

## **Statistical Analysis**

Samples obtained across the different recruitment methods were first compared using GLM ANOVA (with a Bonferroni adjusted p-value of < .05/7 = .007 for significance testing)

and follow up simple contrast tests (using a Bonferroni adjusted p-value of < .05/3 = .017 for significance). Pearson bivariate correlations were used to evaluate the strength and direction of associations among study variables. Analyses were undertaken using IBM Statistical Package for Social Sciences (SPSS) version 22.

Mediation effects and path model fit were tested via SEM using the maximum likelihood (ML) method in IBM SPSS AMOS (version 22) and using 10,000 bootstrap samples to estimate the size of the total, direct, and indirect effects and to yield 95% confidence intervals (CIs) around each effect. Use of this method pre-empts the requirement of multivariate normality required by SEM and has the additional advantages of greater power and more accurate Type I error rates (Byrne, 2010); hence the use of an alpha level of .05 for significance testing. Given that inclusion of BMI and age as covariates in the analyses performed in Study 3A did not result in any variation in the statistical significance of any results, and given the number of variables included and complexity of the models tested in the structural models, BMI and age were not included as covariates in SEM analyses in AMOS.

Overall goodness-of-fit of structural models was assessed using the Tucker-Lewis Index (TLI), Comparative Fit Index (CFI), root mean square error of approximation (RMSEA) and RMSEA closeness of fit (PCLOSE) fit indices. According to commonly accepted recommendations (Byrne, 2010; Schreiber et al., 2006), a priori standards of acceptable goodness-of-fit chosen were TLI greater than .95, CFI greater than .95, RMSEA less than .08, and PCLOSE greater than .05. Chi-square difference ( $\Delta \chi^2$ ) tests were used to compare nested models.

### **Results**

# **Preliminary Analysis**

GLM ANOVA indicated that the student and community samples did not differ significantly on any study variables, but that the web sample scored significantly higher than

both of these with respect to age, ED pathology, internalised shame, body shame and depressive symptoms, and higher than the student sample on body surveillance. There were no significant interactions between recruitment method and any IVs in predicting ED pathology, hence pooling of data across recruitment methods was considered appropriate.

Descriptive statistics for and intercorrelations among study variables are given in Table 4. Of the total sample, 38.5% were classified as being "at risk" of an ED (EAT- $26 \ge 20$ ). SEM Analysis of Mediation Effects

**Model fit.** To test the hypothesis that the association between internalised shame and ED pathology would be mediated by self-objectification, body surveillance, body shame, and depressive symptoms, the hypothesised model with all possible direct and indirect effects included (Model 1) was fitted first (see Figure 1). While this model (Model 1) demonstrated good fit for the data (see Table 5), a number of non-significant paths were revealed. As such, a second model (Model 2) (Figure 2), which constrained to zero all non-significant paths from Model 1, was fitted. This model had equivalent overall goodness of fit as Model 1 ( $\Delta \chi^2_{(df)} = 6.34_{(5)}$ , p > .05), demonstrated very good fit for the data (see Table 5) and explained 69% of the variance in ED pathology.

**Mediation.** Testing of the indirect effects of internalised shame on ED pathology via self-objectification, body surveillance, body shame, and depressive symptoms in the final model (Model 2) indicated that internalised shame had a significant direct effect on self-objectification, body surveillance, body shame, depressive symptoms and ED pathology, and significant indirect effects on ED pathology via self-objectification, body surveillance, body shame, and depressive symptoms (Table 6). Further, the association between each of self-objectification and body surveillance and ED pathology was fully mediated by body shame and depressive symptoms. Finally, body shame had a significant direct effect on ED pathology and a significant indirect effect via depressive symptoms (partial mediation).

Table 4

Study 2: Descriptive Statistics and Correlations Among Study Variables

|   |                          |        |        |        |                    |        |        |        |        |       |     |       |       |      |     | Skewness | ssəi |
|---|--------------------------|--------|--------|--------|--------------------|--------|--------|--------|--------|-------|-----|-------|-------|------|-----|----------|------|
|   |                          | 1      | 2      | 3      | 4                  | 5      | 9      | 7      | 8      | 6     | и   | M     | QS    | Min  | Max | Stat     | SE   |
| 1 | Age                      | 1:     |        |        |                    |        |        |        |        |       | 548 | 22.94 | 6.85  | 18   | 62  | 2.31     | .104 |
| 7 | BMI                      | .199** | ı      |        |                    |        |        |        |        |       | 548 | 22.43 | 4.75  | 14.6 | 51  | 1.79     | .104 |
| 3 | EAT-26                   | .042   | 101*   | .95ª   |                    |        |        |        |        |       | 548 | 19.55 | 17.82 | 0    | 72  | .93      | .104 |
| 4 | EDDS-SC                  | 890.   | .108   | .732** | I                  |        |        |        |        |       | 548 | 29.81 | 19.14 | 0    | 111 | 66:      | .104 |
| 2 | Depressive               | .037   | 002    | .562** | .523**             | .93 a  |        |        |        |       | 548 | 15.27 | 12.49 | 0    | 42  | .590     | .104 |
| 9 | symptoms<br>Internalised | .054   | 015    | .579   | .522**             | .738** | .94ª   |        |        |       | 548 | 68.18 | 22.34 | 22   | 116 | 037      | .104 |
|   | shame                    | *      |        | **     | **                 | *      | *      |        |        |       |     |       | ,     |      |     |          | ,    |
| 7 | SO                       | 089    | .053   | .308   | .267               | .255   | .293   | L      |        |       | 548 | 66.   | 13.39 | -26  | 31  | 073      | .104 |
| 8 | Body                     | 059    | .053   | .429** | .368**             | .314** | .343** | .404   | .82 a  |       | 548 | 4.36  | .95   | -    | 9   | 545      | .104 |
|   | Surveill                 |        |        |        |                    |        |        |        |        |       |     |       |       |      |     |          |      |
| 6 | Body                     | .115** | .157** | .682** | <sub>**</sub> 699. | .554** | .627** | .337** | .548** | .88 a | 548 | 3.56  | 1.26  | 1    | 9   | .029     | .104 |
|   | Shame                    |        |        |        |                    |        |        |        |        |       |     |       |       |      |     |          |      |

\*. Correlation significant at p < .05 (2-tailed). \*\*. Correlation significant at p < .01 (2-tailed). \*. Cronbach's  $\alpha$  in the present study. BMI = Body Mass Index. EAT-26 = Eating Attitudes Test (ED concerns). EDDS-SC = Eating Disorder Diagnostic Scale symptom composite score (ED symptoms). SO = Self-objectification. Body Surveill = Body

surveillance.

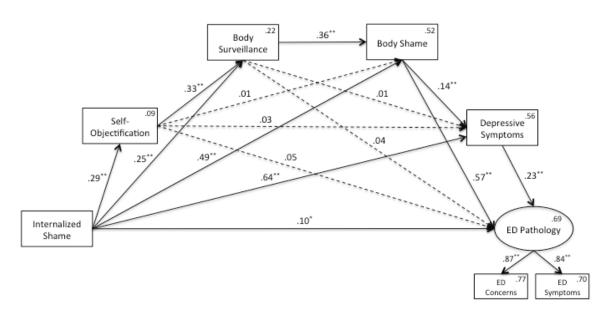


Figure 1. The Hypothesised Mediation Model (Model 1). Model 1 explains 69% of the variance in ED Pathology. Model fit:  $\chi^2 = 7.671$ , df = 4, p = .104, CMIN/df = 1.918, CFI = .998, TLI = .990, RMSEA = .041 (90% CI: <.001, .072, PCLOSE = .568). Dashed line = path not significant (p > .05). \* = p < .05. \*\* = p < .001.

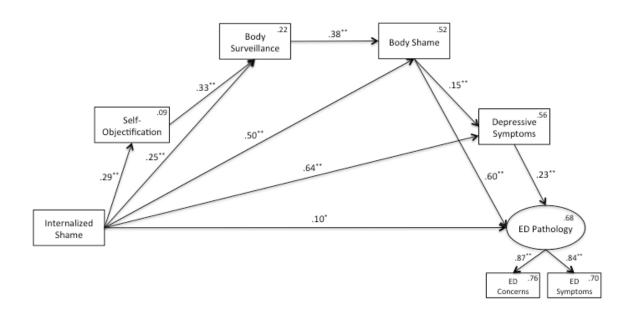


Figure 2. The Final Model (Model 2). All possible indirect effects statistically significant (p <.001). The Final Model explains 68% of the variance in ED Pathology. Model fit:  $\chi^2$  = 14.011, df = 9, p = .122, CMIN/df = 1.557, CFI = .997, TLI = .994, RMSEA = .032 (90% CI: <.001, .063, PCLOSE = .810). Model 1 v 2:  $\Delta \chi^2$ (df) = 6.34(5); p > .05 (ns). \* = p <.05. \*\* = p <.001.

Table 5

Summary of Results of Path Analyses Comparing Model 1 (Hypothesised Model) and Model 2 (Removing all Non-Significant Paths)

| $\chi^{5}$ | df | D    | CMIN/df | CFI  | TLI  | RMSEA | (90% CI)      | PCLOSE | $\Delta\chi^2(df)$ | D    |
|------------|----|------|---------|------|------|-------|---------------|--------|--------------------|------|
| 7.671      | 4  | .104 | 1.918   | 866. | 066. | .041  | (<.001, .072) | .568   |                    |      |
| 14.011     | 6  | .122 | 1.557   | 266. | .994 | .032  | (<.001, .063) | .810   | 6.34(5)            | >.05 |

 $\chi^2$  = Chi-squared. df = Degrees of Freedom. p = Chi-squared significance test. CMIN/df = Chi-squared/degrees of freedom ratio. CFI = Comparative Fit Index. TFI = Tucker-

Lewis Index. RMSEA = Root Mean Square Error of Approximation. 90% CI = 90% Confident Interval around RMSEA. PCLOSE = RMSEA closeness of fit.  $\Delta \chi^2$  = Chi-

square difference.

Table 6

Study 2: Summary of Results of Mediation Analyses

|  | Total effect      | ıt.   | Direct effect     | x     | Indirect          |       |
|--|-------------------|-------|-------------------|-------|-------------------|-------|
|  | $\beta$ (95% CI)  | D     | $\beta$ (95% CI)  | b     | $\beta$ (95% CI)  | d     |
| Model 2 (Final Model)                      |                   |       |                   |       |                   |       |
| Shame→ SO→ Surveill→ Body Shame→Depress→ED | .646 (.587, .699) | <.001 | .103 (.004, .201) | .043  | .544 (.466, .628) | <.001 |
| SO→ Surveill→ Body Shame→ Depress→ ED      | .079 (.054, .112) | <.001 |                   |       | .079 (.054, .112) | <.001 |
| Surveill→ Body Shame→ Depress→ ED          | .239 (.189, .296) | <.001 | I                 |       | .239 (.189, .269) | <.001 |
| Body Shame→ Depress→ ED                    | .631 (.547, .702) | <.001 | .597 (.514, .667) | <.001 | .034 (.013, .066) | <.001 |

 $\beta$  = Standardised Effect Coefficient. 95% CI = Bootstrapped 95% Confidence Intervals around Standardised Effect. p = Two-tailed significance. Shame = Internalised shame.

SO = Self-Objectification. Surveill = Body Surveillance. Depress = Depression. ED = Eating disorder psychopathology latent variable (ED concerns & ED symptoms).

#### Discussion

Study 2 sought to combine elements of objectification theory and displacement theory and test a path model exploring the direct and indirect effects of internalised shame, externalised self-perceptions (self-objectification and body surveillance), body shame, and depressive symptoms on ED pathology. It was hypothesised, first, that the association between internalised shame and ED pathology would be mediated by self-objectification, body surveillance, body shame, and depressive symptoms. Results supported this hypothesis providing evidence of partial mediation and good model fit, with the removal of non-significant paths to create a more parsimonious model not significantly altering the fit of the model, other than resulting in improvement in several fit indices. These results extend the findings of Tylka and Sabik (2010), whose path model included self-esteem, body surveillance, body comparison, body shame and ED pathology, to include internalised shame and depressive symptoms.

It was further hypothesised that body shame would mediate the relationship between internalised shame and depressive symptoms and ED pathology. Results supported this hypothesis, as internalised shame had both a significant indirect effect on ED pathology via body shame (and depressive symptoms) and a small but significant direct effect on ED pathology. These findings support previous evidence in a non-clinical sample that internalised shame was independently associated with ED pathology, over and above body shame (Doran & Lewis, 2012), and that body shame mediates the association between self-esteem and ED pathology (Iannaccone et al., 2016); and are consistent with, and provide preliminary support for a displacement theory-based self/emotion-regulation model of ED pathology. However, these findings diverge somewhat from those of study 1, in which body shame fully mediated the association between internalised shame and ED pathology, and from those of previous research in which internalised shame was not independently associated with ED pathology,

over and above body shame (Burney & Irwin, 2000; Troop & Redshaw, 2012). Further, the strong independent effect of internalised shame on depressive symptoms, over and above body shame and the other predictors in the model, is somewhat surprising. While there was a significant indirect effect of internalised shame on depressive symptoms via body shame (providing evidence of mediation), there remained a strong direct effect of internalised shame on depressive symptoms and depressive symptoms significantly mediated the association between internalised shame and ED pathology. These findings extend those of previous research demonstrating an association between internalised shame and depressive symptoms (Andrews et al., 2002).

Third, it was hypothesised that internalised shame would have a direct effect on both self-objectification and body surveillance. Results supported this hypothesis and extended evidence from previous literature suggesting a possible association between internalised shame and both self-objectification and body surveillance (Goss & Gilbert, 2002; Mercurio & Landry, 2008). Of note, however, is that while internalised shame had a moderate effect on each of self-objectification and body surveillance, internalised shame explained only 9% of the variance in self-objectification and internalised shame and self-objectification together accounted for 22% of the variance in body surveillance. Thus, whilst internalised shame may contribute to both forms of externalised self-perceptions, it seems clear that additional factors not accounted for in the current model likely also contribute to self-objectification (e.g., sexual objectification or internalisation of the thin ideal). Nevertheless, taken together, these findings provide preliminary support for a modified displacement theory of ED pathology whereby negative feelings about the self are externalised (via increased self-objectification and body surveillance) and displaced onto the body, potentially giving rise to ED pathology both directly and indirectly via an increase in depressive symptoms.

Fourth, it was hypothesised that within the model the associations between both selfobjectification and body surveillance and ED pathology would each be mediated by body
shame. This hypothesis was also supported, extending evidence from previous research of the
mediating role of body shame in the relationship between self-objectification and body
surveillance and both depressive symptoms and ED pathology (Tiggemann & Kuring, 2004;
Tiggemann & Williams, 2012), as occurring over and above the direct impact of internalised
shame. Finally, it was hypothesised that depressive symptoms would partially mediate the
association between body shame and ED pathology, but that body shame would have a
significant direct effect on ED pathology. Results again supported this hypothesis and are
consistent with previous evidence of the association between body shame and depressive
symptoms, over and above self-objectification and body surveillance (Tiggemann & Kuring,
2004) and with the results of study 1, in which depressive symptoms were independently
associated with ED pathology, over and above internalised shame and body shame.

### **GENERAL DISCUSSION**

The current research aimed to test a displacement theory-based self/emotionregulation model of ED pathology, whereby negative feelings about the self are externalised
(via increased self-objectification and body surveillance) and displaced onto the body (in the
form of increased body shame), thereby promoting ED pathology. Results of study 1 provided
evidence of the independent roles of internalised shame and low self-esteem in accounting for
variance in ED pathology, over and above the effects of depressive symptoms and one
another, and for full mediation of these associations by body shame. Results of study 2
confirmed the role of internalised shame in independently directly predicting ED pathology,
while also providing evidence of indirect effects on ED pathology via self-objectification,
body surveillance, body shame, and depressive symptoms. Taken together, these results
provide preliminary support for a displacement theory-based self/emotion-regulation model of

ED pathology in which negative feelings about the self are externalised and displaced onto the body in an effort to escape self-awareness (Heatherton & Baumeister, 1991) and also attempt to promote feeling of self-worth through the control of body weight or shape (Goss & Gilbert, 2002).

