Stress, affect systems and eating pathology in problematic weight regulation

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Some ladies smoke too much and some ladies drink too much and some ladies pray too much,

But all ladies think that they weigh too much.

Ogden Nash

"Curl up and diet"

The New Yorker, 1935

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Abstract

Problematic weight regulation as found in obesity and Anorexia Nervosa (AN) are chronic conditions which require long-term management. In order to develop long-term strategies to manage these conditions, a clearer understanding of the factors that can contribute to the development and also recovery from these conditions are a necessity. Although obesity and AN are at the opposite end of the bodyweight spectrum, some shared psychological processes may drive these states.

One factor that has been suggested to contribute to problematic weight regulation is psychosocial stress whilst positive affect systems and affect regulation processes are important for regulating stress-related experiences. Gilbert (2005) describes an affect regulation system which consists of two positive affect systems known as social rank and attachment. Whilst the latter affect system refers to the attachment bond that develops between an infant and its caregiver (which extends to adult relationships), the former is used to form relationships that allow us to compete for limited resources and maintain our status in the social environment. Affect regulation processes in the current research are self-criticism and self-reassurance. Whilst self-critical thoughts and feelings can be triggered by perceptions of being low rank, the idea that people can be self-reassuring or being able to self-reassure at times of difficulty is nested in the positive infant-caregiver attachment bond and a consequence of internalizing parental soothing (Gilbert, 2006). Hence, as stress is suggested to be an important factor in problematic weight regulation and affect systems and processes are central to the regulation of emotional responses to stress-related experiences, the current series of studies examined these factors in relation to problematic weight regulation.

The current research consisted of four studies designed to examine the role of stress and affect regulation in relation to weight change, weight regain following weight loss and recovery versus symptom maintenance in AN in women. A longitudinal study (Study One) was conducted to examine the change trajectories of stress, eating pathology and bodyweight, how these changes influence each other and the role of affect regulation systems and processes on these changes in a

community based sample (*N* = 1157). Study Two examined the role of stress and affect regulation as predictors of weight regain in those who have lost weight (*N* = 42) and Study Three used a measure of life events and difficulties to investigate the role of stressful life changes and affect systems on recovery and relapse following AN (*N* = 30). Finally, in Study four, an expressive writing task which has been demonstrated to have a positive impact on stress-related health outcomes was used to explore the role of stress, affect systems and processes on problematic weight regulation and eating at times of stress (*N* = 57).

The findings of the research studies demonstrated that there is a concurrent link between stress and the regulation of bodyweight and eating in a community-based sample of women. However, the proposed relationship between stress, bodyweight and eating behaviours was not confirmed when examined longitudinally in a community-based sample, over a 7-month period in women who have lost weight or when examined retrospectively as contributing to symptom maintenance in women with AN. However, the main finding of the current series of studies suggested that affect systems and affect regulation processes do have important implications for regulating stress-related experiences, bodyweight and eating behaviours. Perceived low social status, greater insecurity of attachment, more self-critical and less self-reassuring thoughts and feelings were related to increases in stress levels, higher bodyweight and higher levels of dysfunctional eating patterns. In addition, whilst expressive writing did not reduce stress, influence bodyweight or improve affect regulation at times of difficulty, writing about positive experiences had a positive impact on reducing dietary restraint behaviours during a stressful period.

In conclusion, these findings suggest that it may not be stress per se that contributes to unhealthy changes in bodyweight and eating behaviours but how we use our affect systems and processes to manage our emotions at times of difficulty. Consequently, these findings have important implications for practice as weight loss programmes, Eating Disorder prevention programmes and stress management interventions should address the issues of perceived low social status, self-criticism and attachment insecurities.

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Abbreviations

AIC	Akaike's Information Criterion
AN	Anorexia Nervosa
AUC	Area Under the Curve
BED	Binge Eating Disorder
BIC	Bayesian Information Criterion
BMI	Body Mass Index (kg/m^2)
BN	Bulimia Nervosa
BOS	Bristol Online Survey
BT	Behavioural Therapy
CBT	Cognitive Behavioural Therapy
CBT-E	Cognitive Behavioural Therapy-Enhanced
CFA	Confirmatory Factor Analysis
CFI	Comparative Fit Index
CFT	Compassion Focused Therapy
DBS	Dominance Behavioural System
DE	Disordered Eating
DF	Degrees of Freedom
DSM-IV-TR	Diagnostic Statistical Manual of mental disorders IV Text Revision
DSM-V	Diagnostic Statistical Manual of mental disorders V
EDNOS	Eating Disorders Not Otherwise Specified
ED(s)	Eating Disorder(s)
EFA	Exploratory Factor Analysis
FSCRS	Forms of Self-Criticism/Reassurance Scale
IPE(s)	Intensely Positive Experience(s)
IPT	Interpersonal Psychotherapy
LCD	Low Calorie Diet
LEDS	Life Events and Difficulties Schedule
LGCM	Latent Growth Curve Model
MANTRA	Maudsley Model for Treatment of Adults with AN

PSS-4	Perceived Stress Scale-4
RAB	Ritual Agonistic Behaviours
RHP	Resource Holding Power (or Potential)
RMR	Resting Metabolic Rate
RMSEA	Root Mean Square Error of the Approximation
SAHP	Social Attention Holding Power
SCRS	Social Comparison Rating Scale
SDHS	Short Depression-Happiness Scale
SEM	Structural Equation Modeling
SIP	Social Investment Potential
SSCM	Specialist Supportive Clinical Management
Time points	T1 = Time 1; T2 = Time 2; T3 = Time 3; T4 = Time 4
TLI	Tucker Lewis Index
VASQ	Vulnerable Attachment Style Questionnaire
VLCD	Very Low Calorie Diet
WHO	World Health Organization

Thesis overview

The current research programme sought to identify how stress, affect regulation systems (social rank and attachment) and affect regulatory processes (self-criticism/reassurance) can contribute to problematic weight regulation, including weight regain following weight loss and relapse from Anorexia Nervosa. The thesis is divided into nine chapters beginning with an introductory chapter into problematic weight regulation followed by a systematic and narrative review, a methodology chapter, four empirical chapters and an overall discussion.

The introductory chapter provides an overview into problematic weight regulation with a focus on obesity and Eating Disorders. This will be followed by two systematic reviews which provide an appraisal of the literature that has examined the role of stress on weight regain following weight loss and eating pathology. The third chapter is a narrative review of research examining affect regulation systems such as social rank and insecurity of attachment in adulthood and affect regulatory processes such as self-criticism/reassurance and how they influence perceptions of stress and hence problematic weight regulation.

Chapter four describes the general methodology that was adopted to conduct the empirical studies in this research programme. However, each study chapter will also include a methodology section detailing the measures and procedures used. It must also be noted that the methodology chapter will include a description of the development and validation of two self-report measures (Forms of Self-Criticism/Reassuring Scale [FSCRS; Gilbert, Clarke, Hempel, Miles & Irons, 2004] and Vulnerable Attachment Style Questionnaire [VASQ; Bifulco, Mahon, Kwon, Moran & Jacobs, 2003]) used in this research programme. In addition, the validation and development of the FSCRS (Gilbert et al., 2004) has been published in the *British Journal of Clinical Psychology* (Kupeli, Chilcot, Schmidt, Campbell & Troop, 2013a) and the VASQ (Bifulco et al., 2003) is in press for the *Journal of Psychopathology and Behavioral Assessment* (Kupeli, Norton, Chilcot, Schmidt, Campbell & Troop, 2013d).

Chapters five through to eight are empirical chapters which present four studies that examined trajectories of bodyweight, stress and eating pathology,

factors predicting weight regain, the role of life events and affect systems on recovery from AN and the impact of an emotionally expressive writing intervention on changes in stress, bodyweight and eating behaviours. In addition, Study One (Chapter Five) has been prepared for publication in a peer-review journal (Kupeli, Norton, Chilcot, Campbell, Schmidt & Troop, 2013c).

The final chapter is a general discussion of the overall findings of the four studies and how these findings fit in with our existing knowledge. This chapter will also include a research agenda for future research on how these findings can be taken forward to improve our knowledge of the processes of problematic weight regulation.

Chapter 1: Problematic weight regulation from two ends of a spectrum

Introduction

This chapter will provide a brief introduction to problematic weight regulation, i.e., as can be found in obesity and EDs. Although a comprehensive review is beyond the scope of this chapter, here is a brief overview of the prevalence, causes and treatments of obesity and EDs, whilst focusing on two types of problematic weight regulation; failure to maintain successful weight loss following obesity versus failure to maintain successful weight gain following treatment for AN. It must also be highlighted that obesity, while at the opposite end of the weight regulation spectrum to AN, is not a psychiatric disorder whereas AN is, as recognised by the Diagnostic Statistical Manual of mental disorders V (DSM-V; American Psychiatric Association, 2013).

The ability to regulate healthy bodyweight is determined by a combination of factors including genetic, physiological, environmental and psychological mechanisms (Bessesen, 2011; Jéquier & Tappy, 1999). The trajectory of bodyweight is not a stable course but one of a slow but gradual climb, with human adults gaining approximately one pound per year between the ages of 18 and 65 (Bessesen, 2011). However, the ability to regulate bodyweight at a healthy level is not always possible which results in problematic weight regulation. Problematic weight regulation includes excessive weight gain resulting in obesity, being unable to successfully maintain weight lost following obesity, extreme weight loss resulting in AN and being unable to maintain weight gain achieved during treatment for AN. When bodyweight is not regulated at a healthy level, individuals are most likely to fall into one of three categories; underweight, overweight or obese. Quetelet's Body Mass Index (BMI kg/m^2) is the guide often used to determine if an individual falls into one of the categories of being underweight, normal weight, overweight and obese. BMI is calculated by dividing weight in kilograms by the height in metres squared (kg/m^2). BMI for male and female adults are commonly interpreted using the

following categories (*see Table 1.1*), as devised by the World Health Organization (WHO, 2004):

Table 1.1: Classification of adult BMI categories (WHO, 2004)

BMI	Category
Below 18.49	Underweight
18.5 – 24.9	Healthy weight
25 – 29.9	Overweight
30 and above	Obese

Throughout the course of a lifespan, adults are expected to move between these categories, but most commonly adults gain weight over time (Bessesen, 2011; Vardi & Pinhas-Hamiel, 2000). Therefore, weight and weight change are not in themselves problematic. Problematic weight regulation refers to the inability to maintain a weight that is optimally healthy and one that can be detrimental to health. Those who are unable to maintain a healthy bodyweight often report periods of weight cycling which describes those who fluctuate between healthy and unhealthy weights. For example, successful weight loss which is then followed by weight regain in excess of the weight that was initially lost, resulting in a vicious cycle of weight loss and regain (O'Neil, Dietz, DiGirolamo, Kicklighter, Lahmayer, Pi-Sunyer et al., 1991). Wing (1992, 1993) concluded that weight cycling in humans may be associated with mortality but this may not be the case in animals (Reed & Hill, 1993) although many of the studies reviewed had methodological limitations. This highlights the need to investigate why individuals are unable to maintain successful weight loss following periods of obesity or maintain weight gain following a stage of being underweight.

Obesity

Obesity is characterized by greater caloric intake combined with low levels of energy expenditure (Selassie & Sinha, 2011). The prevalence of obesity and disturbed eating

behaviours is on the rise (Darby, Hay, Mond, Quirk, Buttner & Kennedy, 2009), with prevalence of obesity increasing from 13% to 22% in men and from 16% to 24% in women, between 1993 and 2009 (Craig & Hirani, 2010). From an evolutionary perspective, living species may have evolved to gain weight as they age in order to compensate for age-related muscle loss and increase the likelihood of survival and longevity (Vardi & Pinhas-Hamiel, 2000). Despite reports that weight loss following obesity may be associated with an increased risk of mortality in the long run (Sørensen, Rissanen, Korkeila & Kaprio, 2005), maintenance of excess weight has deleterious effects on health and well-being. Obesity has been linked to various health conditions including type 2 diabetes, cardiovascular disease, stroke, osteoporosis, various cancers, depression and sleep disorders (Akinnusi, Saliba, Porhomayon & El-Solh, 2012; Anderson & Wadden, 1999; Kopelman, 2007) and increases the risk of morbidity and mortality (Bellocco, Jia, Ye, & Lagerros, 2010; Birmingham, Muller, Palepu, Spinelli & Anis, 1999; Calle, Thun, Petrelli, Rodriguez & Heath, 1999; Expert panel on the identification, evaluation, and treatment of overweight and obesity in adults, 1998; Manson & VanItallie, 1995). Given that obesity has negative consequences for health and well-being, it also has major costs for employers and the health service. Obesity is associated with lower productivity levels and obese individuals take more and longer sick leave compared to their healthy weight peers (Neovius, Johansson, Kark & Neovius, 2009; Robroek, van den Berg, Plat & Burdorf, 2011; Schmier, Jones & Halpern, 2006; van Duijvenbode, Hoozemans, Van Poppel & Proper, 2009). The obesity epidemic cost the National Health Service (NHS) 2.3-2.6% of the budget in 2001/2002 (McCormick & Stone, 2007) and it has been predicted that treating obesity and the consequences of obesity will cost the NHS £10 billion per year by 2050 (Butland, Jebb, Kopelman, McPherson, Thomas, Mardell et al., 2007).

Risk factors for obesity

The causes of obesity are multifaceted, including a combination of psychological, behavioural, environmental and biological factors. Some of the factors which have been found to contribute to adiposity are stress (e.g., Adam & Epel, 2007; Cizza & Rother, 2012; Delahanty, Meigs, Hayden, Williamson & Nathan,

2002; Kyrou & Tsigos, 2009; Moore & Cunningham, 2012; Wardle, Chida, Gibson, Whitaker & Steptoe, 2011), a perpetuation of bad habits (e.g., Weiss, Galuska, Khan, Gillespie & Serdula, 2007), greater levels of anxiety and low mood (e.g., Bergin, Neale, Eaves, Martin, Heath & Maes, 2012; Chiriboga, Ma, Li, Olendzki, Pagoto, Merriam et al., 2008; Needham, Epel, Adler & Kiefe, 2010; Stunkard, Faith & Allison, 2003), dietary restraint (e.g., Johnson, Pratt & Wardle, 2012) and adverse experiences such as childhood sexual abuse (Gustafson & Sarwer, 2004; Wilson, 2010). Behavioural factors that have been found to result in weight gain and obesity include sedentary lifestyle such as increased TV viewing (Colvin & Olson, 1983; Jakicic, 2011; Mozaffarian, Hao, Rimm, Willett & Hu, 2011; Pearson & Biddle, 2011; Stubbs & Lee, 2004; Wane, van Uffelen & Brown, 2010), consumption of high-calorie, energy dense foods (Ball, Brown & Crawford, 2002; Giskes, Kamphuis, van Lenthe, Kremers, Droomers & Brug, 2007; Lachat, Nago, Verstraeten, Roberfroid, Van Camp & Kolsteren, 2012; Pearson & Biddle, 2011; Stubbs & Lee, 2004; Wane et al., 2010), skipping breakfast (Horikawa, Kodama, Yachi, Heianza, Hirasawa, Ibe et al., 2011) and smoking cessation (Mozaffarian et al., 2011; Wane et al., 2010). Even having a history of attending a weight loss program, dieting and weight cycling can contribute to unhealthy weight gains (Blackburn, Wilson, Kanders, Stein, Lavin, Adler et al., 1989; Brownell, Greenwood, Stellar & Shrager, 1986; Brownell & Rodin, 1994; Coakley, Rimm, Colditz, Kawachi & Willett, 1998; Field, Aneja, Austin, Shrier, Moor & Gordon-Larsen, 2007; Field, Manson, Taylor, Willett & Colditz, 2004; French & Jeffery, 1994; French, Jeffery & Forster, 1994; French, Jeffery, Forster, McGovern, Kelder & Baxter, 1994; Korkeila, Rissanen, Kaprio, Sørensen & Koskenvuo, 1999; Kroke, Liese, Schulz, Bergmann, Klipstein-Grobusch, Hoffmann et al., 2002; Lowe, Annunziato, Markowitz, Didie, Bellace, Riddell et al., 2006; Mann, Tomiyama, Westling, Lew, Samuels & Chatman, 2007; Savage, Hoffman & Birch, 2009; Stevens, Chambless, Tyroler, Harp, Jones & Arnett, 2001; Stice, 1998; Tiggemann, 2004). Prior weight loss was found to be the strongest predictor of subsequent weight gain over and above dietary intake (Colditz, Willett, Stampfer, London, Segal & Speizer, 1990) suggesting that weight regain following weight loss is a common occurrence.

The 'obesogenic' environment which is now a common term in the obesity literature refers to environments that restrict physical activity and promote

excessive energy consumption (Ding & Gebel, 2012; Durand, Andalib, Dunton, Wolch & Pentz, 2011; Lachowycz & Jones, 2011; Lovasi, Hutson, Guerra & Neckerman, 2009; Renalds, Smith & Hale, 2010; Speakman, 2004; Wendel-Vos, Droomers, Kremers, Brug & van Lenthe, 2007). However, situational factors resulting in obesity are not restricted to limited greenspace but can also be influenced by in-store marketing that promotes unhealthy dietary behaviours (Glanz, Baderm & Iyer, 2012). Socio-demographic factors (Ball & Crawford, 2005; Giskes, Avendano & Kunst, 2010; Giskes, van Lenthe, Avendano-Pabon & Brug, 2011; Moore & Cunningham, 2012; Sobal & Stunkard, 1989) and lack of social contacts, especially for women, have been found to contribute to weight gain (Cunningham, Vaquera, Maturo, & Narayan, 2012). Biological factors such as postpartum weight retention (e.g., Gore, Brown & Smith West, 2003), curtailed sleep (Beccuti & Pannain, 2011; Cizza, Requena, Galli & de Jonge, 2011; Horne, 2011; Klingenberg, Sjödin, Holmbäck, Astrup & Chaput, 2012; Knutson, 2012; Knutson & Van Cauter, 2008; Leproult & Van Cauter, 2010; Lucassen, Rother & Cizza, 2012; Marshall, Glozier & Grunstein, 2008; Mavanji, Billington, Kotz & Teske, 2012; Mozaffarian et al., 2011; Nielsen, Danielsen, & Sørensen, 2011; Panossian & Veasey, 2012; Patel & Hu, 2008; Van Cauter & Knutson, 2008), long sleep duration (Marshall et al., 2008; Mozaffarian et al., 2011), antipsychotic drugs (Allison & Casey, 2001), viruses (Atkinson, 2008), impaired immune function (Martí, Marcos & Martínez, 2001) and genetic factors (Astrup, Hill & Rössner, 2004; Loos, 2012; Maes, Neale & Eaves, 1997; Sørensen, 1995; Speakman, 2004; Stunkard, Harris, Pedersen & McCleam, 1990) can also lead to excessive weight gain. The causal and maintaining factors of obesity are complex and interactive which makes treatment of this chronic condition difficult.

Treating obesity

Although a comprehensive review of all the weight management interventions for treating obesity is beyond the scope of this chapter, here is a brief overview of some of the weight loss methods available to obese individuals. Obesity can be managed using methods such as decreasing caloric intake with Low Calorie Diets (LCD) or Very-Low Calorie Diets (VLCD), increasing physical activity, psychosocial interventions such as Behavioural Therapy (BT; which is also commonly

referred to as lifestyle modification or behavioural weight control), pharmacological treatments (i.e., anti-obesity drugs such as orlistat) and surgical techniques such as bariatric surgery.

Weight loss programs such as adopting LCDs and VLCDs require obese individuals to restrict their calorie consumption to 800 – 1800kcal per day or to less than 800kcal per day, respectively. Weight loss programs such as Weight Watchers® and Slimming World® advocate LCDs to those who are overweight or obese but VLCDs are recommended for severely obese individuals who are required to consume a liquid diet which is supplemented with essential vitamins and minerals. A multidisciplinary team of health care professionals carefully monitor severely obese individuals who are selected for one of these intense calorie restriction diets as earlier studies using these regimens showed increased mortality (Isner, Sours, Paris, Ferrans & Roberts, 1979; Sours, Frattali, Brand, Feldman, Forbes, Swanson & Paris, 1981). Despite the early controversy of extreme calorie restricting diets, both LCDs and VLCDs result in similar long-term weight losses (Tsai & Wadden, 2006) but LCDs have been found to induce fewer adverse side effects (Rössner & Flaten, 1997). However, LCDs and VLCDs are not a miracle remedy for the obesity pandemic as dieting is related to depression (Smoller, Wadden & Stunkard, 1987; Stunkard & Rush, 1974) and most weight losers go on to regain approximately 40% to 64% of the weight they initially lost during treatment (Anderson, Hamilton, Crown-Weber, Riddlemoser & Gustafson, 1991; Lowe, Miller-Kovach & Phelan, 2001; National Task Force on the Prevention and Treatment of Obesity, 1993; Wadden, Foster, Letizia & Stunkard, 1992; Wadden, Vogt, Foster & Anderson, 1998). Successful weight loss maintenance has been defined as losing at least 10% of initial bodyweight and maintaining this loss for at least a year (Wing & Hill, 2001; Wing & Phelan, 2005). In addition, combining an LCD with other weight loss methods such as physical activity can increase successful weight loss in the short and longer term (Rippe & Hess, 1998).

Regular physical activity as a form of healthy weight regulation is recommended as a method of increasing energy expenditure. Performing vigorous levels of physical activity or exercise that expends >2500 kcal per week is a key element of short- and long-term weight loss retention (Bray, 2008; Fogelholm &

Kukkonen-Harjula, 2001; Jeffery, Wing, Sherwood & Tate, 2003; Pronk & Wing, 1994; Skender, Goodrick, Del Junco, Reeves, Darnell, Gotto et al., 1996; Tate, Jeffery, Sherwood & Wing, 2007) and is associated with improvement in mood (Wadden et al., 1998) and cardiovascular health (Wadden, Butryn & Wilson, 2007). Physical activity recommendations by the International Association of the Study of Obesity (IASO) for the previously obese and those who are overweight, advise performing 60-90 minutes and 45-60 minutes of moderate intensity exercise per day, respectively (Saris, Blair, Van Baak, Eaton, Davies, Di Pietro et al., 2003). Similar to LCDs, physical activity as a solitary weight loss approach is inadequate, as initial weight losses have been found to be slow (Jakicic & Davis, 2011; Skender et al., 1996) and commonly followed by weight regain (Wadden et al., 2007). Therefore, an approach combining both caloric intake restriction and high levels of physical activity produces far superior weight losses compared to when these methods are adopted in isolation (Jakicic & Davis, 2011; Rippe & Hess, 1998). However, it is also essential for obese individuals to develop strategies such as monitoring weight and behaviours related to weight which would facilitate and motivate them to perform behaviours associated with their dietary and exercise plans (Wadden, Sternberg, Letizia, Stunkard & Foster, 1989).

Psychosocial interventions such as BT advocate lifestyle modification which includes reducing dietary intake, increasing physical activity and developing behavioural strategies to aid weight loss. BT is delivered by an interdisciplinary team of dieticians, psychologists, exercise specialists and other related health care professionals, who educate obese individuals to use core behavioural techniques. BT sessions are designed to teach individuals to set clear, achievable dietary and physical activity goals, regularly self-monitor dietary intake and physical activity and adapt their environment to remove cues associated with problematic weight regulation behaviours to facilitate behaviour change. Other techniques include problem solving, understanding that lapses can occur and developing ways to recognise and deal with situations that precede lapses (relapse prevention) and modifying thoughts related to weight management (cognitive restructuring) (Burke & Wang, 2011; Butryn, Webb & Wadden, 2011; Jones, Wilson & Wadden, 2007; Wadden, Butryn & Byrne, 2004; Wadden et al., 2007). Cognitive restructuring is an

important component of BT as it tackles negative affect by identifying and enabling individuals to develop skills to manage the maladaptive thoughts and behaviours associated with how they control their weight (Van Dorsten & Lindley, 2011; Liao, 2000).

Long-term successful weight loss and maintenance following BT can be attributed to continued adherence to the behavioural strategies developed to adopt healthy eating and regular physical activity during treatment (Perri, 1998). Although on average patients lose approximately 8.4kg (Foreyt & Carlos Poston, 1998) or 8-10% of their initial bodyweight during treatment (Butryn et al., 2011; Wadden et al., 2007), most patients regain the weight that they lost during treatment within 3 to 5 years following treatment (Foreyt & Carlos Poston, 1998; Kramer, Jeffery, Forster & Snell, 1989; Wadden et al., 2007). An explanation for the failure to maintain successful weight loss following BT could be that obese individuals are recommended to only expend 250 kcal per week and gradually increase physical activity to burn a minimum of 1000 kcal per week with no maximum energy expenditure goal being set (Jeffery, Wing, Thorson & Burton, 1998). Therefore, following treatment, reduced-obese individuals may only perform the minimum that is recommended, when research has shown that energy expenditure should be between 1500 to 2000 kcal per week to maintain long-term weight losses (Rippe & Hess, 1998). Experts in the field have emphasised that BT programs should increase the level of physical activity that is recommended to maintain weight loss and prevent relapse (Jeffery et al., 1998; Wing & Hill, 2001). In spite of the disappointing success rates following psychosocial interventions, it has been suggested that BT which includes relapse prevention strategies and continued support and guidance following treatment can improve initial weight loss and aid successful maintenance of weight that has been lost (Perri, Shapiro, Ludwig, Twentyman & McAdoo, 1984). Additionally, a combination of BT and pharmacology has also been suggested to be effective in achieving and maintaining weight losses (Davidson, Hauptman, DiGirolamo, Foreyt, Halsted, Heber et al., 1999; Phelan & Wadden, 2002; Sarwer, von Sydow Green, Vetter & Wadden, 2009; Wadden, Berkowitz, Womble, Sarwer, Phelan, Cato et al., 2005).

Pharmacological treatments such as orlistat have been designed to reduce caloric intake by inhibiting the absorption of dietary fats in the gastrointestinal tract. Orlistat has been found to have modest effects on weight loss when combined with lifestyle modification techniques such as a low-fat diet but side effects such as oily spotting and faecal urgency and/or incontinence are commonly reported (Burke & Wang, 2011; Davidson et al., 1999; Padwal & Majumdar, 2007; Sjöström, Rissanen, Andersen, Boldrin, Golay, Koppeschaar et al., 1998). Side effects associated with orlistat are found to occur when more than 20g of fat is consumed per meal. The unpleasant side effects of orlistat are suggested to act as negative reinforcement encouraging individuals to adhere to a low-fat diet (Wadden, Brownell & Foster, 2002). Notwithstanding these adverse gastrointestinal side effects, orlistat is currently the only obesity management drug on the market and has even been found to improve cardiovascular risk factors (Zhou, Ma, Wu, Lu, Zhang, Guo et al., 2012) unlike appetite suppressants such as sibutramine and rimonabant (Gray, Cooper, Dunkley, Warren, Ara et al., 2012) which were taken off the market due to an increased risk of cardiovascular disease (European Medicines Agency (EMA), 2010; James, Caterson, Coutinho, Finer, Van Gaal, Maggioni et al., 2010) and psychiatric disorders (EMA, 2008), respectively. However, even pharmacotherapy is not a cure of adiposity as 35% to 51% of weight is regained at 2 years post-treatment (Davidson et al., 1999).

When all conventional weight loss interventions fail, morbidly obese individuals are offered weight loss surgery. Despite being a last resort weight loss measure, the number of bariatric operations performed has increased from over 146,000 operations conducted between 2002 and 2003 (Buchwald & Williams, 2004) to over 344,000 operations performed in 2008 worldwide (Buchwald & Oien, 2009). Bariatric surgery involves either gastric banding or a gastric bypass, both of which reduce the size of the stomach and are generally offered to those with a BMI of 40kg/m^2 and above or those who have a BMI of 35kg/m^2 or above and other comorbidities such as diabetes and hypertension (National Institutes of Health Consensus Development Panel, 1991). Obese individuals who undergo a gastric banding have an adjustable band fitted around the top of the stomach to create a small pouch restricting dietary intake. A gastric bypass either involves reducing the

size of the stomach by removing a portion of it or the Roux-en-Y gastric bypass which involves stapling the stomach to create a small pouch and then re-routing the small intestines to the small portion of the stomach (Kissane & Pratt, 2011). Despite the unpleasant experience and the potential complications associated with it, bariatric surgery is a highly effective form of treating obesity. It leads to rapid and significant weight losses with approximately 17% to 67% of weight initially lost following surgery (Christou, Sampalis, Liberman, Look, Auger, McLean et al., 2004; Hsu, Benotti, Dwyer, Roberts, Saltzman, Shikora et al., 1998; Lang, Hauser, Buddeberg & Klaghofer, 2002; Shah, Simha & Garg, 2006; Snyder, Nguyen, Scarbourough, Yu & Wilson, 2009) and has been found to be much more effective than non-surgical treatments (Bray, 2008; Douketis, Macie, Thabane & Williamson, 2005; Moldovan & David, 2011). Additionally, weight loss surgery has been found to improve comorbid conditions such as diabetes, hypertension and obstructive sleep apnea (Buchwald, Avidor, Braunwald, Jensen, Pories, Fahrbach et al., 2004; Christou et al., 2004; Laurino Neto, Herbella, Tauil, Silva & de Lima Jr, 2012; Maggard, Shugarman, Suttorp, Maglione, Sugarman, Livingston et al., 2005).

However, similar to other weight loss approaches like BT and pharmacotherapy, failure to maintain successful weight loss following weight loss surgery seems to be a common occurrence. Approximately 15% to 85% of the weight that is initially lost following surgery is regained (Freire, Borges, Alvarez & Correia, 2012), with weight beginning to creep up from approximately 2 years post-surgery with the amount that is regained increasing with time (Abu Dayyeh, Lautz & Thompson, 2011; Christou, Look & MacLean, 2006; Freire et al., 2012; Hsu et al., 1998; Hsu, Sullivan & Benotti, 2007; Kofman, Lent & Swencionis, 2010; Livhitsm, Mercado, Yermilov, Parikh, Dutson, Mehran, et al., 2011; Magro, Geloneze, Delfini, Pareja, Callejas & Pareja, 2008; Sarzynski, Jacobson, Rankinen, Carlsson, Sjöström, Bouchard et al., 2011). Some of the factors associated with weight regain following surgery include disinhibition, binge eating (consuming large quantities of food in a short period of time), “grazing” (consuming large quantities of food in frequent small doses), low energy expenditure, failure to adhere to lifestyle changes, lack of self-monitoring and neglecting post-surgery follow-ups (Bond, Phelan, Leahey, Hill & Wing, 2009; Freire et al., 2012; Kofman et al., 2010; Grief & Miranda, 2010; Livhitsm

et al., 2011; Odom, Zalesin, Washington, Miller, Hakmeh, Zaremba et al., 2010; Pekkarinen, Koskela, Huikuri & Mustajoki, 1994; Sarwer, Dilks & West-Smith, 2011; van Hout, Verschure, & van Heck, 2005). Therefore, to minimise the risk of weight regain following weight loss surgery, obese individuals should also be provided with another weight loss method such as a psychosocial intervention. Comparative studies of weight loss surgery and psychosocial interventions such as BT have shown that combining weight loss surgery with BT can help improve eating behaviour and aid long-term weight loss maintenance (Kalarchian & Marcus, 2003; Moldovan & David, 2011) by providing obese individuals with the skills to tackle maladaptive behaviours associated with weight regain (Kalarchian, Marcus, Courcoulas, Cheng, Levine, & Josbeno, 2012).

As it can be understood from the above, not one weight loss approach is a "cure" for obesity emphasising the need for long-term management of obesity. So, in summary:

- Short-term weight loss is often followed by a failure to maintain the weight that is lost
- Weight loss methods should not be used in isolation but where possible, combined
- Weight loss methods should be supplemented with long-term care to improve weight loss and minimise the risk of weight regain

So in spite of the number of anti-obesity treatments currently available, why is it that overweight and obese individuals are still unable to maintain the weight they have successfully lost?

Like obesity, the aetiology of weight regain is a complex interaction between individual, environmental and physiological sources, which can have deleterious effects on both psychological and physical well-being (Brownell, Marlatt, Lichtenstein & Wilson, 1986). With increasing prevalence rates of obesity, it is reasonable to suggest that behaviours associated with problematic weight regulation may also be on the rise (Brownell, 1991) as we try to control our bodyweight and shape using precarious methods. Therefore, as researchers, we

must not only attempt to help obese individuals to lose weight but we must also tackle the processes that drive the behaviours associated with problematic weight regulation in order to develop effective prevention methods. Therefore, the next section of this chapter will focus on unhealthy eating behaviours both in terms of initial weight gain as well as weight regain following weight loss and pathological eating behaviours that result in extreme weight loss.