# **Implications**

The results of the current research contribute to theoretical accounts of ED pathology aligning with the view that ED pathology emanates from a disturbance in the sense of self, consistent with both displacement and escape theory models of ED pathology. According to these theories, negative feelings about the self are externalised onto the body and body weight/shape become the focus of self-evaluation and feelings of self-worth. Control of body weight or shape and relentless pursuit of a thin-ideal via control of eating and weight-control behaviours then become a means by which to enhance feelings of self-worth and pride (Goss & Gilbert, 2002), whilst ED behaviour also serving as a means to escape from aversive feelings of self-awareness (Heatherton & Baumeister, 1991). Further, the current findings support an expansion of these theories to include elements of objectification theory, and identify the potential role of deficits in self-development and negative feelings about the self in accounting for individual differences in levels of externalised self-perceptions (both self-objectification and body surveillance).

The findings of the current research also have implications for treatment of EDs and suggest that when negative self evaluations and shame are present in individuals with EDs it may be important to specifically explore the origins and function of these in order to enhance the effectiveness of treatments such as CBT (Goss & Gilbert, 2002). To this end, some promising interventions specifically aimed at reducing shame and self-criticism in EDs have emerged, including compassionate-mind training (Gilbert & Procter, 2006), compassion-focused therapy (Gilbert, 2010), and compassion-focused therapy for EDs (Goss & Allan,

2012). Although these interventions are relatively new, evidence of the importance of self-compassion in EDs is beginning to mount. For instance, in college women self-compassion has been found to be negatively associated with body shame and disordered eating (Breines, Toole, Tu, & Chen, 2013) and self-kindness has been positively associated with body-esteem and negatively associated with ED pathology (Geller, Srikameswaran, & Zelichowska, 2015). Self-compassion has also been negatively associated with shame memories and has been found to moderate the impact of shame memories on ED severity in inpatients with EDs (Ferreira, Matos, Duarte, & Pinto-Gouveia, 2014). In a recent, longitudinal study of intraindividual predictors of change among ED inpatients, periods of increased shame were found to be followed by increased severity of ED pathology while periods of increased self-compassion or decreased ED pathology were followed by decreased levels of shame (Kelly & Tasca, 2016).

Evidence that body acceptance mediates the association between self-compassion and intuitive eating in college women (Schoenefeld & Webb, 2013) further suggests that one of the processes by which self-compassion may alleviate ED pathology is via a reduction in body shame. Furthermore, interventions that focus on reducing avoidance or escape from painful emotions and internal experiences, such as mindfulness training, may be helpful in reducing ED pathology. For instance, in one recent study, also in college women, mindfulness was found to moderate the association between distress levels and ED pathology, such that distress severity predicted ED pathology among participants low in mindfulness but not those high in mindfulness (Geller et al., 2015). Finally, findings from the current research highlight the importance of ED prevention programs targeting negative body image in children and adolescents (for a review see Levine & Smolak, 2009) and the need for interventions aimed at reducing self-objectification in particular, for instance, by enhancing experiences of embodiment and positive body image (Menzel & Levine, 2011).

### Strength, Limitations and Directions for Future Research

At least three limitations of the current research should be noted. First, the research employed a cross-sectional design in each study, which, strictly speaking, cannot be used to establish mediation effects. Nevertheless, A. F. Hayes (2013) argues that where a theoretical foundation exists to support the directionality of effects it is appropriate to use PROCESS analyses and path models to test for mediation effects using cross-sectional data. In the current research strong theoretical support for the direction of effects tested in the models is provided by both the displacement theory of EDs and objectification theory. However, in practice there are likely to be complex, bidirectional associations between the variables examined and clearly longitudinal research would be helpful in elucidating these pathways across time.

Second, the current research relied on self-report measures of all study variables. While online surveys are an efficient means of data collection, potential problems associated with the use of self-report measures of ED pathology in particular are well-known (House et al., 2008). The inclusion of interview data and data obtained from multiple sources would clearly be of interest in future research. Finally, the current research was confined to adult women, thus limiting the generalisability of the findings beyond this population. Although this was reasonable for a study designed to extend previous research conducted in this demographic, the inclusion of adolescent females and of males in future research would be of interest, particularly given increasing attention to ED pathology in males in recent years (e.g., Strother et al., 2012).

There were also important strengths of the current research. Firstly, consideration in unison of the related, but distinct, constructs of internalised shame and low self-esteem and their relative importance in accounting for variance in ED pathology is novel and addressed a gap in the literature; as did our efforts to elucidate the processes by which negative feelings

about the self may contribute to ED pathology, namely, via increases in externalised selfperceptions (self-objectification and body surveillance), body shame, and depressive symptoms. Thereby, moving beyond exploration of direct relationships and contributing new evidence in support of a displacement theory-based self/emotion-regulation model of EDs.

A second notable strength of the current research was the recruitment of participants in each study from several different sources (including ED information websites, general community, and undergraduate students). This method ensured adequate variability on key study measures and allowed sufficient numbers for the use of more sophisticated analytic methods (SEM) to be employed. Finally, whilst the study findings suggest important implications for treatment and prevention of ED pathology, replication of the current findings in a clinical sample would nevertheless be of interest in terms of providing clearer implications for clinical practice.

### Conclusion

Within the limits of a cross-sectional study design, the current findings support the view that negative feelings about the self (low self worth or internalised shame) are externalised (via increased self-objectification and body surveillance) and displaced onto the body (increasing body shame) and in this way may give rise to ED pathology. The findings are consistent with a self/emotion-regulation conceptualisation of EDs and support the role of therapeutic interventions for ED pathology designed to enhance self-directed compassion and reduce self-objectification.

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# Chapter 8

The Role of Attachment Dynamics, Maladaptive Schemas, and Body Shame in Contributing to Eating Pathology in Women

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# **Author Contributions**

First author Kim Woodward was responsible for conceptualisation, design, and conduct of the current study, managed data collection, undertook all statistical analyses, conducted the literature searches, and wrote the first draft of all manuscript. Author Doris McIlwain contributed to the conceptualisation and design of the study at a supervisory level. Author Jonathan Mond contributed to revisions of the manuscript.

#### **Abstract**

**Objective:** To investigate the structural associations among attachment dynamics, core low self-worth (defectiveness/shame), emotional inhibition, unrelenting standards (perfectionism), body shame, depressive symptoms, and eating disorder (ED) pathology. **Methods:** Participants were 724 women (197 recruited via ED support websites, 208 from the community, and 319 undergraduate students; 385 of whom were classified as having no ED and 339 as having a probable DSM-5 ED) who completed questionnaire measures of attachment anxiety and avoidance, early maladaptive schemas of defectiveness/shame, emotional inhibition, and unrelenting standards, body shame, depressive symptoms, and ED pathology via an online survey. **Results:** Structural Equation Modelling (SEM) analysis revealed that a model including attachment-anxiety, attachment-avoidance, defectiveness/shame, emotional inhibition, unrelenting standards, body shame, depressive symptoms and ED pathology demonstrated good fit for the data and entailed significant indirect effects. This model also demonstrated good fit in subgroups of women with and without probable-EDs, albeit with some variations in path significance across subgroups. **Conclusions:** Results support a self/emotion-regulation model of ED pathology grounded in attachment theory, wherein insecure attachment styles underlie the development of negative feelings about the self (defectiveness/shame) and negative beliefs about emotion expression (emotional inhibition). Displacement of these feelings and beliefs onto the body gives rise to the secondary schema of unrelenting standards (perfectionism), body shame, depressive symptoms, and in turn ED pathology. Theoretical and clinical implications are explored.

#### Introduction

Eating disorders (EDs) are among the most disabling mental health problems experienced by women and are notoriously difficult to treat (Agras, 2001). The most frequently used, and recommended, treatment is cognitive behavioural therapy (CBT) for EDs, which focuses on changing body shape and weight concerns, as well as eating and starvation-related beliefs and behaviours (Fairburn et al., 2003). While addressing these issues is integral to recovery, as many as 50 to 75% of women fail to respond adequately to CBT for EDs (Waller et al., 2014; Zipfel et al., 2014). Thus, attempts to enhance the effectiveness of CBT by elucidating etiological and maintenance factors that may inform this goal are needed. One theoretical framework that may be beneficial in this regard, by virtue of its focus on feelings of self-worth and self-development, emotional regulation, interpersonal functioning and other factors believed to be implicated in the development of ED pathology (Tasca & Balfour, 2014b), is attachment theory (Bowlby, 1969, 1973, 1980).

Self-development deficits and difficulties with emotion have long been implicated in the aetiology and maintenance of EDs (Bruch, 1962, 1973). From an emotional-developmental perspective framed in attachment theory, EDs are conceptualised as disorders of self-regulation generally and of emotion-regulation specifically (Amianto et al., 2016; Goodsitt, 1983, 1997). The fundamental principle underlying this view is that ED symptoms serve the maladaptive function of attempting to manage the painful internal experiences emanating from deficits in the self (Amianto et al., 2016). This view is consistent with emotion-regulation models of EDs such as Heatherton and Baumeister (1991) escape theory, where binge eating is seen as serving the function of escaping painful self-awareness via the narrowing of attention to focus on immediate stimuli (namely, food) and the 'blocking' model, which proposes that ED symptoms (e.g., binge eating and extreme weight-control behaviours) are used as maladaptive emotion regulation strategies that provide short-term relief from

unacceptable emotion but which exacerbate distress in the long-term (McManus & Waller, 1995; Root & Fallon, 1989; Waller, Kennerley, et al., 2007).

Similarly, Bruch (1973, 1978) proposed a displacement theory of emotion in EDs suggesting that unexpressed or undifferentiated emotions and negative feelings about the self are displaced onto the body in the form of 'feeling fat'. Like emotion-regulation models of ED pathology, displacement theory views ED symptoms as function as maladaptive attempts to avoid or reduce feelings of shame and also to enhance feelings of pride or self-worth (Goss & Allan, 2009). That is, the displacement of an underdeveloped sense of self and core feelings of worthlessness or shame involves the redirection of ego threatening feelings to a less threatening and more concrete target – the body – and to body weight and shape, things that can be controlled in a more direct fashion via disturbed eating behaviours (Schupak-Neuberg & Nemeroff, 1993). For individuals with EDs, particularly anorexia nervosa (AN), schemas for self-evaluation become intricately connected to the achievement of unrealistically high standards. Striving to achieve these standards becomes a strategy to dispel feelings of defectiveness or emptiness and, to the extent that this aim is achieved, the consequent positive reinforcement serves to maintain the disorder and becomes part of the identity of the individual (Bruch, 1978).

We suggest that these complimentary conceptualisations can be taken together to comprise a self/emotion-regulation model of ED pathology grounded in attachment theory. Such a model implies that attachment dynamics may give rise to negative feelings about the self (feelings of low self-worth and shame) and difficulties expressing emotion (emotional inhibition), which may, in turn, be displaced onto the body, in the form of unrelenting high standards and body shame, and give rise to further low mood (depressive symptoms) and ED pathology. Evidence to support such a model can be found in research investigating the roles

of attachment dynamics, feelings of low self-worth/internalised shame, emotion inhibition, perfectionistic and self-critical beliefs, and body shame in relation to ED pathology.

#### **Individual Difference in Adult attachment**

Attachment theory, first proposed by Bowlby (1969, 1973, 1980), emphasises the importance of the early caregiving environment for later psychological functioning and describes personality development from an interpersonal perspective. According to attachment theory, the capacity to regulate emotion and development of sense of self occur as part of a normative emotional developmental process within the context of the primary caregiving relationship. Information based on the responsiveness of caregivers in close relationships is then organised in the form of internal working models (IWMs) of the self and others, which operate as relationship schemas and enable the individual to plan their own behaviour and to predict the behaviour of others in the context of close relationships (Baldwin. 1992; Baldwin et al., 1996; Kobak & Sceery, 1988). IWMs thus form the implicit memory of the self, self-states, and the self-in-relation to another and are considered to be central elements of personality that guide interactions with others throughout life (Bowlby, 1988; Zimmermann, 1999). In this way attachment processes are theorised to be important across the lifespan (Bowlby, 1988). Although amenable to change, IWMs tend to be stable and selfperpetuating, as they are unconsciously imposed on future relationships often leading to selffulfilling prophecies (Ainsworth, 1990; Collins & Read, 1990; Main et al., 1985).

Adult attachment can be measured along the two dimensions of attachment-avoidance (characterised by discomfort with intimacy and independence seeking) and attachment-anxiety (characterised by fears of rejection and abandonment) (Fraley et al., 2000). In terms of emotion regulation, high attachment-avoidance is associated with the defensive inhibition or suppression of threat-related emotional states (e.g., fear, anxiety, anger, shame and sadness) and incorporates strategies to deactivate the attachment system and minimise

interpersonal closeness and interdependence. Consequences include interference with reappraisal, problem-solving, and support seeking (Mikulincer & Shaver, 2007). Conversely, attachment-anxiety is associated with the intensification of distressing emotions, aimed at activating the attachment system and attracting the attention, care, and protection of attachment figures; a strategy that is often self-defeating and can lead to feelings of helplessness (Shaver & Mikulincer, 2007). Broadly speaking, high attachment-anxiety and/or high attachment-avoidance are seen to constitute insecure attachment styles (Bartholomew & Horowitz, 1991; Fraley et al., 2000).

There is considerable empirical support for an association between insecure attachment and ED pathology in clinical populations. Thus, higher rates of insecure adult attachment have been found in clinical samples of women with EDs compared with non-clinical controls (e.g., Latzer et al., 2002; Troisi et al., 2005), while in other studies of women with EDs between 70 to 100% of participants were classified as insecurely attached (e.g., Ramacciotti et al., 2001; Ringer & Crittenden, 2007). Attachment anxiety and avoidance have each been associated with increased ED pathology in both clinical ED (e.g., Tasca et al., 2013) and undergraduate student samples of women (e.g., Bamford & Halliwell, 2009). Further, evidence suggests that insecure attachment styles may moderate response, and adherence, to treatment among individuals with ED including binge eating disorder (BED) and AN (Tasca et al., 2006; Tasca et al., 2004).

Consistent with a self/emotion-regulation model of EDs grounded in attachment theory, emotion dysregulation has been found to mediate the associations between attachment style and ED pathology in a community sample of women (Ty & Francis, 2013). In a clinical sample of women with EDs, the association between attachment anxiety and ED pathology was fully mediated by emotional reactivity and depressive symptoms, whereas the association between attachment-avoidance and ED pathology was only partially mediated by emotional

deactivation and depressive symptoms and the direct effect of attachment-avoidance on ED pathology remained significant (Tasca et al., 2009a). Findings from these cross-sectional studies, when taken with those demonstrating an association between insecure attachment and ED pathology in adolescent girls (e.g., Sharpe et al., 1998) and those of longitudinal studies demonstrating effects of early attachment insecurity on subsequent ED pathology (Burge et al., 1997; Milan & Acker, 2014), suggest that insecure attachment may contribute to the development and/or maintenance of ED pathology.

# **Early Maladaptive Schemas**

Building upon attachment theory, schema-focused conceptualisations of EDs (Waller, Kennerley, et al., 2007) propose that early interpersonal experiences and environments that fail, for whatever reason, to meet the emotional needs of the child/adolescent (including attachment needs) influence the development of negative/maladaptive unconditional core beliefs about the self, others, and the world (schemas), which may contribute to the development of ED pathology. Schema therapy (Young, 1990), which is based in part of attachment theory, identifies various early maladaptive schemas (EMS) that may operate outside of one's awareness and act as templates for processing later thoughts, feeling, and relationships. It is premised on the need to address maladaptive core beliefs about the self, others, and the world in the treatment of individuals with personality disorders and other chronic forms of psychopathology including, potentially, individuals with EDs.

Evidence suggests that women with EDs possess significantly more negative EMS than women without EDs (Overton et al., 2005; Waller et al., 2000) and that this is associated with the perceived experience of poor parenting bonding (Leung, Thomas, & Waller, 2000) and 'chaotic' family environments during childhood (Ford et al., 2011). Further, schema therapy suggests that some early maladaptive schemas are unconditional (primary) core beliefs about the self and others that develop early on, such as 'defectiveness/shame'.

Whereas others are conditional (secondary) schemas, which develop in response to unconditional (primary) schemas in an attempt to seek some relief from primary schemas (Young et al., 2003); for example, emotional inhibition in response to defectiveness/shame ("If I inhibit expression, I can avoid rejection or feelings of shame") or unrelenting standards in response to defectiveness/shame ("If I am perfect, I will be lovable"). Of particular relevance to a self/emotion-regulation model of EDs grounded in attachment theory are 'defectiveness/shame', 'emotional inhibition' and 'unrelenting standards' schemas, as this model suggests that negative feelings about the self (defectiveness/shame), emotional inhibition, and maladaptive perfectionism may be possible mechanisms through which attachment insecurity may impact ED pathology (Tasca & Balfour, 2014a).

Defectiveness/shame. Defectiveness/shame schemas involve the underlying belief and feeling that one is damaged, bad, unwanted or inferior in important ways or that one would be seen as unlovable if exposed to significant others (Young, 1990). Within a self/emotion-regulation understanding of ED pathology, these feelings of low self-worth and internalised shame may be displaced onto the body. In support of such a theory, associations between internalised shame and ED pathology have been found in both college females (Sanftner et al., 1995) and in clinical ED samples (Troop et al., 2008). These associations have also remained after controlling for depressive symptoms (Gee & Troop, 2003; Hayaki et al., 2002b) and general negative affect (Gupta et al., 2008). Defectiveness/shame beliefs have been found to trigger and predict frequency of episodes of vomiting in women with bulimia nervosa (BN) (Hinrichsen et al., 2007; Waller et al., 2000) and internalised shame has been found to predict depressive symptoms in college students (Andrews et al., 2002).

Defectiveness/shame schemas have been found to be higher in women with AN compared to both psychiatric and healthy controls (Deas et al., 2010), and levels of internalised shame

have been found to be higher among inpatients with AN and BN than those with anxiety and mood disorders (Grabhorn et al., 2006).

Defectiveness/shame schemas are believed to derive in part from compromised early caregiving relationships with evidence that perceptions of low parental care were associated with internalised shame in a clinical sample of women with EDs (L. Keith et al., 2009) and internalised shame fully mediated the association between perceptions of parental overprotection and bulimic symptoms in nonclinical women (Murray et al., 2000). Further, attachment insecurity has been associated with increased experience of shame in children and adolescents (Muris et al., 2013) and young adults (Wei, Shaffer, Young, & Zakalik, 2005), and insecure attachment styles have been found to mediate the association between the experience of childhood trauma (e.g., neglect and sexual abuse) and ED pathology in adults with EDs (Tasca et al., 2013).

Emotional inhibition. Emotional inhibition schemas involve the suppression of spontaneous affect, behaviours, or communication of thoughts/feelings/ideas, in an attempt to avoid feelings of shame, loss of control, or judgment from others (Young, 1990). There is a growing body of evidence linking various forms of emotion inhibition with increased ED pathology in women, including self-silencing (Frank & Thomas, 2003), negative beliefs about emotion (Hambrook et al., 2011), ambivalence over emotional expressiveness (Krause et al., 2000), and expressive suppression (Davies et al., 2012). In clinical samples of women with EDs, emotional inhibition schemas have been found to predict the severity of binge eating (Waller et al., 2000) and levels of drive for thinness (Waller et al., 2002), and to be associated with the perceived experience of a chaotic family environment (Ford et al., 2011). Also in women with EDs, feelings of defectiveness/shame schemas have been associated with increased anger suppression (Waller et al., 2003). Further, within attachment theory, having expectations about the unavailability of significant others (associated with attachment-

avoidance), may increase emotion inhibition (Dozier & Kobak, 1992) and in women with EDs this down-regulation of emotion, together with depressive symptoms, has been found to mediate the effect of attachment-avoidance on ED pathology (Tasca et al., 2009a).

**Unrelenting standards.** Perfectionism involves the propensity to establish and pursue relentlessly high standards, despite negative consequences, and to make stringent self-critical evaluations (Cassin & von Ranson, 2005); and can be operationalised as the holding of unrelenting standards, namely, underlying beliefs that one must strive to meet very high internalised standards in order to avoid criticism (Young, 1990). Such unrelenting standards have been found to be highly correlated with other measures of perfectionism in women with clinical EDs (Overton et al., 2005; Waller et al., 2002). Empirically, perfectionism has consistently been implicated in the development and maintenance of ED pathology (Bardone-Cone et al., 2007; Cassin & von Ranson, 2005; Culbert et al., 2015). Perfectionism and unrelenting standards have been found to be higher in women with AN than in psychiatric control women with mood disorders and healthy control women (Bastiani et al., 1995; Deas et al., 2010) and also controlling for self-esteem, depressive symptoms and global psychiatric severity (Cockell et al., 2002). Unrelenting standards have been associated with increased ED pathology in women with AN (Deas et al., 2010); frequency of binge eating in women with binge eating disorder (BED; Waller, 2003); and bulimic symptoms (La Mela et al., 2015) and ED pathology, controlling for depressive symptoms, in women with BN (Bernert et al., 2013).

Further, perfectionism in young adolescence has been found to predict onset of AN (clinical and subclinical) in young adulthood, controlling for initial ED pathology (Tyrka et al., 2002). There is also evidence of associations with body dissatisfaction in a community sample of women (Wade & Tiggemann, 2013) and body image concerns in clinical-ED samples (Boone et al., 2013). Importantly, perfectionism has also been identified as a factor

that can interfere with response to enhanced CBT treatment for EDs (CBT-E; Fairburn et al., 2008).

In support of a self/emotion-regulation model of EDs grounded in attachment theory, one cross-sectional study using a large clinical sample of women with EDs found that perfectionism mediated the associations between ED pathology and both attachment-anxiety (full mediation) and attachment-avoidance (partial mediation) (Dakanalis et al., 2014). Unrelenting standards have been associated with anger suppression in women with EDs (Waller et al., 2003), emotion inhibition (self-silencing schemas & suppressed anger) in a mixed sample of AN and healthy control women (Geller et al., 2000), and also with emotion inhibition (self-silencing schemas) and depressive symptoms in an undergraduate sample (Flett et al., 2007). Further, two cross-sectional studies of British athletes, using structural equation modelling (SEM), found that a path model in which attachment-avoidance predicted self-critical perfectionism, then self-esteem, depressive symptoms, and then ED pathology demonstrated good fit for the data (Shanmugam et al., 2011); and likewise, using bootstrapping mediation analysis, that the relationship between attachment-anxiety and ED pathology was simultaneously mediated by low self-esteem, self-critical perfectionism, and depressive symptoms (Shanmugam et al., 2012).

Summary of early maladaptive schemas. Considered together, defectiveness/shame, emotional inhibition, and unrelenting standards schemas have all been found to be significantly higher in women with EDs compared with controls (Leung & Price, 2007; Overton et al., 2005) and all three were associated with increased ED pathology in a mixed sample of women (Overton et al., 2005). Such schemas may also play an important role in recovery from EDs and pose a particular challenge for therapy, as core beliefs are believed to be stable constructs that are self-perpetuating and highly resistant to change (Jones et al., 2007). As such, Jones et al. (2007) suggest that maladaptive schemas may be difficult to

challenge via use of conventional CBT. For instance, both emotional inhibition and unrelenting standards were higher in women with current-EDs than in those with recovered-EDs and controls (who did not differ from one another), whereas defectiveness/shame was higher in women with current-ED than those with recovered-EDs, and significantly higher in those with recovered-EDs compared with controls (Jones et al., 2005). Defectiveness/shame and unrelenting standards schemas have also been negatively associated with change in bulimic pathology after 12-weeks of CBT (Leung, Waller, et al., 2000). Taken together, this evidence suggests that defectiveness/shame beliefs and associated feelings of internalised shame may be particularly resistant to change and pose an acute challenge for the effectiveness of traditional CBT for EDs (Goss & Gilbert, 2002; Waller, Kennerley, et al., 2007).

#### **Body Shame**

In addition to considering the association between ED pathology and feelings of defectiveness/shame on a global level, researchers have explored the role of shame specifically related to the body (Troop & Redshaw, 2012). Body shame relates to the experience of one's physical body as unattractive or undesirable in some way (Gilbert, 2002). Internalisation of cultural standards of beauty, such as the 'thin-ideal', together with self-imposed perfectionistic standards, may position women to experience body shame due to the perceived disparity between their actual and their ideal body (McKinley & Hyde, 1996). Women who associate achievement of idealised standards of body weight/shape with their identity, as is theorised to be the case for women who are prone to displace feelings of self-worth onto the body, may be particularly susceptible to adverse effects of body shame in the form of distress and ED pathology (Gilbert, 2002; McKinley & Hyde, 1996).

Empirically, body shame has been found to be associated with ED pathology, over and above internalised shame in a general population sample of women (Burney & Irwin, 2000).

Body shame has also been found to predict ED pathology among women with EDs (Doran & Lewis, 2012) and levels of body shame have been found to be higher among women with EDs compared with a control group of healthy women, controlling for depressive symptoms (Swan & Andrews, 2003). Further, body shame was found to predict symptoms of AN, over and above depressive symptoms and internalised shame, in a longitudinal study of women with a current or past EDs (Troop & Redshaw, 2012). Finally, in support of the expectations of a self/emotion-regulation model of ED pathology, body shame has been found to mediate the association between low self-esteem and ED pathology in high school students (Iannaccone et al., 2016).

## **Depressive Symptoms**

An additional consideration cutting across the theorised associations among insecure attachment, maladaptive core beliefs, negative feelings about the self and body and ED pathology, relates to the potential mediating role of depressive symptoms. Depressive symptoms and negative emotionality more generally have been consistently associated with and considered risk factors for ED pathology (Cassin & von Ranson, 2005; Culbert et al., 2015) (Agras, 2001). Defectiveness/shame, emotional inhibition, and unrelenting standards schemas are strongly associated with both ED pathology and depressive symptoms and markedly elevated among women with comorbid ED pathology and depressive symptoms (Waller, Shah, Ohanian, & Elliott, 2001). Further, and consistent with a self/emotion-regulation model of ED pathology, a recent systematic review and meta-analysis of longitudinal studies of the associations between ED pathology and depression concluded that individuals who experience depressive symptoms may develop ED pathology as a means of reducing negative mood (Puccio et al., 2016). As such, when considering variables potentially associated with ED pathology from a self/emotion-regulation model perspective, it is important to consider the role played by depressive symptoms.

#### **The Present Study**

In summary, a self/emotion regulation model of ED pathology, grounded in attachment theory, suggests that insecure attachment-dynamics give rise to feelings of defectiveness/shame (primary schema) and the development of emotional inhibition and unrelenting standards (secondary schemas) in response. It is proposed that these negative feelings about the self, unexpressed emotions, and unrelenting standards are then displaced onto the body, leading to increases in body shame, depressive symptoms and the use of ED pathology (the pursuit of an idealised body weight/shape, dietary restriction, binge eating, and compensatory behaviours) as a means of moderating distress and enhancing feelings of selfworth (Goss & Gilbert, 2002). To date, however, much of the evidence for this conceptualisation is derived from disparate pieces of research that address only certain components of the model. The goal of the current study was to address this limitation of the current evidence base by testing a path model of the associations among all of the abovementioned variables – attachment-anxiety, attachment-avoidance, defectiveness/shame schemas, emotional inhibition, unrelenting standards, body shame, and depressive symptoms - and ED pathology in a single population, namely, young adult women with varying degrees of ED pathology recruited from several different sources.

It is hypothesised, first, that the associations between attachment-anxiety and/or attachment-avoidance and ED pathology will be mediated by defectiveness/shame, emotional inhibition, unrelenting standards, body shame, and depressive symptoms, such that a path model including these variables will be a good fit for the data. Within such a model, it is hypothesised, second, that there will be full mediation in the case of attachment-anxiety, and partial mediation in the case of attachment-avoidance and that attachment-avoidance will also have a significant direct effect on ED pathology; third, in accordance with schema therapy, the relationships between defectiveness/shame and depressive symptoms and ED pathology

will be mediated by emotional inhibition and unrelenting standards; and fourth, in accordance with displacement theory, body shame will mediate the relationships between defectiveness/shame, emotional inhibition and ED pathology. Finally, it is hypothesised, fifth, that such a path model will demonstrate a good fit for the data in both subgroups of participants with little or no ED pathology and those with high levels of this pathology.

#### Method

# **Participants**

Participants were 724 women aged between 18 to 62 years ( $M_{age} = 22.62$ , SD = 6.52; the "full sample"). Of these, 197 were recruited via advertisements displayed on eating disorder support websites ("web sample"), 208 were recruited via email invitation to participate forwarded by family and friends of the researchers to prospective participants and via flyers displayed at a University campus ("community sample"), and 319 were first-year undergraduate students ("student sample"). Inclusion criteria were being female and 18 years or older, so as to control for potential gender and maturational influences on the associations among ED pathology and other study variables.

#### Measures

Eating Attitudes Test (EAT-26). The EAT-26 (Garner et al., 1982) is a 26-item self-report measure of ED pathology that includes items assessing *dieting*, *bulimia* and *food preoccupation*, and *oral control*. Items are rated on a 6-point scale (where *never*, *rarely*, *sometimes* were = 0, *often* = 1, *usually* = 2 and *always* = 3). The one negatively worded item is reverse scored and items summed to obtain a total score ranging from 0 to 78, with scores of 20 and above taken to indicate high risk of ED pathology (Garner et al., 1982). The EAT-26 is widely used as a screening tool for ED symptoms and concerns in population-based samples and has been found to have good psychometric properties when used for this purpose (Garner et al., 1982; Lee et al., 2002; Mintz & O'Halloran, 2000).

Eating Disorder Diagnostic Scale (EDDS). The EDDS (Stice et al., 2000) is a 22item self-report measure designed to yield probable DSM-IV ED diagnoses of AN, BN, and
BED (American Psychiatric Association, 1994). It contains 4 items assessing the attitudinal
symptoms of AN and BN during the past 3 months, rated on a 7-point scale (from  $\theta = not$  at
all to  $\theta = extremely$ ); 4 items assessing the frequency of binging behaviour in times (BN) and
days (BED) per week; 6 items assessing the experiential elements of binge episodes; and 4
items assessing frequency of purging behaviours (vomiting, use of diuretics or laxatives,
fasting, or excessive exercise). In addition to generating DSM-IV diagnoses, items are
standardised and summed to yield an ED symptom composite score (EDDS-SC). The EDDS
has been shown to have good psychometric properties in non-clinical and clinical samples of
adult women including good criterion validity with interview-based diagnoses, convergent
validity with validated measures of ED pathology, and internal consistency (Krabbenborg et
al., 2012; Stice et al., 2004; Stice et al., 2000).

In the current study ED pathology was assessed using two measures, namely the EAT-26 and the EDDS –SC scores, which were both used as indicators of an ED pathology latent variable in the SEM models, in order to reduce measurement error. Additionally, the algorithm used to generate DSM-IV ED diagnoses from EDDS responses was adapted to align with DSM-5 (American Psychiatric Association, 2013) criteria for ED including AN, BN, BED, and other specified feeding or eating disorders (OSFED). Using this adapted algorithm, participants were categorised as having no ED disturbance ("non-ED" subgroup) or a probable DSM-5 ED diagnosis ("probable-ED" subgroup).

Experience of Close Relationships-Revised (ECR-R) Questionnaire. The ECR-R (Fraley et al., 2000) is a 36-item self-report measure of adult attachment designed to assess the dimensions of attachment-related anxiety and avoidance by means of two 18-item subscales. Items are rated on a 7-point Likert scale (from I = strongly disagree to T = strongly disagree

strongly agree), negatively worded items reverse scored, and items on each subscale averaged to obtain a mean score for each dimension. The ERC-R anxiety and avoidance subscales have demonstrated strong internal consistency (Sibley & Liu, 2004) and good construct validity (Fairchild & Finney, 2006).