Eating pathology

Problematic weight regulation can also result from disordered eating (DE). DE refers to a wide range of disturbed eating behaviours such as extreme fasting, disinhibition, dietary restraint, binge eating, purging and exercising excessively. These disturbed eating patterns are performed in order to control one's bodyweight and shape but have actually been found to result in greater weight gain compared to healthy weight control behaviours like physical activity (Savage & Birch, 2010). Disinhibition refers to over eating in response to dysphoric emotions or situational and environmental cues such as the availability of palatable food (Ruderman, 1986). As expected, greater disinhibition is related to obesity and weight gain over time (Hays, Bathalon, McCrory, Roubenoff, Lipman & Roberts, 2002; Savage et al., 2009; Lindroos, Lissner, Mathiassen, Karlsson, Sullivan, Bengtsson et al., 1997; Provencher, Drapeau, Tremblay, Després & Lemieux, 2003). Dietary restraint refers to the conscious effort to restrict or control food intake and the finding that restrainers gained more weight compared to non-restrainers is in line with restraint theory (Herman & Mack, 1975; Polivy & Herman, 1985, 1992). Restraint theory posits that restrainers whose dietary restraint practices are disrupted are likely to consume more than they would have done if they were not restraining (Herman & Mack, 1975; Polivy & Herman, 1985, 1992) resulting in weight regain in those who had successfully lost weight. Fluctuations in weight as a result of unhealthy eating pathology behaviours such as dietary restraint and disinhibition have been related to increased levels of cholesterol in the blood, hypertension and cardiovascular disease (Hainer, Kunesova, Bellisle, Parizkova, Braunerova, Wagenknecht et al.,

2006; Hays et al., 2002). Substantial fluctuations in weight, specifically weight gain, have been found to trigger binge eating and purging behaviours and even resulting in the onset of EDs (Thomas, Butryn, Stice & Lowe, 2011; Wardle & Beinart, 1981). Binge eating is characterized as consuming excessively large amounts of food (usually high calorie foods such as ice cream, bread, chocolate etc) in a discrete period of time (e.g., 2 hours) and experiencing a sense of lack of control over one's eating (i.e., that one cannot stop). Purging behaviours include vomiting and use of laxatives, diuretics or enemas as weight loss mechanisms. Individuals who take their disturbed eating behaviours to pathological levels may show the diagnostic features of EDs.

Firstly, it must be noted that the current research programme was conducted prior to the publication of the fifth edition of the Diagnostic Statistical Manual of mental disorders (DSM-V; APA, 2013). Therefore, the patients that were recruited for this research have been diagnosed according to the fourth edition of the DSM (DSM-IV; APA, 2000) definition. The DSM-IV (APA, 2000) criteria will be described here and changes in the DSM-V (APA, 2013) will be highlighted below. According to the DSM-IV (APA, 2000), AN is characterized as the refusal to maintain normal bodyweight for age and height by intentionally maintaining BMI at less than 17.5kg/m^2 or at a level that is less than 85% of normal bodyweight for age and height. Diagnostic criteria also include an unhealthy pre-occupation with bodyweight and shape which fuels the fear of gaining weight or becoming fat and reinforces distorted perceptions of shape and weight. A biological marker of AN in post-menarcheal women is when the individual experiences amenorrhea (lack of menstruation). There are varying combinations within AN, with some who lose weight by extreme dieting, fasting or excessive exercise (referred to as restrictive AN) and others who binge-eat and purge by vomiting or using laxatives, diuretics or enemas (referred to as the binge-eat/purge AN), however, others may binge-eat and fast or even purge without binge eating (APA, 2000). Approximately 62% of people with restrictive AN cross-over to binge eating/purging AN (Tong & D'Alessio, 2011). Estimates of the prevalence of AN vary from .3% to 2.2% (Bergh, Brodin, Lindberg & Södersten, 2002; Woodside, 1995; Fairburn & Harrison, 2003; Grave, 2011; Hoek & Van Hoeken, 2003; Kjelsås, Bjørnstrøm, & Götestam, 2004; Machado, Machado,

Gonçlaves & Hoek, 2007; Wade, Treasure & Schmidt, 2011; Zandian, Ioakimidis, Bergh, & Södersten, 2007) with 1% of the population developing the disease between the ages of 14 to 19 (Bergh et al., 2002). Higher prevalence rates of 4.3% for young women who partially meet the current stringent classification of AN have been found (Royal College of Psychiatrists, 2012).

Similar to AN, BN is also characterized by an over-valuation of bodyweight and shape which has been suggested to be the core aspect of the disorder (Fairburn, 1997). BN has been described as experiencing a sense of lack of control and consuming large quantities of food in a short period of time ("bingeing"), on average at least twice a week for at least three months. These recurrent episodes of bingeing are normally followed by inappropriate use of "compensatory" behaviours. Those who fulfill the purging subtype of BN engage in behaviours such as self-induced vomiting or take laxatives, diuretics or enemas to counteract their bingeing behaviour, while those with the non-purging subtype of BN either fast or exercise excessively to prevent weight gain (APA, 2000). Estimates of the prevalence of BN varies from .3% to 4% (Bergh et al., 2002; Woodside, 1995; Fairburn & Harrison, 2003; Hoek, & Van Hoeken, 2003; Kjelsås et al., 2004; Machado et al., 2007; Royal College of Psychiatrists, 2012) with higher prevalence rates for those who partially meet the criteria for BN (7%; Royal College of Psychiatrists, 2012).

EDs of clinical severity that do not meet the specific criteria of AN or BN are characterised as Eating Disorders Not Otherwise Specified (EDNOS; DSM-IV-TR; APA, 2000). These include individuals who meet all of the diagnostic conditions of AN but are still menstruating or those who display symptoms of the binge eating and purging subtype of BN but the behaviours have not occurred more than twice a week or for less than three months (APA, 2000). As more and more individuals are presenting clinical symptoms of DE that do not conform to the strict criteria of AN and BN as set out by the DSM IV-TR (APA, 2000), EDNOS has become the most commonly diagnosed ED (Fairburn, Cooper, Doll, O'Connor, Bohn, Hawker et al., 2009; Schmidt, 2003) with prevalence rates ranging from 2.4% to 24.1% (Kjelsås et al., 2004; Machado et al., 2007; Micali, Hagberg, Petersen & Treasure, 2013). The current criteria used to diagnose EDs as recommended by the DSM IV-TR (APA, 2000) has changed following the publication of the DSM-V (APA, 2013). Therefore,

rather than specifying that weight must be less than 85%, the criterion for AN has changed to "persistent restriction of energy intake leading to significantly low body weight" which encapsulates the behavioural characteristics of AN. In addition, amenorrhea is no longer a criteria for AN allowing the inclusion of females who continue to menstruate even when they have a significantly low bodyweight. Other changes include reducing the twice a week frequency criterion for binge eating and compensatory behaviours to once a week for BN and creating a fourth ED category to encapsulate binge eating disorder (BED). Including a fourth category will enable the diagnosis of individuals who report recurrent episodes of binge eating without the regular use of compensatory behaviours (APA, 2013).

Risk factors for eating pathology

Similar to weight gain, the aetiology of EDs is multifaceted including a combination of behavioural, psychological, environmental, socio-cultural and biological causes (Woodside, 1995; Fairburn & Harrison, 2003; Grave, 2011; Haller, 1992; Patel, Phillips & Pratt, 1998; Polivy & Herman, 2002; Stice, 2002). As there have been many factors that have been implicated in both the development and maintenance of EDs, some of the commonly documented factors are mentioned here. Behavioural causes include dieting (Nielsen, 2001) and environmental factors include problematic relationships within a family (Haller, 1992; Treasure, Sepulveda, MacDonald, Whitaker, Lopez, Zabala et al., 2008), having a parent who suffered from an ED (Hsu, 1997) and childhood sexual abuse (Jáuregui-Garrido & Jáuregui-Lobera, 2012; Wonderlich, Brewerton, Jocic, Dansky & Abbott, 1997). Socio-cultural factors include pressures to conform to inaccurate and unrealistic standards of appearance such as the internalisation of the thin ideal (Stice & Agras, 1998) which may be a result of exposure to mass media that promotes the view that thinness is attractive (Becker, Burwell, Herzog, Hamburg & Gilman, 2002; Levine & Murnen, 2009). Pro-ED websites (Talbot, 2010) promote internalisation of the thin-ideal and even make recommendations on how to maintain a low weight. Some of the psychological factors that have been found to contribute to the development of DE include low self-esteem (Otagaki, Yonezawa, Shiwa, Saito & Nakamura, 2005), body dissatisfaction (Leon, Fulkerson, Perry & Cudeck, 1993), perfectionism (Stice, 2002),

poor interoceptive awareness (difficulty in labeling one's feelings; Leon, Fulkerson, Perry & Early-Zald, 1995), depressive symptoms as a result of the thin ideal (McCarthy, 1990) and stress (Ball & Lee, 2000; Bennett & Cooper, 1999). Finally, biological factors include changes to neurochemical activity such as impaired serotonin activity (Avena & Bocarsly, 2011; Bailer, Price, Meltzer, Mathis, Frank, Weissfeld et al., 2004; Costa, Brennen & Hochgeschwender, 2002), changes in gastrointestinal hormones (Tong & D'Alessio, 2011), and subtle brain damage at birth which may result in early feeding problems and increase the risk of developing AN later in life (Cnattingius, Hultman, Dahl & Sparen, 1999). Other studies suggest that genetics can be a risk factor for EDs as heritability ranges between 33% and 84% as shown by twin and family studies (Costa et al., 2002; Klump, McGue & Iacono, 2000; Hsu, 1997; Treasure, 2007).

Research investigating the psychological factors that contribute to the aetiology and maintenance of ED has led to the development of the transdiagnostic model (Fairburn, Cooper & Shafran, 2003). Fairburn et al. (2003) posit that AN, BN and EDNOS have many common features and could be combined into a single diagnosis. Fairburn and Harrison (2003) suggest that the core psychopathology of AN and BN is the dysfunctional system for over-evaluating self-worth in terms of shape and weight and their ability to control them. The transdiagnostic perspective developed from Cognitive Behavioural Theory (Fairburn et al., 2003). This theory encapsulates a broad range of mechanisms suggested to be involved in the maintenance of AN, BN and EDNOS. Fairburn et al. (2003) proposed four maintaining mechanisms, which are known as clinical perfectionism, core low self-esteem, mood intolerance and interpersonal difficulties. Clinical perfectionism refers to the over-evaluation of self-worth based on the aim for and achievement of personally demanding goals despite adverse consequences. Perfectionism in ED is when an individual attempts to apply high standards to control eating, shape and weight (Shafran, Cooper & Fairburn, 2002). Core low self-esteem refers to a global negative view of the self, which is unconditional and believed to be a part of one's identity. The third maintaining mechanism is mood intolerance, which characterises the inability to cope with certain emotional states such as anger, anxiety or depression. Lastly, interpersonal difficulties refer to an individuals life circumstances

that may have acted as a proximal trigger to the onset of the ED. Examples of interpersonal difficulties include problems with parental and social relations that may intensify the need for dietary control (Fairburn, Shafran & Cooper, 1999). However, this view has been challenged by Birmingham, Touyz and Harbottle (2008) who state that combining AN with BN and EDNOS will reduce the rate of morbidity and mortality of AN, resulting in de-emphasis of the seriousness of the illness. Additionally, the transdiagnostic model of EDs has been used to develop an enhanced form of cognitive-behavioural therapy (CBT-E; Fairburn et al., 2009) aimed at treating all EDs (which will be discussed further in '*Treating Anorexia Nervosa*').

Therefore, the main focus of the present thesis will be on the processes driving both recovery and symptom maintenance from AN in addition to the driving forces behind weight regain following obesity. Unlike obesity, which is now suggested to be a pandemic effecting societies all over the world, AN is rare but it has one of the highest mortality rates which results from either disease-related causes (Jáuregui-Garrido, & Jáuregui-Lobera, 2012; Weiselberg, Gonzalez & Fisher, 2011) or most commonly, as a result of suicide (Dancyger & Fornari, 2005; Latzer & Hochdorf, 2005; Preti, Rocchi, Sisti, Camboni & Miotto, 2011). All-cause mortality rates in AN range between 2.2% to 15% (Bergh et al., 2002; Birmingham, Su, Hlynsky, Goldner, & Gao, 2005; Dancyger & Fornari, 2005; Patel et al., 1998; Polivy & Herman, 2002; Steinhausen, 2002; Zandian et al., 2007), representing a 12-fold increase in mortality for women with AN who are aged between 15 and 24 (Sullivan, 1995). Parasuicidal thoughts and behaviours such as self-harm, suicide ideation, and suicide attempts are also common in the life of those with AN (Dancyger & Fornari, 2005; Lane, 2002; Preti et al., 2011). Despite its rarity, AN has similarities with obesity as it causes many health-related physical and psychological problems. Detrimental comorbidities associated with AN include osteopeina or osteoporosis as a result of bone mineral density loss (BMD; resulting in thin and fragile bones) over time which could be irreversible even after weight restoration and recovery (Jáuregui-Garrido & Jáuregui-Lobera, 2012; Misra, 2008; Morylowska-Topolska & Kośląk, 2010; Treasure & Serpell, 2001), cardiovascular-related problems which can persist even following recovery (Jáuregui-Garrido & Jáuregui-Lobera, 2012) and impaired immune functioning, making sufferers prone to infectious diseases

(Marcos, 1997). Other health problems related to AN include infertility (The EHSRE Capri Workshop Group, 2006), bowel problems (diarrhea, constipation, rectal prolapse) as a result of excessive laxative use (10-60% of those suffering from an ED report abusing laxatives in order to inhibit the absorption of calories; Roerig, Steffen, Mitchell & Zunker, 2010), impaired kidney function (Bouquegneau, Dubois, Krzesinski & Delanaye, 2012), dental erosion (Lo Russo, Campisi, Di Fede, Di Liberto, Panzarella & Lo Muzio, 2008) and dermatological conditions such as acne (Strumia, 2005). AN often co-exists with psychiatric comorbidities such as depression (McCarthy, 1990) and obsessive-compulsive personality disorder including perfectionism (e.g., Serpell, Livingstone, Neiderman & Lask, 2002). However, the causal relationships between AN and these comorbidities may be bidirectional (Foreyt, Carlos Poston & Goodrick, 1996; O'Brien & Vincent, 2003).

Treating Anorexia Nervosa

Treating AN has been difficult and perplexing as individuals with AN can be ambivalent and play down the seriousness of their condition (Treasure, 2005). However, if caught early, AN can be short-lived and treated effectively (Fairburn & Harrison, 2003). There are a number of treatments that are currently available to those who are diagnosed with AN in the UK (Royal College of Psychiatrists, 2012) including nutritional counseling, pharmacotherapy (namely antidepressants such as fluoxetine) and psychological interventions. These psychological interventions include individual and family-based therapies, CBT, Interpersonal Psychotherapy (IPT), Specialist Supportive Clinical Management (SSCM) and a more recently developed approach, the Maudsley Model for Treatment of Adults with AN (MANTRA). Therefore, the following sections will provide a brief overview of some of the therapies currently used to treat AN.

Although an effective approach in treating BN (Bacaltchuk & Hay, 2003; Mauri, Rudelli, Somaschini, Roncoroni, Papa, Mantero et al., 1996; Wilson & Shafran, 2005) the use of antidepressants such as fluoxetine in treating AN is not effective either in the underweight phase, nor in preventing relapse (Capasso, Petrella & Milano, 2009; Kaye, Nagata, Weltzin, Hsu, Sokol, McConaha et al., 2001; Kaye, Weltzin, Hsu & Bulik, 1991; Strober, Freeman, DeAntonio, Lampert &

Diamond, 1997; Walsh, Kaplan, Attia, Olmsted, Parides, Carter et al., 2006). However, it may be beneficial when combined with CBT and could be effective for relapse prevention following weight restoration (Pederson, Roerig & Mitchell, 2003).

CBT is a time-limited intervention which was initially developed to treat BN but has been suggested as an approach that could be used to treat core features of all EDs (Wilson & Fairburn, 1993). The use of CBT in treating AN has been devised to focus on extreme dietary restriction and the cognitive and behavioural patterns which maintain the condition. CBT sessions educate the individual on their condition, provide information regarding nutrition and weight, teach cognitive-behavioural strategies such as self-monitoring and stimulus control (i.e., adapting the environment to facilitate behaviour change and remove any cues which may induce DE behaviours) and challenge dysfunctional thoughts and attitudes regarding shape, weight and eating concerns that contribute to the maintenance of AN. Once individuals have identified these thoughts and attitudes, they are taught to perform behavioural experiments that will challenge them (McIntosh, Jordan, Carter, Luty, McKenzie, Bulik et al., 2005; Wilson & Fairburn, 1993). So far, CBT has been shown to be highly effective in treating BN (Wilson & Shafran, 2005) but studies examining the effectiveness of the conventional CBT approach in treating AN have been inconclusive (Carter, Jordan, McIntosh, Luty, McKenzie, Frampton et al., 2011; McIntosh et al., 2005; Pike, Walsh, Vitousek, Wilson & Bauer, 2003) especially when treating those who present at very low weight (Bulik, Berkman, Brownley, Sedway & Lohr, 2007) or with long illness durations (Bamford & Mountford, 2012). Despite inconclusive findings of the efficacy of using CBT to treat AN, the National Institute for Clinical Excellence (NICE; 2004) made recommendations that outpatient psychological treatments using psychological therapies such as CBT and IPT should be offered to those who present all the symptoms of AN.

However, a more recent study examining the effectiveness of an enhanced version of CBT (CBT-E; Fairburn et al., 2009) in treating AN has shown some promising findings (Fairburn, Cooper, Doll, O'Connor, Palmer & Dalle Grave, 2013). The transdiagnostic model (Fairburn et al., 2003), as described earlier, formed the basis of CBT-E (Fairburn et al., 2009) which is a comprehensive psychological intervention designed to modify the processes that drive all forms of ED

psychopathology. Fairburn et al. (2013) found that 62% of their sample reached the normal BMI range (BMI ≥ 18.5) following treatment and 55% of those who were followed-up maintained the weight they had gained during treatment over the 60-week follow-up period. However, research into the effectiveness of CBT-E (Fairburn et al., 2009) in treating AN is still at the early stages and one study alone is not enough to conclude that this form of treatment is superior to other interventions.

IPT is another time-limited approach which derives from Bowlby's (1977ab) attachment theory that psychological symptoms occur when one's attachment needs are not satisfied from an early age (Rieger, Van Buren, Bishop, Tanofsky-Kraff, Welch & Wilfley, 2010). Failure to form an attachment bond has been suggested to lead to negative social evaluation which leads to core disturbances such as low self-esteem and negative affect (Rieger et al., 2010). Therefore, unlike CBT, IPT takes previous significant life events into account when assessing interpersonal difficulties which are suggested to contribute to psychological symptoms that maintain the ED. The therapist works together with the individual to identify areas of interpersonal difficulty associated with the psychological disturbances such as grief (i.e., death of a person or a relationship), change in life status (i.e., role transition), conflict in a relationship and poor social functioning which contributes to unsatisfying relationships. However, IPT does not directly tackle the disturbed eating behaviours as it considers these behaviours to be a by-product of the negative self-evaluation which developed as a result of failing to form an attachment bond at infancy (Rieger et al., 2010). However, McIntosh et al. (2005) found that SSCM produces superior outcomes when treating AN symptoms compared with both CBT and IPT.

SSCM (sometimes referred to as Nonspecific Supportive Clinical Management [NSCM]) is a three-stage approach which delivers a combination of clinical management and supportive psychotherapy. Clinical management refers to educating people with AN and providing care and support whilst building and maintaining a therapeutic relationship that motivates the individual and promotes adherence. The therapeutic relationship is used to facilitate changes to eating habits to encourage weight restoration. Similar to CBT, SSCM sessions involve identifying the symptoms and setting goals to tackle the symptoms which are then monitored within the supportive, therapeutic relationship. Finally, the therapy concludes with a

discussion of issues related to the end of the therapy and making future plans (McIntosh, Jordan, Luty, Carter, McKenzie, Bulik et al., 2006).

A more recently developed psychological intervention is the MANTRA which evolved from the model of intra- and inter-personal maintenance factors (Schmidt & Treasure, 2006). The model proposes that there are four factors that maintain the starvation regime both intrapersonally (strong positive beliefs regarding the function of AN) and interpersonally (receiving either negative or positive responses from others regarding their condition). These four factors include; 1) perfectionism or cognitive rigidity which refers to the inability to shift perspectives and plans; 2) avoidance of emotions, memories and intimate relationships; 3) positive beliefs about AN and 4) avoidance of close others' critical response to the condition. Similar to other psychological interventions, MANTRA is also a three-stage process which commences with an introduction to the health risks associated with AN and providing feedback to the individual regarding their cognitive style based on a neurocognitive test. The initial phase also includes a discussion of the positive and negative beliefs associated with the function of AN with an overall aim of shifting these beliefs and facilitating change. The second stage involves formulating a treatment plan which is then presented to the individual in the form of a letter and a diagram. Other maintaining features of the condition such as impairments in socio-emotional functioning are also tackled in the second phase. Similar to other psychological interventions, the MANTRA also encourages individuals to experiment with behavioural techniques which are designed to reduce impairments. Finally, the last stage of the treatment focuses on relapse prevention. An initial exploratory study by Wade et al. (2011) found that MANTRA is an effective tool for treating AN following weight restoration. The study found that 48% of patients with AN maintained a BMI >18 and an improvement in eating pathology at both 3- and 12-month follow-ups (Wade et al., 2011). A more recent study by Schmidt, Oldershaw, Jichi, Sternheim, Startup, McIntosh et al. (2012) compared the effectiveness of the newly developed MANTRA with the SSCM. Schmidt et al. (2012) found that both treatment groups improved on vital outcomes such as disturbed eating pathology and BMI but those who were treated with the MANTRA were more likely to require

additional inpatient or outpatient treatment at post-treatment compared to those in the SSCM group.

Although there are several therapeutic options available for the treatment of AN, rates of recovery have been slow with attrition rates ranging from 22% to 73% (Byrne, Fursland, Allen & Watson, 2011; Capasso et al., 2009; DeJong, Broadbent & Schmidt, 2011; Pike et al., 2003; Wade et al., 2011) and 25% to 61% relapsing within a year of treatment (Carter, Blackmore, Sutandar-Pinnock & Woodside, 2004; Carter, Mercer-Lynn, Norwood, Bewell-Weiss, Crosby, Woodside et al., 2012; Eckert, Halmi, Marchi, Grove & Crosby, 1995; Haller, 1992; Herzog, Dorer, Keel, Selwyn, Ekeblad, Flores et al., 1999; Kaplan, Walsh, Olmsted, Attia, Carter, Devlin et al., 2009; Keel, Dorer, Franko, Jackson & Herzog, 2005; Patel et al., 1998; Pike, 1998; Russell, Szmukler, Dare & Eisler, 1987; Strober, Freeman & Morrell, 1997; Wade et al., 2011; Zandian et al., 2007). Some of the factors that hinder treatment outcomes include the frequent crossover from AN to BN with approximately 30-80% of those with BN reporting a history of AN (Haller, 1992). Some of the other factors that have been implicated in predicting poor outcome following treatment is longer length of illness, lower weight at initial presentation, bingeing-purging behaviour, multiple treatments (an indication of severity and/or resistance to treatment), persistent negative views regarding bodyweight and shape, poor relationships, parental history of AN, depressive symptomatology, stress (which will be discussed further in Chapter Two), poor affect regulation (i.e., insecure attachment styles and poor social comparison which will be discussed further in Chapter Three) and sexual or physical abuse (Deter & Herzog, 1994; Eisler, Dare, Russell, Szmukler, le Grange & Dodge, 1997; Haller, 1992; Herzog, Sacks, Keller & Lavori, 1993; Herzog, Schellberg & Deter, 1997; Pike, 1998; Polivy & Herman, 2002; Ratnasuriya, Eisler, Szmukler & Russell, 1991; Russell et al., 1987; Troop, Serpell & Treasure, 2001). However, similar to the treatment of obesity, increasing the length of AN treatment follow-up can increase the likelihood of successful recovery. Remission rates increase from 20-30% following the shortest treatment duration to 70-80% for those attending 8 or more years of follow-up support (Grave, 2011; Steinhausen, 2002).

Obesity and AN – Summary

Both obesity and AN are types of problematic weight regulation that require long-term care management to prevent relapse (Butryn et al., 2011; Carter et al., 2011; Expert panel on the identification, evaluation, and treatment of overweight and obesity in adults, 1998; Perri, 1998; Skender et al., 1996; Tate et al., 2007; Wadden, 1993; Wadden et al., 1989, 2004, 2007). These two types of problematic weight regulation differ on both physical and psychological factors. For example, AN is recognised as a psychiatric condition (DSM-V; APA, 2013) but obesity is a chronic physical condition which is also influenced by psychological factors. However, several studies have established that the obese population are not a homogenuous group of individuals with the psychological aspects of obesity suggesting great heterogeneity within this group (e.g., Fitzgibbon & Kirschenbaum, 1990). In contrast, the psychological features of AN appear to be similar across this group of individuals including the presentation of low self-esteem, body dissatisfaction, perfectionistic attributes, internalisation of the thin-ideal and depression (e.g., Leon et al., 1993; McCarthy, 1990; Otagaki et al., 2005; Stice, 2002).

However, despite obvious differences (i.e., weight, eating patterns), obesity and AN do share some very important similarities including the process of weight change which results in extreme weight gain in the obese individual and severe weight loss in AN. It is proposed by the current programme of research that risk factors that may drive the process of weight change in these two conditions may be similar suggesting that obesity and AN share some similarities in terms of causal factors and factors associated with relapse (e.g., stress which will be discussed in Chapter Two and affect regulatory systems and processes which will be discussed in Chapter Three). The former concerns the inability to lose weight or not being able to maintain successful weight loss and the latter concerns the inability to maintain weight or weight gain. Therefore, based on the common features of these two disabling conditions, this thesis will examine why some formerly overweight or obese women fail to maintain the weight they have successfully lost and why some

women with AN are able to maintain recovery following treatment while others relapse.

Sex differences in problematic weight regulation

Research has shown that sex differences can contribute to problematic weight regulation (Chiriboga et al., 2008). Studies examining sex differences in weight change have established that women report greater weight change variations (Meltzer & Everhart, 1995), with substantial research suggesting that women are at an increased risk of major weight gain compared to men (Ball, Crawford, Ireland & Hodge, 2003; Heitmann & Garby, 1999; Jackson, Ball & Crawford, 2001; Van Lenthe, Droomers, Schrijvers & Mackenbach, 2000; Williamson, 1993; Williamson, Kahn, Remington & Anda, 1990). Younger adults aged between 25 and 44 years old are more likely to report weight gain over time (St Jeor, Brunner, Harrington, Scott, Cutter, Brownell et al., 1995) and overweight or obese women in this age group are at particular risk (Ball et al., 2003; Kuczmarski, 1992; Stevens, Knapp, Keil & Verdugo, 1991; Williamson et al., 1990). Other sex differences in problematic weight regulation include findings that indicate that middle-aged women, especially those from a low education background, experience greater weight gains over time compared to men and to women educated to degree level (Siu, Giskes, Shaw & Turrell, 2011).

Although both men and women gained weight over a 10-year period, women who were initially heavier and those who reported lower drive for thinness scores gained more weight (Heatherton, Mahamedi, Striepe, Field & Keel, 1997) providing further support for research which has demonstrated that women are more concerned about their bodyweight and shape compared to men (Rolls, Fedoroff & Guthrie, 1991).

While women are more likely to be concerned with healthy eating compared to men, they are also more likely to diet (e.g., Kiefer, Rathmanner & Kunze, 2005; Wardle, Haase, Steptoe, Nillapun, Jonwutiwes & Bellisie, 2004). In addition, as men and women age, women report more DE behaviours than men (Lewinsohn, Seeley,

Moerk & Striegel-Moore, 2002) and as a result are more likely to gain weight (Field et al., 2007). Although a decrease in eating pathology was observed amongst male and female college students, men and women have different trajectories of eating pathology; women reported a decline in bulimic scores, binge eating, fasting, and diuretic, laxative and diet pill use, whereas men reported a decrease in bulimic scores, binge eating and fasting (Heatherton, Nichols, Mahamedi & Keel, 1995). Similarly, dietary restraint strategies appear to affect men and women differently as high dietary restraint is associated with weight gain in women but with weight loss in men (Drapeau, Provencher, Lemieux, Despres, Bouchard & Tremblay, 2003; Klesges, Isbell & Klesges, 1992). An explanation of why women experience weight gain is because restrictive behaviour is difficult to maintain over long periods of time and lapses in dietary restraint can lead to overeating to compensate for the periods spent restraining, resulting in weight gain (Heatherton, Polivy & Herman, 1991) and even weight cycling (Lowe, 1993). Therefore, studies that have found women are more likely to engage in dysfunctional eating patterns may be one reason why EDs are more prevalent amongst women than men (e.g., Hoek & Van Hoeken, 2003).

These studies have demonstrated that men and women differ in the ability to regulate bodyweight and eating behaviours at a healthy level and therefore men and women should be studied separately. Consequently, the studies conducted as part of this research programme include only women.

Conclusion

This chapter provided an introduction to the epidemiology and aetiology of obesity and AN and the methods used to treat these chronic conditions. The accumulation of knowledge regarding both types of problematic weight regulation suggests that the road to recovery isn't straightforward and relapse rates are alarmingly high. Some of the processes that trigger relapse following weight loss in obesity or recovery in AN appear to be similar. This chapter has also demonstrated that, in comparison to men, women may be at an increased risk of problematic weight regulation. For this reason, the overarching agenda of the current thesis is to

present research investigating the processes of weight change, with a focus on factors that have been suggested to cause and/or maintain problematic weight regulation in women. Although other common factors such as eating pathology, caloric intake and mood levels will be taken into account, the cardinal processes that this thesis will focus on are stress, affect regulatory *systems* (social rank and vulnerable attachment styles) and affect regulatory *processes* (self-criticism/reassurance) for which a more thorough systematic and narrative review will be provided in the next two chapters.

Chapter 2: A systematic review of the role of stress and life events on weight regain and eating pathology

Introduction

As pointed out in Chapter one, treatments for both obesity and AN are prone to high relapse rates. The present thesis examines the possibility that there are similar processes that drive unhealthy changes in weight in both obesity and AN, specifically stress and affect systems. Therefore, the aim of the current Chapter is to review the literature on the role of stress in relation to weight regain following a period of obesity and eating pathology.

Stress and life events

Since the early pioneering work by Hans Selye (1936) into the role of stress on health and well-being, there have been many definitions of stress. For example, Folkman and Lazarus (1985) defined stress from a psychological approach as the "relationship between the person and the environment that is appraised by the person as relevant to his or her well-being and in which the person's resources are taxed or exceeded" (p. 152). This definition focuses on the outcome resulting from an interaction between the individual and his/her environment whereby stress is determined by the individual's appraisal of the situation that he/she has encountered. There are different types of stressors including physical, psychological and emotional stressors and the effects of these stressors on psychological well-being can be measured using various methods. These include self-report scales, measures designed to objectively identify stressful life events and biological indicators such as elevated cortisol levels.

Physiological manifestation of stress comes in the form of cortisol production which is regulated by the hypothalamic-pituitary-adrenocortical (HPA) axis. The adaptive function of cortisol production has been established to trigger fight or flight responses at times of threat and help the body to deal with stressful experiences by suppressing the immune function and increasing blood sugar levels to provide

energy. However, exposure to chronic stress can result in excessive cortisol production which can have detrimental effects on psychological and biological well-being (e.g., Miller, Chen & Zhou, 2007). Cortisol can be found in saliva which can be extracted using non-invasive methods. The advantage of measuring cortisol levels as a physiological marker of stress is that it is a reliable measure of stress and overcomes problems associated with self-report measures (e.g., social desirability bias, styles of responding, lack of insight). However, the process of assessing salivary cortisol is expensive and time-consuming, hence most research examining the role of stress has opted for self-report or interviewer-based measures of stress and life events.

Over the years, researchers have devised many tools to measure psychological stress and the experience of life events. Self-report measures of stress have been developed to obtain a global sense of the stress one is experiencing. In contrast, the life events method of examining stress is based on the idea that any experience of life change can contain an element of stress. Therefore, life events instruments focus on determining the accumulation and amount of adjustment that is required as a result of stressful events that contribute to feelings of stress. Several differences are present between self-report measures of psychological stress and life events measures. Self-report measures of psychological stress provide a subjective measure of stress whilst certain life events instruments can be used to obtain a more objective assessment of stressful experiences. The majority of self-report measures of stress require the individual to appraise their feelings of stress based over a few weeks whilst life events measures can be used to cover a longer period of time. For example, the stress section of the Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995) covers experiences of stress over the last week whilst the Perceived Stress Scale (PSS-14 [Cohen, Karmack & Mermelstein, 1983]; PSS-10 or PSS-4 [Cohen & Williamson, 1988]) requires individuals to appraise their feelings of stress based on the last month. In contrast, both self-report and interview-based measures of life events have been designed to cover a longer period. For example, the Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978) can be adapted to cover several years and self-report measures of life events such as the Social Readjustment Rating Scale (SRRS; Holmes & Rahe, 1967), the Life

Experiences Survey (LES; Sarason, Johnson & Siegel, 1978) and the List of Threatening Experiences (Brugha, Bebbington, Tennant & Hurry, 1985) focus on the occurrence of specific life events that have occurred over the past year. The experience of psychological stress is related to an array of psychological and physical conditions (e.g., Cohen, Janicki-Deverts & Miller, 2007) including changes in bodyweight.