Young Schema Questionnaire – Short Form (YSQ-S3). The YSQ-S3 (Young, 2007) is a 90-item, self-report measure of 18 early maladaptive schemas (EMS) within five domains, namely *Disconnection & Rejection, Impaired Autonomy & Performance, Impaired Limits, Other-Directedness*, and *Overvigilance & Inhibition*. Of relevance for the current study were the subscales measuring defectiveness/shame, emotional inhibition, and unrelenting standards schemas. Each of these subscales contains 5-items rated on a 6-point scale from 1 (*completely untrue of me*) to 6 (*described me perfectly*), which are averaged to obtain a mean score for each EMS. The YSQ-S3 has been found to have good psychometric properties in both non-clinical and clinical samples of women (including with BN) (Waller, Meyer, & Ohanian, 2001; Wellburn, Coristine, Dagg, Pontefract, & Jordan, 2002), including high subscale internal consistency and good discriminant and construct validity (Lyrakos, 2014; Trip, 2006).

Body Shame subscale of the Objectified Body Consciousness Scale (OBCS). The OBCS (McKinley & Hyde, 1996) is a 24-item self-report measure designed to assess the three dimensions of body surveillance, body shame, and control beliefs. The body shame subscale contains 8-items assessing feelings of shame regarding one's body not conforming to cultural standards. This subscale has been found to have good test-retest reliability, acceptable internal consistency ( $\alpha = .84$  & .70, for undergraduate and middle-aged women, respectively), and construct validity (McKinley & Hyde, 1996).

**Depression, Anxiety and Stress Scale (DASS-21) Depression subscale.** Depression was measured using the depression subscale of the DASS-21 (Lovibond & Lovibond, 1995a),

which includes 7-items assessing symptoms of depression during the past week. Items are rated on a 4-point scale ( $0 = did \ not \ apply \ to \ me \ at \ all$  to  $3 = applied \ to \ me \ very \ much, \ or most \ of the time)$  and summed and then multiplied by two to yield a score ranging from 0 to 42. The depression subscale has demonstrated adequate internal consistency (Cronbach's  $\alpha = .81$ ) and good construct validity in a broad range of community-based samples (Lovibond & Lovibond, 1995b).

#### Procedure

Data were collected via an online survey using Qualtrics software. A brief description of the research was provided prior to commencement and informed consent to participate obtained. Participants were first asked their age, height, and weight before being presented with the abovementioned measures in randomised order (presentation of items within each scale was also randomised). Participants in the student sample participated in return for course credit, while those in the web and community samples were entitled to enter a prize draw for one of ten AUD\$100 ColesMyer Gift Cards. The Macquarie University Human Research Ethics Committee approved all aspects of the study design and methods.

### **Statistical Analysis**

Study samples were first compared across recruitment methods using General Linear Model Analysis of Variance (GLM ANOVA) with simple contrast tests (using a Bonferroni adjusted *p*-value of < .017 for significance). Pearson bivariate correlations were used to determine the strength and direction of associations between study variables. An alpha level of .05 was set to test significance, missing data were excluded pairwise, and all analyses were conducted using IBM Statistical Package for Social Sciences (SPSS) version 24.

Mediation effects and path model fit were tested via SEM using the maximum likelihood method (ML) in IBM SPSS AMOS (version 22) and using 10,000 bootstrap samples to estimate the size of the total, direct, and indirect effect and give 95% confidence

intervals (CIs) around each effect to test for statistical significance. Advantages of this method include the use of sampling distributions that are free from the requirement of multivariate normality that underlies SEM, increased statistical power and greater accuracy in the assessment of parameter estimates (Byrne, 2010). It should be noted that in previous literature BMI is a variable of interest to ED concerns and had a small positive association with ED symptoms and a small negative association with ED concerns in the current study. However, given the complexity of the theoretical models being assessed via SEM Analyses in AMOS and the number of variables in each model in the current study, BMI was excluded from these analyses.

The overall goodness-of-fit of structural models was assessed using the Tucker-Lewis Index (TLI), Comparative Fit Index (CFI), root mean square error of approximation (RMSEA) and RMSEA closeness of fit (PCLOSE) fit indices. According to commonly accepted criteria (Byrne, 2010; Schreiber et al., 2006), acceptable goodness-of-fit is indicated by TLI greater than .95, CFI greater than .95, RMSEA less than .08, and PCLOSE greater than .05. In addition, chi-square difference ( $\Delta \chi^2$ ) tests were used to compare nested models.

#### Results

#### **Preliminary Analysis**

Results of the GLM ANOVA contrast tests indicated that the web sample scored higher than the community and student samples on all study variables, whereas the community sample scored higher than the student sample on measures of defectiveness/shame, emotional inhibition, unrelenting standards, and body shame. These differences were expected given the different recruitment methods. Additional analysis indicated that there were no significant interactions between recruitment method and any of the study variables in predicting ED pathology. Hence, pooling of data across the recruitment methods was deemed appropriate.

Descriptive statistics and correlations between study variables are given in Tables 1 and 2. According to the EDDS, 385 participants (53.2% of the total sample) were classified as having no probable ED diagnosis ('non-ED' subgroup), while the remaining 339 participants met operational criteria for a probable DSM-5 ED diagnosis ('probable-ED' group). Of these, 67 were classified as probable cases of AN, 130 probable BN, 30 probable BED, and 112 OSFED (56 'purging disorder' and 56 'restrained eating disorder'). As can be seen in Table 1, the probable-ED subgroup scored significantly higher than the non-ED subgroup on all study variables with the exception of age and BMI.

### **SEM Analysis of Model Fit and Mediation Effects**

**Hypothesis 1.** A full model specifying that the associations among attachment anxiety and avoidance and ED pathology would be mediated by defectiveness/shame, emotional inhibition, unrelenting standards, body shame, and depressive symptoms, the hypothesised model (Figure 1), was first tested in the full sample. This model (Model 1) was found to have very good fit to the data (Table 3), although a number of non-significant paths were apparent. As such Model 2 (Figure 2), which constrained to zero all non-significant paths from Model 1, was fitted. It demonstrated equivalent overall goodness of fit as Model 1 (no significant  $\Delta \chi^2$ ) and was thus deemed a more parsimonious model. The final model (Model 2) was found to have very good fit for the data and explained 69% of the variance in ED pathology.

Hypotheses 2 to 4. Within the final model, attachment-anxiety and attachment-avoidance each had significant indirect effects on ED pathology via defectiveness/shame, emotional inhibition, unrelenting standards, body shame, and depressive symptoms (Table 4). The direct effect of attachment-avoidance on ED pathology remained significant (partial mediation), whilst there was no significant direct effect of attachment-anxiety on ED pathology (full mediation). Defectiveness/shame was directly associated with each of emotional inhibition, unrelenting standards, and depressive symptoms. Defectiveness/shame

Table 1

Descriptive Statistics for Study Variables in the Full Sample and Comparison in the Non-ED and Probable-ED Subgroups

|                       |     |       | Full  | Sample |       |          |      | Non-ED subgroup | group | Probable-El | e-ED  |         |       |
|-----------------------|-----|-------|-------|--------|-------|----------|------|-----------------|-------|-------------|-------|---------|-------|
|                       |     |       |       |        |       | Skewness | ness |                 |       | sapgro      | dno   |         |       |
|                       | и   | M     | QS    | Min    | Max   | Stat     | SE   | M               | SD    | M           | SD    | t       | D     |
| Age                   | 724 | 22.62 | 6.58  | 18     | 62    | 2.35     | .091 | 22.53           | 6.67  | 22.72       | 6.35  | l       | .704  |
| BMI                   | 724 | 22.28 | 4.71  | 12.88  | 51.02 | 1.88     | .091 | 22.19           | 4.65  | 22.39       | 4.78  |         | .575  |
| ED symptoms           | 724 | 29.80 | 19.07 | 0      | 111   | 1.04     | .091 | 18.26           | 12.11 | 42.91       | 16.94 | -22.254 | <.001 |
| ED concerns (EAT-26)  | 724 | 20.07 | 18.19 | 0      | 75    | .91      | .091 | 10.07           | 10.75 | 31.43       | 18.26 | •       | <.001 |
| Attachment avoidance  | 721 | 3.49  | 1.37  | -      | 7     | .18      | .091 | 10.20           | 9.78  | 21.10       | 12.54 | •       | <.001 |
| Attachment anxiety    | 721 | 3.75  | 1.34  | -      | 7     | 07       | .091 | 3.13            | 1.25  | 3.90        | 1.37  |         | <.001 |
| Defectiveness/shame   | 707 | 2.76  | 1.53  | -      | 9     | .55      | .092 | 3.33            | 1.28  | 4.22        | 1.25  |         | <.001 |
| Emotional inhibition  | 707 | 2.98  | 1.27  | -      | 9     | .23      | .092 | 2.14            | 1.20  | 3.49        | 1.55  |         | <.001 |
| Unrelenting standards | 707 | 3.84  | 1.13  | -      | 9     | 10       | .092 | 2.54            | 1.11  | 3.50        | 1.25  | •       | <.001 |
| Body shame            | 721 | 3.58  | 1.24  | -      | 9     | .04      | .091 | 3.52            | 1.08  | 4.20        | 1.08  |         | <.001 |
| Depressive Symptoms   | 724 | 15.30 | 12.41 | 0      | 42    | .588     | .091 | 2.92            | 1.03  | 4.33        | 1.00  | ٠,      | <.001 |

BMI = Body Mass Index. EAT-26 = Eating Attitudes Test. ED symptoms = Eating Disorder Diagnostic Scale ED symptom composite score (EDDS-SC).

Table 2

Correlations Among Study Variables in the Full Sample

|    |                       | П    | 2    | 3      | 4                | 5                | 9         | 7                | 8        | 6                | 10               | 11               |
|----|-----------------------|------|------|--------|------------------|------------------|-----------|------------------|----------|------------------|------------------|------------------|
| 1  | Age                   | I    |      |        |                  |                  |           |                  |          |                  |                  |                  |
| 7  | BMI                   | .195 | I    |        |                  |                  |           |                  |          |                  |                  |                  |
| 3  | ED symptoms (EDDS-SC) | .078 | .080 | I      |                  |                  |           |                  |          |                  |                  |                  |
| 4  | ED concerns (EAT-26)  | .062 | 103  | .738   | .95 <sup>a</sup> |                  |           |                  |          |                  |                  |                  |
| 5  | Attachment avoidance  | .012 | 031  | .407   | .416**           | .95 <sup>a</sup> |           |                  |          |                  |                  |                  |
| 9  | Attachment anxiety    | 041  | .040 | .380   | .357**           | .431             | $.93^{a}$ |                  |          |                  |                  |                  |
| 7  | Defectiveness/shame   | .005 | 010  | .523   | .556             | .595             | .590      | .93 <sup>a</sup> |          |                  |                  |                  |
| ∞  | Emotional inhibition  | 010  | 022  | .455   | .502             | .588             | .497      | .742             | $80^{a}$ |                  |                  |                  |
| 6  | Unrelenting standards | 0200 | 019  | .400°° | .477             | .348             | .381      | .543             | .551     | .81 <sup>a</sup> |                  |                  |
| 10 | Body shame            | .109 | .164 | .655   | 869              | ÷404.            | .439      | .5963.           | .522     | .508             | .87 <sup>a</sup> |                  |
| 11 | Depressive symptoms   | .037 | 019  | .518** | .577             | .460°°           | .506      | £629.            | .605     | .434             | .567**           | .93 <sup>a</sup> |
|    |                       |      |      |        |                  |                  |           |                  |          |                  |                  |                  |

\*. Correlation significant at p < .05 (2-tailed). \*\*. Correlation significant at p < .01 (2-tailed). \*. Cronbach's  $\alpha$  in the present study. BMI = Body Mass Index.

Table 3

Summary of Results of Path Analysis Comparing Nested Model 1 (Hypothesised Model) and Model 2 (with Non-Significant Paths Removed;

Final Model) in the Full Sample and Nested Models 2 and 2a/2b in each of the Subgroups Separately

|                      | $\chi^{2}$ | df | р    | p CMIN/df | CFI  | TLI  | TLI RMSEA | (90% CI)     | (90% CI) PCLOSE | $\Delta\chi^2(df)$ | р    |
|----------------------|------------|----|------|-----------|------|------|-----------|--------------|-----------------|--------------------|------|
| Full Sample          |            |    |      |           |      |      |           |              |                 |                    |      |
| Model 1              | 13.225     | 9  | .040 | 2.204     | 866. | 886. | .041      | (.009, .072) | .638            |                    |      |
| Model 2              | 27.719     | 15 | .023 | 1.848     | 966  | .992 | .035      | (.013, .055) | .891            | 14.494 (9)         | >.05 |
| Probable-ED subgroup |            |    |      |           |      |      |           |              |                 |                    |      |
| Model 2              | 18.037     | 15 | .261 | 1.202     | 866  | .994 | .025      | (.000, .061) | .855            |                    |      |
| Model 2a             | 22.050     | 18 | .230 | 1.225     | 766. | .993 | .026      | (.000, .059) | .870            | 4.013(3)           | >.05 |
| Non-ED subgroup      |            |    |      |           |      |      |           |              |                 |                    |      |
| Model 2              | 33.336     | 15 | .004 | 2.222     | 986  | 996. | .057      | (.031, .083) | .302            |                    |      |
| Model 2b             | 38.092     | 17 | .002 | 2.241     | .984 | 996. | .057      | (.033, .082) | .285            | 4.756 (2)          | >.05 |

 $\chi^2$  = Chi-square. df = Degrees of Freedom. p = Chi-square significance test. CMIN/df = Chi-square/degrees of freedom ratio. CFI = Comparative Fit Index. TFI = Tucker-Lewis Index. RMSEA = Root Mean Square Error of Approximation. 90% CI = 90% Confident Interval around RMSEA. PCLOSE = RMSEA closeness of fit.  $\Delta \chi^2$  = Chi-

squared difference.

Table 4

Summary of Results of Mediation Analyses in Full Sample

|   | Total effect      |       | Direct effect           |       | Indirect                |       | Result         |
|---|-------------------|-------|-------------------------|-------|-------------------------|-------|----------------|
|   | $\beta$ (95% CI)  | d     | $\beta$ (95% CI)        | d     | $\beta$ (95% CI)        | р     |                |
| Model 2 (Final Model)                     |                   |       |                         |       |                         |       |                |
| Avoid→Shame→EmoInh→UnStand→               | .332 (.264, .392) | <.001 | .109 (.049, .165) <.001 | <.001 | .223 (.185, .265) <.001 | <.001 | Mediation      |
| BodSha→Dep→ED                             |                   |       |                         |       |                         |       |                |
| Anxiety→Shame→EmoInh→UnStand→             | .293 (.240, .346) | <.001 | I                       |       | .293 (.240, .346) <.001 | <.001 | Full mediation |
| BodSha→Dep→ED                             |                   |       |                         |       |                         |       |                |
| Shame→EmoInh→UnStand→ BodSha→             | .470 (.412, .527) | <.001 | ı                       |       | .470 (.412, .527) <.001 | <.001 | Full Mediation |
| Dep→ED                                    | ,                 |       |                         |       |                         |       |                |
| EmoInh→UnStand→ BodSha→Dep→ED             | .121 (.088, .160) | <.001 | I                       |       | .121 (.088, .160)       | <.001 | Full mediation |
| UnStand→ BodSha→Dep→ED                    | .250 (.181, .318) | <.001 | .092 (.031, .150)       | .004  | .158 (.115, .204)       | <.001 | Mediation      |
| BodSha $\rightarrow$ Dep $\rightarrow$ ED | .624 (.557, .684) | <.001 | .579 (.508, .644)       | <.001 | .045 (.027, .070)       | <.001 | Mediation      |

 $\beta$  = Standardised Effect Coefficient. 95% CI = Bootstrapped 95% Confidence Intervals around Standardised Effect. p = Two-tailed significance. Shame =

Defectiveness/Shame Early Maladaptive Schema (EMS). BodSha = Body Shame. Dep = Depression. ED = Eating disorder psychopathology latent variable (ED concerns &

ED symptoms). Avoid = Attachment avoidance. EmoInh = Emotional Inhibition EMS. UnStand = Unrelenting Standards EMS.

also had significant indirect effects on depressive symptoms via emotional inhibition (partial mediation) and on ED pathology via emotional inhibition, unrelenting standards, body shame, and depressive symptoms (full mediation). The variables significantly directly associated with ED pathology were attachment-avoidance, unrelenting standards, body shame, and depressive symptoms (partial mediation for each).

Hypothesis 5. The final model was found to have good fit in each of the non-ED and probable-ED subgroups considered separately (Table 4), although a number of non-significant paths were apparent in each subgroup (Figure 3). In the probable-ED subgroup, the three paths between attachment-anxiety and emotional inhibition, body shame, and depressive symptoms were each not significant. An alternate model (Model 2a) constraining these paths to zero demonstrated equivalence of overall model fit (no significant  $\Delta \chi^2$ ) and excellent fit for the data. In the non-ED subgroup, the two direct paths between each of attachment-avoidance and unrelenting standards and ED pathology were not significant. An alternate model (Model 2b) constraining these paths to zero demonstrated equivalent overall goodness of fit (no significant  $\Delta \chi^2$ ) and good fit for the data.

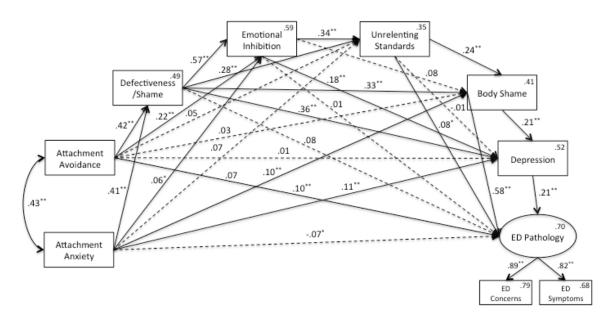


Figure 1. The Hypothesised Mediation Model (Model 1) Fitted in the Full Sample. Model 1 explains 70% of the variance in ED Pathology. Model fit:  $\chi^2 = 13.225$ , df = 6, p = .040, CMIN/df = 2.204, CFI = .998, TLI = .988, RMSEA = .041 (90% CI: .009, .072, PCLOSE = .638). Dashed line = path not significant (p > .05). \* = p < .05. \*\* = p < .001.

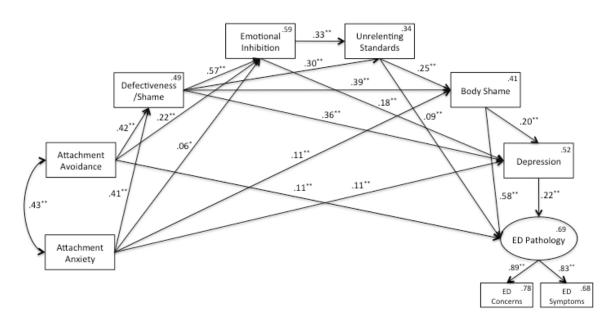


Figure 2. The Final Model (Model 2) fitted in the Full Sample. All possible indirect effects statistically significant (p < .001). The Final Model explains 69% of the variance in ED pathology. Model fit:  $\chi^2 = 27.719$ , df = 15, p = .023, CMIN/df = 1.848, CFI = .996, TLI = .992, RMSEA = .035 (90%CI: .013, .055, PCLOSE = .891). Model 1 v 2:  $\Delta \chi^2$ (df) = 14.494(9); p > .05 (ns). \*\* = p < .001.

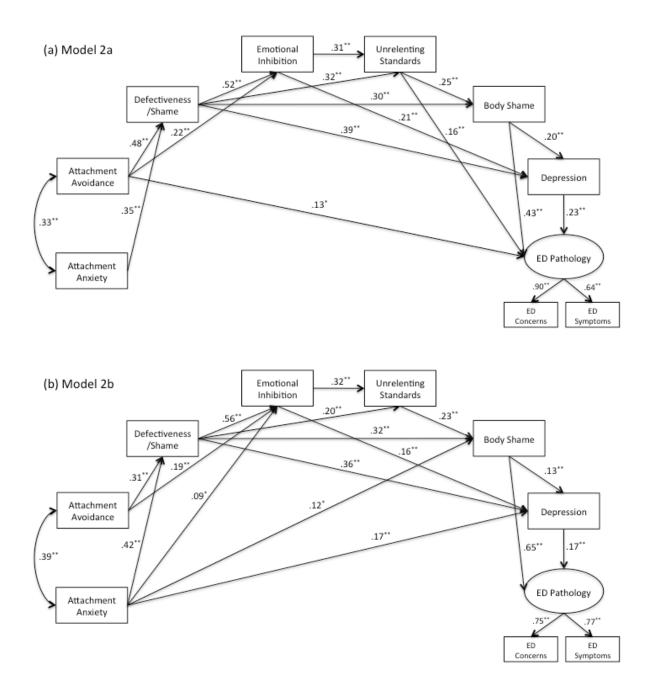


Figure 3. Comparison of standardised path coefficients in the final reduced models fitted separately in each of the Probable-ED (Model 2a) and Non-ED (Model 2b) subgroups.

\* = p < .05.\*\* = p < .001.

#### **Discussion**

The current study aimed to investigate, by means of SEM, a self/emotion-regulation model of EDs grounded in attachment theory in a large, diverse sample of young adult women. As hypothesised, a path model exploring the associations among attachment-anxiety,

attachment-avoidance, defectiveness/shame, emotional inhibition, unrelenting standards, body shame, depressive symptoms and ED pathology demonstrated excellent fit for the data and explained a substantial proportion of the variance in ED pathology. This accords with and extends previous research examining the contributions to variance in ED pathology of low self-esteem, perfectionism and interpersonal difficulties (Lampard, Tasca, Balfour, & Bissada, 2013); interpersonal problems, core low self-worth, perfectionism and mood intolerance (Dakanalis et al., 2015); and attachment-avoidance, perfectionism, low self-esteem and depressive symptoms (Shanmugam et al., 2011, 2012).

Also consistent with theoretical prediction and previous research (Dakanalis et al., 2014; Tasca et al., 2009a), it was hypothesised that the associations among both attachmentanxiety and attachment-avoidance and ED pathology would be mediated by the other study variables but that in the case of attachment avoidance a direct effect on ED pathology would remain. These hypotheses were also confirmed, suggesting that the impact of attachmentanxiety on ED pathology is accounted for by feelings of defectiveness/shame, emotional inhibition, unrelenting standards, body shame, and depressive symptoms. The impact of attachment-avoidance on ED pathology, by contrast, appears to occur both indirectly, via feelings of defectiveness/shame and emotional inhibition, and directly. It is possible that, in addition to its impact on feelings of self-worth and emotional expression, attachmentavoidance influences ED pathology through its adverse impact on interpersonal behaviour, by interfering with the individual's ability to access social support and to regulate emotions interpersonally. Consistent with this view, evidence suggests that women with high attachment-avoidance tend to be reticent or ineffective in seeking social support during times of distress or when dealing with difficult life events (e.g., Collins & Feeney, 2000; DeFronzo, Panzarella, & Bulter, 2001).

It was further hypothesised, consistent with a schema therapy model (Young et al., 2003), that the association between defectiveness/shame and ED pathology would be mediated by emotional inhibition and unrelenting standards schemas and that, in accordance with displacement theory (Bruch, 1962), the associations between defectiveness/shame, emotional inhibition and unrelenting standards and ED pathology would be mediated by body shame. These hypotheses were also supported, namely, defectiveness/shame predicted both emotional inhibition and unrelenting standards but did not have a direct effect on ED pathology after controlling for all other indirect effects. Interestingly, unrelenting standards fully mediated any effect of emotional inhibition on body shame and ED pathology, whereas emotional inhibition had a significant direct effect on depressive symptoms. The effects on ED pathology of attachment-anxiety, defectiveness/shame, and emotional inhibition were each fully mediated by unrelenting standards, body shame, and depressive symptoms, while the effects of attachment-avoidance and emotional inhibition were each partially mediated by unrelenting standards and body shame. These findings extend those of previous research (Burney & Irwin, 2000; Dakanalis et al., 2014; Troop & Redshaw, 2012) in suggesting that negative feelings about the self and unexpressed emotions are displaced onto the body, thereby giving rise to further negative mood and ED pathology.

Finally, it was hypothesised that the final model would demonstrate good fit for the data in both non-ED and probable-ED subgroups. There was partial support for this hypothesis, in that the final model demonstrated adequate goodness of fit in both subgroups, while a number of distinct non-significant pathways were observed in each case. Among women with a probable-ED, defectiveness/shame fully mediated the association between attachment-anxiety and each of the other study variables: emotional inhibition, unrelenting standards, body shame, depressive symptoms, and ED pathology. In other words, attachment anxiety influenced these variables via feelings of defectiveness/shame. Among asymptomatic

women, by contrast, attachment-anxiety was found to influence emotional inhibition, body shame, and depressive symptoms directly while indirectly influencing ED pathology via these variables and defectiveness/shame. Further, within this subgroup the impact of attachment-avoidance on ED symptoms was fully mediated by defectiveness/shame and emotional inhibition (and each of their indirect effects).

# **Study Implications**

Within the limits of the cross-sectional design, the findings of the current study provide support for a self/emotion-regulation model of EDs grounded in attachment theory, wherein insecure attachment dynamics (anxiety and avoidance) drive the development of negative feelings about the self (defectiveness/shame) and negative beliefs about emotional expression (emotional inhibition). Negative feeling and beliefs about the self and unexpressed emotions are then displaced onto the body, in the form of unrealistically high standards relating to body weight and shape and consequent body shame, thereby giving rise to low mood (depressive symptoms) and ED pathology. Thus, within this model, the body becomes the locus of feelings of low self-worth and control of body weight/shape and food becomes the means by which to both block feelings of defectiveness/shame and negative emotions and to attempt to enhance feelings of self-worth (Bruch, 1978; Goss & Gilbert, 2002).

The current findings suggest that difficulties with attachment, feelings of defectiveness/shame and emotional expression, along with unrealistically high standards and undue self-criticism, may warrant greater consideration in the clinical management of ED pathology (Goss & Gilbert, 2002; Lawson & Waller, 2012). To this end schema-focused CBT for EDs (SFCBT; Waller, Cordery, et al., 2007), has been suggested as a means of targeting some of these underlying features contributing to ED pathology. While there is evidence of promising outcomes of the use of SFCBT for EDs from several case reports (Ohanian, 2002) and a pilot study of group schema therapy for EDs (Simpson, Morrow, van Vreeswijk, &

Reid, 2010), systematic evidence from RCTs is needed to support the efficacy of schemafocused CBT for EDs and compare its efficacy with other available treatment modalities.

Additionally, some potentially promising interventions aimed explicitly at reducing shame and self-criticism among individuals with EDs also have recently emerged, including compassionate-mind training (Gilbert & Procter, 2006), compassion-focused therapy (Gilbert, 2009), and compassion-focused therapy for EDs specifically (Goss & Allan, 2012). While research addressing the utility of these approaches in the treatment of ED pathology is also in its infancy, evidence of the importance of self-compassion in moderating ED pathology and risk factors for this, is growing in both non-clinical (Breines et al., 2013; Geller et al., 2015; Schoenefeld & Webb, 2013) and clinical ED samples of women (Ferreira et al., 2014; Kelly & Tasca, 2016).

The findings of the current study also support the use of treatments that specifically target perfectionism among women with ED pathology (Lloyd, Fleming, Schmidt, & Tchanturia, 2014) and treatments that focus on reducing inhibition or avoidance of painful emotions and internal experiences. The latter include CBT-E (Corstorphine, 2006), integrative cognitive-affective therapy for BN (Wonderlich et al., 2010), and mindfulness-based approaches, such as DBT for BN and BED (Safer et al., 2009), emotion acceptance behaviour therapy for AN (EABT; Wildes, Marcus, Cheng, McCabe, & Gaskill, 2014), and acceptance and commitment therapy (ACT) for EDs (Sandoz et al., 2010). Finally, given the important role of attachment anxiety and avoidance in directly and indirectly predicting ED pathology, treatments that specifically address underlying issues with attachment insecurity and the emotional and interpersonal styles associated with these, such as emotion-focused therapy for EDs (EFT; Dolhanty & Greenberg, 2007), may warrant greater consideration.

# Study strengths, limitations and directions for future research

A notable strength of the current study lies in its effort to explore in an integrated model the structural relationships among insecure attachment dynamics (anxiety and avoidance), defectiveness/shame, emotional inhibition, perfectionism (unrelenting standards), body shame, and depressive symptoms in explaining variance in ED pathology. The study sought to bring together a number of highly-related and overlapping theoretical models proposed to explain the development and maintenance of EDs, to combine these in a comprehensive self/emotion-regulation model of ED pathology grounded in attachment theory, and to test such a model using relatively sophisticated statistical analyses (SEM and Bootstrapping procedures); thereby addressing a gap in the literature. Another strength of the study was the recruitment of a relatively large sample of participants from a number of sources. Thus, ensuring a broad range of values on each of the study variables, enhancing the generalisability of the findings, and permitting the identification of a subgroup of probable-ED cases. The fact that close to half of the sample was identified with probable-ED diagnoses suggests that the findings may have clinical relevance. Nevertheless, future research replicating the current findings in a treatment-seeking sample of individuals with EDs would have more direct implications for clinical practice.

The most notable limitation of the current study was the use of a cross-sectional correlational design. Correlations cannot provide evidence of direction of effect and mediational processes, such as those suggested in the path models here, do imply direction of effect and causality. However, despite this limitation, it has been suggested that where a theoretical foundation exists to support the directionality of effects it may be appropriate to test for mediation effects in cross-sectional data using path analyses (A. F Hayes, 2013). Given the strong theoretical basis for the path model proposed and tested in the current study – based on attachment theory (Bowlby, 1969), displacement theory (Bruch, 1962), escape

from self-awareness (Heatherton & Baumeister, 1991), and 'blocking' (McManus & Waller, 1995; Root & Fallon, 1989) theories of self and emotion regulation in EDs – it was deemed appropriate at a preliminary stage of instigation to test such a structural model using cross-sectional data. Nevertheless, there are likely to be complex, bidirectional pathways underpinning the associations among insecure attachment styles, defectiveness/shame, emotional inhibition, unrelenting standards, body shame, depressive symptoms, and ED pathology and clearly replication of these findings in longitudinal research will be needed to elucidate the direction and nature of these pathways across time. Additionally, future research may be served by also including such variables as age and BMI as covariates.

Another notable limitation of the current study is that the algorithm used to categorize participants as having no probable-ED or a probable diagnosis of DSM-5 EDs from EDDS responses, is unvalidated for DSM-5 and future research using a validated measure, or ideally, an interview measure to diagnose DSM-5 EDs, to replicate similarities and differences found across the subgroups herein will be necessary. Indeed reliance on self-report measures of the study variables is a general limitation of the current study. While the use of online surveys are a highly efficient means of data collection, potential problems with the use of self-report measures of ED symptoms, in particular, are well-known (House et al., 2008). The inclusion of interview data, for example, from the Eating Disorder Examination (EDE-17.0D; Fairburn et al., 2014) for ED pathology and the Adult Attachment Interview (Kaplan & Main, 1985) or a relationship interview technique, such as the Current Relationship Interview (CRI; Crowell & Owens, 1996; Crowell et al., 2002), which explores representations of adult attachment within adult romantic relationships, for adult attachment, would be of interest in future research.

It should be noted also that the recruitment methods employed in the current study are susceptible to selection bias, which may detract from the generalisability of the findings. For

example, it is possible that emotionally inhibited and avoidantly-attached individuals are less likely to volunteer to participate in research aimed at assessing emotional and interpersonal experiences and styles. The relatively high proportion of participants with high levels of ED pathology is another example of selection bias, albeit an asset in the current context. Finally, the fact that the current research was confined to adult females needs to be acknowledged as a limitation to the generalisability of the current findings. Although this was reasonable for a study designed to extend research previously conducted in this demographic, the replication of these findings in future research using samples including adolescent girls and men would be of interest.

## Conclusion

Within the limits of a cross-sectional design, the current findings support the proposed self/emotion-regulation model of EDs grounded in attachment theory, and suggest that insecure attachment styles (anxiety and avoidance) contribute to negative feelings about the self (defectiveness/shame), emotional inhibition, and unrealistically high standards, which are externalised onto the body (in the form of body shame), and thereby contribute to depressive symptoms and, in turn, increased ED pathology.