A recent meta-analysis by Wardle et al. (2011) found a small but significant effect of stress on weight gain. For example, one of the studies included in this meta-analysis was that of Roberts, Troop, Connan, Treasure and Campbell (2007) which examined the effect of stress on weight change in 71 student nurses over a 12-week course ending with an exam. This study measured stress in two ways, which included a self-report psychological measure and the collection of saliva samples for the assessment of cortisol secretion as a physiological measure of stress. Roberts et al. (2007) found that increases in cortisol (indicating increased stress) predicted weight gain, causing women to overeat and to consume more foods that were higher in saturated fats. These findings support previous research which has shown that cortisol and its regulatory network enhance the motivation to eat and the ability to store or mobilise energy stores (Dallman, la Fleur, Pecoraro, Gomez, Houshyar & Akana, 2004; Dallman, Pecoraro, Akana, la Fleur, Gomez, Houshya et al., 2003). This is because during acute stress, feedback from corticosteroids reduces circulating cortisol but, in chronic stress, this feedback inhibition is diminished causing high levels of circulating cortisol. Increased levels of cortisol account for the differences in depositing or losing fat in stressed individuals. Physiological evidence of stress-induced eating has also been demonstrated in the laboratory. Epel, Lapidus, McEwen and Brownell (2001) found that high cortisol secretion was related to increases in consumption of high-fat foods following exposure to a laboratory stressor. Furthermore, Roberts, Campbell and Troop (2013) found that a large percentage of the variance (73%) in changes in BMI during a stressful period was explained by a decrease in dietary restraint and an increase in cortisol production and caloric intake. However, further analyses revealed that an increase in the consumption of saturated fats and carbohydrates mediated the link between an increase in cortisol production and increases in bodyweight. These

findings indicate that not only do individuals relax their restrictive diets during stressful periods, but they also consume more foods that are high in saturated fats and carbohydrates (Roberts et al., 2013).

Similarly, Habhab, Sheldon and Loeb (2009) found that highly stressed women preferred sweet, high-fat food in comparison to low stressed women who ate more low-fat foods. High stress levels have also been related to a greater tendency to eat as a distraction from the stress experienced prior to an examination (Macht & Simons, 2000). Sex differences have also been found with overweight women reporting higher stress scores in comparison to their male counterparts (Cachelin, Striegel-Moore & Brownell, 1998). Therefore, these studies suggest that stress levels play an important role in weight gain, as compared to men, women report higher stress levels (Cachelin et al., 1998; Green & Pope, 1999; Koopmans & Lamers, 2007), rate stressful life events as more negative and out of their control (Matud, 2004), eat as a way to "cope" with stress (Macht & Simons, 2000) and also change what they eat as a result of stress (Habhab et al., 2009; Roberts et al., 2007, 2013). Stress, therefore appears to relate to weight gain through physiological and behavioural changes.

However, stress not only contributes to changes to eating and bodyweight but it has also been suggested to play an important role in eating pathology. Ball and Lee (2000) reviewed the literature examining the relationship between stress, as measured by questionnaire-based assessments and experience of life events, and eating pathology. The authors concluded that there is an association between perceived stress, life events and eating pathology but longitudinal studies with large samples are required to clarify some of the inconsistencies in the literature and confirm this relationship (Ball & Lee, 2000).

Therefore, the remainder of this chapter will focus on a systematic review which will appraise the literature examining the role of stress and life events on problematic weight regulation. The systematic review is split into two main sections. As a thorough meta-analysis by Wardle et al. (2010) established that stress does have a small but positive effect on bodyweight, the first section of this systematic review will focus on the role of stress in relation to weight regain in those who were previously obese. Secondly, as the review by Ball and Lee (2000) was conducted

more than a decade ago, the second section will provide an update and appraise the studies which have examined the role of stress in eating pathology since this review. These reviews will include research studies that have examined both stress and life events as a measure of stress in relation to weight regain and DE.

The role of stress and life events on weight regain in obesity

Before providing an appraisal of the research examining stress and life events on weight regain, it must be highlighted that definitions of weight loss maintenance have varied in the literature. For example, these include losing $\geq$ 10% of bodyweight and maintaining the weight loss for a year (Foster & Kendall, 1994; Wing & Hill, 2001) and losing $\geq$ 5% of bodyweight and maintaining the weight loss for a minimum of a year (Stern, Hirsch, Blair, Foreyt, Frank, Kumanyika et al., 1995) or 2 years (Crawford, Jeffery & French, 2000). Failure to meet the criteria for weight loss maintenance has also been used to define weight regain, for example, achieving a weight change of < 3% (Stevens, Truesdale, McClain & Cai, 2005) or < 2.5% compared to pre-weight loss (Makoundou, Bobbioni-Harsch, Gachoud, Habicht, Pataky & Golay, 2010). Weight regain has therefore been defined as the failure to meet the criteria for weight loss maintenance. This review will incorporate all studies which have examined weight regain but the differences in the definitions used will be taken into consideration during the review process.

Literature Search

A search using Thomson Reuters Web of Knowledge (databases used include Web of Science, MEDLINE and the BIOSIS Citation Index) was conducted to identify research studies examining the role of perceived stress and life events on the failure to maintain successful weight loss (please note that a search was also conducted using the term “cortisol” to identify any studies which may have employed cortisol as a measure of stress in relation to weight regain, but no relevant studies were located). These were conducted separately (as described below) and results were combined in the search table (*see Table 2.1*). Additional limits were also applied to

narrow the search to research studies published in the research domain of Social Sciences, as the research areas covered under this domain included psychology, psychiatry and medicine. Only papers published in English were included in the review. Reference lists of relevant articles were also scrutinized.

Table 2.1: Results of the search terms used to locate studies examining the role of stress and life events on weight regain

No.	Search term used	Additional limits	Number of results	Located	Relevant	Excluded
1.	Stress AND Weight	None	173,363	0	0	0
	Stress AND Weight	Social Sciences	10,266	0	0	0
2.	Stress AND Weight gain	None	12,755	0	0	0
	Stress AND Weight gain	Social Sciences	893	22	1	21
3.	Stress AND Weight regain†	None	200	14	11	3
4.	Stress AND Weight maintenance	None	1,353	0	0	0
	Stress AND Weight maintenance	Social Sciences	279	2	2	2
5.	Stress AND Weight loss	None	8,295	0	0	0
	Stress AND Weight loss	Social Sciences	697	1	0	1
6.	Stress AND Weight loss maintenance	None	18	1	0	1
7.	Life events AND Weight	None	9,424	0	0	0
	Life events AND Weight	Social Sciences	1,427	0	0	0
8.	Life events AND Weight gain	None	638	3	0	3
9.	Life events AND Weight regain	None	14	1	1	0
10.	Life events AND Weight maintenance	None	204	2	1	1
11.	Life events AND Weight loss	None	783	1	0	1
12.	Life events AND Weight loss maintenance	None	63	0	0	0

Note. †Reviews by Byrne (2002), Elfhag and Rössner (2005), Jeffery, Drewnowski, Epstein, Stunkard, Wilson, Wing et al. (2000) and Wing and Hill (2001) were located at this stage of the search which revealed an additional 10 research articles.

From the above search process, various research studies, which were deemed relevant initially, were later excluded due to several reasons. The reasons for excluding papers from the current review were because the articles:

- Examined the role of stress on weight change (Adams & Rini, 2007; Epel, Jimenez, Brownell, Stroud, Stoney & Niaura, 2004; Fogel, 1993; Foreyt, Brunner, Goodrick, Cutter, Brownell & St Jeor, 1995; Fowler-Brown, Bennett, Goodman, Wee, Corbie-Smith & James, 2009; Iversen, Strandberg-Larsen, Prescott, Schnohr & Rod, 2012; Kivimäki, Head, Ferrie, Shipley, Brunner, Vahtera et al., 2006; Parham, 1990; Provencher, Polivy, Wintre, Pratt, Pancer, Birnie-Lefcovitch et al., 2009; Serlachius, Hamer, & Wardle, 2007)
- Investigated the role of stress or life events on weight gain (Berset, Semmer, Elfering, Jacobshagen & Meier, 2011; Block, He, Zaslavsky, Ding & Ayanian, 2009; Cachelin et al., 1998; Gerace & George, 1996; Groesz, McCoy, Carl, Saslow, Stewart, Adler et al. 2012; Korkeila, Kaprio, Rissanen, Koskenvuo & Sörensen, 1998; Kouvonen, Kivimäki, Cox, Cox & Vahtera, 2005; Ogden, Stavrinaki & Stubbs, 2009; Roberts et al., 2007; Rookus, Burema & Frijters, 1988; Tseng & Fang, 2011; Van Strien, Rookus, Bergers, Frijters & Defares, 1986)
- Examined the role of stress on weight gain in those taking part in an obesity prevention programme (Barrington, Ceballos, Bishop, McGregor & Beresford, 2012; Chang, Brown & Nitzke, 2009; Foreyt & Goodrick, 1994)
- Examined beliefs regarding reasons for weight loss relapse rather than stress, per se (Cachelin et al., 1998)
- Provided an overview of several papers examining weight loss maintenance (Sarlio-Lähteenkorva, 2001)
- Included a measure of stress but did not assess weight regain following weight loss surgery (Ray, Nickels, Sayeed & Sax, 2003)
- Examined the role of sudden life events that trigger weight loss efforts and how these may sustain weight loss maintenance rather than how life events may contribute to weight regain (Epiphaniou & Ogden, 2010)

This search process also includes a review of the factors that are associated with weight regain (Elfhag & Rössner, 2005) which included three review papers by Byrne (2002), Jeffery et al. (2000) and Wing and Hill (2001). The reference lists of these reviews were scrutinized and an additional 10 relevant papers were located. Overall, a total of 16 research papers were found, of which four were found to be investigating the role of stress on weight regain (Bond et al., 2009; DePue, Clark, Ruggiero, Medeiros & Pera, 1995; Dohm, Beattie, Aibel & Striegel-Moore, 2001; Sarlio-Lähteenkorva, Rissanen & Kaprio, 2000) and 12 studies exploring the impact of life events on failure to maintain successful weight loss (Byrne, Cooper & Fairburn, 2003; Byrne, Cooper & Fairburn, 2004; Dubbert & Wilson, 1984; Foster, Wadden, Kendall, Stunkard & Vogt, 1996; Gormally & Rardin, 1981; Gormally, Rardin & Black, 1980; Hartz, Kalkhoff, Rimm & McCall, 1979; Jeffery, Bjornson-Benson, Rosenthal, Lindquist, Kurth & Johnson, 1984b; Kayman, Bruvold & Stern, 1990; Marston & Criss, 1984; Tinker & Tucker, 1997; Turk, Sereika, Yang, Ewing, Hravnak & Burke, 2012).

Before providing an appraisal of the literature, this section will begin with a description of the different instruments that have been used to assess levels of stress in relation weight regain, including both assessments measuring perceptions of stress and experiences of life events as a measure of stress.

Instruments used to examine stress

Each of the four studies investigating the role of stress on weight regain in this literature review used different measures of stress. Only one study (Bond et al., 2009) used a validated measure of stress which was the PSS-14 (Cohen et al., 1983). The PSS-14 (Cohen et al., 1983) is a 14-item self-report measure assessing perceptions of stress experienced in the past month and respondents must rate on a 5-point Likert scale (“Very often” to “Never”) the degree to which they appraise situations as stressful. The PSS-14 (Cohen et al., 1983) has been found to be a single factor measure of stress that has good reliability and validity with a Cronbach’s alpha of .86 (Bekker & Boselie, 2002). However, other studies reviewed here have either developed a measure of stress for the purpose of their research (DePue et al., 1995), taken a selection of items measuring stress from a previous study (Dohm et

al., 2001) or used a brief 4-item measure assessing the stress of daily activities (Sarlio-Lähteenkorva et al., 2000). Finally, perceived stress refers to the subjective psychological stress experienced by the individual and is not reliant on the number of life events or stressors that may have taken place prior to the onset of the condition under study. Therefore, it is important to also collate evidence from studies which have examined the role of life events on weight regain.

Instruments used to examine life events

Similar to the studies examining the role of stress on weight regain, the research that has examined the impact of life events on weight regain has also not been uniform in their assessment of life events. Only three studies (Foster et al., 1996; Gormally et al., 1980; Tinker & Tucker, 1997) used a validated measure of life events. Foster et al. (1996) used the full 57-item Life Experiences Survey (LES; Sarason et al., 1978) which is a self-report measure covering a broad range of life events such as illness, relationship problems and stressors at work. The LES (Sarason et al., 1978) provides positive, negative and total scores as participants are required to rate each life event on a 7-point Likert scale from extremely positive (3) to extremely negative (-3). Gormally et al. (1980) used a modified version of the LES (Sarason et al., 1978) and supplemented the self-report measure with an interview designed to assess how each life event impacted weight control efforts. Finally, Tinker and Tucker (1997) used the Life Events Questionnaire (LEQ; Tucker, Vuchinich & Pukish, 1995) to assess nine categories of life events such as work, residence, intimate relationships, family relations, friendship/social, physical health and weight-related events that had occurred over the last four years. For each reported event, participants were required to indicate when it occurred and if the event had a positive or a negative impact. However, the majority of the studies that will be described in this review have developed an interview or a self-report measure of life events specifically for the purposes of their study.

Literature review

The first section of this review will examine the literature on *stress* and weight regain which will be followed by an appraisal of the literature assessing the

relationship between *life events* and weight regain. A summary of all the papers included in this review can be found in *Table 2.2*.

Stress and weight regain

The current review located only four studies which have examined the role of stress on weight regain. An appraisal of the four studies indicated inconsistencies in the findings.

Only one study (DePue et al., 1995) reported that stress is an impeding factor for those who are trying to maintain successful weight loss. DePue et al. (1995) followed up 178 participants who had taken part in a VLCD (approximately <800 kcal per day) to examine potential facilitators (e.g., regular weigh-ins, attending maintenance program, regular exercise) and obstacles (e.g., personal or family stress, low motivation, eating in restaurants) to weight loss maintenance. Those who regained weight post-VLCD were more likely to rate personal and family stress as an obstacle in comparison to those who successfully maintained the weight they had lost.

The relationship between stress and weight regain was partly supported by Sarlio-Lähteenkorva et al. (2000), who found that stress levels differ amongst men who are successful and unsuccessful weight loss maintainers, but not in women. Stress was measured at three time points using four self-report items tapping into the stress of daily activities (Koskenvuo, Langinvainio, Kaprio, Rantasalo & Sarna, 1979). A higher percentage of men who were weight loss maintainers reported a stress-free life compared to overweight and weight regainers. Also, compared to weight regainers, weight loss maintainers reported lower stress scores at both follow-ups. This longitudinal study spanning over 15 years highlights the need to examine men and women separately in order to determine the processes that predispose different sexes to relapse during their weight loss maintenance journey. However, Sarlio-Lähteenkorva et al. (2000) did not provide information as to how these participants initially lost weight which may be another contributing factor to how weight loss is maintained. Research has shown that, compared to men, women are more likely to diet and use physical activity strategies in order to lose weight (Lemon, Rosal, Zapka, Borg & Andersen, 2009).

However, the remaining two studies did not find an effect of stress on weight regain (Bond et al., 2009; Dohm et al., 2001) even when participants were recruited based on losing weight either following weight loss surgery or through non-surgical methods (Bond et al., 2009). In addition, despite including two separate measures of stress, Dohm et al. (2001) did not find stress to be an important factor in explaining why relapsers failed to maintain successful weight loss. However, the first stress measure used was a single item amongst a list of items developed using obesity expert opinion on the causes of obesity (Bray, York & DeLany, 1992). This broad, single measure of stress required participants to rate on a 5-point Likert scale on the degree to which they thought their experience of stress could be the reason as to why they had gained weight in the past. In retrospect, participants may not attribute stress as a reason why they gained weight every single time, but at the time of experiencing stress they may have acted on this stress by either eating more or exercising less, hence resulting in weight gain. The second measure of stress required individuals to rate how stressed they had felt over the past six months and over the past 24 hours. However, Dohm et al. (2001) classified regainers as those who had lost and regained a minimum of 10 to 19lbs at least once but did not request information on when weight losses and subsequent regains occurred. Therefore, assessing stress levels over the past six months or the last 24 hours is arbitrary as these individuals may have experienced weight loss and regain several years ago.

In conclusion, the four studies presented in this part of the review highlight the urgent need for more good quality research examining the role of stress on weight regain. Interestingly, only one retrospective study (DePue et al., 1995) which examined stress using a multiple item measure specific to weight loss maintenance efforts found an effect of stress on weight regain. Although Bond et al. (2009) used a single item that was specific to how stress contributes to weight loss maintenance efforts, this item may not have been sensitive enough to detect the experience of stress on weight regain. Importantly, even a longitudinal study spanning over 15 years found an effect of stress on weight regain only in men (Sarlio-Lähteenkorva et al., 2000). Therefore, these fours studies suggest that maybe only weight specific stress measures are sensitive enough to detect the influence of stress on weight

regain. However, more research with better quality methods are needed to determine if this is the case.

Life events and weight regain

In comparison to the literature on general perceptions of stress on weight regain, more studies (*n* = 12) have investigated the role of life events on weight regain.

In contrast to the above findings, seven of the twelve studies examining the role of life events on weight regain suggest that life events experienced during efforts aimed at maintaining weight loss can predispose a person to relapse and regain the weight they had previously lost (Byrne et al., 2003; Dubbert & Wilson, 1984; Gormally & Radin, 1981; Gormally et al., 1980; Kayman et al., 1990; Tinker & Tucker, 1997; Turk et al., 2012). Out of these seven studies, three found a clear link between stressful life events and weight regain. Dubbert and Wilson (1984), Tinker and Tucker (1997) and Turk et al. (2012) all found that a greater proportion of those who regained the weight they had lost reported experiencing stressful life events compared to those who had successfully maintained their weight loss. All three studies examined stress retrospectively covering the period following weight loss but the length of period that was covered ranged between 1.5 to 4 years. In addition, despite all these studies using different (and mostly unvalidated) measures, there appears to be a consistent relationship between life events and weight regain. Finally, these studies also indicated that the types of life events that are typically reported by weight regainers were illness-related stresses or problems of an interpersonal nature (i.e., relationship conflicts). Therefore, despite methodological differences, these studies indicate that stressful life events are related to weight regain following successful weight loss (Dubbert & Wilson, 1984; Tinker & Tucker, 1997; Turk et al., 2012).

However, four of the seven studies (Byrne et al., 2003; Gormally & Radin, 1981; Gormally et al., 1980; Kayman et al., 1990) that examined the relationship between life events and weight regain portray slightly different findings. These studies found that even though the number of people reporting events didn't differ between maintainers and regainers, those who regained weight *attributed* their

weight gain to changes in eating in response to these events. In other words, life events per se were not necessarily directly causal but some people appeared to be more affected than others. These findings suggest that the experience of stressful life events may encourage comfort eating, a decline in physical activity or even engaging in unhealthy behaviours such as smoking and drinking as a way of dealing with their problems in some people but not others. In addition, Kayman et al. (1990) found significantly fewer relapsers (33%) sought social support when faced with problems compared with maintainers (70%) and healthy weight controls (80%). These findings indicated that it may be not the occurrence of life events per se that contribute to unsuccessful weight loss maintenance but how one *responds* to adversity is what influences weight regain.

However, a caveat of these findings is that all four of the studies mentioned here examined the role of stressful life experiences on weight regain in females only (Byrne et al., 2003; Gormally & Radin, 1981; Gormally et al., 1980; Kayman et al., 1990). Therefore, although it is difficult to provide a causal chain of events with certainty, based on the findings of the first seven studies reviewed here, it is possible to suggest that the experience of negative life events following weight loss may induce stress, which could hinder one's ability to successfully maintain weight loss, especially in women.

However, out of the twelve studies located during the literature search, five studies (Byrne et al., 2004; Foster et al., 1996; Hartz et al., 1979; Jeffery et al., 1984b; Marston & Criss, 1984) did not find an association between adverse life events and weight regain in those who were formerly overweight. An important difference between these studies is the samples that were recruited; three studies employed an all-female sample (Byrne et al., 2004; Foster et al., 1996; Hartz et al., 1979) and the other two studies employed either a predominantly female sample (Marston & Criss, 1984) or an all-male sample (Jeffery et al., 1984b). In addition, these findings should be accepted with caution as, out of the five studies, only one (Foster et al., 1996) used a well-validated measure of life events (LES; Sarason et al., 1978). Whilst the other authors briefly describe the life events measure developed specifically for their research purposes, they do not provide any explanation as to how the measure was developed or if the measure has been validated by previous

studies (Byrne et al., 2004; Hartz et al., 1979; Jeffery et al., 1984b; Marston & Criss 1984). For example, Hartz et al. (1979) assessed stress as responses to three items regarding the number of diets previously attempted, if participants considered themselves to be overweight and if they had ever lost a child before. It can be argued that these items are not a true indication of stress levels experienced during the weight loss maintenance phase when relapse occurs. Similarly, Marston and Criss (1984) just state that they used a 17-item measure of life events and stress and do not provide any additional information such as item examples from the measure or how the tool was developed.

Summary

The current review found that studies examining the role of stress and life events on weight regain produced inconsistent findings. Additionally, this review has highlighted the need for good quality research into the relationship between stress and weight regain as only four studies located in this review attempted to examine this association (Bond et al., 2009; DePue et al., 1995; Dohm et al., 2001; Sarlio-Lähteenkorva et al., 2000). Similarly, although more studies (*n* = 12) were found to assess the relationship between life events and weight regain, a comprehensive appraisal of these studies revealed that the failure to use a well validated measure of life events may have contributed to inconsistent findings. Therefore, further investigation is required to disentangle these inconsistent findings and provide a clearer understanding of how stressful experiences may influence weight regain. As a result, Chapter Six will present a research study that will examine the relationship between stress and problematic weight regulation in those who have lost weight.

Table 2.2: Research studies examining the role of stress and life events failure to maintain successful weight loss

Author	*N* *(% Female)*	Sample Characteristics	Assessment Measure	Study Design	Results	Support for Relationship
a) Studies examining the role of stress on weight regain						
Bond et al. (2009)	315 (86)	Weight losers following surgery: 105 (*M* age = 45.8, *SD* = 10.3) Matched non-surgical weight losers: 210 (*M* age = 44.6, *SD* = 11.2)	Stress: PSS-14 (Cohen et al., 1983) Weight regain: gained ≥ 5kg at 1 year follow-up	Stress measured at two time points, one-year apart	Stress was not a significant predictor of weight regain in both groups	No
DePue et al. (1995)	107 (75)	Regainers: 76 Maintainers: 31 (*M* age = 43)	Stress: survey developed for study which involved rating potential weight management obstacles (e.g. personal or family stress) Weight regain: follow-up weight ≥ 10lbs compared to weight immediately post-treatment	Stress measured at follow-up (2 years post-treatment)	Weight regainers more likely to rate stress as an obstacle to weight management compared to weight loss maintainers ($p < .05$)	Yes

Dohm et al. (2001)	1212 (54.3)	Maintainers: 606 Regainers: 606 (Each group consisted of 329 women [*M* age = 41.21, *SD* = 11.51] and 277 men [*M* age = 49.75, *SD* = 12.74])	Stress: survey of items taken from Bray et al. (1992) including stress as a reason for weight gain and stress questions assessing level of stress over the past 6 months and the past 24 hours Weight regain: regained a minimum of 10-19lbs	Cross-sectional	Maintainers and regainers did not differ on any of the stress-related questions	No
Sarlio-Lähteenkorva et al. (2000)	717 (15.5)	Regainers: 54 Maintainers: 55 Overweight: 608	Stress: 4-item measure assessing stress of daily activities (Koskenvuo et al., 1979) Weight regain: lost ≥ 5% of their weight between baseline and first follow-up (6 years later) but then gained more than they initially lost at second follow-up (9 years after first follow-up)	Longitudinal: stress levels assessed at 3 time points	Higher % of men who were weight loss maintainers reported a more stress-free life than overweight men and weight regainers at baseline. Weight loss maintainers reported lower stress scores compared to weight regainers at both follow-ups. No differences in women's stress scores between groups	Partial

b) Studies examining the role of life events on weight regain						
Byrne et al. (2003)	76 (100)	Regainers: 28 (*M* age = 44.4, *SD* = 9.3) Maintainers: 28 (*M* age = 44.7, *SD* = 11.5) Healthy stable weight: 20 (*M* age = 35.9, *SD* = 8.6)	Life events: interview developed for study which focused on recent weight loss attempt Weight regain: regained the weight they had lost and were within 3.2kg of their original weight	Cross-sectional	Higher percentage of regainers overate in response to an adverse event	Partial
Byrne et al. (2004)	50 (100)	Regainers: 20 Maintainers: 21 Continued to lose weight: 9 (*M* age = 42.5, *SD* = 10.1)	Life events: interview developed for study using the factors derived from Byrne et al. (2003) Weight regain: lost ≥ 10% of their bodyweight but regained more than 3.2kg at final follow-up (1 year later)	Cross-sectional but life events measured retrospectively covering the past 4 weeks	Maintainers and regainers did not differ on the mean number of adverse life events reported (*U* = 184.5, *p* = .89)	No
Dubbert & Wilson (1984)	62 (77.4)	No descriptive information provided	Life events: no clear description of the scale used Weight regain: follow-up weight >	Longitudinal but life events assessed	43% of regainers reported stressful events such as major illnesses, bereavements and other	Yes

			weight immediately post-treatment	retrospectively at follow-up (30 months post-treatment)	environmental stressors compared to 15% of those who continued to lose weight	
Foster et al. (1996)	48 (100)	*M* age = 41.0, *SD* = 9.4	Life events: 57-item Life Experiences Survey (LES; Sarason et al., 1978) Weight regain: weight at follow-up 5kg above pre-treatment weight	Longitudinal but life events assessed retrospectively at follow-up (58 months post-treatment)	Life events scores were not related to the percentage of weight regained over time	No
Gormally & Rardin (1981)	100 (100)	*M* age = 39.4, *SD* = 7.6	Life events: interview developed for study which included assessment of major life changes Weight regain: either regained > 50% of initial weight lost, regained	Longitudinal but life events assessed retrospectively at follow-up (7	Higher percentage of relapsers (84%) reported losing complete control of their dietary intake and physical activity in response to an adverse life event compared to	Partial

			more than 7lbs (3.2kg) during last 3 months of follow-up or showed a steady month to month regain	months post-treatment)	weight loss maintainers (20%)	
Gormally et al. (1980)	40 (100)	Maintainers: 7 Relapsers: 13 (*M* age of entire sample = 35)	Life events: modified version of the Life Experiences Survey (LES; Sarason et al., 1978) Weight regain: regained > 50% of initial weight lost	Longitudinal but life events only assessed retrospectively at follow-up (7 months post-treatment)	No difference between the number of life changes reported by regainers and maintainers, $t(18) = 1.58$, $p > .05$ Regainers reported more negative impact of life changes on weight control, $t(18) = 2.83$, $p < .01$	Partial
Hartz et al. (1979)	175 (100)	(*M* age = 42.4)	Life events: responses to items about losing a child, the number of diets previously attempted and if participants considered themselves to be overweight Weight regain: any amount of weight regained from baseline to follow-up (18-24 months post-	Longitudinal but life events assessed only at baseline	No relationship between the three stress variables and weight change over time	No

			treatment)			
Jeffery et al. (1984b)	89 (0)	(*M* age = 52.8)	Life events: life events schedule covering events such as marriage, divorce, bereavement and loss of job Weight regain: no clear definition of weight regain provided	Longitudinal but life events assessed at follow-up (1 year post-treatment)	Life events experienced during the year following treatment were not associated to weight loss maintenance	No
Kayman et al. (1990)	108 (100)	Regainers: 44 (*M* age = 47) Maintainers: 30 (*M* age = 41) Healthy stable weight: 34 (Age range 21-73)	Life events: interview developed for study which included questions regarding recent troubling situations or events Weight regain: had previously lost 20% of bodyweight one or more times but regained it and ≥ 20% overweight on study entry	Cross-sectional but life events assessed retrospectively	77% of regainers reported regaining in response to adverse life event Similar % of relapsers, maintainers and control participants reported a stressful event or situation (87%, 80% and 76%, respectively) More relapsers (56%) reported experiencing stress due to being overweight and/or as a result of their health than maintainers	Partial

					(10%) and controls (2%)	
Marston & Criss (1984)	47 (80.9)	Regainers: 42% Maintainers: 58% (*M* age = 37)	Life events: 17-item measure of life events and stress Weight regain: regained ≥ 20% of lost weight	Longitudinal but life events only assessed at baseline	Life events did not discriminate between relapsers and maintainers	No
Tinker & Tucker (1997)	41 (71%)	Currently obese: 20 Maintainers: 21 (*M* age = 34.9, *SD* = 8.6)	Life events: LEQ (Tucker et al., 1995) Weight regain: currently obese individuals who had previously lost weight but then regained all of the weight they had lost	Cross-sectional but life events measured retrospectively covering 4 years	Reduction in the number of negative events reported by recovered participants during and after weight loss Obese group reported more family, social and weight-related events	Yes
Turk et al. (2012)	107 (86%)	Relapsers: 61 Maintainers: 46 (*M* age = 46.3, *SD* = 6.9)	Life events: 4-item measure assessing the impact of stress and life events on weight management Weight regain: gained > 5%	Longitudinal but life events only measured at follow-up (18 months post-treatment)	Stressful life events associated with weight regain in both linear (*b* = .51, SE = .24, *p* = .04) and logistic regression (OR = 1.30, 95%CI = 1.04–1.63, *p* = .02) 64% of participants reported experiencing 1 or more life event	Yes

Note. LES = Life Experiences Survey (Sarason et al., 1978); LEQ = Life Events Questionnaire (Tucker et al., 1995)

The role of stress and life events on eating pathology

Similar to unsuccessful weight loss maintenance efforts described in the first section of this chapter, therapeutic interventions aimed at treating AN have been met with high relapse rates (Carter et al., 2004, 2012; Eckert et al., 1995; Haller, 1992; Herzog et al., 1999; Kaplan et al., 2009; Keel et al., 2005; Patel et al., 1998; Pike, 1998; Russell et al., 1987; Strober et al., 1997b; Wade et al., 2011; Zandian et al., 2007). Chapter One provided a brief overview of the factors that can contribute to the development and maintenance of AN, therefore, this section will focus on the role of stress on dysfunctional eating patterns. The important aspect of this literature review is to bring together empirical research that has examined the role of stress and life events on DE. Firstly, this review will describe the literature search process, followed by a description of the instruments that have been used to examine stress, life events and eating pathology before finally providing an appraisal of the research in this area.

Literature Search

Similar to the literature search for research papers examining the role of perceived stress and life events on the failure to maintain successful weight loss in obesity, a literature search was conducted using Thomson Reuters Web of Knowledge (databases used include Web of Science, MEDLINE and the BIOSIS Citation Index) to identify research studies examining the role of stress and life events on eating pathology (please note once again that a search was also conducted using the term "cortisol" to identify any studies which may have employed cortisol as a measure of stress in relation to DE, but no relevant studies were located). These were conducted separately (as described below) and results were combined in the search table (*see Table 2.3*). Additional limits were also applied to narrow the search to research studies published in Social Sciences and only papers published in English were included in the review. Reference lists of relevant articles were also scrutinized.

Table 2.3: Results of the search terms used to locate studies examining the role of stress and life events on eating pathology

No.	Search term used	Additional limits	Number of results	Located	Relevant	Excluded
1.	Stress AND Disordered eating†	None	194	17	17	0
2.	Life events AND Disordered eating	None	24	1	1	0
3.	Stress AND Eating Disorders	None	2,716	0	0	0
	Stress AND Eating Disorders	Social Sciences	800	17	16	1
4.	Life events AND Eating Disorders	None	135	6	6	0

Note. †Reviews by Ball and Lee (2000) and Bennett and Cooper (1999) were located at this stage of the search with the latter review producing an additional 4 research articles.