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# Chapter 9

**General Discussion** 

### SUMMARY OF RESEARCH AIMS AND MAIN FINDINGS

The broad goals of the current research were to explore both emotion-regulation and self-regulation models of eating disorder (ED) pathology and to test an integrated self/emotion-regulation model of ED pathology grounded in attachment theory. These goals were addressed across four papers – in which four more specific aims were addressed – using two distinct diverse samples of women.

In accordance with emotion-regulation models of ED pathology, the research project aimed, first, to explore the individual and unique roles of several forms of emotion inhibition (as one specific maladaptive form of emotion regulation) – namely, negative beliefs about emotion, ambivalence over emotional expressiveness, silencing-the-self and divided-self schemas – in accounting for variance ED pathology, while also examining any mediating and/or moderating role of negative emotionality in the associations among each form of emotion inhibition and ED pathology. Results of the first empirical study (Chapter 5) were consistent with previous evidence in suggesting that each form of emotion inhibition was individually associated with ED pathology when controlling for depressive symptoms. However, when considered together, only the construct of negative beliefs about emotion uniquely contributed to the variance in ED pathology, controlling for depressive symptoms. The prediction that negative emotionality would moderate any associations was not supported by the findings, suggesting that lower negative emotionality may not be protective in mitigating the association between emotion inhibition and ED pathology.

However, the results of the first study extended previous research in suggesting that negative emotionality partially mediates the bivariate associations between negative beliefs about emotion, silencing-the-self, and divided-self and ED pathology, and fully mediates the association between ambivalence over emotional expressiveness and ED pathology, taking account of depressive symptoms. These findings converge with evidence that emotion

inhibition, in the form of expressive suppression (Gross & John, 2003) and ambivalence over emotional expressiveness (King & Emmons, 1990), is associated with increased experience of negative emotion and provides new evidence of one of the potential processes through which these various forms of emotion inhibition may influence ED pathology via negative emotionality.

The second aim of the current research was to investigate the direct and indirect roles of various emotion-regulation strategies – cognitive reappraisal ('reappraisal'), expressive suppression ('suppression'), rumination, reflection, and emotional reflection – negative beliefs about emotion, negative emotionality, and depressive symptoms in explaining variance in ED pathology. Results of the second empirical study (Chapter 6) confirm and extend previous findings regarding associations between each of these emotion-regulation strategies and negative beliefs about emotion with ED pathology (e.g., Hambrook et al., 2011; McLean et al., 2007; Rawal et al., 2010). Whilst emotional reflection was uniquely associated with ED pathology, over and above general reflection, when all emotion-regulation strategies were considered together, neither reflection nor emotional reflection were uniquely associated with ED pathology.

Further, findings of the second study revealed that negative emotionality and depressive symptoms each mediated the associations between reappraisal, rumination, and suppression and ED pathology (full mediation in the case of rumination and reappraisal). These findings provide an important and novel contribution concerning one of the possible mechanisms by which both maladaptive emotion-regulation strategies and the adaptive emotion-regulation strategy of reappraisal (inversely) may influence ED pathology via increases in the experience of negative emotion and depressive symptoms. Additionally, structural equation modelling (SEM) analysis indicated that the maladaptive emotion-regulation strategies of suppression and rumination, together with negative emotionality and

depressive symptoms each mediated the association between negative beliefs about emotion and ED pathology. These findings contribute new evidence that possessing negative beliefs about emotion may contribute to the use of maladaptive emotion-regulation strategies (suppression and rumination) thereby increasing the experience of negative emotion, depressive symptoms, and ED pathology. These findings extend the body of empirical evidence on the associations between maladaptive emotion-regulation strategies and ED pathology, by identifying the possible contribution of underlying maladaptive beliefs about emotion. Further, these results were consistent when this structural model was fitted in subgroups of women with no probable ED (non-ED) and probable-ED diagnoses separately, suggesting that these associations apply in both clinical and non-clinical populations.

The third aim of the current research was to test a combined displacement theory-based self/emotion-regulation model of ED pathology, whereby feelings of low self-worth (internalised shame and low self-esteem), externalised self-perceptions (self-objectification and body surveillance), body shame, and depressive symptoms are both directly and indirectly associated with ED pathology. Results of Study 3A (Chapter 7) indicated that internalised shame and low self-esteem were each independently associated with ED pathology, over and above the effects of depressive symptoms; and that these associations were each fully mediated through body shame. Study 3B (Chapter 7) results indicated that internalised shame independently directly contributed to variance in ED pathology, whilst also having indirect effects on ED pathology via self-objectification, body surveillance, body shame, and depressive symptoms. Taken together these results provide preliminary support for a displacement theory-based self/emotion-regulation model of ED pathology in which negative feelings about the self are externalised and displaced onto the body and that ED pathology may provide both an escape from self-awareness (Heatherton & Baumeister, 1991)

and an attempt to promote feelings of pride and self-worth through the control of body weight or shape (Goss & Gilbert, 2002).

The final aim of the current research was to evaluate a self/emotion-regulation model of ED pathology grounded in attachment theory. Supporting this theoretical model, a structural model comprising attachment-anxiety, attachment-avoidance, defectiveness/shame, emotional inhibition, unrelenting standards, body shame, depressive symptoms and ED pathology demonstrated an excellent fit for the data and explained a substantial proportion (69%) of the variance in ED pathology (Study 4; Chapter 8). Consistent with previous research (Dakanalis et al., 2014; Tasca et al., 2009a), within this structural model the association between attachment-anxiety and ED pathology was fully explained by indirect associations through feelings of defectiveness/shame, emotional inhibition, unrelenting standards, body shame, and depressive symptoms. Whereas attachment-avoidance had both a direct effect on ED pathology and indirect effects via increases in feelings of defectiveness/shame, emotional inhibition, unrelenting standards, body shame, and depressive symptoms.

One possible explanation for these results is that in addition to the negative impact of attachment-avoidance on feelings of self-worth and emotional expression, the isolation and lack of social support characteristic of those with an avoidant attachment style may increase the experience of ED pathology. Social support has been found to moderate the impact of subclinical levels of disordered eating on quality of life in young women (Wade et al., 2012). Accordingly, attachment-avoidance may give rise to or maintain ED pathology by interfering with the individual's ability to access social support and to regulate emotions through interpersonal relationships. Such an explanation accords with evidence that women with high attachment-avoidance tend to be reluctant or ineffective in seeking social support during times

of distress or when dealing with difficult life events (e.g., Collins & Feeney, 2000; DeFronzo et al., 2001).

Also of note, in relation to the findings of Study 4, is that when the final model was fitted in subgroups of women with and without probable-EDs separately, while adequate fit for the data was demonstrated in each subgroup, there were some differences in path significance across these subgroups. Among women with a probable-ED, defectiveness/shame fully accounted for the associations of attachment-anxiety and each of the other study variables: emotional inhibition, unrelenting standards, body shame, depressive symptoms, and ED pathology. In other words, attachment anxiety influenced these variables solely via feelings of defectiveness/shame. In women with no probable ED, by contrast, attachment-anxiety was found to influence emotional inhibition, body shame, and depressive symptoms directly while also indirectly influencing ED pathology via these variables and defectiveness/shame. Further, within non-ED women the impact of attachment-avoidance on ED symptoms was fully accounted for by defectiveness/shame and emotional inhibition (and each of their indirect effects).

# DISCUSSION OF FINDINGS AND UNIQUE CONTRIBUTIONS

Taken together the results of Studies 1, 2, and 4 suggest that emotion inhibition is an ineffective means of dealing with emotion, which paradoxically leads to increases in the experience of negative emotion. Further, emotion inhibition may contribute indirectly to increases in ED pathology through these increases in negative emotion. Such findings are consistent with the predictions of both self-silencing theory (Jack, 1991) and Corstorphine's (2006) affect-regulation model of ED pathology and extend findings from previous research demonstrating the counterproductive effects of suppression on negative emotion (Gross & John, 2003). These findings also provide support for the self/emotion-regulation model of ED pathology grounded in attachment theory proposed by this thesis.

The results of studies 3 and 4 together are consistent with the view that negative feelings about the self (internalised shame, low self-esteem and shame schemas) are externalised and displaced onto the body, contributing to increased body shame. In accordance with displacement theory (Bruch, 1973), such body shame mediates the association between deficits in sense of self and ED pathology. The results also extend predictions of both displacement and objectification theories (Fredrickson & Roberts, 1997) and provide new information about one of the factors that may underlie self-objectification and body surveillance, namely, internalised shame. Additionally, they suggest that self-objectification and body surveillance may each mediate the associations among internalised shame and body shame, depressive symptoms, and ED pathology. Further, unrelenting standards schemas were found to mediate the association between defectiveness/shame schemas and ED pathology. These findings together are consistent with suggestions that women with impairments in identity development and lack of a clearly defined sense of self may be particularly vulnerable to internalising cultural messages about "ideal" body weight and shape (Stein & Corte, 2003; Vartanian, 2009).

The findings of all four studies taken together, support the proposed self/emotion-regulation model of ED pathology grounded in attachment theory, whereby insecure attachment styles (anxiety and avoidance) are suggested to underlie both feelings of low self-worth (internalised shame and defectiveness/shame schemas) and maladaptive emotion inhibition beliefs. These in turn are then displaced onto the body, contributing to unrelenting standards, body shame, depressive symptoms, and, in turn, ED pathology. The displacement of negative feelings about the self and unexpressed emotions onto the more concrete target of the body, is believed to serve the maladaptive dual function of attempting to enhance feelings of self-worth through the pursuit of an idealised body weight or shape via extreme weight-control behaviours and temporarily escaping self-awareness (in the case of binge eating

behaviours). Results of the current research provide support for this integrated self/emotion regulation model of ED pathology.

An additional implication of the current findings is that emotion inhibition beliefs (i.e., negative beliefs about emotion and emotion inhibition schemas) are associated with the use of maladaptive emotion-regulation strategies, such as suppression and rumination, which may give rise to ED pathology by compounding the experience of negative emotionality and depressive symptoms. This pattern of associations is also consistent with the proposed self/emotion regulation model, wherein ED pathology is believed to function as a means of blocking or avoiding aversive emotional experience. The suggestion that negative beliefs about the acceptability of experiencing and expressing negative emotions may underlie the use of the maladaptive emotion-regulation strategies and thereby contribute to increased negative emotionality, depressive symptoms and, in turn, ED pathology, is novel and adds to existing evidence bearing on the role of difficulties with emotion-regulation in ED pathology (e.g., Aldao & Nolen-Hoeksema, 2010; Lavender et al., 2015).

Emotion inhibition is a broad construct that has been defined and operationalised in a variety of ways across the literature and variously associated with ED pathology via a number of distinct conceptualisations: for instance, within self-silencing theory as silencing-the-self and divided-self schemas (e.g., Buckholz et al., 2007; Ross & Wade, 2004), expressive suppression (e.g., Davies et al., 2012; McLean et al., 2007), ambivalence over emotional expressiveness (e.g., Forbush & Watson, 2006; Krause et al., 2000), within schema therapy as emotional inhibition schemas (Waller et al., 2002; Waller et al., 2000), and as negative beliefs about emotion (Hambrook et al., 2011; Oldershaw et al., 2012). The current research sought to make a unique contribution by teasing out the relative importance of these constructs when considered together (in study 1). Results revealed that negative beliefs about emotion and

suppression, in particular, seem to be important in their contribution to explaining variance in ED pathology.

Another broad construct that has been variously defined and operationalised in the ED literature is that of self-worth and how deficits in sense of self relate to ED pathology. The current research project explored the role of sense of self (incorporating feelings of self-worth) in contributing to ED pathology and attempted to tease out the relative contributions of several low self-worth-related constructs, such as low self-esteem, internalised shame, and defectiveness/shame schemas, in contributing to ED pathology. Results support the unique contribution of both self-esteem and internalised shame in explaining variance in ED pathology, over and above depressive symptoms. Furthermore, results support the importance of defectiveness/shame schemas in contributing to ED pathology both directly and indirectly within the integrated self/emotion-regulation model grounded in attachment theory.

### IMPLICATIONS FOR THEORY

As has been noted, the various emotion-regulation and self-regulation conceptualisations of ED pathology considered in the background to this research (Chapters 2 and 3) can be seen to comprise a number of closely overlapping concepts, all of which may arguably be subsumed within the overarching theoretical framework of attachment theory. In particular there is extensive overlap between the various conceptualisations of ED pathology deriving from emotion and self-regulation theories. Integrating these conceptualisations within the overarching theoretical framework of attachment theory may, it is suggested, be the most parsimonious way to address the complimentary contributions of sense of self and emotion-regulation processes in the development and maintenance of ED pathology. The findings of the current research support this contention and are consistent with a self/emotion-regulation model of EDs grounded in attachment theory.

According to attachment theory, insecure attachment styles (anxiety and avoidance) are seen as driving negative internal working models of self and others that are believed to underlie a developing sense of self characterised by internalised shame schemas. The coregulation of emotions through close relationships, and development of adaptive self-regulation strategies are at the heart of attachment theory and insecure attachment styles are theorised to underlie difficulties with emotional-development and capacities for emotion-regulation (both intrapersonally and interpersonally). The findings of the current research accord with and extend previous evidence on the importance of both negative feelings about the self and difficulties with emotion-regulation in contributing to ED pathology, and provide evidence for the role of attachment-avoidance and attachment-anxiety in underpinning such contributions.

The results of the current research also have implications for objectification theory (Fredrickson & Roberts, 1997), in providing preliminary evidence that internalised shame may be a factor contributing to increases in self-objectification and body surveillance.

Furthermore, findings suggest that self-objectification and body surveillance may each mediate the associations among internalised shame and body shame, depressive symptoms, and ED pathology. Such findings lend support to the notion that women with deficits in sense of self may be particularly vulnerable to internalising cultural messages about "ideal" body weight and shape, and thereby contribute to the development of ED pathology (Stein & Corte, 2003; Vartanian, 2009).

Finally, the results of the current research lend support to the theoretical propositions of schema therapy (Young et al., 2003) that defectiveness/shame, as a primary schema, may contribute to the development of secondary schemas, such as emotional inhibition and unrelenting standards schemas. Current findings extend this notion to provide preliminary evidence that emotional inhibition and unrelenting standards schemas in part account for the

associations among defectiveness/shame schemas and body shame, depressive symptoms and ED pathology in women.

While causal inference and direction of effect cannot be concluded from the current cross-sectional studies, and thus it is unclear whether disturbances in sense of self, feelings of low self-worth and difficulties with emotion-regulation precede ED pathology or are a consequence of it, nevertheless, it seems clear that such disruptions in sense of self and emotion-regulation need to be addressed therapeutically within treatment of EDs (Ewell et al., 1996).

## IMPLICATIONS FOR TREATMENT

The current findings suggest that disruptions in sense of self, insecure attachment styles, and maladaptive emotion regulation strategies may warrant greater consideration in the clinical management of ED pathology, not only as a target of treatment but also in terms of promoting treatment engagement. For instance, previous research has identified attachment-avoidance to be associated with treatment drop-out while high attachment-anxiety was associated with treatment completion in women with anorexia nervosa (Tasca et al., 2004) and binge eating disorder (Tasca et al., 2006). Further different treatment approaches may need to be tailored to different attachment styles, given evidence that high attachment-anxiety was associated with better outcomes using group psychodynamic interpersonal therapy rather than group-CBT at 12-month follow-up, whereas low attachment anxiety was predictive of better outcomes using group-CBT at 12-month follow-up (Tasca et al., 2006).

Treatment engagement may also be compromised by deficits in emotional awareness.

Impairments in the ability to recognise, identify, and describe emotional experience and to distinguish between various emotional and associated physical states may significantly interfere with the ability of individuals to engage in cognitive-behavioural therapies (CBT) which requires simple emotional self-monitoring and the capacity to link thoughts, behaviours,

and emotions (Jansch et al., 2009). Thus, addressing such difficulties with emotion and emotion-regulation may form an integral component of therapy and additionally enhance the efficacy of CBT for EDs, currently the most well supported treatment for ED pathology.