Amongst these research papers, review papers by Ball and Lee (2000) and Bennett and Cooper (1999) were located. Ball and Lee (2000) provide a thorough appraisal of the literature examining the relationship between stress, as measured by questionnaire-based assessments and experience of life events, on eating pathology. Ball and Lee (2000) conclude that there is an association between perceived stress, life events and eating pathology but longitudinal studies with large samples are required to clarify some of the inconsistencies in the literature and confirm this relationship. This comprehensive review includes research studies investigating the relationship between perceived stress and eating pathology published between 1983 to 1995 and between 1954 to 1996 for studies examining the role of life events on eating pathology. Therefore, it was decided to focus on research studies published after 1995. The reason for focusing on 18 years worth of research is to firstly assess if studies published after the Ball and Lee (2000) review continue to produce inconsistent findings or firmly establish the relationship between stress and DE. Secondly, an appraisal of research that spans over a longer

period of time rather than just focusing on more recent findings will highlight if any changes in the stress and DE relationship have occurred over the years. Although it was decided to focus on studies published after 1995, different stages of the literature revealed three papers which were published before 1995 but not included in the Ball and Lee (2002) review. These papers (Cattanach, Phil, Malley & Rodin, 1988; Mitchell, Davis & Goff, 1985; Råstam & Gillberg, 1992) have been included in the present review in order to determine whether they are consistent with the studies that have been reviewed already. The review by Bennett and Cooper (1999) is not an exhaustive review but was found to describe four studies (Fornari, Kent, Kabo & Goodman, 1994; McFarlane, McFarlane & Gilchrist, 1988; Nygaard, 1990; Sohlberg, 1990) that were not included in the Ball and Lee (2000) paper which will also be included in the present review. All four research studies were found to examine the role of life events in relation to EDs.

In addition to the four papers located from the Bennett and Cooper (1999) review, a further 13 suitable papers were found using the key terms “stress” and “disordered eating”. From this selection of papers, 10 studies were found to examine the role of stress on eating pathology (Ball & Lee, 2002; Ball, Lee & Brown, 1999; Beukes, Walker & Esterhuyse, 2010; Cattanach et al., 1988; Chen, Wang, Guo, Arcelus, Zhang, Jia, et al., 2012; Cohen & Petrie, 2005; Costarelli & Patsai, 2012; King, Vidourek & Schwiebert, 2009; Mussap, 2007; Shea & Pritchard, 2007). The remaining three studies were found to assess the impact of life events on disturbed eating behaviours (Bodell, Smith, Holm-Denoma, Gordon & Joiner, 2011; Loth, van den Berg, Eisenberg & Neumark-Sztainer, 2008; Smyth, Heron, Wonderlich, Crosby & Thompson, 2008).

The remainder of the search process located another 23 relevant articles with five examining the role of stress on DE (Bekker & Boselie, 2002; Berg, Frazier & Sherr, 2009; Fryer, Waller & Kroese, 1997; Martz, Handley & Eisler, 1995; Striegel-Moore, Dohm, Kraemer, Schreiber, Taylor & Daniels, 2007), 17 studies examined the role of stressful life events on eating pathology (Berge, Loth, Hanson, Croll-Lampert & Neumark-Sztainer, 2012; Cooley, Toray, Valdez & Tee, 2007; Grilo, Pagano, Stout, Markowitz, Ansell, Pinto et al., 2012; Karwautz, Rabe-Hesketh, Hu, Zhao, Sham, Collier et al., 2001; Karwautz, Wagner, Waldherr, Nader, Fernandez-Aranda, Estivill

et al., 2011; Mitchell et al., 1985; Pike, Hilbert, Wilfley, Fairburn, Dohm, Walsh et al., 2008; Pike, Wilfley, Hilbert, Fairburn, Dohm & Striegel-Moore, 2006; Raffi, Rondini, Grandi & Fava, 2000; Råstam & Gillberg, 1992; Rojo, Conesa, Bermudez & Livianos, 2006; Schmidt, Tiller, Blanchard, Andrews & Treasure, 1997; Sharpe, Ryst, Hinshaw & Steiner, 1997; Steinhausen, Gavez & Winkler Metzke, 2005; Taylor, Bryson, Altman, Abascal, Celio, Cunning et al., 2003; Troop & Treasure, 1997; Welch, Doll & Fairburn, 1997) and one study assessed both perceptions of stress and adverse life events on disturbed eating behaviours (Blaase & Elklit, 2001).

From the search process, one study was excluded as it examined the role of perceived stress on eating pathology but this study combined the stress scores with scores of depression and social support to create a global measure of negative well-being which makes it difficult to determine the exact role of stress on eating behaviours (Provencher et al., 2009).

Overall, a total of 41 research papers were identified, of which 16 investigated the role of stress on disturbed eating behaviours, 25 studies examined the impact of life events on eating pathology and one study investigated both perceived levels of stress and life events on eating behaviours.

Instruments used to examine stress

Identical to the research presented in the first section of the review, most of the studies that will be described in this section have used the PSS-14 (Cohen et al., 1983) or PSS-10 (Cohen & Williamson, 1988) as a general measure of stress. However, some studies also investigated the role of feminine gender role stress on eating pathology (Bekker & Boselie, 2002; Martz et al., 1995; Mussap, 2007). These studies used the Feminine Gender Role Stress (FGRS; Gillespie & Eider, 1992) scale which measures five aspects of feminine gender role stress including unassertiveness, insecurities about being in an unemotional relationship, being found unattractive, victimized and the inability to be nurturing. The present review will also describe measures of stress that have been developed by study authors for the purpose of their research.

Instruments used to examine life events

The majority of the research studies discussed in this paper used the Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978). The LEDS (Brown & Harris, 1978) is a semi-structured interview designed to elicit information regarding events and difficulties that occurred over a specified time period. A wide range of areas are covered in the interview such as education, work, reproduction, housing, money/possessions, legal, health, marital/partner relationships, other relations (e.g., child, parent), and other events (including bereavement). Contextual information of the life experiences is used to rate the severity of the event. Each event is rated in terms of threat or unpleasantness on a 4-point scale ('marked', 'moderate', 'some' and 'little/none'). The short and long-term severity of each event is rated, with short-term referring to the threat/unpleasantness of the event during the first few days and long-term severity referring to the threat/unpleasantness for 10-14 days after the event. A severe event refers to an event that is rated as 'marked' or 'moderate' on the long-term severity. Problems lasting four weeks or more are classified as difficulties, which are rated on a 7-point scale and marked difficulties are those rated on the top three scale points. A marked difficulty lasting two or more years is labeled as a major difficulty. The LEDS (Brown & Harris, 1978) is a valuable tool to use when assessing life events and difficulties as it does not rely on subjective reporting but determines severity of events based on contextual information. This allows researchers to account for life circumstances and the goals and commitments of the respondent.

Four studies in this review used the Oxford Risk Factor Interview (RFI; Fairburn, Doll, Welch, Hay, Davies & O'Connor, 1998) to assess exposure to life events prior to onset (Karwautz et al., 2001, 2011; Pike et al., 2006, 2008). In addition to the assessment of putative risk factors related to EDs, the RFI (Fairburn et al., 1998) also inquires about life events that precede onset. This semi-structured interview enables the specific timing and circumstances of each life event that is identified by the respondent to be determined. However, other measures of life events and difficulties have also been used, which are described in the literature review section.

Instruments used to examine eating pathology

The majority of research studies discussed in this section of the literature review have employed clinical samples. However, the studies which recruited healthy controls used a variety of self-report scales to measure DE behaviours. These include EDE-Q (Fairburn & Beglin, 1994), Eating Disorders Inventory (EDI; Garner, Olmstead & Polivy, 1983) and earlier versions, Eating Attitudes Test-40 (EAT-40; Garner & Garfinkel, 1979) and the EAT-26 (Garner, Olmsted, Bohr & Garfinkel, 1982) which is the shortened version of the EAT-40.

The EDE-Q (Fairburn & Beglin, 1994) was developed from a semi-structured interview (Eating Disorder Examination [EDE]; Cooper, Cooper & Fairburn, 1989) into a 36-item questionnaire consisting of 4 subscales assessing dietary restraint and weight, shape and eating concerns as well as diagnostic items (these items have been left out of the analysis in the papers reviewed here). The EDE-Q (Fairburn & Beglin, 1994) total score and subscales have been reported to have adequate levels of internal consistency. The total EDE-Q score was estimated to have a Cronbach alpha of .90, whilst the dietary restraint and weight, shape and eating concerns subscales had Cronbach alphas of .70, .72, .83 and .73, respectively (Peterson, Crosby, Wonderlich, Joiner, Crow, Mitchell et al., 2007).

The EDI (Garner et al., 1983) is a 64-item instrument that measures the psychological characteristics of ED and has been used to distinguish between people with anorexia and bulimia. The EDI (Garner et al., 1983) consists of three DE subscales labeled as drive for thinness, bulimia and body dissatisfaction and five psychological constructs commonly found in people with EDs (referred to as ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness [difficulty labeling one's feelings] and maturity fears). The total EDI score was estimated to have a Cronbach alpha of .92, whilst the drive for thinness, bulimia and body dissatisfaction subscales had Cronbach alphas of .86, .82 and .89 (Lee, Lee, Leung & Yu, 1997). The five psychological constructs also had acceptable internal consistencies as measured by Cronbach's alpha (ineffectiveness α = .85, perfectionism α = .70, interpersonal distrust α = .78, interoceptive awareness α = .77 and maturity fears α = .77; Lee et al., 1997). EDI-2 (Garner, 1991) and EDI-3 (Garner,

2004) which are modified versions of the EDI (Garner et al., 1983) have also been used.

The EAT-40 (Garner & Garfinkel, 1979) is a 40-item self-report scale which measures AN symptomatology reflecting food preoccupation, body image for thinness, vomiting and laxative abuse, dieting, slow eating, secretive eating and perceived social pressure to gain weight. The EAT-40 has good internal consistency for both AN respondents (Cronbach's α = .79) and healthy controls (Cronbach's α = .94) as reported by Garner and Garfinkel (1979). Garner et al. (1982) used the EAT-40 to create an abbreviated version of 26 items which consists of three subscales measuring dieting behaviour, bulimia and food preoccupation and oral control. Similar to the EAT-40, the EAT-26 (Garner et al., 1982) also has high internal consistency for a sample of AN respondents (Cronbach's α = .90) and healthy controls (Cronbach's α = .92; Rudiger, Cash, Roehrig & Thompson, 2007). Research studies that have not used any of the measures that have been mentioned here have either developed their own measure of DE by using a mixture of items from various scales or assigned respondents into groups according to clinical assessment which will be described during the review process.

Literature Review

The review of the role of stress and life events and difficulties on DE will be presented in three sections. The cross-sectional findings from studies examining the role of stress on DE will be presented first, followed by the longitudinal findings in this area. The final section will discuss studies which have examined the relationship between stressful life events and eating pathology. A summary of the research studies included in this review can be found in *Table 2.4*.

Stress and DE: Cross-sectional findings

From the 41 studies which were located from the literature search, 16 examined the role of stress on DE. Out of these 16 studies, 13 presented cross-sectional findings, two examined the relationship between stress and DE over time and one presented both cross-sectional and longitudinal results.

The cross-sectional findings suggest that there is a relationship between stress and eating pathology with 13 out of the 14 studies showing this link. Out of these 13 studies, six (Ball & Lee, 2002; Bekker & Boselie, 2002; Beukes et al., 2010; Blasse & Elklit, 2001; Cattanach et al., 1988; Chen et al., 2012) used a version of Cohen and colleagues PSS as a measure of stress (Cohen et al., 1988; Cohen & Williamson, 1983). These studies showed that those who reported DE behaviours also reported higher scores on the PSS. Similarly, studies which examined the relationship between feminine gender role stress and DE also revealed that stress experienced as a result of being a female is also related to dysfunctional eating behaviours (Bekker & Boselie, 2002; Martz et al., 1995; Mussap, 2007).

The cross-sectional link between stress and DE was also found when other measures of stress were used. For example, Cohen and Petrie (2005) used a 7-item measure assessing depression, happiness, shame, guilt, confidence, anxiety and stress with one item per construct (measure taken from Stice and Shaw, 1994) and found that higher stress levels were related to DE. Similarly, Fryer et al. (1997) used a version of the LES (Sarason et al., 1978) adapted for adolescents and showed that lower levels of stress are related to less disturbed eating behaviours. A recent study by Costarelli and Patsai (2012) also demonstrated that DE behaviours are higher during stressful periods. The authors measured eating behaviours during an exam period and a control period and found that female students reported higher levels of disturbed eating behaviours during an exam period compared to a control period. In addition, female students also reported consuming more highly palatable food items such as chocolate, crisps and ice-cream during the exam period (Costarelli & Patsai, 2012).

The studies which have examined the cross-sectional link between stress and DE seem to find a positive relationship when community-based (Ball & Lee, 2002; Beukes et al., 2010; Cattanach et al., 1988; Chen et al., 2012; Costarelli & Patsai, 2012; Fryer et al., 1997; King et al., 2009; Mussap, 2007; Shea & Pritchard, 2007) or clinical samples (Bekker & Boselie, 2002; Blasse & Elklit, 2001; Cohen & Petrie, 2005; Martz et al., 1995) are recruited even when different measures of stress are used.

From the studies which examined the relationship between stress and DE at one time point, only one study did not establish this link (Ball et al., 1999). This

exploratory study used a single item question taken from the Canadian Bureau of Statistics (Statistics Canada, 1991), which was "How often do you feel under stress?" and participants were required to respond on a 5-point Likert scale ("not applicable", "never", "sometimes", "often" and "all the time"). The results of this study did not find a significant correlation between perceived stress and either DE behaviours or concerns. The authors suggest that a reason for not detecting the expected relationship was a result of the low proportion of young women who reported DE behaviours in this sample with only 33% engaging in at least one DE behaviour. However, the authors also conducted some additional descriptive analyses to compare the DE behaviours of women who felt stressed "all the time" and women who "never" felt stressed. The findings showed that 47% of women in the high stress group reported engaging in at least one DE behaviour in comparison to only 18% of women in the low stress group. Ball et al. (1999) used this finding to suggest that high stress levels can be linked to DE. However, one potential problem with this final set of analyses is that the authors completely discarded those respondents who stated that they felt stressed "sometimes" and "often". Therefore, these middle range groups should have been used as a comparison group against the two extreme groups to establish if varying levels of stress are associated with DE.

Cross-sectional studies examining the role of stress on eating pathology have revealed that there is a relationship between stress and disturbed eating behaviours regardless of the measure of stress employed. However, as causality cannot be determined in cross-sectional studies it is possible that it is DE behaviours that actually drive increased levels of stress and not vice versa. Therefore, longitudinal studies which have examined stress and eating pathology over a period of time need to be appraised.

Stress and DE: Longitudinal findings

Compared to the number of studies that have investigated the cross-sectional relationship between stress and DE, far fewer studies have examined this link over time. Ball and Lee (2002) examined the role of psychological stress in a community sample of young women with symptoms of DE both cross-sectionally and at two time points, six months apart. Levels of perceived stress were examined using

the PSS-14 (Cohen et al., 1983) and DE behaviours were assessed using the EDI (Garner et al., 1983). To examine the role of stress on women with symptoms of DE, the researchers selected women who engaged in one or more of the five DE behaviours (binge eating, vomiting, fasting and misuse of laxatives or diuretics). Although cross-sectional findings of this study (as mentioned above) suggest that there is a link between stress and DE, the longitudinal analysis revealed that perceived stress levels at baseline did not make a significant contribution to DE scores at T2 (six months later) when controlling for baseline DE scores. However, DE scores at baseline were found to account for a significant proportion of the variance in perceived stress levels at T2 when modeled as an addition to perceived stress levels at baseline. Therefore, these findings suggest that the effects of perceived stress on DE may only be cross-sectional, only observable when examined over shorter periods or that stress may be a consequence rather than the cause of DE. Similarly, Berg et al. (2009) revealed that changes in academic stress experienced by students did not correlate with or predict changes in their DE behaviours over time.

Although, studies by Ball and Lee (2002) and Berg et al. (2009) examined stress and eating pathology at two-time points, it is still not possible to determine direction of causality. Striegel-Moore et al. (2007) followed 1,560 young girls from the age of 9-10 years old to the age of 20-21 and found that 45 of them developed BN or BED at some point over the 10-year period. Those with a maximum PSS-14 (Cohen et al., 1983) score before the age of 14 were more likely to develop an ED. These valuable findings reveal that stress is an important factor in the development of EDs.

A précis of the literature examining the role of stress and DE suggests that there is a cross-sectional relationship between stress and eating pathology but this relationship may not be as simple as stress causing DE when studies adopt a longitudinal design. However, only one longitudinal study which spans over a decade has been conducted indicating that the causal relationship may be as suggested by the cross-sectional studies with stress resulting in more DE behaviours (Striegel-Moore et al., 2007) but one study is not enough to make a definite case for this direction of causality. Therefore, the present review emphasises the need for more longitudinal studies to replicate and confirm these findings. Additionally, a limitation

of this area of research is that all of these studies have used self-report measures of stress, which do not provide an insight into the severity of the events that took place to induce high levels of stress. For example, the PSS-14 (Cohen et al., 1983) is a measure of the symptoms of stress rather than the presence of a stressor. Hence, research examining the role of life events in relation to DE will provide an insight into the severity of each event and the difficulties experienced over a specified time period.

Life events and DE

This section of the review will focus on the 25 studies that examined the role of stressful life events and difficulties on eating pathology. Out of the 25 studies located during the search process, five employed nonclinical samples (Bodell et al., 2011; Cooley et al., 2007; Loth et al., 2008; Smyth et al., 2008; Steinhausen et al., 2005) and the remaining 20 studies which examined the effect of life events on eating behaviours included clinical samples of ED participants (Berge et al., 2012; Blaase & Elklit, 2001; Fornari et al., 1994; Grilo et al., 2012; Karwautz et al., 2001, 2011; McFarlane et al., 2008; Mitchell et al., 1985; Nygaard, 1990; Pike et al., 2006, 2008; Raffi et al., 2000; Råstam & Gillberg, 1992; Rojo et al., 2006; Schmidt et al., 1997; Sharpe et al., 1997; Sohlberg, 1990; Taylor et al., 2003; Troop & Treasure, 1997; Welch et al., 1997). A small number of these studies (*n* = 4) which examined the role of life events on EDs were based on case studies (Fornari et al., 1994; McFarlane et al., 2008; Nygaard, 1990) or qualitative analysis (Berge et al., 2012). Although these studies did not employ a control group for comparison and hence, do not provide evidence for or against the relationship between negative life events and eating pathology, they do provide important information on the *types* of life events that were reported prior to onset of an ED (and so will be discussed on the basis of this).

A similarity between all of the studies located during the search process is that information on stressful life events were collected retrospectively and examined in relation to eating pathology in terms of events that occurred prior to interview, prior to the onset of the ED or prior to and surrounding relapse following recovery. Unlike the studies which examined the relationship between stress and DE,

retrospective studies examining the role of stress on dysfunctional eating patterns have found a more consistent relationship. This review will present the findings from this body of literature based on nonclinical samples, case notes of patients who were diagnosed with an ED and larger clinical samples to determine the role of life events on eating pathology.

The five studies (Bodell et al., 2011; Cooley et al., 2007; Loth et al., 2008; Smyth et al., 2008; Steinhausen et al., 2005) which employed nonclinical samples to examine the relationship between life events and DE were mostly supportive of this link. Smyth et al. (2008) used a self-report measure of trauma and adverse life events to examine the nature (such as death of a loved one, divorce/separation of parents and sexual or violent event), quantity and severity of traumatic events that took place prior to baseline on DE in students. The authors used the data on traumatic events to examine the relationship between stressful life experiences in relation to eating pathology at two time points, three months apart. Cross-sectional findings revealed that both trauma type and overall trauma severity significantly predicted both restricted eating and bingeing/purging at T1. Prospective analysis showed that overall trauma severity predicted both restrictive eating and bingeing/purging at T2. Also, various trauma types including death of a loved one, divorce/separation of parents and other trauma were found to significantly predict bingeing/purging at T2. These findings indicate that previously experienced traumatic events do have a lasting effect on later eating pathology.

Similarly, both Cooley et al. (2007) and Loth et al. (2008) showed that previously experienced life events are associated with DE scores even though these studies differed on the time period they covered when assessing life events (8 and 12 months, respectively). However, not all studies have revealed the same pattern of results. Bodell et al. (2011) examined the relationship between life events and eating pathology in 270 female students across two time points, 8 weeks apart. The study findings revealed that negative life events that occurred during the 8-week period between baseline and follow-up did not predict T2 symptoms of bulimia but the interaction between life events and low social support did. Steinhausen et al. (2005) also found inconsistent findings when comparing groups of adolescents who were at high risk for developing EDs with matched controls. The study consisted of three

time points with each phase consisting of a group of high-risk individuals and a group of matched controls. The findings indicated that the number of life events reported by the high risk group and the healthy controls did not differ at T1 but the groups at T2 and T3 did differ with high risk groups reporting more life events. There appears to be some inconsistencies in the findings based on nonclinical samples which may be due to the differences in the measures that have been used to assess life events. Out of the five studies, those that did not find a relationship between life events and DE used previously developed scales (Bodell et al., 2011; Steinhausen et al., 2005). Bodell et al. (2011) employed the Negative Life Events Questionnaire (NLEQ; Metalsky & Joiner, 1992) and Steinhausen et al. (2005) used an adapted version of the Life Events Scale (LES; Goodyer, 1990). However, although the remaining three studies found a more consistent relationship between traumatic experiences and eating pathology, these studies did not use validated measures of life events but instead developed a life events measure for their research needs (Cooley et al., 2007; Loth et al., 2008; Smyth et al., 2008). In addition, the findings of these studies must be taken with caution as all five studies recruited student samples which have been shown to report greater levels of disturbed eating patterns compared to nonstudent samples (e.g., Seymour, Hoerr & Huang, 1997). Also, as these studies did not recruit a clinical sample, it is not possible to determine if adverse life events are a causal factor in the development of EDs.

Unlike the studies which recruited nonclinical samples, the studies which examined the role of stressful experiences on eating behaviours in clinical samples presented some unequivocal findings. From the 16 studies which examined the role of adverse life experiences on eating pathology in clinical samples, 12 support this relationship (Blaase & Elklit, 2001; Grilo et al., 2012; Karwautz et al., 2001, 2011; Mitchell et al., 1985; Pike et al., 2006; Raffi et al., 2000; Rojo et al., 2006; Schmidt et al., 1997; Sohlberg, 1990; Troop & Treasure, 1997; Welch et al., 1997), two are partially supportive (Råstam & Gillberg, 1992; Taylor et al., 2003) and two are unsupportive of this link (Pike et al., 2008; Sharpe et al., 1997).

The studies which are supportive of the relationship between life events and eating pathology in clinical samples found similar proportions of patients reporting life events. Schmidt et al. (1997) found that 67% of AN patients and 76% of BN

patients experienced at least one negative life event or difficulty in the year before onset. In addition, although Schmidt et al. (1997) did not find any differences in the proportion of AN (43%) and BN (48%) patients who reported severe life *events* prior to onset when compared with healthy controls (32%), they did find that more patients reported *difficulties* that were rated high on the severity of threat/unpleasantness scale and had been experiencing this difficulty continuously for two or more years compared to controls. These findings are similar to that of Rojo et al. (2006) who found that although patients and controls did not differ in regards to life events, the ED group did report significantly more difficulties in the time preceding onset. Both study findings suggest that enduring long periods of stress is a precursor for the onset of EDs.

Also using the LEDS (Brown & Harris, 1978), Troop and Treasure (1997) reported similar rates of AN (58%) and BN (77%) patients reporting at least one severe life event or marked difficulty in the year preceding onset. Although the rate of severe events and difficulties in the control group was also very high (75%), this study was specifically interested in coping responses to stressful events and so deliberately biased its control group by advertising for participants who had experienced stress. It also included life events and difficulties over a 5-year period in controls versus 12 months in patients in order to identify stresses on which to compare groups in terms of their coping responses. Although Troop and Treasure (1997) did not have healthy controls for comparison and Schmidt et al. (1997) did not have control data for all of their variables of interest, these findings can be compared to a review of studies using the LEDS (Brown & Harris, 1978), which found that approximately 30% of women without psychopathology report a severe event or difficulty in any 12-month period (Brown & Harris, 1986). On that basis, the findings of Troop and Treasure (1997) and Schmidt et al. (1997) are also supportive of the link between life events and EDs. These findings are also comparable to that of Blaase and Elklit (2001) who reported that a high number of currently ill ED patients (85%) and those with a history of EDs (70%) indicated that they had experienced at least one negative life event in the six months prior to onset. Similarly, both Raffi et al. (2000) and Welch et al. (1997) found that experiencing stressful life events is a precipitating factor to the onset of BN. Importantly, twin studies have also

emphasized the importance of the environment such as the experience of negative life events as a risk factor for the development of EDs, specifically AN, even possibly over and above the influence of genetic factors (Karwautz et al., 2001, 2011).

The majority of the research that has been presented so far has focused on investigating the stressful life experiences that have preceded onset of EDs. However, it is also imperative to develop our understanding of the life events and difficulties that precede relapse following recovery from an ED. The present review located three studies which also assessed the role of life events on relapse following recovery from EDs (Grilo et al., 2012; Mitchell et al., 1985; Sohlberg, 1990). Sohlberg (1990) found that negative life events experienced during the follow-up year predicted worse outcome at follow-up in a sample of patients which consisted of different ED diagnoses. Similarly, other studies have also suggested that stressful life experiences may play an important role in triggering relapse following remission from EDs. Grilo et al.'s (2012) recent study used the Life Events Assessment (LEA; Pagano, Skodol, Stout, Shea, Yen, Grilo et al., 2004) to examine life events annually over a 6-year period. The authors found that 16 of the 35 BN patients and 34 out of the 82 EDNOS patients relapsed following remission and it was negative life events, in particular work-related and social/relationship stressors, that predicted relapse. Similarly, Mitchell et al. (1985) reported that 80% of their BN sample reported experiencing a stressful life event prior to their first relapse. However, the findings of both of these studies must be taken with caution as Grilo et al.'s (2012) findings are not generalisable to those with a diagnosis of AN and Mitchell et al.'s (1985) findings are based on a single interview question developed for the study.

However, not all studies were completely supportive of the role that life events may play in triggering onset or relapse following recovery from EDs. For example, although Råstam and Gillberg (1992) report that 14% of the AN group experienced a major life event (i.e., death of a parent or sibling, parent diagnosed with malignant tumor, assault including rape, loss of a close friend) prior to onset compared to the healthy controls, who did not report any similar events, for four of the patients, the adversity occurred either before they were born or before they were three months old. Similarly, Taylor et al. (2003) also reported inconsistent findings from a twin centre study which found that, over the course of the study, 32

girls developed BN (*n* = 1), partial syndrome BN (*n* = 26) and BED (*n* = 5). The results based on the sample recruited in Arizona indicated that those who developed an ED reported more life events compared to healthy controls but the sample recruited at the California site did not report any differences. However, a limitation of the study is that the sample of respondents who went onto develop a full ED was extremely small (*n* = 6) limiting the statistical power of the overall findings. Therefore, the findings of Råstam and Gillberg (1992) and Taylor et al. (2003) should be taken with caution.

Finally, out of the 20 studies which examined the impact of life events on eating behaviours in clinical samples of ED participants, only two did not find evidence for the relationship between life events and eating pathology. Pike et al. (2008) found that when the AN group and a psychiatric group which consisted of patients diagnosed with various conditions such as mood, anxiety or substance disorder were combined, they reported significantly more life events compared to healthy controls. Although the AN and psychiatric group did not differ on the number of negative life events reported, the AN group did report encountering significantly more critical comments about shape, weight or eating prior to onset. Similarly, Sharpe et al. (1997) found that the number of stressful life events only differed between the ED group and controls when events involving EDs were included. The authors classified ED related events as those that were related directly to the individuals' ED, for example, going to hospital for treatment (which of course cannot be considered causal as they would have occurred *after* onset).

Although the majority of the studies have demonstrated that stressful life experiences do play a role in triggering EDs or precede relapse following recovery from EDs, there are some general limitations and methodological variability that need to be highlighted. Firstly, life events research, in general, entails an element of vulnerability to recall bias as each individual's ability to remember events will differ. However, in comparison to studies which have recruited nonclinical samples to examine the relationship between life events and DE, a large number of the studies which recruited clinical samples have used previously developed measures (Grilo et al., 2012; Karwautz et al., 2001, 2011; Pike et al., 2006; Raffi et al., 2000; Rojo et al., 2006; Schmidt et al., 1997; Sohlberg, 1990; Troop & Treasure, 1997). Interestingly,

most of the studies that have used the LEDS (Brown & Harris, 1978) or the RFI (Fairburn et al., 1998) as a measure of life events were largely supportive of the link between negative life experiences and eating pathology (Karwautz et al., 2001, 2011; Pike et al., 2006; Rojo et al., 2006; Troop & Treasure, 1997; Schmidt et al., 1997).

Another limitation is the problem of defining the onset of EDs. Although some of the studies examining the role of life events and difficulties on EDs reviewed in this paper have provided an operational definition of onset of EDs (Schmidt et al., 1997; Troop & Treasure, 1997; Welch et al., 1997), these definitions have differed. For example, Schmidt et al. (1997) defined the onset of AN as the point at which BMI has reached 17.5kg/m^2 or the respondent has lost menses, whichever occurred first. In contrast, Troop and Treasure (1997) defined onset of AN as the start of weight loss where the individual continued to lose weight until a BMI less than 17.5kg/m^2 was reached and/or amenorrhea occurred. Studies have also used slightly different definitions of the onset of BN. Troop and Treasure (1997) and Schmidt et al. (1997) define the onset of BN as the point of when continuous bingeing had begun and continued for at least three months. For example, if an individual binged and purged a few times but then her eating behaviours normalized for a couple of months, this would not be enough to fulfill a diagnosis of BN. However, once binge eating behaviours begun and continued for at least three months, it was this episode that was taken as the onset of BN. In contrast, Pike et al. (2008) identified the age of onset as the point at which the individual first started to diet which continued for at least three consecutive months, overate at least once a week for three months or performed purging behaviours at least once a week for at least three months. Therefore, the lack of consensus on the definition of onset results in variability in the periods being assessed for life events hence, making it difficult to make comparisons between studies. However, despite these limitations, studies assessing life events and difficulties have shown that these experiences do play an important role in eating pathology even when different measures of life events are used.

Additionally, studies examining the role of adverse life events on eating pathology differ greatly on various design aspects, such as whether healthy controls were recruited for comparative measures, the number of patients and control participants recruited and the types of ED diagnoses that were used to form clinical

groups. Out of the 12 supportive studies, four studies did not recruit healthy controls for comparison (Grilo et al., 2012; Mitchell et al., 1985; Sohlberg, 1990; Troop & Treasure, 1997) and one study did not have comparison data for all of their variables of interest (Schmidt et al., 1997). Also, out of the studies which provided some evidence for the link between adverse experiences and eating pathology, seven focused on one type of ED (Karwautz et al., 2001, 2011; Mitchell et al., 1985; Pike et al., 2006 Raffi et al., 2000; Råstam & Gillberg, 1992; Welch et al., 1997), two studies compared those with AN to those with BN (Schmidt et al., 1997; Troop & Treasure, 1997) and five studies recruited a combination of patients to create an ED group (Blaase & Elklit, 2001; Grilo et al., 2012; Rojo et al., 2006; Sohlberg, 1990; Taylor et al. 2003). Similarly, the two studies which were unsupportive of this relationship, one study focused on life events and AN (Pike et al., 2008) whilst the other study created an ED group consisting of those diagnosed with AN or BN (Sharpe et al., 1997). There is also great variability in the sample sizes, with the number of patients recruited ranging between 22 (Blaase & Elklit, 2001; Sharpe et al., 1997) and 162 (Pike et al., 2006). Therefore, methodological differences in this area of research make comparisons between studies very difficult.

Although not all of the studies examining the relationship between stressful life events and eating behaviours have recruited healthy controls for comparison purposes (as mentioned above), some of the studies which have recruited controls have matched them to ED patients by age and sex (Karwautz et al., 2001, 2011; Pike et al., 2006; Raffi et al., 2000; Råstam & Gillberg, 1992; Rojo et al., 2006; Steinhausen et al., 2005; Welch et al., 1997). Therefore, despite the methodological inconsistencies in this area of research, the studies which have used a well-validated measure of life events (such as the LEDS [Brown & Harris, 1978] and the RFI [Fairburn et al., 1998]) and recruited healthy matched controls have provided strong evidence for the link between stressful life experiences and EDs (Karwautz et al., 2001, 2011; Pike et al., 2006; Rojo et al., 2006).

The studies that have been presented in the current review highlighted the importance of the stress process in DE behaviours as reported by nonclinical samples but also as a triggering mechanism of a variety of EDs including AN, BN, EDNOS and BED and even provoking relapse following recovery from EDs. An advantage of life

events research using interviewer-based measures such as the LEDS (Brown & Harris, 1978) allows researchers to understand the range and timing of events and difficulties experienced by an individual in relation to onset. This avoids problems with rationalisation, as individuals are not asked to provide an explanation of what may have caused their DE (more information regarding the utility of the LEDS will be provided in Chapter Seven).

In addition, life event and difficulties research also allows one to understand the *types* of events that are most commonly reported by those with disturbed eating behaviours. Both qualitative and quantitative studies provided an insight into the types of events that were commonly reported in the period preceding onset and relapse following recovery from EDs. Several studies reported events such death of a family member, relationship problems, changes in the home or at work, abuse and even more extreme adverse events such as witnessing a murder (Berge et al., 2010; Fornari et al., 1994; Grilo et al., 2012; McFarlane et al., 1988; Nygaard, 1990; Råstam & Gillberg, 1992; Rojo et al., 2006; Schmidt et al., 1997; Troop & Treasure, 1997). A qualitative study by Berge et al. (2012) also revealed six themes of events that preceded the onset of EDs. These include events such as school transition, death of a family member, relationship changes, illness/hospitalization, home and job transitions, and abuse, sexual assault or incest. Although qualitative and case studies do not employ comparison groups which limits the generalisaibility of findings, they do provide a wealth of knowledge regarding the types of events that are experienced by patients prior to clinical onset and relapse following recovery. Also, these findings are supported by Schmidt et al. (1997) who found that the most common problems that occurred prior to onset were interpersonal problems. Both AN (36%) and BN (41%) patients reported experiencing relationship difficulties with family and friends such as parental problems. These findings are comparable to Rojo et al. (2006) who found that 41% of ED participants reported experiencing at least one difficulty as a result of their relationship with their parents in comparison to 19% of the matched controls. Therefore, this review has demonstrated that adverse life events play an important role in eating pathology and that there are specific events that are prominent in triggering pathological eating patterns.

Summary

Overall, the current review suggests that the studies which have been published since the Ball and Lee (2002) review support the role of stress and negative life events on eating pathology. Similar to the findings of Ball and Lee (2002), the current review confirmed that stress and DE behaviours do coexist but longitudinal findings are very limited in establishing this link. However, only three studies have examined the longitudinal relationship between stress and DE (Ball & Lee, 1999; Berg et al., 2009; Striegel-Moore et al., 2007).

In contrast, studies which have examined the role of life events on dysfunctional eating behaviours are largely supportive of this relationship. In summary, three of the five studies (Cooley et al., 2007; Loth et al., 2008; Smyth et al., 2008) which recruited nonclinical samples confirmed that negative life experiences do play a role in eating pathology and similarly, 12 of the 20 studies (Blaase & Elklit, 2001, Grilo et al., 2012; Karwautz et al., 2001, 2011; Mitchell et al., 1985; Pike et al., 2006; Raffi et al., 2000; Rojo et al., 2006; Schmidt et al., 1997; Sohlberg, 1990; Troop & Treasure, 1997; Welch et al., 1997) which recruited clinical samples also support this relationship. Therefore, returning to the conclusions made by Ball and Lee (2000) following their review, this update of the literature in this area suggests that studies conducted since this review have improved in the methods that have been used to examine this relationship and are largely supportive of the notion that stress and negative life experiences do play a pivotal role in disturbed eating behaviours. Also, despite great methodological variability between these studies, the majority of the studies produced some consistent findings suggesting that these findings are particularly robust. In conclusion, this review suggests that these findings make a firm argument for the role of life events in triggering EDs. However, the current review located only three studies which examined the role of life events in triggering relapse following recovery from EDs (Grilo et al., 2012; Mitchell et al., 1985; Sohlberg, 1990). Therefore, this area of research needs more methodologically strong studies to determine if stress and adverse life events are precipitating factors to relapse following remission from EDs.

A final caveat that must be addressed is that the studies which have examined the role of stress and life events on eating behaviours mainly consist of all-

female samples with only eight out of the 40 studies employing a mixed sex sample (Berge et al., 2012; King et al., 2009; Loth et al., 2008; Nygaard, 1990; Råstam & Gillberg, 1992; Shea & Pritchard, 2007; Smyth et al., 2008 Steinhausen et al., 2005). Even where studies recruited both men and women, these consisted of over 80% women (Berge et al., 2012; King et al., 2009; Nygaard, 1990; Råstam & Gillberg, 1992; Steinhausen et al., 2005). Therefore, the findings that have been presented here are mainly based on the role of stress on eating pathology in women.

Table 2.4: Research studies examining the role of stress and life events on DE

Author	*N* *(% Female)*	Sample Characteristics	Assessment Measures	Study Design	Results	Support for Relationship
a) Studies examining the role of stress on DE						
Ball & Lee (2002)	T1: 552 T2: 415 (100)	T1: 552 DE: 254 Controls: 298 T2: 411 DE: 162 Controls: 249 (Age range = 19-24)	Stress: PSS-14 (Cohen et al., 1983) DE: EDI (Garner et al., 1983)	Cross sectional and at two time points	Cross-sectional findings: Compared with controls, DE groups reported higher stress levels at T1, $F(2, 550) = 77.21$, $p < .001$ and T2, $F(2, 413) = 71.55$, $p < .001$ Stress predicted DE in T1 (β= .42, $p < .001$) and T2 (β = .42, $p < .001$) Longitudinal findings: stress at T1 did not contribute to DE scores at T2 ($p > .05$)	Partial
Ball et al. (1999)	212 (100)	(Age range = 18-22)	Stress: Single item measuring perceived stress DE: EDE-Q (Fairburn & Beglin, 1994)	Cross-sectional	No significant correlations between stress and DE behaviours and concerns	No
Bekker &	88	BN: 36 (*M* age = 25.8)	Stress (used two): PSS-14	Cross-sectional	BN group reported more recently experienced	Yes

Boselie (2002)	(100)	Controls: 52 (*M* age = 21.2)	(Cohen et al., 1983) & FGRS (Gillespie & Eider, 1992) DE: EDI (Garner et al., 1983)		stress compared to controls, $t(2, 86) = -5.95$, $p < .001$ and stress predicted eating disordered behaviour, ($\beta = .47$, $p < .001$) BN group reported higher feminine gender role stress compared to controls, $t(2, 86) = 2.19$, $p = .03$ Fear of non-emotional relationships, $t(2, 86) = -2.44$, $p = .02$, and physical unattractiveness, $t(2, 86) = 5.46$, $p < .001$, predicted ED symptoms	
Berg et al. (2009)	186 (100)	Age range: 18-21	Stress: 6-item scale measuring academic stress developed for study DE: EDI-2 (Garner, 1991)	Two-time points (2 months apart)	Change in stress did not correlate or predict changes in DE	No
Beukes et al. (2010)	349 (100)	(*M* age = 18.9, *SD* = .9)	Stress: PSS-14 (Cohen et al., 1983) DE: EDI-3 (Garner, 2004)	Cross-sectional	Stress significantly correlated with drive for thinness ($r = .22$), bulimia ($r = .30$) and body dissatisfaction ($r = .26$)	Yes

Blaase & Elklit (2001)	75 (100)	Current ED: 22 (*M* age = 24.6, *SD* = 4.6) History of ED: 20 (*M* age = 26.0, *SD* = 4.3) Controls: 33 (*M* age = 26.2, *SD* = 5.8)	Stress: PSS-14 (Cohen et al., 1983) DE: EDI (Garner et al., 1983)	Cross-sectional	Current ED group reported higher levels of perceived stress compared to controls (Kruskal-Wallis' test $p < .001$)	Yes
Cattanach et al. (1988)	30 (100)	DE: 15 Control: 15 (Age range = 17-21)	Stress: PSS-14 (Cohen et al., 1983) DE: EDI (Garner et al., 1983) used to assign participants into groups	Cross-sectional	DE group reported more global perceived stress over the past month compared to control group, $t(2, 28) = 2.14$, $p < .05$)	Yes
Chen et al. (2012)	245 (100)	*M* age = 21.1, *SD* = 1.3	Stress: Chinese version of PSS-10 (Cohen et al., 1988) DE: EAT-26 (Garner et al., 1982)	Cross-sectional	Stress correlated with DE ($r = .31$, $p < .001$) No direct effect of stress on DE (standardized coefficient = –.01, 95% CI: –.04**,** .01, $p = 0.36$) Indirect effect of stress on DE via depression (Standardized indirect coefficient = .04, 95% CI: .02**,** .04, $p = 0.001$) and anxiety (Standardized indirect coefficient = .02, 95%	Partial

					CI: .01**,** .02, *p* **=** .01)	
Cohen & Petrie (2005)	334 (100)	ED: 32 Symptomatic: 130 Asymptomatic: 172 (*M* age 20.8, *SD* = 4.1)	Stress: 7-item scale assessing depression, happiness, shame, guilt, confidence, anxiety & stress with 1 item per affective state (Stice & Shaw, 1994) DE: Q-EDD (Mintz, O'Halloran, Mulholland & Schneider, 1997)	Cross-sectional	ED and symptomatic groups reported higher levels of stress compared to the asymptomatic group ($p \leq .005$)	Yes
Costarelli & Patsai (2012)	60 (100)	*M* age = 19.9, *SD* = 1.7	Stress: Exam period DE: EAT-26 (Garner et al., 1982)	Cross-sectional but assessed during exam and control period	Higher levels of DE during exam period compared to control period ($p = .01$)	Yes
Fryer et al. (1997)	286 (100)	*M* age = 14.7, *SD* = .6	Stress: Life Experiences Survey (LES; Sarason et	Cross-sectional	Stress negatively associated with DE ($r = -.42$, $p < .001$)	Yes

			al., 1978) adapted for adolescents DE: EAT-26 (Garner et al., 1982)		Regression models: Stress predicted DE (b = −.48 p = .0001) and this remained significant when self-esteem and perfectionism were included in the model (b = −.27, p = .0003) Path analysis: Stress predicted DE (standardised coefficient = .25, p < .05)	
King et al. (2009)	430 (95)	87% ≥ 31 years old 13% ≤ 30 years old	Stress: 12-item scale developed for study assessing different aspects of a nurses job used to assign participants into groups DE: 15-item scale developed for study	Cross-sectional	High job stress group had higher DE than low stress group, F(15, 402) = 1.81, p = .03	Yes
Martz et al. (1995)	355 (100)	ED: 12 Psychiatric inpatients: 33 Controls: 310	Stress: FGRS (Gillespie & Eider, 1992)	Cross-sectional	FGRS interacted with diagnostic category F(6, 338) = 2.27, p < .05 FGRS discriminated EDs from other psychiatric conditions t(354) = 3.6, p < .001 and healthy	Yes

					controls $t(354) = 2.9$, $p < .004$	
Mussap (2007)	124 (100)	*M* age = 24.5, *SD* = 4.9	Stress: FGRS (Gillespie & Eider, 1992) DE (several used): EDE-Q (Fairburn & Beglin, 1994); EDI-3 (Garner, 2004); QEWP (Spitzer Devline, Walsh, Hasin, Wing, Marcus et al., 1992)	Cross-sectional	Overall feminine gender role stress correlated with overcontrol ($r = .27$), body dissatisfaction ($r = .41$), dietary restraint ($r = .27$) and eating concerns ($r = .30$) Body dissatisfaction and overcontrol mediate the relationship between fear of unattractiveness and eating concerns and binge eating ($p < .01$) Body dissatisfaction and overcontrol mediate the relationship between fear of assertiveness and eating concerns ($p < .01$)	Yes
Shea & Pritchard (2007)	459 (57)	*M* age = 23.2, *SD* = 7.0	Stress: Inventory of College Student Recent Life Experiences (Kohn, Lafreniere & Gurevhich, 1990) DE: EDI (Garner et al.,	Cross-sectional	Stress predicted bulimic symptoms ($\beta = .12$, $p < .01$) and body dissatisfaction ($\beta = .10$, $p < .05$) but not drive for thinness	Partial

			1983)			
Striegel-Moore et al. (2007)	1560 (100)	History of BN: 20 History of BN/BED: 25 Controls: 1,515 (*M* age = 21.4, *SD* = .74)	Stress: PSS-14 (Cohen et al., 1983) DE: Screening Interview for Eating Disorders (SIED; Striegel-Moore, Wilfley, Pike, Dohm & Fairburn, 2000)	Longitudinal	Those with a stress score > 40 before the age of 14 were more likely to develop BED/BN 39 BED/BN group scored > 40 on the PSS-14 before the age of 14 compared to only 6 BED/BN group who scored < 40	Yes
b) Studies examining the role of life events on DE						
Berge et al. (2012)	27 (96)	AN: 9 AN/BN: 1 BN: 3 EDNOS: 14 (Median age = 27, *M* age = 34.6, *SD* = 13.7)	Life events: semi-structured interview assessing social and environmental factors developed for study	Retrospective (Covered complete history)	Qualitative analysis revealed 6 themes of events preceding onset of EDs including school transition, death of a family member, relationship changes, home and job transitions, illness/hospitalization and abuse, sexual assault or incest	Yes
Blaase & Elklit (2001)	75 (100)	Current ED: 22 (*M* age = 24.6, *SD* = 4.6) History of ED: 20 (*M*	Life events: Measure developed for study which assessed stressful	Retrospective (Covered complete	Compared to controls, more women with a history of EDs and those currently with an EDs reported life events during childhood, χ^2(df =	Yes

		age = 26.0, *SD* = 4.3) Controls: 33 (*M* age = 26.2, *SD* = 5.8)	life events experienced since childhood DE: EDI (Garner et al., 1983)	history)	2) = 6.88, *p* <. 05 and had experienced more than one or chronic event χ^2(df = 2) = 6.23, *p* <. 05 High proportion of current ED group (85%) and those with a history of EDs (70%) reported an adverse life event in the 6 months prior to onset	
Bodell et al. (2011)	270 (100)	*M* age = 18.7, *SD* = 1.4	Life events: NLEQ (Metalsky & Joiner, 1992) DE: EDI (Garner et al., 1983)	Two time points (Covered life events during 8 weeks between T1 and T2)	Negative life events did not predict bulimia at T2 (β < .001, *t* = .004, *p* = 1.00) Interaction between life events and low social support predicted T2 bulimic symptoms (β = −.10, *t* = −2.12, *p* = .04) but not on restrictive tendencies (β = .001, *t* = .02, *p* = .98)	Partial
Cooley et al. (2007)	117 (100)	Aged 18 years old	Stress: Negative Life Events Scale developed for study DE: Composite Eating	Retrospective (Covered 8 months prior to baseline)	T1 life events correlated positively with baseline DE (*r* = .33) and DE at follow-up (*r* = .25) T1 life events predicted DE at follow-up (β =	Yes

			Disorders Scale (Klemchuk, Hutchinson & Frank, 1990)		.28, *p* = .004)	
Fornari et al. (1994)	3 (100)	AN: 3 (aged 32, 35 and 39)	Case studies	Retrospective (Covered complete history)	Life events reported include witnessing a murder at the age of 16, severe physical abuse as a child and beginning a strict exercise regime after being told that "everything falls apart at 40"	Yes
Grilo et al. (2012)	117 (100)	BN: 35 EDNOS: 82 (*M* age 31.2, *SD* = 8.1)	Life events: LEA (Pagano et al., 2004)	Retrospective (Covered 6 months and 12 months prior to interviews)	Negative life events predicted ED relapse (hazard ratio (HR) = 1.52, $p < 0.04$) in particular, increased work (HR = 3.04, $p < 0.01$) and social/relationship stressors (HR) = 3.13, $p < 0.04$)	Yes
Karwautz et al. (2001)	90 (100)	AN Twin = 45 (*M* age = 27.7, *SD* = 8.5) Healthy control twin = 45 (*M* age = 27.4, *SD* = 9.7)	Life events: RFI (Fairburn et al., 1998)	Retrospective (Covered complete history to onset)	Twin sisters diagnosed with AN revealed a greater level of exposure to disruptive events prior to onset compared with the healthy twin sisters ($p < .05$)	Yes

Karwautz et al. (2011)	256 (100)	AN Twin = 128 (*M* age = 25.4, *SD* = 8.1) Healthy control twin = 45 (*M* age = 25.9, *SD* = 8.7)	Life events: RFI (Fairburn et al., 1998)	Retrospective (Covered complete history to onset)	Disruptive events and interpersonal problems preceding AN onset increased the risk independently of genetic factors (p = .02)	Yes
Loth et al. (2008)	1708 (55)	*M* age = 20.4, *SD* = .8	Life events: 12-item scale measuring life events developed for study DE: Scale measuring DE behaviours developed for study	Retrospective (Covered a 12-month period between T1 and T2)	Number of stressful life events positively related to DE at T2 (p < .05)	Yes
McFarlane et al. (1988)	3 (100)	AN: 3 (Aged 11, 23 and 26)	Case studies	Retrospective (Covered complete history)	Reported life events such as rape and bushfire disaster destroying home	Yes
Mitchell et al. (1985)	30 (100)	BN: 30 (*M* age = 24.8)	Life events: single interview question developed for study	Retrospective (Covered from end of	80% reported experiencing a stressful or difficult situation prior to first relapse	Yes

				treatment to relapse)		
Nygaard (1990)	84 (94)	AN = 84 (55/84 were < 16 years old)	Case studies	Retrospective (Covered complete history to onset)	Events such as loss of a close family member, problems at school of work, broken homes, abortion, moving to another environment and martial problems reported to trigger AN	Yes
Pike et al. (2008)	150 (100)	AN = 50 (*M* age = 26.7, *SD* = 6.2) Psychiatric matched controls = 50 (*M* age = 27.0, *SD* = 6.1) Healthy controls = 50 (*M* age = 26.6, *SD* = 5.5)	Life events: RFI (Fairburn et al., 1998)	Retrospective (Covered complete history to onset)	Groups did not differ on events such as separation from parent (p = .47) or parental absence or death (p = .07) prior to onset AN and psychiatric controls (combined) reported more life events in the year preceding onset compared to healthy controls (p = .001)	Partial
Pike et al. (2006)	538 (100)	Group 1: 162 BED (*M* age = 30.8, *SD* = 5.8) & 162 Non-psychiatric	Life events: RFI (Fairburn et al., 1998)	Retrospective (Covered 12 months prior	BED group reported more life events preceding onset compared to non-psychiatric controls (Stuart Maxwell χ_3^2=64.19, p < 0.001)	Yes

		matched controls (*M* age 30.0, *SD* = 5.6) Group 2: 107 BED (*M* age = 30.6, *SD* = 5.9) & 107 Psychiatric matched group (*M* age = 29.5, *SD* = 6.7)		to onset)	BED group reported more life events preceding onset compared with psychiatric controls (Stuart Maxwell χ_3^2=22.28, $p < 0.001$)	
Raffi et al. (2000)	60 (100)	BN: 30 Controls: 30 (*M* age = 26.2, *SD* = 5.1)	Life events: Interview for Recent Life Events (Paykel, 1997)	Retrospective (Covered 6 months prior to onset)	BN reported 46 events compared to 24 events reported by controls, $t(58) = 2.38$, $p < .05$ Contextual threat rating showed that 26 events experienced by BN were negative compared to only 9 events reported by controls, $t(58) = 2.64$, $p < .05$	Yes
Råstam & Gillberg (1992)	102 (94)	AN: 51 (*M* age = 16.0) Controls: 51 (*M* age = 16.2)	Life events: paper does not provide details regarding assessment tool used	Retrospective (Covered 3 months prior to onset)	No differences in the number of chronic events reported by AN & controls 7 AN experienced death of father, brother or sister prior to onset of AN compared to no reporting of such events by controls ($p < .01$)	Partial

					7 AN experienced major life events 3 months prior to onset compared with controls who did not report any such events (p <. 01)	
Rojo et al. (2006)	64 (100)	EDs: 29 females (M age = 15.0, SD = 1.8) & 3 males (M age = 14.3, SD = 1.5) Matched controls: 32	Life events: LEDS (Brown & Harris, 1978) DE: EAT-40 (Garner & Garfinkel, 1979)	Retrospective (Covered 12 months prior to onset)	6% (2/32) of the EDs group reported at least one severe event compared to 0% (0/32) of the control group (nonsignificant) EDs group reported higher average number of difficulties (M = 2.88, SD = 1.99) than controls (M = 1.26, SD = 1.05), t(62) = 4.31, p < .001	Yes
Schmidt et al. (1997)	129 (100)	AN: 72 (Age range = 13-42) BN: 29 (Age range = 16-29) Controls: 28 (Age range = 18-35)	Life events: LEDS (Brown & Harris, 1978)	Retrospective (Covered 12 months prior to onset)	No difference between groups in relation to the proportion of individuals who reported at least one severe event (p = .44) More EDs individuals (AN = 22% and BN = 34%) reported at least one major difficulty compared to the controls (4%) (p = .02) No difference between AN and BN groups (data only available for DE groups) in the proportion of those who had experienced at	Yes

					least one marked difficulty (p = .19) or at least one severe event or marked difficulty (p = .75)	
Sharpe et al. (1997)	71 (100)	EDs: 13 AN & 9 BN (*M* age = 15.9, *SD* = 1.7) Controls: 49 (*M* age = 16.1, *SD* = 1.2)	Life events: Life Events and Coping Inventory (LECI; Dise-Lewis, 1988)	Retrospective (Covered complete history)	ED group reported more stressful life events reported compared to controls, $t(69) = -1.80$, p = .08, but significance disappeared when ED events were excluded	No
Smyth et al. (2008)	249 (55)	(Age range = 18-19)	Life events: self-report of trauma and adverse event history developed using categories derived from Smyth, Hockemeyer, Heron, Wonderlich and Pennebaker (2008) DE: EDE-Q (Fairburn & Beglin, 1994)	Retrospective (Covered complete history)	Trauma type predicted restricted eating at T1, $F(6, 242) = 3.30$, p = .004 and at T2, $F(7, 211) = 71.42$, $p < .0001$. Trauma type predicted bingeing/purging at T1, $F(6, 242) = 5.85$, $p < .001$ and at T2, $F(7, 211) = 26.62$, $p < .001$ Overall trauma severity predicted T1 restrictive eating, $F(3, 238) = 8.10$, $p < .001$ and bingeing/purging, $F(3, 238) = 5.43$, p = .001 Overall trauma severity predicted bingeing/purging at T2, $F(4, 208) = 30.47$, $p <$	Yes

					.001 and restrictive eating at T2, $F(4, 208) = 91.35$, $p < .0001$ but relationship entirely accounted for by restricted eating at T1, $t = 18.39$ $p < .001$	
Sohlberg (1990)	37	AN: 8 AN/BN: 2 BN: 19 EDNOS: 8 (*M* age = 24.2, *SD* = 6.6)	Life events: shortened version of the PERI Life Events Scale (Dohrenwend, Askenasy, Krasnoff & Dohrenwend, 1978)	Retrospective (Covered 12 months prior to 1- and 2-year follow-up)	Events that occurred leading up to the 1-year follow-up correlated with the presence of an ED (r=.30, $p < .05$) and with overall ED status (r=.32, $p < .05$). Similar findings found at 2-year follow-up	Yes
Steinhausen et al. (2005)	T1: 128 (77%) T2: 504 (81%) T3: 328 (86%)	T1: 64 high-risk (*M* age = 15.0, *SD* = 1.0) & 64 matched controls (*M* age = 14.8, *SD* = 1.0) T2: 252 high-risk (*M* age = 16.2 *SD* = 1.5) & 252 matched controls	Life events: 36 items selected from the LES (Goodyer, 1990) DE: EDE-S (Beglin & Fairburn, 1992)	Retrospective (Covered 12 months prior to interview)	High-risk and controls did not differ on total life events score at T1, $F(df = 1) = 2.99$ but did differ at T2, Kruskal–Wallis $\chi^2(df = 1) = 11.52$, $p < .001$ and at T3, $F(df = 1) = 4.05$, $p < .05$ High-risk groups reported higher unpleasantness scores at T2, Kruskal–Wallis $\chi^2(df = 1) = 18.05$, $p < .001$ and at T3, Kruskal–Wallis $\chi^2(df = 1) = 15.82$, $p < .001$ but not at T1,	Partial

		(*M* age = 16.0, *SD* = 1.5) T3: 164 high-risk (*M* age = 19.7, *SD* = 1.6) & 164 matched controls (*M* age = 19.6, *SD* = 1.6)			*F*(*df* = 1) = 3.54	
Taylor et al. (2003)	1,103 (100)	BN: 1 Partial syndrome bulimia: 26 BED: 5 (Students in Grades 6-9 – typically aged 11-15)	Life events: McKnight Risk Factor Survey IV (Shisslak, Renger, Sharpe, Crago, McKnight, Gray et al., 1999) DE: McKnight Eating Disorder Examination is a semi-structured interview based on the EDE (Cooper et al., 1989)	Longitudinal (Covered complete history)	Arizona site (*n* = 19): change in negative life events predicted onset of EDs (*W* = 9.1, *p* < .01) and new onset EDs reported more life events (*M* = .39, *SD* = 1.2), whereas healthy controls showed a decrease (*M* = –.25, *SD* = 1.0; effect size for differences = .65) California site (*n* = 13): above findings not replicated	Partial
Troop &	32	AN: 19 (*M* age = 23.3)	Life events: LEDS (Brown	Retrospective	77% of BN patients and 58% of AN group	Yes

Treasure (1997)	(100)	BN: 13 (*M* age = 25.4)	& Harris, 1978)	(Covered 12 months prior to onset)	reported a severe event or marked difficulty	
Welch et al. (1997)	306 (100)	BN: 102 Matched controls: 204 (*M* age = 23.7)	Life events: semi-structured interview designed for study	Retrospective (Covered 12 months prior to onset)	Greater number of events related to increased likelihood of being bulimic, χ_1^2 for linear trend = 17.48, *df* = 1, *p* < .001	Yes

Note. APES = Adolescent Perceived Events Scale (Compas et al., 1987); DSP = Derogatis Stress Profile (Derogatis, 1987); EAT-40 = Eating Attitudes Test-40 (Garner & Garfinkel, 1979); EAT-26 = Eating Attitudes Test-26 (Garner et al., 1982); EDE = Eating Disorder Examination (Cooper et al., 1989); EDE-S = Eating Disorder Examination Screening (Beglin & Fairburn, 1992); EDE-Q = Eating Disorders Examination Questionnaire (Fairburn & Beglin, 1994); EDI = Eating Disorders Inventory (Garner et al., 1983); EDI-2 = Eating Disorder Inventory-2 (Garner, 1991); EDI-3 = Eating Disorder Inventory-3 (Garner, 2004); FGRS = Feminine Gender Role Stress (Gillespie & Eider, 1992); LEA = Life Events Assessment (LEA; Pagano et al., 2004); LEDS = Life Events and Difficulties Schedule (Brown & Harris, 1978); NLEQ = Negative Life Events Questionnaire (Metalsky & Joiner, 1992); PSS-14 = Perceived Stress Scale-14 (Cohen et al., 1983); PSS-10 = Perceived Stress Scale-10 (Cohen, 1988); Q-EDD = Questionnaire for Eating Disorder Diagnosis (Mintz et al., 1997); QEWP = Questionnaire of Eating and Weight Patterns-Revised (Spitzer et al., 1992); RFI = Oxford Risk Factor Interview (Fairburn et al., 1998)

Systematic review summary

The present systematic review provides an appraisal of the research that has examined the role of stress on weight regain in obesity and on eating pathology.

Amongst the four papers that examined the relationship between stress and weight regain in obesity, only one paper revealed that stress may hinder the weight loss maintenance process (DePue et al., 1995). However, the authors did not actually assess stress levels as these findings were based on participant ratings on what they viewed to be an obstacle to their weight loss maintenance efforts (DePue et al., 1995). Furthermore, studies that assessed stress levels in relation to weight regain reported null findings (Bond et al., 2009; Dohm et al., 2001).

In contrast to the area of research focusing on *stress* and weight regain, the current review was able to locate more studies which examined the role of *adverse life events* on the failure to successfully maintain weight loss. However, this body of research revealed some inconsistent findings. Some studies demonstrated that weight regainers reported a greater number of negative life events compared with those who successfully maintained their weight losses (Byrne et al., 2003; Dubbert & Wilson, 1984; Gormally & Rardin, 1981; Kayman et al., 1990; Tinker & Tucker, 1997; Turk et al., 2012) whereas other studies did not report such differences (Byrne et al., 2004; Foster et al., 1996; Hartz et al., 1979; Jeffery et al., 1984b; Marston & Criss, 1984).

The inconsistent findings of studies examining the role of stress and life events on weight regain may be a result of two limitations in this area of research. Firstly, only two of four studies examining the role of stress and three of the twelve studies examining negative life events used validated measures (Bond et al., 2009; Foster et al., 1996; Gormally et al., 1980; Sarlio-Lähteenkorva et al., 2000; Tinker & Tucker, 1997) whilst the other studies developed measures for their research needs. Secondly, several definitions of weight regain have been used by studies discussed in this review. These definitions include regaining ≥ 5kg (Bond et al., 2009; Foster et al., 1996; Turk et al., 2012), ≥ 10lbs (DePue et al., 1995; Dohm et al., 2001) and even gaining more than the weight that was originally lost (Sarlio-Lähteenkorva et al., 2000). Although Crawford et al. (2000), Foster and Kendall (1994) and Wing and Hill (2001) provide researchers with definitions of weight regain, the majority of

researchers have not adopted these recommendations. Therefore, future research should use a clear and consistent definition of weight regain when examining the factors related to unsuccessful weight loss maintenance.

The second section of this review presented the literature examining the role of stress and life events on DE behaviours and EDs. A previous review by Ball and Lee (2000), which provided a thorough appraisal of the literature published between 1954 and 1995, suggested that there is a relationship between stress and eating pathology. However, the authors did propose that longitudinal, prospective studies with large samples were required to clarify some of the inconsistencies in the literature and confirm this relationship (Ball & Lee, 2000). Therefore, the present review located research studies which were conducted post-1995 to determine if studies that have been conducted in the last 18 years also reported similar findings.

Firstly, the research studies that have investigated the influence of perceived stress on DE suggest that there is a strong, concurrent link between stress and DE. Despite the use of different measures to assess stress and DE, all but one study (Ball et al., 1999) in this literature review displayed strong cross-sectional evidence for the relationship between stress and DE. Therefore, it is possible to conclude that people with DE report higher levels of stress in comparison to those individuals who do not display eating pathology. However, studies which examined stress and DE behaviours over two time points indicate that stress at baseline does not predict DE behaviours at follow-up (Ball & Lee, 2002) and changes in stress do not predict changes in eating pathology (Berg et al., 2009). Similarly, observational studies that consist of two time points still do not provide information regarding the direction of causality in this relationship. Overall, these studies suggest that stress and DE may coexist and the direction of causality in this relationship may not be as expected. However, longitudinal studies which assess stress and eating behaviours at multiple occasions would shed light on this association. Therefore, longitudinal studies that map both changes in stress and changes in dysfunctional eating patterns are needed to determine if it is the change in stress that predicts changes in DE or vice versa. The present review was only able to locate one longitudinal, prospective study which examined the influence of stress on eating pathology and showed that high levels of stress experienced by adolescents can increase the risk of developing EDs (Striegel-

Moore et al., 2007). However, caution must be taken when applying these findings to the entire spectrum of EDs as the study examined the risk factors for binge eating disorders and not other EDs such as AN and EDNOS (Striegel-Moore et al., 2007). Hence, although cross-sectional findings suggest that there is a relationship between stress and DE, more longitudinal studies are required to determine whether stress drives eating pathology or vice versa.

The current review also evaluated research studies that investigated the role of life events and difficulties on disturbed eating behaviours. Unlike the studies which have examined the role of stress on DE, research which has explored the relationship between negative life events and eating pathology have demonstrated that stressful life events and difficulties do play an important role in the onset of eating pathology and can even be suggested to have an enduring influence (Smyth et al., 2008). This relationship has been demonstrated by twin studies (Karwautz et al., 2001, 2011), eating disordered samples in treatment (Berge et al., 2012; Blasse & Elklit, 2001; Pike et al., 2006; Råstam & Gillberg, 1992; Schmidt et al., 1997; Troop & Treasure, 1997), community-based samples with BN (Raffi et al., 2000; Taylor et al., 2003; Welch et al., 1997) and non-eating disordered samples that report DE symptoms (Cooley et al., 2007; Loth et al., 2008; Smyth et al., 2008). Importantly, these studies have also revealed that the most frequently reported events that precede the onset of ED are interpersonal problems. Karwautz et al. (2011), Rojo et al. (2006), Schmidt et al. (1997) and Troop and Treasure (1997) all found a large proportion of respondents with an ED reporting relationship problems with family and friends. This is consistent with research on difficulties in affect regulatory processes which are reviewed in the next chapter.

Therefore, it is possible to conclude that these findings strengthen the overall conclusions proposed by Ball and Lee (2000). There is a relationship between stressful life experiences and eating pathology but studies examining the role of stress in clinical samples need to become more consistent in their definition of onset of EDs in order for comparisons between studies to be possible. In addition, more studies are needed to assess the role of stressful life events on relapse following remission from EDs as the current review located only three studies examining this relationship (Grilo et al., 2012; Mitchell et al., 1985; Sohlberg, 1990).