Efforts to revise established CBT protocols for ED treatment along these lines have been made in recent years. Thus, Fairburn and colleagues' "enhanced CBT" protocol (CBT-E) is designed to give greater attention to interpersonal difficulties, core low self-esteem, mood intolerance, and clinical perfectionism as factors in the maintenance of ED pathology (Fairburn et al., 2003; Fairburn et al., 2008). Whilst CBT-E has demonstrated efficacy in the treatment of some EDs, controlled studies of real-world effectiveness suggest that 50% or more of individuals with BN – the ED for which CBT is considered the "gold-standard" treatment – remain symptomatic following treatment, up to one third still meet criteria for BN (Poulsen et al., 2014; Waller et al., 2014), and a third may relapse (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000). Outcomes for individuals with AN are poorer still (Zipfel et al., 2014).

A possible explanation for these unsatisfactory results is that CBT models of ED pathology are largely maintenance models that fail to address certain factors implicated in the development of EDs, such as attachment dynamics and feelings of internalised shame, for example. Further, it is apparent that many of the maladaptive core beliefs that underlie ED pathology, such as those relating to defectiveness/shame, emotional inhibition and self-silencing, perfectionism and self-criticism, and negative beliefs about emotion, extend beyond those which form the primary focus of CBT for EDs, namely, beliefs relating to body weight/shape and food and the control of these. Importantly, these more pervasive underlying maladaptive schemas appear to be stable constructs, that are resistant to change, and may be particularly difficult to challenge via conventional CBT (Jones et al., 2007; Pugh, 2015). Furthermore, including consideration of core beliefs in CBT for EDs has not resulted in

improvement in treatment outcomes. This may indicate that more specialised and targeted treatments for identifying, challenging, and changing such core beliefs (e.g., defectiveness/shame, unrelenting standards, and emotional inhibition) may be needed.

Findings from the current research support this view that these maladaptive schemas and strategies need to be specifically addressed in therapy (Goss & Gilbert, 2002; Lawson & Waller, 2012). To this end a number of treatment alternatives (or adjuncts to CBT) may be indicated including schema-focused CBT for EDs (SFCBT; Waller, Cordery, et al., 2007), compassion-focused therapy for EDs (CFT-E; Goss & Allan, 2012), mindfulness-based therapies (e.g., Alberts et al., 2012; Kristeller et al., 2013), and treatment programs focusing on incorporating skills for enhancing interpersonal orientation (e.g., Rieger et al., 2010; Tanofsky-Kraff & Wilfley, 2010) and adaptive emotion-regulation/expression (e.g., Dolhanty & Greenberg, 2007; Ivanova & Watson, 2014).

#### **Schema Therapy**

Schema-focused CBT for EDs (Waller, Cordery, et al., 2007), has been suggested as a means of targeting the early maladaptive schemas that may underlie ED pathology. The findings of the current research suggest that addressing these underlying maladaptive schemas could be of great importance in ED therapy. Schema therapy includes elements of cognitive therapy and the challenging of maladaptive core beliefs, as is done in CBT for EDs. However, schema therapy goes beyond the use of cognitive strategies to also include experiential strategies (e.g., imagery rescripting), behavioural-pattern breaking, and use of the therapeutic relationship for limited reparenting, as a means of changing maladaptive schemas (Young, 1990). With regard to efficacy, there is some evidence available of promising outcomes from this approach in several case reports (Ohanian, 2002) and a pilot study of group schema therapy for EDs (Simpson et al., 2010). However, further systematic evidence from RCTs is

now needed to support the efficacy of schema-focused CBT for treating ED pathology and to compare its efficacy with other more established treatment modalities.

## **Compassion-Focused Therapy**

Recently, some promising interventions aimed directly at reducing internalised shame and self-criticism have also emerged, including compassionate-mind training (Gilbert & Procter, 2006), compassion-focused therapy (CFT) (Gilbert, 2009), compassion-focused therapy for EDs specifically (CFT-E; Goss & Allan, 2012), and self-compassion (Neff, 2011). According to CFT insecure attachment styles (anxious and avoidant) underlie the development of shame-based harsh self-criticism. This attachment-based model borrows heavily from Buddhist influences and builds on traditional CBT-type approaches. Thus, in addition to a focus on the therapeutic relationship, its tools include: psychoeducation, thought/emotion/body/behaviour monitoring, structured formulations, functional analyses, understanding safety strategies, exposure, and behaviour experiments, all typical of CBT. Most notably, CFT also involves the development of a capacity for compassionate-self correction, as opposed to shame-based self-criticism via chair work, emotional tolerance training, including understanding complex emotions and developing coping skills for dealing with conflicts, mentalizing capacities, mindfulness, expressive letter writing, and forgiveness (Gilbert, 2010). Thus CFT is said to be "doing CBT and emotion work with a compassion (kindness) focus" (Gilbert, 2010, p. 7). Importantly, CFT, compassionate-mind training, and self-compassion are all designed to teach individuals skills for activating their own neurobiological attachment-systems to enhance self-soothing capacities.

One recent study evaluated the impact of including compassion focused therapy in a standard CBT group-based outpatient programme for individuals with EDs and found promising results with regard to BN (73% recovered, 4% improved), but less favourable results for AN (21% recovered, 37% improved) and eating disorders not otherwise specified

(30% recovered and 30% improved) (Gale, Gilbert, Read, & Goss, 2014). More generally, group compassionate-mind training has also been found to reduce depression, shame, self-criticism, and feelings of inferiority and increase participant's capacity to self-sooth and cultivate feelings of warmth and reassurance for self in a small uncontrolled trial of individuals with chronic mental health concerns (Gilbert & Procter, 2006).

However, research on the efficacy of compassion-focused interventions is in its infancy and evaluation of compassion-focused therapy for EDs (CFT-E), specifically, in RCTs is needed. Despite this gap in treatment outcome research, empirical evidence of the apparent importance of self-compassion in moderating ED pathology and risk factors for EDs is mounting. For instance, self-compassion has been found to be negatively associated with body shame and disordered eating in a diary study of college women (Breines et al., 2013) and self-kindness has been found to be positively associated with body-esteem and negatively associated with ED pathology (Geller et al., 2015). Self-compassion has also been negatively associated with shame memories and to moderate the impact of shame memories on ED severity in inpatients diagnosed with EDs (Ferreira et al., 2014). Evidence that body acceptance mediates the relationship between self-compassion and intuitive eating in a cross-sectional study of undergraduate women (Schoenefeld & Webb, 2013) suggests that one of the processes by which self-compassion may alleviate ED pathology is via a reduction in body shame and enhancement in body acceptance.

In a similar vain to CFT's aim of reducing self-criticism, albeit in a less holistic fashion, findings from the current research also suggest that treatments specifically targeting perfectionism among women with ED pathology may be beneficial (Lloyd et al., 2014). In this regard, it might be noted that the use of expressive writing has been suggested as a tool for reducing levels of self-criticism in university students (Troop, Chilcot, Hutchings, & Varnaite, 2013).

#### **Mindfulness-Based Therapies**

In highlighting the contribution of maladaptive emotion inhibition beliefs, and other maladaptive emotion-regulation strategies, to ED pathology, the current findings support the potential utility of interventions specifically targeting avoidance of emotions. This includes mindfulness-based interventions. As the direct experience of emotion, rather than its avoidance via suppression or rumination, is more psychologically adaptive (Watkins, 2004, 2008) and use of expressive suppression has also been linked to lower levels of social support and closeness with others (Srivastava et al., 2009).

Empirically, mindfulness has been found to moderate the relationship between global distress and ED pathology, such that global distress severity predicted increased ED pathology in undergraduate students who were low in mindfulness, but not those high in mindfulness (Geller et al., 2015). Mindfulness-based cognitive therapy (MBCT) has been found to reduce body image concerns, food cravings, dichotomous thinking, and emotional eating in a community based sample of women with disordered eating (Alberts et al., 2012). Mindfulness-based eating awareness training (MB-EAT) has been found to reduce the number of binge eating days, size of binges, binge eating scale scores, depressive symptoms, and enhance self-esteem compared to waitlist in a randomised controlled trial of overweight and obese individuals, two thirds of whom met criteria for binge eating disorder (BED) (Kristeller et al., 2013). Mindfulness-action based cognitive behaviour therapy (MACBT) has also been found to reduce objective binge eating episodes, ED attitudes, depressive symptoms, and severity of drug and alcohol abuse in individuals with comorbid BED and substance use disorders (Courbasson et al., 2011). Further, teaching experiential processing-based selfawareness has been associated with self and body-acceptance in a group of women with AN (Rawal et al., 2009). Mindfulness training has also been found to reduce rumination and improve negative mood in people with mood disorders (Broderick, 2005; Ramel et al., 2004).

There is also evidence that naturally occurring trait mindfulness features of awareness, non-reactivity, and non-judgment are uniquely inversely associated with ED pathology, over and above depression and anxiety (Lavender et al., 2011). Hence these facets of mindfulness may represent intrinsic protective factors against ED pathology, but may also be skills that can be taught/enhanced therapeutically, as suggested by evidence on the efficacy of mindfulness-based interventions discussed above (and see also the discussion below of dialectical behaviour therapy and acceptance and commitment therapies, which incorporate core mindfulness skills) and also self-compassion training for enhancing non-judgmental awareness, in the treatment of ED pathology. Importantly, there is promising preliminary evidence that mindfulness-based interventions may also be useful in the *prevention* of ED pathology in young women (Atkinson & Wade, 2016) and adolescent girls (Atkinson & Wade, 2015).

#### **Adaptive Emotion-Regulation and Interpersonal Skills Interventions**

While mindfulness training is promising, treatments that focus more specifically on reducing emotion inhibition or avoidance of painful emotions and beliefs about emotions may also be helpful in reducing ED pathology. These approaches include cognitive-emotional-behaviour therapy for EDs (Corstorphine, 2006), integrative cognitive-affective therapy for BN (Wonderlich et al., 2010), and emotion acceptance behaviour therapy for AN (EABT; Wildes et al., 2014). Dialectical behaviour therapy (DBT) for BN and BED (Safer et al., 2009), and acceptance and commitment therapy (ACT) for EDs (Sandoz et al., 2010) also include emotion regulation skill development that goes beyond mindfulness training. As is the case for the approaches considered above, however, evidence for the utility of these approaches in the clinical management of ED pathology is largely confined to uncontrolled trials at present.

Finally, given current findings highlighting the contribution of attachment-anxiety and attachment-avoidance in directly and indirectly contributing to variance in ED pathology, treatments that specifically address underlying issues with attachment insecurity and the emotional and interpersonal styles associated with these may be beneficial. Examples of such treatments include, interpersonal psychotherapy for BN and BED (Tanofsky-Kraff & Wilfley, 2010), interpersonal psychotherapy for EDs (IPT-ED) (Rieger et al., 2010), and emotion-focused therapy for EDs (Dolhanty & Greenberg, 2007; Ivanova & Watson, 2014), which may warrant further consideration. More generally, the findings of the current research also highlight the importance of programs aimed at prevention of negative body image and eating disorders (for a review see Levine & Smolak, 2009) and also the need for interventions aimed at reducing self-objectification, for instance, via enhancing experiences of embodiment and positive body image (Menzel & Levine, 2011).

# RESEARCH STRENGTHS, LIMITATIONS, AND FUTURE DIRECTIONS

# Strengths

The most notable strength of the current research lies in its attempt to draw together two broad theoretical accounts of the development and maintenance of ED pathology, namely emotion-regulation theories and self-regulation theories, and to integrate these within the overarching theoretical framework of attachment theory. Having adopted this approach, the research then sought to consider a number of closely related and frequently overlapping constructs in an attempt to tease out their unique and shared contributions to variance in ED pathology within the proposed, integrated self/emotion-regulation framework.

Broad constructs such as emotion inhibition and low self-worth were considered in more nuanced ways by incorporating various conceptually inter-related definitions and operationalisations of these constructs and considering the relative importance of each variable when considered in unison. Further, the research replicated findings obtained using

different operationalisations of these separate constructs across the various studies in the current thesis. For instance, various conceptualisations of emotion inhibition were considered together in Study 1 and the importance of the two highly conceptually related constructs of negative beliefs about emotion and emotional inhibition schemas were demonstrated across Studies 1, 2 and 4. Likewise, the relative importance of low self-esteem and internalised shame were considered in Study 3A and the importance of the two highly conceptually related variables of internalised shame and defectiveness/shame schemas were shown in Studies 3B and 4.

Furthermore, ED pathology was also considered in a more complex and nuanced fashion across Studies 2, 3B, and 4 by operationalizing ED pathology using two measures, the Eating Attitudes Test (EAT-26; Garner et al., 1982) and the Eating Disorder Diagnostic Scale Symptom Composite score (EDDS-SC; Stice et al., 2000), to yield a latent overarching construct of ED pathology with greater accuracy and reliability of measurement. It is also a strength that both adaptive and maladaptive emotion-regulation strategies were considered concurrently in Study 2.

The recruitment of two relatively large samples of participants from a number of sources for each of the surveys was also a strength. This ensured variance on each of the key study variables. The strategy of combining studies into two broader surveys (the second with two parts) facilitated the recruitment of large enough samples to allow for the application of relatively sophisticated analytic techniques (i.e., SEM) and enabled consideration of subgroups of women with and without probable ED diagnoses separately in both Studies 2 and 4. Use of women recruited cross two samples also allowed for the follow up investigation of some findings from the first phase of research in the second phase of the research (e.g., from Study 1 to Study 2 and across Study 3A and Study 3B). Recruiting women from a broad number of sources also served to enhance the generalisability of the findings.

#### Limitations

There were also, however, substantial limitations. The use of cross-sectional designs for all studies is a major limitation. The thesis was interested in exploring some of the processes by which several psychosocial indicators impact ED pathology, both directly and indirectly. However, such mediational processes are, by definition, causal and directional in nature and thus cannot, strictly speaking, be reliably explored in cross-sectional data. Whilst longitudinal designs are necessary in order to obtain evidence as to direction of effects, such research is extremely challenging in relation to the variables of interest in this study and difficult to achieve in the timeframe of a doctoral project. Further, it has been suggested that where a theoretical foundation exists to support the assumptions of direction of effect, it may be appropriate to test mediation analyses with cross-sectional data (A. F Hayes, 2013). One of the strengths of the current research lies in the strong theoretical developmental foundation underlying the proposed overarching self/emotion-reregulation model of ED pathology based in attachment theory. There is also strong theoretical support, from numerous emotionregulation and self-regulation-based theories of EDs, provided to support each of the structural models tested in the current research. As such the findings of the current research, whilst requiring replication in both longitudinal and experimental designs, may provide preliminary evidence in support of the proposed models and structural associations tested herein.

A second notable limitation of the current research is the reliance on self-report measures for all study variables. While the use of online surveys are an efficient means of data collection that allows for high levels of participant privacy and anonymity, potential problems with the use of self-report measures, in general, exist, such as the limits of self-reflective capacities, willingness to disclose, and memory or other biases. Further, problems with the use of self-report measures of ED symptoms in particular are well recognised: for

instance, individuals experiencing ED pathology have been found to frequently minimise certain ED symptoms (e.g., dietary restriction, particularly in individuals with AN); over or under estimation and reporting of body weight and height; and significant differences found between self-report measures and interview measures of ED pathology (House et al., 2008). It should also be noted that classification of participants into probable-ED and no probable-ED subgroups relied on a measure of ED pathology (the EDDS) designed to generate operational diagnoses according to DSM-IV criteria and validated for this purpose. The adapted algorithm employed in the current study, which permitted the generation of probable DSM-5 ED diagnoses, has not yet been validated. It should be noted that comparing model fit across non-ED and probable-ED subgroups was not a primary aim of the current research. Nevertheless, the current findings may provide preliminary evidence suggestive of processes applying in subgroups of women with and without diagnosed EDs.

Another limitation concerns the recruitment methods, which are susceptible to selection bias, as this may detract from the generalisability of the findings. For example, it is possible that emotionally avoidant and insecurely attached individuals are less likely to volunteer to participate in research aimed at assessing emotional and interpersonal experiences. Alternatively, those with an interest in or who identify with having difficulties with emotion or disordered eating may also be specifically attracted to participation in such research. Indeed, the relatively high proportion of participants with high levels of ED pathology in both samples in this research is an example of such bias, albeit arguably an asset in the current context. Further, the fact that the current research was confined to young adult women clearly limits the generalisability of findings. This step was taken because the models tested in the current research were based almost entirely on research conducted in young adult women and because the recruitment of an adequately sized sample of male participants with similarly variable levels of ED and associated pathology was deemed impractical.