In conclusion, both stress and life events have been shown to play a role in weight regain and eating pathology, although the effect of stress on DE is stronger compared to the evidence that stress influences weight regain. The latter studies produced some inconsistent findings due to poor methodologies. However, despite these inconsistent findings, subsequent research should examine the factors that may influence the relationship between stress and weight regain and DE symptoms. Previous research has mainly focused on the role of stress and coping on DE (e.g., Ball & Lee, 2000; Troop, Holbrey & Treasure, 1998) but have not accounted for other mediating and moderating effects. Factors such as mood, affect systems (i.e., social rank and attachment insecurity) and self-criticism/reassurance could also have an influence on the stress process. The next chapter will provide a narrative review of how these affect systems and affect regulatory processes can contribute to the link between stress and problematic weight regulation.

Chapter 3: Affect regulation and problematic weight regulation

Introduction

This chapter will provide a narrative review of the literature examining the role of affect systems and affect regulation processes in relation to the stress process and, thus, how they may contribute to the relationship between stress and problematic weight regulation described in the previous chapter. The affect systems and processes that will be evaluated in the current review are social rank, insecurity of attachment in adulthood and self-criticism/reassurance. Before providing a summary of the historical background of affect regulation, this chapter will describe the core model for affect regulation that has provided the basis for the studies that will be presented in this thesis.

Early work based on evolutionary theory suggested that our ultimate biosocial goal is to survive in an environment that can provide us with warmth and safety but can also threaten our well-being (Gilbert, 1995). Gilbert (2005) proposed the Social Mentality Theory which posits that our ability to survive stems from an interaction between our motives, emotions, behaviours and information processing abilities. Gilbert (2005) suggested that the array of interactions lead the way to "different patterns of neurophysiological activity" (p. 15) known as social mentalities that aid the ability to form and operate in reciprocal relationships (Liotti & Gilbert, 2011). These social mentalities include the ability to form relationships with those who seek our care and to whom we provide care (infant-carer interactions) in order to enhance their chances of survival (e.g., an infant elicits care from a caregiver by smiling which represents affiliative signals or sends out distress calls which indicates that the infant is seeking the carer). These types of social mentalities refer to the attachment bond that develops between an infant and its caregiver. Similarly, the social ranking mentality is used to form relationships that allow us to compete for limited resources and maintain our status in the social environment (Gilbert, 2005). These social mentalities form the basis for different types of positive affect systems.

Gilbert (2005) proposed an affect regulation system based on an interactive model consisting of the threat-defence system and two positive affect systems (*see Figure 3.1).* Positive affect refers to one's high level of engagement with one's social environment (Gilbert, Allan, Brough, Melley & Miles, 2002). These two positive affect systems capture resource/achievement- and affiliation-focused processes whilst the threat-focused component of this model refers to behaviours that are activated in response to threat and harm. The two positive affect systems, which are known as social rank and attachment, respectively, form the basis of this review. Although the model emphasizes reciprocal effects between these systems, the positive affect systems have been suggested to encourage the activation and immobilization of the threat-defence system. The threat-defence system can be activated in response to perceived and actual danger. For example, when efforts to be valued by others are unsuccessful, this can result in a perception of low social rank which can activate the threat-defence system. However, a well developed attachment system at times of threat can promote the ability to self-soothe by easily recalling memories of the safe and loving environment provided by the caregiver in childhood which can alleviate feelings of threat (Gilbert, 2005).

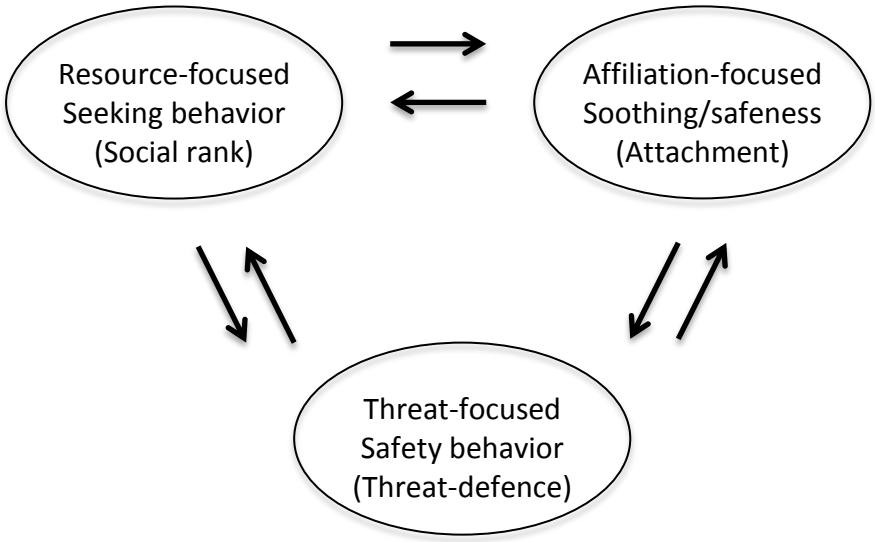


Figure 3.1: Affect regulation system (*source: adapted from Gilbert, 2005, p. 26*)

Social rank

This first section of the narrative review will provide an overview of the history of social rank and the historical and modern day research that has shown how social rank can influence human emotion and behaviour. For the purpose of this review, the term social rank system will be used throughout although some researchers have referred to this as the Dominance Behavioural System (DBS; Johnson, Leedom & Muhtadie, 2012; Zuroff, Fournier, Patall & Leybman, 2010).

The construct of social rank is based on the early works of Festinger (1954) who proposed social comparison theory as way of explaining behaviour in the social world. Social comparison theory posits that humans have an innate drive to perform evaluative judgements to obtain information regarding their abilities and own opinions in order to determine how they compare to others in their social environment (Festinger, 1954). Festinger (1954) proposed that humans prefer to be objective in their comparisons by striving to obtain an accurate and fair comparison. They do this by comparing themselves to those who are similar to them, but sometimes they engage in upward and downward comparisons which serve different functions (Festinger, 1954; Taylor & Lobel, 1989). Engaging in an upward comparison involves comparing oneself to someone who is more capable which serves to motivate the individual to improve, whilst the drive to compare oneself to those who are less fortunate (downward comparison) is a self-enhancement and coping strategy (Festinger, 1954; Taylor & Lobel, 1989). Additionally, our social behaviours differ depending on the circumstances that surround us, for example, at times of adversity such as when we encounter conflict or threat, we are more likely to make downward comparisons in order to enhance our well-being (Taylor & Lobel, 1989; Wills, 1981).

The evolutionary perspective suggests that social comparison strategies are adaptive features in a world where species strive for the same goals and limited resources (Buss, 1991). Classic ethological findings by Lorenz (1981) have provided an insight into how species use ritual agonistic behaviours (RAB) to determine social hierarchies and promote group cohesiveness within a competitive environment. RAB is a process of signaling behaviour to determine a social hierarchy by creating or reinforcing the asymmetry of social relationships within the group (Lorenz, 1981).

Although RAB is a survival mechanism which maintains social order in an environment with limited resources and multiple opponents, this process of organising the group into a social hierarchy can also have adverse effects on well-being. An example of this has been documented when two hens are introduced into the same environment, a pecking fight commences to determine one winner and one loser. If the losing hen is blocked from escaping the situation, it exhibits depressive behaviours such as drooping wings and head in the dust (Price & Sloman, 1987; Price, Sloman, Gardner, Gilbert & Rohde, 1994). Price and colleagues (1987, 1994) applied these findings to the aetiology of depression and hypothesized that depressive symptomatology is a consequence of this loss of rank and the inability to escape. When the option to flee a stressful situation is prevented (i.e., arrested flight; Gilbert, 2001), this can result in a sense of helplessness (referred to as learned helplessness) which has been established as a fundamental contributing factor to the development of depression (e.g., Abramson, Seligman & Teasdale, 1978; Miller & Seligman, 1975).

Gilbert (1997) referred to rank as an individual's position in a hierarchical structure which consists of dominant and subordinate individuals at different levels of the system. Strategies used by humans to gain rank and status can be either threat or attraction-based (Gilbert, 1997). Parker (1974) described Resource Holding Power (or Potential; RHP) which requires the use of fear eliciting social signals such as aggression and intimidation to demonstrate fighting ability and to prevent lower rank opponents from fighting for limited resources. However, throughout history, the use of aggression and hostility to obtain resources and social status has become less crucial for survival in humans (Gilbert, 1997). Humans use attributes that are socially attractive to others by using what Gilbert (1997) referred to as Social Attention Holding Power (SAHP). SAHP (Gilbert, 1997) is an attraction-based approach which involves a display of positive social behaviours designed to obtain resources and positive social rewards such as praise and acceptance and is used to avoid being rejected or ostracised if viewed as being inferior to others (Gilbert, 2005). The overarching aim of using SAHP is to gain and maintain vital resources such as mates and one's own position in the social hierarchy (Gilbert, Price & Allan, 1995). Evolution has shaped the human brain not only to recognise and respond to

social cues but also to have an innate desire to stimulate positive affect in others in order to form attachment bonds and cooperative relationships (Gilbert, 2005).

Gilbert (2001) suggested that there are two types of estimating one's SAHP, which he referred to as internal and external. The external method of estimating one's SAHP is based on how much attention and interest we can arouse from others, which may be positive or negative. The internal method refers to estimating our SAHP by focusing on our own attributes which we may like or dislike (i.e., our judgements about our own relative attractiveness and competence; Gilbert, 2001). However, when RHP and (internal and/or external) SAHP are not successful in provoking the desired response, defensive mechanisms such as depression can ensue as a result of feeling inferior to others or perceiving oneself to be of a lower rank compared to others (Gilbert, 1997).

However, it is not just how others in our social world perceive us that is important for the way we compare ourself to others but also how much we invest in others and how much others invest in us. This refers to our Social Investment Potential (SIP; Allen & Badcock, 2003). Humans have the capacity to ensure that their SIP does not exceed what we provide to others and when we perceive that we are investing more in a relationship than the other person, we will either demand more investment from the other person or limit our investment. In the same way that perceiving oneself to be of a lower rank to others can increase vulnerability to psychopathology, behaviours related to SIP can also result in defeat or rejection and lead to psychopathology such as depressive symptomatology and social withdrawal (Allen & Badcock, 2003).

Research has shown that perceived low social status is associated with various psychological and behavioural characteristics including personality traits, the ability to communicate effectively and even alcohol consumption. Cancer patients who score high on neuroticism report comparing themselves to other patients who are doing worse (engaging in downward comparisons), whereas those who indicate an extraverted personality have the tendency to compare themselves to those who are doing better than themselves (Van Der Zee, Buunk, Sanderman, Botke & Van Den Bergh, 1999). Studies have shown that both upward and downward comparisons can relate to positive and negative affect depending on the nature of the comparison

(Buunk, Collins, Taylor, VanYperen & Dakof, 1990). Buunk and colleagues (Buunk et al., 1990; Buunk & Ybema, 1997; Ybema & Buunk, 1995) developed the identification-contrast model to explain our need to identify ourselves with those who are better off than us (upward identification) and perceive ourselves to be different from those who are worse off (downward contrast). The process of adopting upward identification or downward contrast is suggested to result in positive affect, whereas downward identification or upward contrast can evoke negative thoughts and feelings (Collins, 1996). This is because identifying with those who are worse off than us presents us with a negative image of our self in the future whilst comparing ourselves to others who are doing better than us can instill negative self-evaluations (Ybema & Buunk, 1995). Similarly, perfectionistic features are also associated with unfavourable social comparison (Wyatt & Gilbert, 1998) because those who constantly try to live up to high and unrealistic standards would engage in upward comparisons resulting in persistent negative self-evaluations (e.g., upward contrast). On the other hand, favourable social comparison has also been shown to be maladaptive as it is related to higher alcohol consumption in students (McShane & Cunningham, 2003; Novak & Crawford, 2001). Together these findings suggest that engaging in social comparisons is an integral part of us which can aid our survival but also create uncertainty and maladaptive behaviours.

However, social comparison does not stand alone as a rank-related concept. To date, a great deal of research has shown that unfavourable social comparison is associated with other rank-related constructs (*see Table 3.1*). The table below provides a brief definition of each rank-related construct and studies which have provided evidence for the relationship between each rank-related concept and unfavourable social comparison.

Table 3.1: Brief description of rank-related constructs and studies showing the relationship between different rank-related concepts and unfavourable social comparison

Rank-related construct	Definition	Evidence for relationship with unfavourable social comparison
Entrapment	Perceived or actual inability to escape negative circumstances	Gilbert & Allan (1998)
Feelings of inferiority	Perceiving oneself to be subordinate to others	Gilbert, Broomhead, Irons, McEwan, Bellew, Mills et al. (2007) Gilbert, McEwan, Bellew, Mills & Gale (2009)
Helplessness	Feeling powerless (i.e., to escape an uncontrollable adverse situation)	Brewin & Furnham (1986)
Low self-esteem	Negative self-evaluation of our own competence in areas of life that are deemed important to us	Dagnan & Sandhu (1999) Gilbert et al. (1995)
Self-blame	Act of attributing fault or responsibility to one's character	Gilbert, Irons, Olsen, Gilbert & McEwan (2006)
Self-concealment	Conscious effort to conceal information about the self that is perceived to be unattractive to others	Cruddas, Gilbert & McEwan (2012)
Self-criticism	Negative self-evaluations regarding various characteristics of the self	Gilbert & Miles (2000b) Gilbert, Price & Allan (1995)
Shame	Negative evaluations that certain characteristics will be perceived as unattractive or lead to being rejected by others	Gilbert, Allan & Goss (1996) Gilbert & McGuire (1998) Gilbert et al. (1995) Goss, Gilbert & Allan (1994)
Social defeat	Losing a battle which results in the loss of social status and access to resources and mates	Gilbert & Allan (1998) Gilbert et al. (2002)
Submissiveness	Behaviours such as eye gaze avoidance and backing down when challenged which are employed to inhibit or reduce the risk of being attacked	Allan & Gilbert (1997) Gilbert (2000)

The similarities between some of these concepts have spurred Sturman (2011) to propose a higher-order factor referred to as involuntary subordination which consists of social comparison, submissive behaviour, defeat and entrapment. Involuntary subordination is a concept introduced by Price and Sloman (1987) which refers to the act of withdrawing from a competitive scenario to prevent further conflict and accept a new status in the social environment. Although Sturman and Mongrain (2005, 2008) found that involuntary subordination is related to depression, several studies have demonstrated that these individual social rank constructs are related to different pathologies. For example, unfavourable social comparison and submissive behaviour has been reported in EDs (Pinto-Gouveia, Ferreira & Duarte, 2012; Troop, Andrews, Hiskey & Treasure, 2013; Troop & Baker, 2008) and social defeat in post-traumatic stress disorder (Troop & Hiskey, 2013) suggesting the need to examine these constructs separately.

Gilbert et al. (1995) have suggested that these rank-related constructs are separate concepts which are strongly linked. For example, engaging in unfavourable social comparison results in submissive behaviours such as gaze aversion and retreating when challenged, which from an evolutionary perspective, is a damage limitation mechanism designed to prevent being attacked (Gilbert, 2000). Similarly, shame is an emotion that can emerge as a result of negative evaluations that certain characteristics will be perceived as unattractive or lead to being rejected by others (Gilbert & McGuire, 1998) and so motivate the individual not to engage in that behaviour (Miller, 1997). External feelings of shame, which refer to perceived negative evaluations based on how others may view the self, may become internalised (Gilbert, 2000). Once these external feelings of shame are deep-rooted internally (internal shame), self-criticism which refers to self-attacking thoughts and feelings, can ensue (Pinto-Gouveia et al., 2012; Gilbert, 1997; Gilbert, Baldwin, Irons, Baccus & Palmer, 2006; Gilbert & Miles, 2000b). Self-criticism will be described further in section four of this chapter.

Social rank and stress

The majority of studies of social rank have been based on the link between unfavourable social comparison and depression (Allen & Badcock, 2003; Allan &

Gilbert, 1995, 1997, 2004; Brown, Harris & Hepworth, 1995; Dagnan & Sandhu, 1999; Gilbert, 2001; Gilbert & Allan, 1998; Gilbert et al., 2002, 2006; Horowitz & Vitkus, 1986; Johnson et al., 2012; Price et al., 1994; Swallow & Kuiper, 1988; Thwaites & Dagnan, 2004; Zuroff et al., 2010). Gilbert (2001) proposed that depression ensues when stress activates the threat-focused system but the ability to cope (i.e., defence mechanisms) with the traumatic experience is either blocked or ineffective resulting in feelings of helplessness and depressive symptomatology. Additionally, clinical research has also shown that those who display depressive symptoms present a more suppressed affiliation-focused soothing system and a more developed resource- (rank) and threat-focused systems (Gilbert, 2005). Therefore, when faced with a stressful experience, they are unable to self-soothe to buffer the effects of stress on psychological well-being. However, depression is not the only psychological disorder that has been found to relate to perceptions of low rank because as a Westernised society that thrives on competition, we are at an increased risk of developing various psychological disorders (Arrindell, Steptoe & Wardle, 2003). Evidence for this comes from studies which have found that rank-related constructs are associated with depression (e.g., Allan & Gilbert, 1997; Gilbert, 2000; Gilbert & Allan, 1998; Gilbert, Allan & Trent, 1995; O'Connor, Berry, Weiss & Gilbert, 2002), anxiety (Aderka, Weisman, Shahar & Gilboa-Schechtman, 2009; Gilbert, Boxall, Cheung & Irons, 2005; Gilbert et al., 2006; Trower & Gilbert, 1989), post-traumatic stress disorder (Troop & Hiskey, 2013), psychosis and auditory hallucinations (Birchwood, Meaden, Trower, Gilbert & Plaistow, 2000), self-harm (Gilbert, McEwan, Irons, Bhundia, Christie, Broomhead et al., 2010), and even predicts burnout in teachers (Buunk, Peíró, Rodríguez & Bravo, 2007).

However, social rank not only influences psychological and behavioural characteristics but also physiological changes in animals (Sapolsky, 2005) and humans (Henry, 1982) including changes in cortisol secretion. A meta-analysis on the relationship between social rank status and cortisol levels in primate species revealed that subordinate conspecifics who encountered more stressors and did not have access to support from other primates exhibited higher levels of cortisol (Abbott, Keverne, Bercovitch, Shively, Mendoza, Saltzman et al., 2003). However, animals who achieve high social status can also experience elevated stress levels,

especially when they must constantly exert their physical abilities to maintain their social standing in the hierarchy (Sapolsky, 2005).

Although the methods used to obtain social status differs between animals and humans, research has shown that, similar to primates, humans also live within a hierarchical existence. For example, socio-economic status (SES) is an obvious example of how Western societies maintain different levels of hierarchy which consists of dominant and subordinate groups of people. Although a pecking order system is proposed to be necessary to maintain a certain level of cohesion and promote affiliation, findings suggest that SES predicts an increase in the incidence of diseases and mortality (Adler, Epel, Castellazzo & Ickovics, 2000). Therefore, similar to animals, humans who view themselves to be inferior or strive to avoid being viewed as inferior by others can experience feelings of stress (Ferreira, Pinto-Gouveia & Duarte, 2013a; Gilbert, 2005). Insecure striving is a maladaptive function of social comparison as it refers to a perceived need to constantly demonstrate and gain acceptance for one's attributes in order to avoid feelings of inferiority and gain a place in the hierarchy (Gilbert et al., 2007). Rank-related constructs such as submissiveness and insecure striving are also related to high stress levels (Gilbert et al., 2007). In contrast, feeling accepted within one's social environment (referred to as secure non-striving) is negatively correlated with stress (Gilbert et al., 2007). However, situations that are either uncontrollable or entail an element of social threat can stimulate a stress-related physiological response (Dickerson & Kemeny, 2004). Interestingly, when compared with men, feelings of social rejection appear to induce higher levels of cortisol production in women, who experience higher stress levels when faced with achievement-based challenges (Stroud, Salovey & Epel, 2002). These studies have clearly established that social rank is a contributing factor to the experience of stress. Therefore, as Chapter Two has presented a systematic review of how stress can play an important role in problematic weight regulation, it is essential to consider how social comparison can also influence eating behaviours.

Social comparison and eating behaviours

Firstly, animal studies have shown that changes in status/rank within the their social environment can have an effect on eating behaviours. Change to a social

rank system amongst pigs has been suggested to contribute to anorexic like behaviours known as 'thin sow syndrome' (Treasure & Owen, 1997). Although wasting diseases are common in animals, pigs were found to exhibit anorexic behaviours such as loss of appetite in the presence of freely available food and hyperactivity when they were defeated in their competition for a place in the social hierarchy (Treasure & Owen, 1997). Importantly, behaviours associated with thin sow syndrome bear a remarkable resemblance to behaviours such as self-starvation, exhibited by humans with AN.

Making social comparisons in a highly competitive culture can also be based on one's physical attractiveness which can be used to define one's social status within a group (Ferreira et al., 2013b). As previously mentioned in Chapter One, we currently live in a society that promotes the thin ideal (Stice, 2002; Stice & Agras, 1998) so combining the internalisation of the thin ideal with a highly competitive world that bases a thin female silhouette with beauty, health and success (Feingold, 1992; Kanazawa & Kovar, 2004; Sypeck, Gray, Etu, Ahrens, Mosimann & Wiseman, 2006), it is no surprise that social comparison has been linked to disturbed eating behaviours. Studies have shown that unfavourable social comparison is associated with DE behaviours in nonclinical samples (Bellew, Gilbert, Mills, McEwan & Gale, 2006; Stormer & Thompson, 1996; Thompson, Coovert & Stormer, 1999; Troop & Baker, 2008), clinical sample of EDs (Connan, Troop, Landau, Campbell & Treasure, 2007; Troop, Allan, Treasure & Katzman, 2003) and even changes in ED symptoms over time (Troop et al., 2013). The maintenance of eating pathology can also be related to evidence which suggests that those with EDs are more likely to experience negative self-evaluations as a result of engaging in upward comparisons by making comparisons with those who are thinner and perceived to be more attractive (Corning, Krumm & Smitham, 2006). Not only do those diagnosed with EDs or who have a history of EDs engage in upward comparisons in relation to body size and shape, but they also frequently recall feelings of inferiority during adolescence (Troop & Bifulco, 2002) and inadequacy compared to others (Butow, Beumont & Touyz, 1993; Williams, Power, Millar, Freeman, Yellowlees, Dowds et al., 1993).

However, the link between social comparison and dysfunctional processes related to eating and weight is applicable to both genders as Halliwell and Harvey

(2006) demonstrated that engaging in social comparison is associated with body dissatisfaction, internalisation of the thin ideal and DE in male and female adolescents. The link between social comparison and eating behaviours is not just based on our perception of how the world perceives our body as a way of determining our physical attractiveness, but also how we compare our physical attributes to others in order to estimate our status in the social environment (Jones, 2001). Women have been found to engage in more upward comparisons than men when describing their appearance (Strahan, Wilson, Cressman & Buote, 2006) by making unrealistic comparisons with targets such as models and actresses which results in dissatisfaction with one's physical appearance (Engeln-Maddox, 2005; Neziroglu, Khemlani-Patel & Veale, 2008). Additionally, the link between unfavourable social comparison and dysfunctional eating patterns can be viewed together with research that negative self-evaluations are associated with perfectionistic features (Wyatt & Gilbert, 1998) which in turn has been established as a fundamental risk factor for the development of EDs (Stice, 2002). Therefore, it can be suggested that a toxic combination of pursuit for thinness, perfectionistic characteristics and negative self-evaluations increase the risk of developing dysfunctional eating patterns. These concepts can be linked to social comparison as the desire to be thinner or have 'perfect' characteristics can result in negative self-evaluations. Hence, unfavourable social comparison can ensue when one believes that one's features are not as attractive as those of others and one is therefore of a lower status compared to others in the social environment.

Additionally, a recent study has also applied social comparison to weight loss efforts (Matos, Duarte, Stubbs, Gilbert & Morris, In press). Matos et al., (In press) conducted a large cross-sectional study with 2236 obese individuals who were enrolled onto a weight management programme and found that unfavourable social comparison predicted greater levels of disinhibition of dietary restraint.

However, social rank/status alone is not enough to explain behaviour, especially those related to problematic weight regulation. Both Allen and Badock (2003) and Gilbert (1995) have argued that psychology requires an integrative approach, especially for treatments offered at a clinical setting. The Social Risk Hypothesis proposed by Allen and Badcock (2003) is an integrative view

encapsulating the human capacity to conserve resources, maintain high social standing and form and maintain affectional bonds. The biopsychosocial approach which is guided by evolutionary theory, recommends that humans develop mental models of functioning which operate as a defence mechanism at times of conflict or loss or provide a sense of security (Gilbert, 1993, 1995). Gilbert (1993, 1995) proposed two different types of strategies referred to as defence and safety. The defence mechanism operates on a "better safe than sorry" principle and is stimulated when the individual experiences threat or loss. For example, when danger signals are detected, the individual may retreat in order to minimise the risk of being attacked. The safety system develops through positive infant and caregiver interactions which provides the infant with a sense of security. However, if the infant is reared in a negative and hostile environment, the infant experiences distress from an early age and develops an insecure attachment style predisposing the individual to an easily activated defence system.

The interactive relationship between attachment and social rank can be related back to theories of interpersonal behavior which suggest that affiliation and the need for power and status function together to form the basis of behaviours crucial for social relations between people (Horowitz & Vitkus, 1986). Bugental (2000) also proposed that evolution has prepared humans to use five domains of social behaviour which include developing alliances, ensuring reciprocal investment, selecting and protecting mates, maintaining status in a social hierarchy and developing a secure attachment as a way of maintaining proximity with a caregiver for safety and survival. Hostile interactions that take place between a caregiver and a child earlier in life can increase vulnerability to psychopathology in adulthood. However, the two positive affect systems provide different explanations as to how this may occur. The social rank system would suggest that vulnerability to psychopathology in adulthood is a result of the caregiver forcing the child into a subordinate position, whereas the attachment system posits that it is the lack of warmth that hinders the development of a secure attachment resulting in psychological problems later on (Gilbert et al., 1996; Gilbert, Cheung, Grandfield, Campey & Irons, 2003). Therefore, the next section of this review will provide an

overview of how attachment can contribute to our ability to manage stress-related responses.

Attachment

Attachment refers to the bond that develops between an infant and its caregiver to provide young children not only with a sense of security and to aid survival but to develop patterns of emotion regulation (Bowlby, 1969, 1973, 1980). Using an ethological-evolutionary approach, Bowlby (1977a,b) suggested that the cardinal function of forming an attachment bond during early life is to keep the young infant safe and secure away from danger. Bowlby (1977a,b) described two types of behaviours related to the formation of attachment bonds which he referred to as care-giving and exploration. Care-giving refers to the availability and responsiveness of the caregiver to the infant's needs and their willingness and ability to provide protection at times of danger (Bowlby, 1977a,b). However, care-giving is not just about satisfying basic needs such as hunger or physical protection, but also refers to the psychological needs of the child. Harlow's (Harlow, Rowland & Griffin, 1964) seminal work with primates provided a clear example of this as monkeys demonstrated a preference for interacting with a cloth-covered makeshift 'mother' substitute rather than with a wire mesh surrogate 'mother' who provided milk at times of stress (e.g., when a toy robot was put into their cage). Therefore, when a caregiver instills a sense of security in the infant, the child will engage in exploratory activities of its environment and then return to the secure base and its attachment figure (Bowlby, 1977a,b). These dyadic interactions lead to the construction of what Bowlby (1973, 1980) referred to as internal working models. Bowlby (1973, 1980) postulated that infants develop internal working models of the world, self and their attachment figure which are a set of representations that are useful for guiding emotions and behaviour later in life. Therefore, if a child is reared in a safe environment by a responsive caregiver, the child will go onto develop a working model of the caregiver as loving and the self as a valued individual who is worthy of love. However, if a caregiver is indifferent to the infant's needs for interaction and comfort, then the infant will develop a working model of the caregiver as rejecting and the self as not deserving of reassurance and attention (Bowlby, 1973). Hence, it

is the development of negative internal working models that can be the root of pathological behaviours over the lifespan (Bretherton, 1985).

Bowlby's early theoretical ideas on attachment were shaped by his experiences as a child psychiatrist (Bowlby, 1958, 1960; Bowlby, Ainsworth, Boston & Rosenbluth, 1956) and observations of institutionalized children (Robertson & Bowlby, 1952). However, it was the work of Mary Ainsworth and her colleagues that provided empirical evidence in support of Bowlby's theory of attachment (Ainsworth, 1964; Ainsworth & Bell, 1970; Ainsworth, Blehar, Waters & Wall, 1978; Ainsworth & Wittig, 1969).

Ainsworth (1964) first applied the theory of attachment to infants and their caregivers in Uganda. Based on these findings, Ainsworth and Wittig (1969) devised laboratory-based naturalistic experiments to observe young infants and how they interacted with the environment and their caregivers. Although the term the Strange Situation is commonly associated with the work of Ainsworth and colleagues (Ainsworth, 1964; Ainsworth & Bell, 1970; Ainsworth et al., 1978; Ainsworth & Wittig, 1969; Bell, 1970; Blehar, Lieberman & Ainsworth, 1977), it was actually earlier work by Arsenian (1943) that introduced the idea of examining attachment-related behaviours by observing infants under various conditions. Arsenian (1943) used naturalistic experiments similar to that of the Strange Situation to observe infants and caregivers during various situations.

Ainsworth et al. (1978) used Arsenian's (1943) method of observing caregiver and infant interactions to develop and create situations during which the infant is exposed to unfamiliar surroundings (i.e., laboratory playroom) with the caregiver and then later in the absence of the caregiver. Ainsworth and colleagues (1978) conducted meticulous observations of infants once they are introduced into the playroom with their caregiver, when a stranger enters the room, when the infant is left alone with the stranger, when the caregiver returns, when the infant is left alone in the playroom and then finally when the stranger and caregiver return. These episodes of drama were designed to expose the infant to stress and arouse changes in behaviour in order to observe how the infant explored the unfamiliar surroundings whilst using the caregiver as a secure base. Ainsworth and Bell (1970) found that infants engaged in more exploratory behaviours whilst in the presence of

the caregiver compared to when an unfamiliar figure was introduced into the playroom. However, what Ainsworth and Bell (1970) found most intriguing was that some infants became ambivalent or detached when reunited with the caregiver. Those who presented ambivalent behaviours were found to seek physical contact with the caregiver upon reunion whilst also displaying aggression such as kicking the caregiver. Those who displayed detached behaviours, avoided contact with the caregiver altogether (Ainsworth & Bell, 1970).

Ainsworth et al. (1978) used their observational findings to formulate three types of attachment styles known as secure, anxious/ambivalent and avoidant, with the latter two referring to insecure attachment styles. Secure infants were found to use their caregiver as a secure base from which they would explore the unfamiliar surroundings and show signs of distress in the caregivers absence but, once reunited, the infant would seek contact with the caregiver and become calm. In contrast, anxious/ambivalent infants would display intense signs of distress upon separation. Upon reunion, the infant would seek contact but resist interaction with the caregiver at the same time. Lastly, avoidant infants rarely cried in the absence of the caregiver and would avoid the caregiver upon reunion despite the caregiver's efforts to engage with the infant (Ainsworth et al., 1978). These findings provided further support for Bowlby's (1969, 1973, 1980) theory of attachment which suggests that caregivers, who are responsive to the infant's care-eliciting behaviours and provide the infant with a safe and secure environment, promote independence in the child who develops the ability to relax or safely explore the environment. Early research also demonstrated that the caregivers' maternal sensitivity to the infants' needs shapes the type of bond that develops in the infant-caregiver dyad (Bell & Ainsworth, 1972; Blehar et al., 1977; Sroufe & Waters, 1977). For example, a receptive caregiver who encourages interaction with the infant will instill a secure attachment style in the child who will be more responsive and positive during interactions resulting in a positive relationship (Bell & Ainsworth, 1972; Blehar et al., 1977).

Although early work on attachment was based on the types of bonds that develop between an infant and its caregiver, Bowlby (1977a,b) described the attachment bond created during early childhood as lasting "from the cradle to the

grave" (p. 203). Similarly, Ainsworth (1985) also suggested that the attachment between an infant and its caregiver can persist and even shape later affectional bonds formed during adulthood. Support for the idea that early childhood attachment patterns can shape social relations beyond infancy comes from longitudinal research which has found that the type of attachment bond formed within the infant-caregiver dyad predicts social behaviour during early school years (Bretherton, 1985). This idea later spurred the application of infant attachment patterns to adult attachment relations starting with Main and colleagues (George, Kaplan & Main, 1984; Main & Goldwyn, 1991; Main, Kaplan & Cassidy, 1985) who devised the first measure of adult attachment patterns referred to as the Adult Attachment Interview (AAI). The AAI (George et al., 1984; Main & Goldwyn, 1991) is an interview-based measure which consists of open-ended questions regarding the type of attachment patterns an adult had with their caregiver(s) during childhood. George et al. (1984) described three types of attachment patterns which were secure, preoccupied and dismissing. Secure individuals were those who reported attachment-related memories as having an influence on their adult personality and who provided a clear and coherent account of their childhood attachments even if their experiences were not satisfying. Preoccupied individuals reported inconsistent memories of their childhood and were unable to draw together a coherent narrative. Finally, those who were indifferent about the influence that their childhood attachment bond had on their adult personality or were unable to remember much about their childhood were classified as dismissing (George et al., 1984).

Further support for the attachment patterns derived using the AAI (George et al., 1984) came from Kobak and Sceery (1988) who used the interview to examine attachment styles of adolescents. This study demonstrated that the type of attachment bond that develops between an infant and its caregiver can influence affect regulation during adolescence (Kobak & Sceery, 1988). The study showed that those who were classified as securely attached reported having a supportive social network and were rated as less anxious and less hostile by their peers. In contrast, those who were regarded as preoccupied or dismissing reported having low family support and, in addition, the former were also more anxious, whilst the latter group showed higher levels of hostility (Kobak & Sceery, 1988). Kobak and Sceery (1988) go

on to suggest that the activation of the attachment system is evident during stressful life experiences and how feelings of stress are regulated depends on the internal working models of the self and the attachment figure. The authors posit that during a stressful experience, a securely attached individual will use his/her positive memories of supportive parents to manage negative feelings. In contrast, the preoccupied group who displayed signs of anxiety were suggested to continually strive to obtain support from parents. Lastly, difficulty in recalling attachment-related memories was suggested to be indicative of the dismissing group's attempt to block out stress-related thoughts and feelings. Therefore, these findings demonstrated the importance of attachment on later affect regulatory processes.

Hazan and Shaver (1987) also used the idea that infant-caregiver attachment styles can provide a plausible framework for understanding attachments formed in adulthood. Secure attachment patterns in adulthood characterise adults who are comfortable with getting close to and depending on others. However, those with an avoidant attachment style have difficulty trusting others and are uncomfortable with becoming intimate with others. Similarly, anxious/ambivalent adults worry that others do not really care about them and are often characterised as being highly dependent on others.

Bartholomew & Horowitz (1991) developed our knowledge further by using Bowlby's (1973, 1980) internal working models of the self and others to devise a four-category model of attachment organisation in adulthood. The four-factor model covers the idea that every individual has a positive or negative view of the self and a positive or negative view of others and different combinations of these dichotomies resulted in four attachment styles labeled as secure, dismissing, preoccupied and fearful. Securely attached individuals have a positive view of the self and others, which is characterized by an internal self-worth and seeking close relationships. Individuals with a dismissing attachment style have a positive view of the self but a negative view of others and are identified by the avoidance of close relationships due to negative expectations. Preoccupied individuals have a negative view of the self but a positive view of others. Hence, an individual who seeks excessive closeness due to a deep sense of unworthiness portrays a preoccupied attachment style. Lastly, individuals with a fearful attachment style have a negative view of the self

and of others. These individuals experience feelings of unworthiness and expect others to be untrustworthy and rejecting. Both Bartholomew and Horowitz (1991) and Hazan and Shaver (1987) provided further support for the notion that childhood attachment patterns can be useful in identifying attachment styles in adulthood. Both studies found links between attachment styles in adulthood and attachment styles in childhood suggesting that there is a continuation of attachment patterns throughout the lifespan (Bartholomew & Horowitz, 1991; Hazan & Shaver, 1987)

The theoretical foundations of attachment theory as devised by Bowlby (1969, 1973, 1980) and Ainsworth et al. (1978) have been used to develop many other measures of attachment behavior in adulthood (see reviews: Bradford & Lyddon, 1994; Crowell & Treboux, 1995; Lyddon, Bradford & Nelson, 1993; Ravitz, Maunder, Hunter, Sthankiya & Lancee, 2010). These include both interview-based assessments and self-report measures. Importantly, as the development of attachment in early infancy is important for emotion regulation processes throughout the lifespan, measures assessing adult attachment styles have been used to evaluate the impact of adult attachments (specifically insecurity of attachment) on psychopathology, for example, depression (Allen, Coyne & Huntoon, 1998; Bifulco, Kwon, Jacobs, Moran, Bunn & Beer, 2006; Carnelley, Pietromonaco & Jaffe, 1994; Cooper, Shaver & Collins, 1998; Haaga, Yarmus, Hubbard, Brody, Solomon, Kirk et al., 2002; Hazan & Shaver; 1990; Kobak, Sudler & Gamble, 1991; Mickelson, Kessler & Shaver, 1997; Murphy & Bates, 1997; Pettem, West, Mahoney & Keller, 1993; Roberts, Gotlib & Kassel, 1996; Shorey & Snyder, 2006; Wei, Vogel, Ku & Zakalik, 2005; Zuroff & Fitzpatrick, 1995), anxiety (Aderka et al., 2009; Bifulco et al., 2006; Eng, Heimberg, Hart, Schneier & Liebowitz, 2001; Meredith, Strong & Feeney, 2006; Mickelson et al., 1997; Watt, McWilliams & Campbell, 2005; Weems, Berman, Silverman & Rodriquez, 2002), EDs (O'Kearney, 1996; O'Shaughnessy & Dallos, 2009; Ward, Ramsay & Treasure, 2000; Zachrisson & Skårderud, 2010) and stress (Ditzen, Schmidt, Strauss, Nater, Ehlert & Heinrichs, 2008; Kidd, Hamer & Steptoe, 2011).

Bowlby (1973) suggested that the attachment system is designed to reduce stress and promote safety. This takes us back to Gilbert's (2005) tripartite system of affect regulation. Gilbert (1997) posits that caregivers who provide a safe and soothing environment for their infant encourage the development of a positive

internal SAHP allowing them to refer back to these memories at times of stress in order to regulate their emotions better. Support for this notion comes in the form of research studies which have demonstrated that attachment is important for the regulation of physiological responses (Gilbert, 2001). Infants who are exposed to inadequate care and adverse life circumstances during childhood experience immune and stress problems (Cacioppo, Berntson, Sheridan & McClintock, 2000; Gilbert & Miles, 2000a; Perry, Pollard, Blakley, Baker & Vigilante, 1995; Uchino, Cacioppo & Kiecolt-Glaser, 1996). Those who present insecure attachment behaviours display greater cortisol levels (Spangler & Grossmann, 1993). Additionally, Field (1998) also demonstrated that physical contact during early life has soothing effects on physiological changes, in particular the production of cortisol demonstrating that close proximity within the caregiver-infant dyad has important implications for regulating stress-related experiences.

So far, the present chapter has demonstrated that both social rank and attachment are important for managing stress-related experiences. As presented in the first section of this review, unfavourable social comparison is related to high levels of stress. Similarly, our ability to regulate our affective responses during adverse life circumstances can be influenced by our early interactions with our caregiver. Therefore, how do these systems interact to help us to manage stress-related responses? Given the proposed link between stress and weight/eating behaviours, when these systems are maladaptive, can they hinder our ability to maintain our bodyweight and eating behaviours at a healthy level?

Affect regulation

Sloman and colleagues (Sloman, 2008; Sloman, Atkinson, Milligan & Liotti, 2002; Sloman, Gilbert & Hasey, 2003) bring together the two positive affect systems to explain their role in affect regulation. Research on attachment-related behaviours during infant-caregiver separation has established that when an infant is separated from its caregiver, it has an innate ability to send out distress calls to try and reinstate the caregiver in its presence (Robertson & Bowlby, 1952; Bowlby, 1960). This is because infants have an intrinsic desire to maintain proximity with the caregiver as a process of maintaining emotional equilibrium (Mikulincer, Florian &

Tolmacz, 1990). Sloman (2008) suggested that displays of anger and protest are indicative of the infants seeking-behaviour (i.e., searching for the caregiver) which suggests activation of the social rank system as this positive affect system is a resource-seeking facility. The absence of the caregiver can also stimulate the attachment system as crying and protesting are attachment-related behaviours. However, once the caregiver is available and responsive to the infants needs, the attachment system is again activated and the infant is soothed leading to demobilization of the social rank system (Sloman, 2008). However, if the attachment figure remains unresponsive to the infant's needs, this results in the activation of the defence system and the infant will internalize a sense of insecurity and adopt a resource-seeking method of assessing the environment rather than an affiliation-focused outlook (Gilbert, 1993, 1995). Therefore, as an adult, the infant will be highly competitive and increasingly concerned with obtaining acceptance and striving to avoid being rejected by others (Gilbert, 1993, 1995). Those who are reared within a positive infant-caregiver dyad develop a secure attachment style enabling them to feel comfortable to form alliances in their social world (Gilbert, 2001). However, those who adopt an insecure style of attachment-related behaviours will avoid forming relationships due to the fear of being rejected. Additionally, despite being reliant on others, avoidant individuals do not seek support from others at times of stress (Mikulincer, Birnbaum, Woddis & Nachmias, 2000). Forming positive affiliations provides the individual with a social support network that can be used at times of adversity and even minimise the risk of developing psychopathology (Gilbert, 2001). Therefore, the development of secure attachments is proposed to help us to learn the skills needed to manage difficult emotions at times of stress, being able to self-soothe rather than be self-critical (a form of self-attacking, associated with shame and a perceived low status; Gilbert, 2006). Research examining the relationship between affect regulation systems and stress has shown that subordinate primates who encountered more stressors and lacked access to social support exhibited higher levels of cortisol (Abbott et al., 2003).

Both affect systems are related to psychopathology but Gilbert et al. (2003) have suggested that there is a difference between an activated resource-seeking

system and a suppressed affiliation-focused system on adulthood psychopathology. The former refers to a focus on threats to social status suggesting that psychopathology ensues when an individual adopts a sense of low rank compared to those in its social environment whilst the latter indicates the inability to self-soothe at times of stress due to a lack of parental attentiveness during childhood (Gilbert et al., 2003). However, these systems are not stand-alone mechanisms but function interactively. Irons and Gilbert (2005) have demonstrated that insecurity of attachment during adolescence is related to feelings of unfavourable social comparison. Securely attached individuals, however tended to rate themselves as similar or slightly above others on rank, attractiveness and how they fit in with others. These findings provide further support for the notion that insecurely attached individuals are sensitive to rank-related threats.

Therefore, activation of the social rank system or suppression of the attachment system is related to later psychopathology such as anxiety and depression (Irons & Gilbert, 2005; Sloman, 2008). The role of early life relationships on later social comparison and adaptability has been shown by Benn, Harvey, Gilbert and Irons (2005). These authors found that first year undergraduate students who indicated that their parents were supportive during childhood also reported lower levels of homesickness, greater interpersonal trust and more favourable social comparison compared to those who recalled parents who were rejecting. These findings demonstrate the importance of parental behaviour during childhood on later social comparison and the ability to be able to adapt.

But how do the social rank and attachment systems relate to the processes of affect regulation? As suggested by Sloman et al. (2003) securely attached individuals (as opposed to insecurely attached) develop the ability to self-soothe and regulate their emotions as a result of a positive infant-caregiver dyad. In contrast, adults who recall memories of parents who were rejecting or overprotective during childhood reported greater thoughts and feelings of inadequacy whilst overprotective parenting was associated with higher levels of self-hatred (Irons, Gilbert, Baldwin, Baccus & Palmer, 2006), which are considered types of self-criticism (Gilbert et al., 2004). Similarly, negative self-evaluations can be triggered as a result of unfavourable social comparison which occurs when one regards the self to be of a

lower social standing compared to others in their social environment (Gilbert, 1997). Therefore, the remainder of this review will focus on the role of self-criticism and reassurance as a form of affect regulation and its association with the ability to manage stress-related responses.

Self-criticism/reassurance

Blatt and Homann (1992) referred to self-criticism in relation to achievement failure as the "constant and harsh self-scrutiny and evaluation … a chronic fear of disapproval, criticism, and of losing the acceptance and love of significant others" (p. 49). This definition can be suggested to relate back to Gilbert's (2001, 2005) social rank system and the notion of internal and external SAHP. Blatt and Homann's (1992) definition of self-criticism can be applied to the idea that self-critical thoughts and feelings can ensue when the social rank system is activated. This is because self-evaluations (either internally or externally generated) are an important aspect of determining competencies of individuals who are competing for positions on a social hierarchy or for limited resources and mates (Gilbert, Clarke, Hempel, Miles & Irons, 2004). For example, when an individual experiences unfavourable social comparison or other forms of threat to social status, they can become highly self-critical. Thus, when negative SAHP is experienced, defensive mechanisms such as self-criticism can ensue as a result of feeling inferior to others or perceiving oneself to be of a lower rank compared to others (Gilbert, 1997). However, the relationship between perceptions of low rank and self-critical thoughts and feelings may even be bi-directional as research has shown that self-critical women appear to be especially sensitive to social status threats (Santor & Zuroff, 1997).

In contrast, the ability to self-reassure at times of difficulty may reflect a learned ability to be compassionate directed internally (Gilbert, 2005). Gilbert (2005) suggested that the ability to self-reassure is nested in the positive infant-caregiver attachment bond and that develops as a consequence of internalizing parental soothing (Gilbert, 2006). Empirical evidence suggests this is the case, with self-reassurance being related to security of attachments (Irons et al., 2006) and early memories of warmth and safeness (Richter, Gilbert, & McEwan, 2009). Whilst self-critical thoughts and feelings can be triggered by perceptions of being low rank, the

idea that people can be self-reassuring or self-soothing comes from the idea that they were able to build secure attachments at an early age (Gilbert, 2005). Therefore, both self-criticism and self-reassurance are products of the positive affect systems.

Self-criticism has also been referred to as self-condemnation (Driscoll, 1989) and self-blame (Garnefski, Van Den Kommer, Kraaij, Teerds, Legerstee & Onstein, 2002) and found to relate to other forms of rank-related constructs including internal and external entrapment, defeat and shame (Sturman & Mongrain, 2005, 2008; Tangney, Wagner & Gramzow, 1992). The inability to self-reassure has also been linked to rank-related constructs such as feelings of inferiority, submissive behaviour and shame (Gilbert et al., 2010).

There is some suggestion that self-criticism and self-reassurance may form two ends of a single spectrum, known as self-compassion (Neff, 2003a,b). Neff (2003a,b) proposed three components of self-compassion: (1) kindness towards the self rather than critical self-judgement (similar to the constructs of self-reassurance and self-criticism, respectively), (2) common humanity in which it is accepted that failure and pain are part of the human experience (rather than a perceived isolation from this human experience), and (3) mindful awareness of negative emotional states in which these are faced and accepted rather than denied or exaggerated (as in over-identification of emotional states). Higher levels of self-compassion (incorporating high levels of self-kindness and low levels of critical self-judgement) predict better psychological health (e.g., Neely, Schallert, Mohammed, Roberts, & Chen, 2009; Neff, 2003a, 2004; Neff, Kirkpatrick, & Rude, 2007; Neff, Rude, & Kirkpatrick, 2007; Neff & Vonk, 2009). However, although self-kindness and self-judgement are similar to the constructs of self-reassurance and self-criticism, respectively, a recent fMRI study has shown that inducing self-critical and self-reassuring responses led to activation in different areas of the brain (Longe, Maratos, Gilbert, Evans, Volker, Rockliff et al., 2010). Clearly, though related, self-criticism, and self-reassurance appear to be distinct processes.

Self-criticism is related to various forms of psychopathology including depression, anxiety, self-harm, suicidal ideation and PTSD (e.g., Blatt & Zuroff, 1992; Dunkley, Sanislow, Grilo, & McGlashan, 2009; Fazaa & Page, 2003; Gilbert et al.,

1995, 2010; Gilbert, McEwan, Matos & Rivis, 2011; Lee, 2005; Sturman & Mongrain, 2005) and is suggested to interfere with progress towards important goals (Powers, Koestner, Lacaille, Kwan & Zuroff, 2009). Similar to unfavourable social comparison and insecurity of attachment, self-criticism is also positively related to stress (Gruen, Silva, Ehrlich, Schweitzer & Friedhoff, 1997). Additionally, self-criticism is related to DE behaviours in community-based samples (Feinson & Meir, 2012; Fennig, Hadas, Itzhaky, Roe, Apter & Shahar, 2008; Pinto-Gouveia et al., 2012; Williams et al., 1993), and those who are enrolled onto a weight loss programme (Matos et al., In press). However, the similarities do not end there as those diagnosed with EDs report high levels of negative self-evaluations and self-criticism (Dunkley & Grilo, 2007; Fairburn, Cooper, Doll & Welch, 1999; Fairburn, Welch, Doll, Davies & O'Connor, 1997). Since low levels of self-compassion are associated with greater levels of shame and eating pathology in ED patients (Kelly, Carter, Zuroff & Borairi, 2013) and those who are highly self-critical express difficulty in generating self-reassuring thoughts and feelings (Gilbert et al., 2011), these findings may have implications for treatment and recovery.

Therefore, as self-critical evaluations are a fundamental component of EDs, several researchers (Gale, Gilbert, Read & Goss, 2012; Goss & Allan, 2010; Kelly, Carter & Borairi, 2013) have suggested that ED patients may benefit from Compassion Focused Therapy (CFT; Gilbert, 2005, 2009). CFT is a therapeutic process of guiding individuals to develop the ability to self-soothe at times of stress by improving accessibility to the affiliation-focused affect system. This allows us to return back to Gilbert's (1997) suggestion that caregivers who provide a safe and soothing environment for their infant encourage the development of a positive internal SAHP allowing them to refer back to these memories at times of stress in order to regulate their emotions better. However, those infants who are exposed to inadequate care and adverse life circumstances during childhood internalize a sense of insecurity and adopt a resource-seeking method of assessing the environment rather than an affiliation-focused outlook (Gilbert, 1993, 1995), thus greeting the self with critical evaluations. Therefore, the primary aim of CFT is to train individuals to reduce self-critical evaluations and instead, rejuvenate their attachment system in order to develop a self-soothing method of alleviating feelings of stress. Studies that

have employed CFT have shown this therapeutic intervention to be effective in various clinical settings. For example, CFT has been shown to alleviate auditory hallucinations (Mayhew & Gilbert, 2008), improve depression and self-esteem following brain injury (Ashworth, Gracey & Gilbert, 2011), enhance recovery treatment for psychosis (Laithwaite, O'Hanlon, Collins, Doyle, Abraham, Porter et al., 2009) and increase the ability to be self-soothing in those with chronic mental health difficulties (Gilbert & Procter, 2006). In addition, experimental adaptation of CFT (Gilbert, 2009) has been shown to reduce number of cigarettes smoked per day (Kelly, Zuroff, Foa & Gilbert, 2010) and other experimental studies which have induced self-compassion showed a reduction in stress (e.g., Neely et al., 2009; Rockliff, Gilbert, McEwan, Lightman & Glover, 2008). In conclusion, CFT is a useful method for developing a self-soothing approach in order to improve psychological well-being in psychiatric settings and reduce unhealthy behaviours in community-based samples.

Narrative review summary

The current section of this chapter has provided a descriptive overview of the affect regulatory systems and processes that contribute to the stress process.

So far, the current thesis has demonstrated how affect regulatory systems and processes can play an important role in psychopathology. However, these interactive positive affect systems are not only related to various psychopathologies but also to behaviours. As the principal objective of the current research is to understand the processes behind problematic weight regulation, it is important to describe how these positive affect systems can contribute to the inability to maintain bodyweight at an optimal level. So far, the research presented in this review suggests that both unfavourable social comparison, insecurity of attachment and self-critical thoughts and feelings are related to various patterns of dysfunctional eating behaviours (Bellew et al., 2006; Connan et al., 2007; Feinson & Meir, 2012; Fennig et al., 2008; O'Kearney, 1996; O'Shaughnessy & Dallos, 2009; Pinto-Gouveia et al., 2012; Schmidt & Treasure, 2006; Stormer & Thompson, 1996; Troop et al.,

2003, 2013; Troop & Baker, 2008; Thompson et al., 1999; Ward et al., 2000; Williams et al., 1993; Zachrisson & Skårderud, 2010). Also, unfavourable social comparison has been related to greater levels of disinhibition in those attending a weight loss programme (Matos et al., In press). Additionally, both positive affect systems and self-criticism have been implicated in the stress literature (Cacioppo et al., 2000; Dickerson & Kemeny, 2004; Field, 1998; Gilbert, 2001, 2005; Gilbert et al., 2007; Gilbert & Miles, 2000a; Gruen et al., 1997; Perry et al., 1995; Spangler & Grossmann, 1993; Stroud et al., 2002; Uchino et al., 1996) which has been suggested to contribute to problematic weight regulation (as presented in Chapter Two). Therefore, it can be postulated that, together, stress and affect systems, can hinder our ability to regulate our bodyweight at a healthy level. However, none of these studies have attempted to examine the role of these affect regulation systems and processes together with stress in relation to problematic weight regulation.

Justification for the current research

So far, the current thesis has provided evidence from an extensive literature within the wide spectrum that is problematic weight regulation starting with a general overview of the causes and treatments of weight-related disorders, specifically, obesity and AN (*see Chapter One*). This introductory chapter was followed by two reviews, firstly an appraisal of the research examining the role of stress on weight regain and AN and, secondly, how affect systems (social rank and attachment) and affect regulatory processes such as self-criticism/reassurance can contribute to the development of stress. The systematic review revealed that there have been a limited number of studies which have examined the role of stress on weight regain following weight loss and these have produced some inconsistent findings. Similarly, although a review by Ball and Lee (2002) found that studies conducted pre-1995 suggest that stress is a contributing factor of DE when examined cross-sectionally, there are still inconsistent findings when this relationship is examined over time. Therefore, longitudinal studies which employ large samples are required to eliminate the inconsistencies that are still present in the literature. In addition, the narrative

review presented in Chapter Three has established that affect regulation plays an important role in regulating emotions triggered as a result of stressful experiences. Therefore, if this is the case, then it is important to determine if and how stress, affect systems and affect regulatory processes contribute to our ability to regulate our bodyweight and eating behaviours. In order to do this, the following model will form the underpinning of the research that will be presented in this thesis (*see Figure 3.2*):

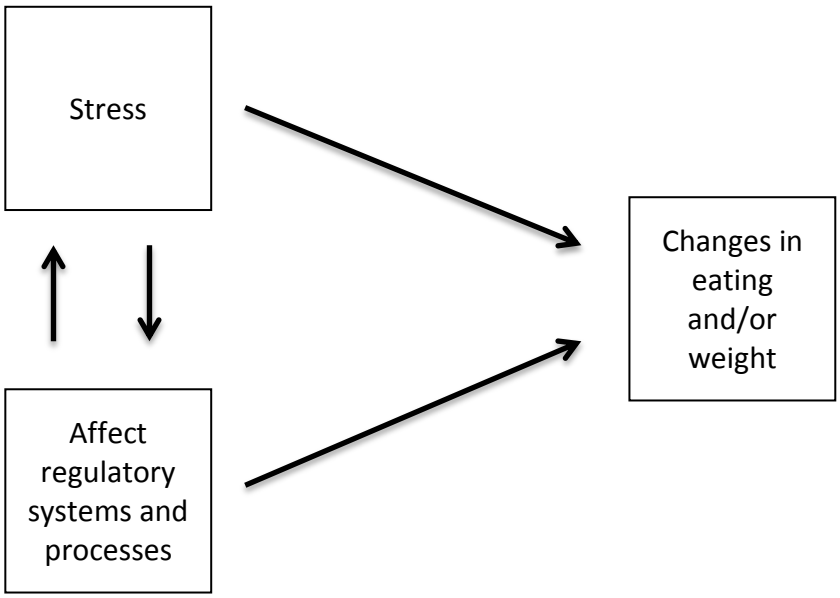


Figure 3.2: Proposed model to be tested in the research programme

Based on the systematic reviews (*see Chapter Two*) and narrative review (*see Chapter Three*) presented so far, it is possible that it is ineffective affect regulatory systems and processes are detrimental to how emotions are regulated at times of stress which is what results in changes in eating behaviours and weight. However, it can also be proposed that affect regulatory systems and processes mediate the relationship between stressful experiences and changes in eating and weight. Therefore, different aspects of this model will be tested during the four empirical studies that will be presented in this thesis.

Objectives

Based on the above model, the focus of the thesis will be to answer the following research questions which are broken down into research questions set for the overall research programme and the broad research questions that will be addressed by each study. Each empirical chapter will also consist of more specific questions that will drive the statistical analyses of each study.

<u>Overall research questions</u>

1. How do longitudinal changes in stress, bodyweight and DE behaviours impact on each other and do affect regulatory systems and processes contribute to these changes?
2. Can stress and affect regulation predict weight regain in those who have lost weight?
3. What is the role of stress and affect regulation in relapse and recovery from AN?
4. Can an intervention addressing the regulation of emotions alleviate the effects of stress on changes in bodyweight and eating behaviours during a stressful period?

<u>Broad research questions addressed by each study</u>

Study One: The role of mood and affect systems on the trajectories of BMI, disordered eating, eating and stress

1. What are the trajectories of BMI, DE and stress and how do these trajectories influence each other in a community-based sample of women?
2. What is the impact of mood and affect regulatory systems (social rank, attachment) and processes (self-criticism/reassurance) on changes in BMI, DE and perceived stress?

Study Two: Predictors of weight regain

1. Do stress and affect regulatory systems and processes contribute to changes in weight and eating pathology in a community-based sample of women who have previously lost weight?
2. Do affect regulatory systems and processes also contribute to changes in perceived and physiological levels of stress in this sample of women?

Study Three: The role of life events and difficulties on relapse and recovery from AN

1. What is the role of stressful life changes on recovery and maintenance of ED symptoms following treatment for AN?
2. Do affect regulatory systems and processes contribute to recovery and maintenance of ED symptoms following treatment for AN?

Study Four: The impact of an emotionally expressive writing intervention on bodyweight, eating and eating pathology

1. Will writing about IPEs influence changes in stress, bodyweight, eating behaviours and affect systems and processes during a stressful period?
2. Will specific types of language used by expressive writers determine the changes in stress, bodyweight, DE and affect regulatory systems and processes?

Chapter 4: General Methodology

Introduction

This chapter will present an overview of the methodology that was used to accomplish the research studies in this thesis. Throughout the current research programme certain decisions were made in light of previous research and this Chapter will provide a justification as to why these decisions were made. Therefore, this section will begin with a brief explanation as to why only female data are presented in the current thesis. This will be followed by a summary of the constructs that were measured in this research, the tools that were used to measure these constructs and then a brief description of the four empirical studies that will be presented in this thesis. Finally, because there is as yet only limited psychometric evaluation for the two of the questionnaires used throughout this thesis (the Forms of Self-Criticising/Attacking and Self-Reassuring Scale [FSCRS; Gilbert et al., 2004] and Vulnerable Attachment Style Questionnaire [VASQ; Bifulco et al., 2003]), further psychometric evaluation of the two scales will be described, in particular exploratory (EFA) and confirmatory factor analyses (CFA) of their structure as well as reliability and validity.

Rationale for studying women

Firstly, the evidence discussed in Chapters One and Two demonstrated that there are distinct differences between men and women when it comes to the ability to regulate bodyweight, eating patterns and stress at an optimally healthy level. In summary, women are at an increased risk of experiencing excessive weight gain, frequently report dieting and performing DE behaviours and are more likely to develop an ED. Chapter Two also revealed that, compared to men, women report higher stress levels (Cachelin et al., 1998; Green & Pope, 1999; Koopmans & Lamers, 2007), rate stressful life events as more negative and out of their control (Matud, 2004), eat as a way to cope with stress (Macht & Simons, 2000) and also change

what they eat as a result of stress (Habhab et al., 2009; Roberts et al., 2007, 2013). Therefore, these issues appear to be more salient for women. It was decided, therefore, that this thesis would focus on stress and affect systems in problematic weight regulation in women only. The majority of the studies that are presented in this thesis recruited only women. Where data from male participants were collected (Study 1; Chapter Five), they were not included in the analysis.

Constructs

The current thesis will assess several psychological and behavioural constructs. These include bodyweight, caloric intake, DE, stress, adult attachment, social comparison and self-criticism and reassurance. Problematic weight regulation will be assessed using bodyweight and DE behaviours. Bodyweight will be determined using Quetelet's BMI (kg/m^2) which is a convenient and reliable measure of adiposity (Garrow & Webster, 1985; Kraemer, Berkowitz & Hammer, 1990). The correlation between BMI and a precise measure of body fatness is .70 (Keys, Fidanza, Karvonen, Kimura & Taylor, 1972). Also, as the current study will use self-report and actual measurements of bodyweight, it must be noted here that although actual assessments of weight and height are preferable, the correlation coefficient between self-reported and actual measure of height and bodyweight is around .95 (Rowland, 1990).

The other component of problematic weight regulation is eating pathology which refers to behaviours such as binge eating, dietary restraint and disinhibited eating. The factors that will be examined in relation to problematic weight regulation are stress, affect systems and affect regulatory processes. As described in Chapter Two, stress has been found to contribute to changes in weight, eating and eating pathology. Chapter Three presented social rank and attachment as the two evolved affect systems that influence the ability to manage stress and have been suggested to contribute to eating behaviours. For the purpose of this research, the current thesis will present findings using social comparison as a rank-related construct in relation to stress and problematic weight regulation. Similarly, self-critical and self-

reassuring thoughts and feelings are products of matured social rank and attachment systems, respectively, and will also be examined in relation to stress and problematic weight regulation. Therefore, the current thesis will present a selection of studies which will explore the role of stress and affect regulation in problematic weight regulation.

Measures

Bodyweight and height data were either collected as self-report information or measured by an experimenter. Bodyweight was either converted to or measured in kilograms and height information was either provided as or converted into metres. Where the researcher measured participant weight and height, an electronic Salter Body Analyser Scale (Salter, 2012) and a Seca Leicester Height Measure (Seca, 2012) were used, respectively. Height was measured without footwear and weight was measured without any heavy clothing such as jumpers and jackets and footwear such as boots. These measurements were then used to calculate each participant's BMI by dividing weight in kilograms by height in metres squared (kg/m^2).

Caloric intake was measured using the Food Frequency Questionnaire (FFQ; Cade & Margetts, 1988) *(see Appendix A)*. The FFQ is a 63-item scale which requires individuals to state how frequently they consume different food types such as meats, fruits, cereals etc. Each item was rated on a 6-point Likert scale from "Rarely/Never" to "2 or more times a day". In addition to total caloric intake (which also include alcohol consumption), the FFQ also provides information on calories from key nutrients such as protein, total fat, saturated fatty acids, polyunsaturated fatty acids, carbohydrate, starch, sugars and fibre. For the purpose of the current research, total caloric intake including alcohol was used.

Disordered eating was assessed using the 16th version of the Eating Disorder Examination (EDE; Fairburn, Cooper & O'Connor, 2008) and Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994) *(see Appendix B for the EDE-Q)*. The EDE is a semi-structured interview designed to measure the severity and frequency of disturbed eating behaviours over the past four weeks. The interview

consists of 62 items measuring dietary restraint, bulimic behaviours and eating, weight and shape concerns. Each item is rated on a 7-point scale with higher scores indicating greater severity or frequency. The EDE-Q is the self-report measure derived from the EDE (Cooper & Fairburn, 1987). The EDE-Q is a 36-item questionnaire consisting of 4 subscales assessing dietary restraint and weight, shape and eating concerns. Of the 36 items, 23 are rated on a 7-point Likert scale from "No days" to "Every day" with items such as "Have you tried to avoid eating food which you like in order to influence your shape or weight?" and "Have you had a strong desire to lose weight?" From the remaining 13 items, 6 require a "Yes/No" answer and each of these questions are followed-up with another question requiring the individual to quantify how many times they have experienced the event, e.g., binge eating, vomiting etc. These are not included in the total EDE-Q score. The total score is computed from the dietary restraint and weight, shape and eating concerns subscales with higher scores indicating more DE.

Insecurity of attachment was measured using the Vulnerable Attachment Style Questionnaire (VASQ; Bifulco et al., 2003) *(see Appendix C)*. The VASQ is a 22-item scale assessing behaviours, emotions and attitudes relating to adult attachment. Participants are asked to rate each statement on a 5-point Likert scale ("Strongly disagree" to "Strongly agree"). Items include "I rely on others to help me make decisions" and "I find it hard to trust others". The VASQ can be used to compute a total score reflecting vulnerable attachment and two separate subscales indicating insecure and proximity-seeking attachment patterns. Higher scores indicate a more vulnerable attachment when computing a total score and more insecurity and proximity-seeking attachment when using the subscales. However, the current research programme further developed the VASQ and this is described below (*see Psychometric evaluation of the FSCRS and VASQ*).