Finally, whilst the current research has been anchored within a self/emotion-regulation model of ED pathology grounded in attachment theory and results provide preliminary support for such a model within young adult women, the findings do not necessarily preclude alternate theoretical conceptions of ED pathology. Nor do the current findings preclude the possibility of alternate structural models indicating differing directions of effects among the variables considered in the current research. Indeed each of the theoretical models explored herein and much of the research evidence examined in support, suggests that reciprocal relationships and feedback loops may well exist among many of the study variables included in these models.

### **Directions for Future Research**

As suggested above, given the cross-sectional nature of the current research, current findings need to be replicated in future research using longitudinal designs, in order to obtain evidence as to direction of effects. The use of experimental designs would also enable causal inferences. Furthermore, there are likely to be complex, bidirectional pathways underpinning the associations among constructs such as insecure attachment styles, defectiveness/shame schemas and feelings, emotional inhibition, unrelenting standards, body shame, negative emotionality, depressive symptoms, and ED pathology and clearly future longitudinal research exploring multiple potential pathways across time will be needed to elucidate the direction and nature of such relationships.

A novel way in which experimental studies could be used to test some of the associations in the current research might be to make use of security priming or paradigms that introduce a stressor to activate the attachment system. Such experiments could measure state-based attachment feelings, perhaps via the State Adult Attachment Measure (SAAM; Gillath, Hart, Noftle, & Stockdale, 2009), and assess the impact of primes/stressors. Further, the Secure Base Scoring System (SBSS) (Crowell et al., 2002) could be used to measure

attachment behaviours in response to such primes/stressors and the use of various ER strategies, emotional experience, body shame, mood, and/or ED pathology could be measured following such primes. Such experiments would be useful in assessing the causal relationships among adult attachment representations and the potential mediating variables and/or ED pathology.

Future research would also be well served by investigating alternate models testing different directions of effects among the current study variables, in order to provide further support for the models presented here and to rule out alternate explanations and directions of effects. For instance, the bidirectional relationship suggested in Corstorphine's (2006) model between ED pathology and negative emotion via a feedback loop, or the bidirectional relationships suggested among ED behaviours and mood intolerance and perfectionism suggested in the schematic representation of the transdiagnostic maintenance model of EDs (Fairburn et al., 2003) could be tested.

Additionally, use of alternate methods, such as ecological momentary assessments (EMA), in future research would be beneficial to track state-based experience of attachment behaviour, emotional experience, use of ER strategies, depressive symptoms, body shame, and/or experience of ED pathology (both ED cognitions and ED behaviours). Such methods offer the benefit of limiting recall biases and maximising ecological validity, and may provide insight into the direction of associations among such variables across time. Of particular interest could be the use of increasingly innovative smart phone applications that might allow increasingly convenient options for data collection within EMS-based research.

Proliferating models of EDs within the literature have been argued to pose a challenge to future research with regard to deciding which models are worthy of further research via longitudinal analysis of changes in causal and maintaining factors across time and which treatments should be investigated (Pennesi & Wade, 2016). However, the self/emotion-

regulation model proposed in the current thesis and the findings of the current research seeks to support the integration of several theories and models of EDs and embed them in an overarching theoretical background. Use of such an integrated model seeks to reduce the number of extant models and inform integration of several promising therapeutic options in order to augment CBT and current treatment efficacy for individuals experiencing ED pathology. The current findings thus suggest that future research should be directed at exploring treatment outcomes for the use of schema-focused therapy for EDs (Waller et al., 2007), self-compassion-focused therapy for EDs (Goss & Allen, 2012), emotion-focused therapy for EDs (Dolhanty & Greenberg, 2007; Ivanov & Watson, 2014), and mindfulness-based approaches for EDs, in particular.

Additionally, whilst it has been identified as a strength that recruitment methods used for the second phase of the current research (studies 2 and 4) allowed for the identification of subgroups of women with and without probable-ED diagnoses, future research needs to replicate these findings in clinical samples of women with diagnosed EDs. Furthermore, the use of interview data to measure study constructs would be of interest in future research, for example, use of the Eating Disorder Examination (EDE-17.0D; Fairburn et al., 2014) for ED pathology, the Adult Attachment Interview (Kaplan & Main, 1985) or a relationship interview technique, such as the CRI and the SBSS (Crowell & Owens, 1996; Crowell et al., 2002), for adult attachment representations, and the Emotion Regulation Interview (Werner et al., 2011) for ER strategies.

Finally, recruitment of participants from a number of sources has been cited as a strength, however, generalisability of the current findings is limited to adult women. Given that the current research was designed to extend research previously conducted in this demographic, it was reasonable to focus solely on adult women. However, future research will need to consider how best to replicate and extend the current findings in other

demographic subgroups, namely, adolescent girls, older females, and males of different ages and thereby test the viability of the proposed theoretical framework in these different subgroups. While research of this kind has traditionally proved challenging, given the relatively low prevalence of EDs in males in particular, findings from recent epidemiological studies, including research addressing muscularity-oriented ED attitudes and behaviours, suggest that research of this kind may be increasingly feasible and of interest (Mitchison, Hay, Slewa-Younan, & Mond, 2014; Mitchison & Mond, 2015).

#### GENERAL CONCLUSIONS

Within the limits of a cross-sectional design, the current findings support a broad self/emotion-regulation model of EDs, which incorporates both emotion-regulation and selfregulation models of ED pathology and is grounded in attachment theory. Specifically, in support of self-regulation models of ED pathology, the findings suggest that negative feelings about the self (low self esteem, internalised shame, and shame schemas) are externalised (via increased self-objectification and body surveillance) and displaced onto the body (increasing body shame) and in this way may give rise to depressive symptoms and ED pathology. Findings also support emotion-regulation models of ED pathology and the view that various forms of emotion inhibition, negative beliefs about emotion, in particular, and other maladaptive emotion-regulation strategies may serve to increase the experience of negative emotion, which in turn may trigger ED pathology as a means of alleviating this experience. Finally, findings suggest that within a broad self/emotion-regulation model insecure attachment dynamics contribute to negative feelings about the self (defectiveness/shame), emotional inhibition, and unrealistically high standards, which are externalised onto the body (in the form of body shame), and thereby contribute to depressive symptoms and, in turn, increased ED pathology.

Such evidence highlights the need for therapeutic interventions for ED pathology to also address underlying difficulties with insecure attachment, feelings of low self-worth, difficulties with emotion regulation and other maladaptive schemas in order to enhance the efficacy of current treatments and improve therapy outcomes for women with EDs.

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# Appendices

## Appendix A

## Eating Disorder Diagnostic Scale (EDDS)\* Scoring Algorithm Adapted for DSM-5

If edds5=0 and edds6=0 edds9=0. If edds5=0 and edds6=0 edds10=0.

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If edds5=0 and edds6=0 edds11=0.
If edds5=0 and edds6=0 edds12=0.
If edds5=0 and edds6=0 edds13=0.
If edds5=0 and edds6=0 edds14=0.
Compute eddsBMI=(Edds19 / 2.205) / ((Edds20 / 39.37) ** 2).
If edds19 < 50 eddsBMI=999.
If edds20 < 40 eddsBMI=999.
RECODE eddsBMI (999=SYSMIS).
Compute feature=sum(edds9, edds10, edds11, edds12, edds13).
Compute bedbing=999.
If edds5>=0 bedbing=0.
If edds5=1 and edds6=1 and edds8 >=1 and feature >=3 and edds14=1 bedbing=1.
Compute bulbing=999.
If edds5>=0 bulbing=0.
If edds5=1 and edds6=1 and edds8 \ge 1 bulbing=1.
RECODE bedbing bulbing (999=SYSMIS).
Compute compsum=999.
Compute compsum=sum(edds15, edds16, edds17, edds18).
RECODE compsum (999=SYSMIS).
Compute wtshap=999.
If edds3 < 7 or edds4 < 7 wtshap=0.
If edds3 \ge 3 or edds4 \ge 3 wtshap=1.
If edds3 >4 or edds4 >4 wtshap=2.
Compute EDDS dsm5=999.
If edds1 < 8 EDDS dsm5=0.
If bulbing =1 and compsum \geq=1 and wtshap \geq=1 EDDS dsm5=7.
If eddsBMI<18.5 and (edds2 >=3 or compsum >=1) and (edds1 >=3 or wtshap >=1) EDDS dsm5=6.
If bulbing=0 and edds17 \ge 1 and wtshap = 2 EDDS dsm5=5.
If bulbing =0 and (edds15>=1 or edds16>=1) and wtshap =2 EDDS dsm5=4.
If bedbing =1 and compsum=0 EDDS dsm5=3.
If bulbing =1 and compsum >=1 and edds3 >4 and edds4 >4 EDDS dsm5=2.
If eddsBMI< 18.5 and (edds2 >4 or compsum>=1) and (edds1>4 or wtshap=2) EDDS dsm5=1.
RECODE EDDS dsm5 (999=SYSMIS).
EXECUTE.
Note. Where EDDS dsm5 category 1 = AN, 2 = BN, 3 = BED, 4 = Purging disorder, 5 = Restrained eating
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disorder, 6 = sub-threshold AN, and 7 = sub-threshold BN.

\* Stice, E., Telch, Christy F., & Rizvi, Shireen L. (2000). Development and validation of the Eating Disorder Diagnostic Scale: A brief self-report measure of anorexia, bulimia, and binge-eating disorder. *Psychological Assessment*, *12*, 123-131. doi: 10.1037//1040-3590.12.2.123

### Appendix B

From: "Ethics Secretariat" <ethics.secretariat@mq.edu.au>

Subject: Approved- Ethics application- McIlwain (Ref No: 5201200615)

Date: 15 October 2012 4:21:42 PM AEDT

To: "Dr Doris Mctlwain" <doris.mcilwain@mq.edu.au>

Cc: "Ms Kim Woodward" <kim.woodward@students.mq.edu.au>

#### Dear Dr McIlwain

Re: "Development and validation of the Differential Anorexia and Bulimia Scale (DABS): A self-report measure of differential anorexic and bulimic characteristics in eating disturbances" (Ethics Ref: 5201200615)

Thank you for your recent correspondence. Your response has addressed the issues raised by the Human Research Ethics Committee and you may now commence your research.

This research meets the requirements of the National Statement on Ethical Conduct in Human Research (2007). The National Statement is available at the following web site:

http://www.nhmrc.gov.au/\_files\_nhmrc/publications/attachments/e72.pdf.

The following personnel are authorised to conduct this research:

Dr Doris Mellwain Ms Kim Woodward

NB. STUDENTS: IT IS YOUR RESPONSIBILITY TO KEEP A COPY OF THIS APPROVAL EMAIL TO SUBMIT WITH YOUR THESIS.

Please note the following standard requirements of approval:

- The approval of this project is conditional upon your continuing compliance with the National Statement on Ethical Conduct in Human Research (2007).
- Approval will be for a period of five (5) years subject to the provision of annual reports.

Progress Report 1 Due: 15 October 2013 Progress Report 2 Due: 15 October 2014 Progress Report 3 Due: 15 October 2015 Progress Report 4 Due: 15 October 2016 Final Report Due: 15 October 2017

NB. If you complete the work earlier than you had planned you must submit a Final Report as soon as the work is completed. If the project has been discontinued or not commenced for any reason, you are also required to submit a Final Report for the project.

Progress reports and Final Reports are available at the following website:

http://www.research.mq.edu.au/for/researchers/how\_to\_obtain\_ethics\_approval/human\_research\_ethics/forms

- 3. If the project has run for more than five (5) years you cannot renew approval for the project. You will need to complete and submit a Final Report and submit a new application for the project. (The five year limit on renewal of approvals allows the Committee to fully re-review research in an environment where legislation, guidelines and requirements are continually changing, for example, new child protection and privacy laws).
- 4. All amendments to the project must be reviewed and approved by the Committee before implementation. Please complete and submit a Request for Amendment Form available at the following website:

http://www.research.mq.edu.au/for/researchers/how\_to\_obtain\_ethics\_approval/human\_research\_ethics/forms

Please notify the Committee immediately in the event of any adverse effects on participants or of any unforeseen events that affect the continued ethical acceptability of the project.  At all times you are responsible for the ethical conduct of your research in accordance with the guidelines established by the University. This information is available at the following websites:

http://www.mq.edu.au/policy/

http://www.research.mq.edu.au/for/researchers/how\_to\_obtain\_ethics\_approval/human\_research\_ethics/policy

If you will be applying for or have applied for internal or external funding for the above project it is your responsibility to provide the Macquarie University's Research Grants Management Assistant with a copy of this email as soon as possible. Internal and External funding agencies will not be informed that you have final approval for your project and funds will not be released until the Research Grants Management Assistant has received a copy of this email.

Please retain a copy of this email as this is your official notification of final ethics approval.

Yours sincerely Dr Karolyn White Director of Research Ethics Chair, Human Research Ethics Committee

### Appendix C

From: "Ethics Secretariat" <ethics.secretariat@mq.edu.au>

Subject: Final Approval- Ethics application reference-5201100185

Date: 5 April 2011 11:14:42 AM AEST

To: "Dr Doris McIlwain" <doris.mcIlwain@mq.edu.au>

Cc: "Ms Kim Woodward" <kim.woodward@students.mg.edu.au>

#### Dear Dr McIlwain

Re: "Emotional experience and eating attitudes: The role of attachment style and shame" (Ethics Ref: 5201100185)

The above application was reviewed by the Human Research Ethics Committee at its meeting on 25/03/11. Final Approval of the above application is granted, effective 05 April 2011, and you may now commence your research.

The following personnel are authorised to conduct this research:

Dr Dorts McIlwain- Chief Investigator/Supervisor Ms Kim Woodward- Co-Investigator

NB. STUDENTS: IT IS YOUR RESPONSIBILITY TO KEEP A COPY OF THIS APPROVAL EMAIL TO SUBMIT WITH YOUR THESIS.

Please note the following standard requirements of approval:

- The approval of this project is conditional upon your continuing compilance with the National Statement on Ethical Conduct in Human Research (2007).
- Approval will be for a period of five (5) years subject to the provision of annual reports. Your first progress report is due on 05 April 2012.

If you complete the work earlier than you had planned you must submit a Final Report as soon as the work is completed. If the project has been discontinued or not commenced for any reason, you are also required to submit a Final Report for the project.

Progress reports and Final Reports are available at the following website:

http://www.research.mq.edu.au/for/researchers/how\_to\_obtain\_ethics\_approval/ human\_research\_ethics/forms

- 3. If the project has run for more than five (5) years you cannot renew approval for the project. You will need to complete and submit a Final Report and submit a new application for the project. (The five year limit on renewal of approvals allows the Committee to fully re-review research in an environment where legislation, guidelines and requirements are continually changing, for example, new child protection and privacy laws).
- 4. All amendments to the project must be reviewed and approved by the Committee before implementation. Please complete and submit a Request for Amendment Form available at the following website:

http://www.research.mq.edu.au/for/researchers/how\_to\_obtain\_ethics\_approval/ human\_research\_ethics/forms

Please notify the Committee immediately in the event of any adverse effects on participants or of any unforeseen events that affect the continued ethical acceptability of the project.  At all times you are responsible for the ethical conduct of your research in accordance with the guidelines established by the University. This information is available at the following websites:

http://www.mq.edu.au/policy/

http://www.research.mq.edu.au/for/researchers/how\_to\_obtain\_ethics\_approval/ human\_research\_ethics/policy

If you will be applying for or have applied for internal or external funding for the above project it is your responsibility to provide the Macquarie University's Research Grants Management Assistant with a copy of this email as soon as possible. Internal and External funding agencies will not be informed that you have final approval for your project and funds will not be released until the Research Grants Management Assistant has received a copy of this email.

If you need to provide a hard copy letter of Final Approval to an external organisation as evidence that you have Final Approval, please do not hesitate to contact the Ethics Secretariat at the address below.

Please retain a copy of this email as this is your official notification of final ethics approval.

Yours sincerely Dr Karolyn White Director of Research Ethics Chair, Human Research Ethics Committee