Mood was measured using the Short Depression-Happiness Scale (SDHS; Joseph, Linley, Harwood, Lewis & McCollam, 2004) *(see Appendix D)*. The SDHS consists of 6 statements, which required respondents to rate how they have felt over the last 7 days on a 4-point Likert scale ("Never" to "Often"). Items include "I felt dissatisfied with my life" and "I felt that life was meaningless". The SDHS is a bi-

directional scale with lower scores indicating low mood and higher scores indicate greater happiness.

Self-criticism/reassurance was assessed using the Forms of Self-Criticism/Reassuring Scale (FSCRS; Gilbert et al., 2004) *(see Appendix E)*. The FSCRS is a 22-item measure requiring respondents to rate a selection of positive and negative statements on a 5-point Likert scale ("Not at all like me" to "Extremely like me"). Items include "I am easily disappointed with myself" and "I am gentle and supportive with myself". Positive items reflect the ability to self-reassure (referred to as reassured-self) and negative items indicate self-critical thoughts and feelings (split into subscales of inadequate-self and hated-self). The current research programme further developed the FSCRS and this version is described below (*see Psychometric evaluation of the FSCRS and VASQ*).

Social rank was measured using the Social Comparison Rating Scale (SCRS; Allan & Gilbert, 1995) *(see Appendix F)*. The SCRS is an 11-item measure, which required respondents to rate how they judge themselves in comparison to others on rank, attractiveness and how they fit in with others. The items state 'In relation to others I feel…' and each item is rated on a 10-point Likert scale with dimensions such as inferior-superior, left out-accepted and unattractive-more attractive. An example of an item is:

Inferior 1 2 3 4 5 6 7 8 9 10 Superior

Scores below 50 indicate an unfavourable social comparison in which the individual perceives him/herself to be lower status compared to others and scores above 60 indicate a favourable social comparison in which the individual perceives him/herself to be higher status compared to others.

Stress

The current thesis used the following three methods of assessing stress:

Self-report measure

The Perceived Stress Scale-4 (PSS-4; Cohen & Williamson, 1988) is a 4-item self-report measure of stress perceptions *(see Appendix G)*. Individuals were required to rate on a 5-point Likert scale ("Very often" to "Never") the degree to which they appraise situations as stressful. Items include "In the last month, how often have you felt that you were unable to control the important things in your life?" and "In the last month, how often have you felt that things were going your way?" Positive items were reversed and high scores indicated more stress.

Physiological measure

Saliva samples were collected to assess cortisol levels which is a biomarker of stress. Salivary cortisol sampling provides a reliable and stress free estimate of circulating free cortisol. Collection of salivary cortisol using the salivette sampling device provides an optimal method for hygienic collection of saliva. The salivette is a tube which consists of a synthetic swab specifically designed for cortisol determination. All participants were instructed to remove the swab from the salivette and place the swab in the mouth and chew it for about two minutes to stimulate salivation. Participants were also required to complete a short food diary of everything they ate and drank on the day that they took their saliva samples. The food diary enabled us to monitor if participants ate or drank any fluids 30 minutes prior to taking a saliva sample. Participants were provided with re-sealable specimen bags, a permanent marker pen to record the time the saliva sample was taken and a return envelope to post them securely to the researcher. In two of the studies, collection of saliva samples took place at two time points, therefore, all saliva samples collected at T1 were stored at -70°C until samples from T2 were returned. Once all the samples had been collected, saliva samples were transported to KingsPath Clinical Diagnostic Pathology Services for analysis using Radioimmunoassay. Only the samples of those participants who completed all study phases were analysed for cortisol.

Cortisol secretion was determined using area under the curve (AUC), calculated using the trapezoid rule as this formula is widely used in the literature to assess cortisol secretion throughout a specific time period (e.g., Roberts et al., 2007), i.e., AUC = (0.5 x first time-point) + sum of second to penultimate time-point + (0.5 x last time-point). The formula was amended accordingly to account for the different number of saliva samples that were collected across the day in the different studies.

Life events measure

As described in Chapter Two, the current study used the Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978). The LEDS is a semi-structured interview designed to elicit information regarding events and difficulties that occurred over a specified time period (a more detailed description of the LEDS will be provided in Chapter Seven). A wide range of areas are covered in the interview such as education, work, reproduction, housing, money/possessions, legal, health, martial/partner relationships, other relations (e.g., child, parent), and other events (including bereavement). The current study used the definition of severe life events and marked difficulties as set out by Brown and Harris (1978). Therefore, only severe life events that are classified as focused on the participant and rated as 'marked' or 'moderate' on long-term contextual threat were examined. Marked difficulties were determined as those that are focused on the participant and rated as 'high marked', 'low marked' or 'high moderate' for two or more months.

For the current study, the researcher and principal supervisor (who was blind to the participant group assignments) met for consensus meetings and rated the life events and difficulties. Upon any confusion or difficulty in rating a life event or difficulty, advice was sought from Dr. Tirril Harris, who was unaware of the medical status of the participant. The LEDS has been shown to have acceptable levels of reliability and validity (Brown, 1989) and has been used to assess the role of life events and difficulties on a range of physical and psychological disorders.

Additionally, once a severe life event or difficulty was identified during the interview process, the interviewer also asked questions regarding self-critical and reassuring thoughts and feelings in relation to the way the individual responded to the experience *(see Appendix H)*. This enabled the assessment of affect regulation

processes. Additional questioning regarding self-critical and self-reassuring thoughts and feelings in relation to the way individuals responded to severe life events and difficulties were developed by N. A. Troop for the current study and incorporated as part of the LEDS. Questions included "Sometimes when things go wrong, people can be very critical of themselves – were you very critical of yourself?" If participants stated that they were self-critical, additional probing questions were used to elicit further information about the types of things they said or did to themselves. Participants were also asked, "On the other hand, sometimes when people experience problems they can be very self-soothing or very self-reassuring – is this something you did?" and if they replied positively, they were asked "In what way were you soothing or self-reassuring?" These questions were included in the LEDS to gain an understanding if and how participants were self-reassuring at times of difficulty.

Studies

This section will provide a brief description of the four studies that were conducted in this research programme and the measures that were used in each of the studies (*see Table 4.1*).

Study 1: The role of mood and affect systems on the trajectories of BMI, disordered eating and stress

The first study (Chapter Five) was a large, longitudinal study examining the change trajectories of BMI, DE and stress over an 18-month period in a community-based sample. Importantly, this study also examined how changes in BMI, DE and stress influence each other and how mood and affect regulatory systems (social rank and attachment) and processes (self-criticism and self-reassurance) contributed to these changes. The findings of this study have been submitted for publication in a peer-review journal (Kupeli, Norton, Chilcot, Campbell, Schmidt & Troop, 2013).

Study 2: Predictors of weight regain

The second study (Chapter Six) in this thesis examined the role of stress, eating behaviours and affect regulation on weight regain in a small sample of community-dwelling women. In order to achieve this, a group of women who were at least three BMI points lighter than their heaviest adult weight on entry to Study One (see above) were invited to take part in Study Two. Participants completed the same self-report measures as Study One at two time points, six months apart. However, in addition to the self-report measures, this sample of women also gave saliva samples (as a physiological measure of stress) at both time points.

Study 3: The role of life events and difficulties on relapse and recovery from AN

Study three (Chapter Seven) of this research programme employed the LEDS (Brown & Harris, 1978) to examine the role of life events and difficulties on relapse and recovery from AN. In order to achieve this, a group of women who have recovered from AN for at least two years were interviewed on the life events and difficulties they experienced in the two years prior to interview. The group of women who had recovered from AN were compared to a group of patients who completed therapy at the Section of Eating Disorders, Maudsley Hospital. The latter group consisted of those who were in remission following therapy and those who maintained their ED symptoms throughout and following treatment. Therefore, the patients recruited from Maudsley Hospital were interviewed regarding the life events and difficulties they experienced in the two years prior to interview which covers the year of completing therapy and the follow-up year. In line with Studies One and Two, the role of affect regulation on symptom maintenance, relapse and recovery from AN were also examined.

Study 4: The impact of an emotionally expressive writing intervention on bodyweight, eating and eating pathology

The final study (Chapter Eight) of this research programme was an intervention study examining the impact of an emotionally expressive writing task on bodyweight, eating and eating pathology. Expressive writing traditionally involves

writing for 15-20 minutes for 3-5 consecutive days either about a stressful experience or a neutral topic (Pennebaker & Beall, 1986) and has been found to lead to improved health outcomes (e.g., Smyth, 1998). This study used an adapted version of the expressive writing task which involved writing about an intensely positive experience (see Burton & King, 2004) to examine the role of therapeutic writing on changes in bodyweight and eating pathology at times of stress. The current study used a naturally occurring stressor (exams) to explore the role of expressive writing on stress-related changes and affect regulatory systems and processes.

Table 4.1: Instruments/methods of collecting data during each study

	Study 1 Longitudinal	**Study 2 Weight regain**	**Study 3 Anorexia Study**	**Study 4 Intervention**
BMI[1]	✓	✓	✓	✓
FFQ	✓	✓		✓
EDE[2]			✓	
EDE-Q	✓	✓		✓
VASQ	✓	✓	✓	✓
SDHS	✓	✓		✓
FSCRS[3]	✓	✓		✓
SCRS	✓	✓	✓	✓
Stress				
PSS-4	✓	✓		✓
Cortisol		✓		✓
LEDS[3]			✓	

Note:

- [1] Self-report weight and height data were collected in Studies 1, 2 and from the recovered AN participants in Study 3 but actual measurements were taken by clinicians in Study 3 (from AN patients) and by the experimenter in Study 4
- [2] Patients with AN who completed the therapy were assessed using the EDE

- [3] Study 3 also contained interviewer-based questions about self-criticism and self-reassurance

Increasing participation/retention

To increase participation and retention in Studies Two and Four, Amazon vouchers were offered as a gesture of gratitude. For Study Two, participants were awarded £20 worth of Amazon vouchers upon completion of both time points. Similarly, participants who completed all phases of Study Four received a £15 Amazon voucher at the end of the study.

Data collection methods used

The self-report measures in Studies One and Four were completed online using the Bristol Online Survey (BOS; University of Bristol, 2013) facility, whilst Studies Two and Three required participants to complete hardcopy versions of the self-report measures. Please also note that as two of the studies conducted in this research programme required the collection of participant contact information such as email addresses, the BOS facility adheres to ethical requirements as an individual researcher account is used and therefore only the researcher and supervisor had access (i.e., it is not a shared account where other students or staff can access data).

Ethical responsibility

Ethical approval for all of the studies undertaken as part of this research are in accordance with the University of Hertfordshire (UH) Psychology Ethics Committee with Delegated Authority (ECDA) and/or NHS Research Ethics Committee (REC) where relevant (i.e., collection of human tissue or recruitment of patients).

The investigator was trained in the Human Tissue Act 2004 (HTA), therefore, where human tissue (i.e., saliva samples) was collected, this was done in accordance with the HTA 2004. In accordance with the guidelines set in place by the UH Psychology ECDA, all participants who completed sensitive questionnaires, such as those measuring mood and DE were provided with a leaflet of specialist services set up to assist individuals who suffer from mental health problems such as depression or EDs. All participants were also provided with a final debriefing letter explaining

that all participants received an information sheet on specialist services regardless of their scores on the respective scales. Hence, in the event of low moods or eating and/or weight concerns, participants would have details of relevant organisations to contact for advice and support.

Psychometric evaluation of the FSCRS and VASQ

Baseline data collected from Study One were used to conduct both independent EFA and CFA to determine the factor structure of the measures assessing self-criticism/reassurance (FSCRS; Gilbert et al., 2004) and insecurity of attachment (VASQ; Bifulco et al., 2003). In contrast to the other measures used in this thesis, these two scales have not been used extensively and there is limited psychometric evaluation and development. The factor structure of the FSCRS has only been examined once in the original paper describing the development of the measure using a sample of 246 female students (Gilbert et al., 2004). Similarly, the factor structure of the VASQ has only been examined once during its development by Bifulco et al. (2003) in a sample of 262 community-based participants which consisted of middle-aged women and their family members. Importantly, Bifulco et al. (2003) found the VASQ to be a better predictor of the subsequent onset of depression than another widely used measure of attachment styles, the Relationship Questionnaire (RQ; Bartholomew & Horowitz, 1991). A recent review of adult attachment scales (Ravitz et al., 2010) suggested it is a promising measure but in need of further psychometric evaluation.

EFA and CFA are part of a collection of Structural Equation Modelling (SEM) techniques used to develop and refine psychometric assessments. Multiple item assessments in the world of social sciences have been designed to measure at least one construct though, more often, several constructs. Techniques such as EFA and CFA have been developed to explore and determine the relationship between a set of observed and latent variables in order to derive a factor structure that can be readily interpreted. An important distinction between EFA and CFA is that EFA is predominantly a data-driven approach whereas CFA is theory-driven (Fabrigar,

Wegener, MacCallum & Strahan, 1999). EFA is, as the name states, of an exploratory nature as statistical analysis is conducted on the basis that there is limited theoretical reasoning underlying a particular factor structure. In contrast, CFA is used to refine or validate previously determined models in the literature (Flora & Curran, 2004). Fabrigar et al. (1999) recommended that if data from a large sample are readily available, then the dataset should be randomly split into two and an EFA conducted using the first half of the dataset followed by a CFA using the second half of the data. This would enable the EFA to provide the rationale for specifying a specific factor structure for the CFA that follows. Recommended guidelines regarding the number of observed variables sufficient to explain a construct of interest is approximately 3-5 variables per factor (Fabrigar et al., 1999). Several guidelines for the number of participants required per observed variable have been proposed. Everitt's (1975) rule of thumb for the ratio of participants to each observed variable is 10:1, whereas Streiner (1994) advised that 5 participants per observed variable with a minimum sample of 100 participants is adequate. The following section will describe the procedure that was adopted for the development of these two scales. Both procedures were similar except where stated.

EFA

The data were randomly split to perform EFA using the first half of the randomly split dataset (the training sample) in Mplus version 6 (Muthén & Muthén, 2010). Oblique Geomin rotation was employed since the extracted factors were expected to correlate. The number of factors extracted was based on the Kaiser criterion (eigenvalues > 1) and the examination of the Scree plot. In addition, EFA using Mplus allows standard SEM goodness-of-fit indices to be evaluated across several factor solutions. Items were removed from the EFA if their factor loadings were non-significant or if they loaded significantly but weakly (i.e., <.40) onto more than one factor.

The best fitting models identified from the EFA (after removal of non-significant and double loadings) were subsequently selected for CFA using the second half of the dataset (the testing sample) from which post-hoc modifications could be sought and evaluated.

CFA

CFA was also conducted using Mplus version 6 (Muthén & Muthén, 2010). As the responses were approximately normally distributed for the VASQ, analyses were computed using the Maximum Likelihood estimator (ML). However, for the FSCRS, due to polychoric correlations (which refers to using ordinal data to measure a linear relationship between two latent continuous variables), the method of estimation used was Weighted Least-Squares with mean and variance adjustment (WLSMV). Mplus generates several fit indices to assess how well the proposed model fits the sample data. Firstly, the Chi^2 statistic may be used as a measure of fit between the sample covariance and fitted covariance matrices (Byrne, 1998). A non-significant Chi^2 is desired suggesting that the observed and reproduced covariance matrices do not differ significantly and, thus, demonstrate a good model fit. However, due to the large sample size of the current study, a significant Chi^2 is expected based on standard statistical theory of how sample size, power and significance are associated (Cohen, 1992). The corresponding degrees of freedom for each Chi^2 will also be reported. Therefore, in addition to the Chi^2 statistic, several fit indices were evaluated including the Bayesian Information Criterion (BIC), Comparative Fit Index (CFI) and the Tucker Lewis Index (TLI). The model with the lowest BIC is preferred (Raftery, 1995). The CFI and TLI values range between 0 and 1 and values > .95 indicate a reasonable fit (Hu & Bentler, 1999). Root Mean Square Error of the Approximation (RMSEA) is another fit index which takes into account the error of approximation in the population and values $\leq$ .06 indicate a good model fit (Hu & Bentler, 1999).

FSCRS (Gilbert et al., 2004)

Kupeli et al. (2013a) argued that one-, two- and three-factor solutions for the FSCRS were feasible. The rationale behind a one-factor model containing both self-criticism (inadequate- and hated-self) and self-reassurance was based on the similarity between these scales and the self-judgement and self-kindness components of the Self-Compassion Scale (SCS; Neff, 2003a,b), respectively which were combined to represent opposite ends of a single factor. The two-factor solution is based on previous research (Gilbert, Baldwin, Irons, Baccus & Palmer, 2006) which

combined the inadequate- and hated-self subscales to create a single self-criticism factor (because of high intercorrelations) suggesting that the FSCRS contains only two factors, self-criticism and self-reassurance. Finally, the three-factor structure is based on the original principal components analyses conducted by Gilbert et al. (2004) which revealed two separate self-criticism factors (inadequate- and hated-self) and one self-reassurance (reassured-self) factor.

An EFA was initially conducted on half of the data to assess three different factor solutions which revealed that a one-factor (χ^2 (209) = 2307.9, p <.001; CFI = .786, TLI = .763, RMSEA = .115) and two-factor solution (χ^2 (188) = 1268.1, p <.001; CFI = .890, TLI = .864, RMSEA = .087) had poor fit to the data as evidenced by significant Chi^2 tests and fit indices above threshold. A three-factor (χ^2 (168) = 634.6, p <.001; CFI = .952, TLI = .934, RMSEA = .06) solution had acceptable fit to the data. Item-factor loadings for the three-factor solution were examined which led to the removal of Item 4 ("I find it difficult to control my anger and frustration at myself ") due to a factor loading <.40. Items 18 ("I think I deserve my self-criticism") and 20 ("There is a part of me that wants to get rid of the bits I don't like") were also removed since their factor loadings were weak (<.40) and were double loaded thus limiting their interpretation. Removal of these items did not influence the overall value of the measures of sampling adequacy (MSA = .948). The three-factor solution was re-tested after excluding items 4, 18 and 20 and it was found that this model had superior fit to the data (χ^2 (117) = 481.6, p <.001; CFI = .958, TLI = .938, RMSEA = .06). Items loading on the three factors were essentially the same as those in the original solution described by Gilbert et al. (2004). Factor 1 contained 6 items and was labelled *Inadequate-self* (Eigen Value = 9.0, variance explained = 47.5%). Factor 2 contained 5 items and was labelled *Hated-self* (Eigen Value = 1.7, variance explained = 8.8%). The final factor contained 8 items and was labelled *Reassured-self* (Eigen Value = 1.3, variance explained = 6.7%).

The three-factor model derived from the EFA was then evaluated in the second sample's dataset using CFA. This model (χ^2 (98) = 959.1, p < .001; CFI = .958, TLI = .952, RMSEA = .08) demonstrated a reasonable fit to the data as evidenced by the fit indices. However, there were issues regarding item 22 ("I do not like being

me"). In the original solution item 22 loaded onto the HS factor but examination of the modification indices suggested this item should also be predicted by the latent factor *Reassured-self*. When item 22 item is permitted to load onto both HS and RS scales (χ^2 (99) = 800.3, p <.001; CFI = .966, TLI = .961, RMSEA = .07), the fit is significantly better than when it is constrained to load only on the HS scale as shown by a Chi^2 Difference test (DIFFTEST; $\Delta\chi^2$ (1) = 79.5, p <.01). This modified model is shown in *Figure 4.1* accompanied by standardized model coefficients.

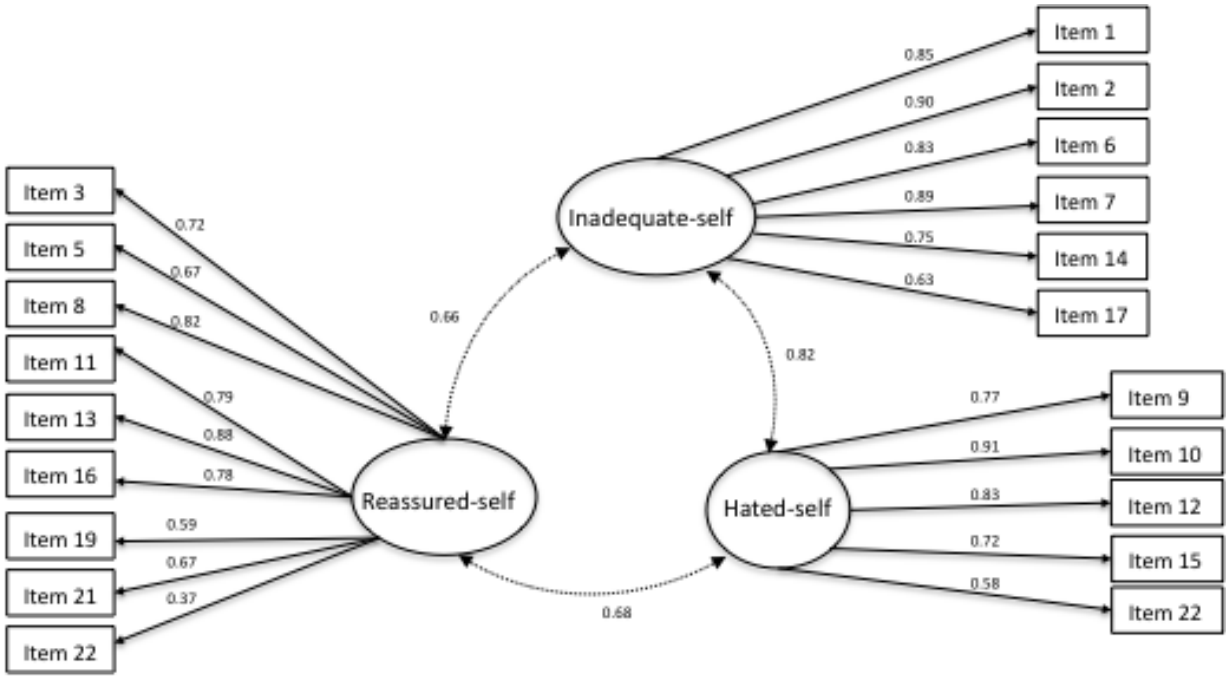


Figure 4.1: CFA: Modified FSCRS model

The three factors all showed logical interrelationships. Inadequate-self correlated highly with hated-self (r = .82, p <.001) and reassured-self (r = .66, p <.001). Hated-self and reassured-self also correlated highly (r = .62, p <.001). Given the double loading of item 22, and to achieve simple structure, a further CFA was carried out removing item 22 completely from both the RS and HS subscales. When item 22 is removed completely, there is a slight improvement in the fit compared to the model in which it is allowed to double-load (χ^2 (93) = 712.4, p <.001; CFI = .967, TLI = .962, RMSEA = .07). In conclusion, a shorter 18-item version of the FSCRS was an improvement on the original 22-item version and was used throughout this thesis.

VASQ (Bifulco et al., 2003)

Kupeli et al. (2013d) suggested that the structure of the VASQ could be presented as a two-, three- and four-factor model. The two-factor structure was based on the original factor analysis conducted by Bifulco et al. (2003) which identifies the insecurity and proximity-seeking subscales, whilst the possibility of the three- and four-factor models was largely exploratory (i.e., based on the Kaiser criterion (eigenvalues > 1) and the examination of the Scree plot.

EFA models extracting between two and four factors were considered. The two factor model revealed the expected (based on the original paper) insecurity and proximity-seeking structure, however the fit of the model was poor using standard SEM criteria (χ^2 (188) = 946.5, p <.001; CFI = .765, TLI = .711, RMSEA = .08). The three factor model split the proximity-seeking scale into separate factors, but still exhibited poor model fit (χ^2 (168) = 982.3, p <.001; CFI = .840, TLI = .781, RMSEA = .07). The four factor model further split the insecure factor into two subscales (χ^2 (149) = 513.3, p <.001; CFI = .887, TLI = .825, RMSEA = .06). The fit of this model was still outside acceptable limits but was chosen for further analysis because it provided a closer fit to the data than the two or three factor solutions and the pattern of loadings made theoretical sense.

Using the criteria outlined above, items 1, 3, 4, 9, 14, 17 and 21 were excluded and the four-factor solution re-estimated. The fit of the model was within acceptable limits (χ^2 (51) = 98.1, p <.001; CFI = .978, TLI = .955, RMSEA = .04). The loading pattern was similar to the original solution described by Bifulco et al. (2003) but with the original Insecurity and Proximity-seeking scales each split into two further subscales and item 13 (“I am clingy with others”) loading onto one of the insecurity subscales instead of a proximity-seeking subscale. Of the two subscales spilt from the original proximity-seeking subscale, one reflected overreliance and difficulty making decisions while the other reflected dependence and fear of abandonment. These were labelled *lack of autonomy* (common variance explained = 19.8%) and *anxious-dependent* (common variance explained = 20.7%), respectively. Of the two subscales that split from the original insecure subscale, one reflected dismissiveness and mistrust, while the other reflected antagonism and clinginess.

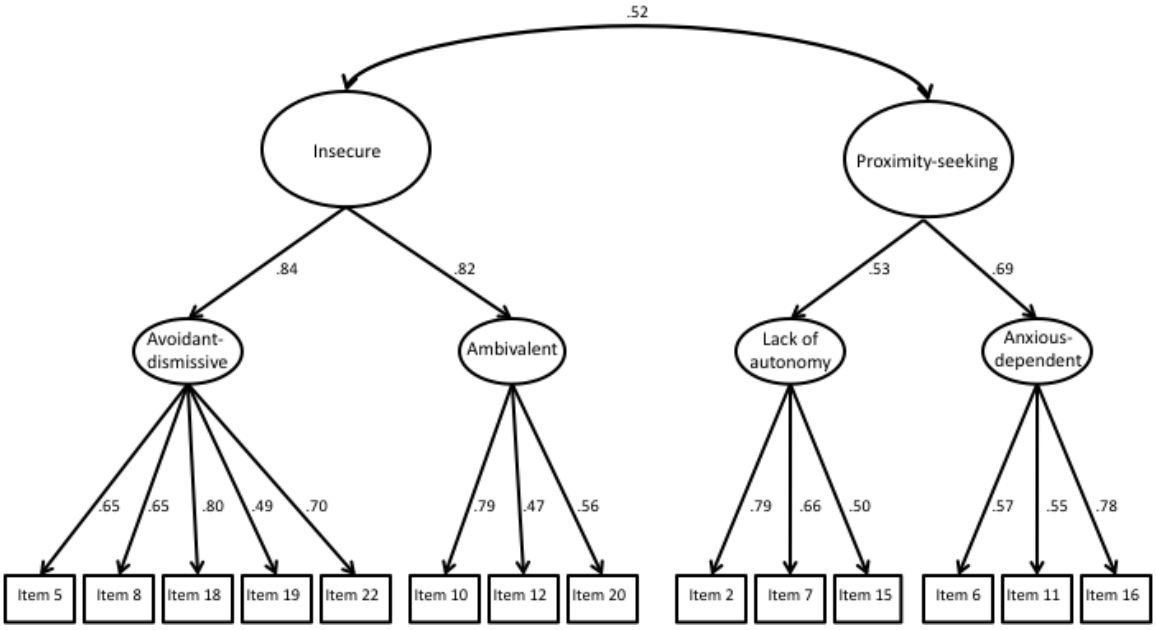
These were labelled *avoidant-dismissive* (common variance explained = 29.6%) and *ambivalent* (common variance explained = 30.0%), respectively.

Prior to evaluating the four-factor model derived from the EFA, the original factor structure proposed by Bifulco et al. (2003) was examined in a CFA in the testing sample and again revealed poor fit to the data since all fit indices were outside of their recommended cut-off ranges (χ^2 (208) = 1407.2, p <.001; CFI = .675, TLI = .639, RMSEA = .10).

Following the exclusion of several items (see above), the four-factor model derived from the EFA was evaluated in the testing sample using CFA. This model demonstrated a reasonable fit to the data as evidenced by the fit indices (χ^2 (84) = 303.6, p <.001; CFI = .905, TLI = .881, RMSEA = .06). However, there were issues regarding item 13, as in the EFA solution: item 13 loaded onto the *Ambivalent* factor but examination of the modification indices suggested this item should also load on the other three factors. Given the multiple loading of item 13 and, as the wording of this item seemed to be best described by the *Ambivalent* subscale, a further CFA was carried out removing item 13 completely. The fit indices of this modified model were within acceptable levels (χ^2 (71) = 186.7, p <.001; CFI = .945, TLI = .929, RMSEA = .05).

While a four-factor solution has been found here, the original factor structure described by Bifulco et al. (2003) suggests that a two-factor solution, combining the two Proximity-seeking subscales (*Lack of autonomy* and *Anxious-dependent* [6 items]) together and the two Insecurity subscales (*Avoidant-dismissive* and *Ambivalent* [8 items]) together, might also be sufficient (i.e., adopting a two-factor solution proposed by Bifulco et al. [2003] but including only those items retained in the CFA presented above). This alternative higher-order model was tested and revealed a slight decline in all fit indices but still had a reasonable fit to the data, providing support for Bifulco et al.'s (2003) two-factor structure (χ^2 (74) = 212.0, p <.001; CFI = .934, TLI = .919, RMSEA = .05), albeit with a smaller number of items (n = 14). The higher-order model is shown in *Figure 4.2* accompanied by standardised model coefficients. In conclusion, the shorter 14-item version of the VASQ was an

improvement on the original 22-item version and this is the version that was used throughout this thesis.



Note. All standardised coefficients are significant at $p < .001$

Figure 4.2: CFA: Higher-order VASQ model

Reliability analysis of scales

Internal reliabilities of each of the scales used during each time points of the four studies were assessed using Cronbach's alpha (*see Tables 4.2, 4.3 and 4.4*). The first table presents the results of the reliability analysis for Studies One, Two and Four. The EDE-Q and its subscales, SDHS, SCRS, PSS-4 and the FSCRS were all found to have good internal reliability across Studies One, Two and Four (*see Table 4.2*). Although the overall VASQ and the higher order factors were found to have acceptable internal reliability across Studies One, Two and Four, the Anxious-dependent and Ambivalent subscales of the four-factor structure did not fare so well.

Table 4.2: Cronbachs alpha for scales at each time point during Studies One, Two and Four

	Study 1				Study 2		Study 4	
Variable	**Time 1**	**Time 2**	**Time 3**	**Time 4**	**Time 1**	**Time 2**	**Time 1**	**Time 2**
(Number of items)	**(*N* = 1155)**	**(*N* = 320)**	**(*N* = 247)**	**(*N* = 228)**	**(*N* = 42)**	**(*N* = 38)**	**(*N* = 76)**	**(*N* = 57)**
EDE-Q								
Total score *(22)*	.95	.95	.94	.95	.94	.93	.95	.96
EDE-Q: Subscales								
Dietary restraint *(5)*	.83	.84	.82	.82	.80	.79	.84	.92
Eating concerns *(5)*	.84	.84	.85	.86	.85	.83	.84	.90
Weight concerns *(5)*	.84	.84	.81	.84	.81	.78	.82	.89
Shape concerns *(8)*	.92	.93	.91	.92	.88	.88	.92	.94
SDHS *(6)*	.89	.88	.89	.91	.91	.91	.89	.84
SCRS *(11)*	.93	.93	.92	.93	.91	.93	.94	.93
PSS-4 *(4)*	.80	.82	.83	.87	.81	.76	.82	.85
VASQ								
Total score *(14)*	.77	.76	.78	.76	.78	.77	.81	.81
VASQ: Two-factor structure								
Insecurity *(8)*	.80	.81	.82	.84	.88	.77	.82	.84
Proximity-seeking *(6)*	.65	.67	.71	.69	.69	.73	.56	.68

VASQ: Four-factor structure								
Lack of autonomy *(3)*	.70	.77	.73	.74	.83	.82	.68	.65
Anxious-dependent *(3)*	.68	.68	.76	.78	.75	.83	.54	.63
Ambivalent *(3)*	.66	.62	.65	.62	.60	.42	.52	.55
Avoidant-dismissive *(5)*	.78	.80	.83	.85	.89	.80	.78	.83
FSCRS								
Inadequate-self *(6)*	.89	.92	.91	.94	.88	.93	.92	.91
Reassured-self *(8)*	.89	.88	.90	.92	.91	.92	.90	.90
Hated-self *(4)*	.84	.86	.83	.83	.79	.86	.88	.78

Table 4.3 presents the reliability analysis for the SCRS, VASQ and VASQ subscales for those who have recovered from AN and those who completed therapy at the Section of Eating Disorders, Maudsley Hospital during Study Three. The patient group completed the self-report measures (SCRS and VASQ) on four occasions, once at baseline (pre-treatment), T4 (end of treatment), T5 (1-year follow-up) and T6 (following completion of the LEDS which took place shortly after T5). The patients were also contacted during the therapy (T2) but this was a short telephone follow-up and patients did not complete self-report measures at this time point. As the current study was designed to examine recovery and relapse, T3 data for the self-report measures was not used as part of this study as T3 took place six months after baseline which was whilst patients were still attending monthly follow-up therapy sessions. The recovered group completed the self-report measures once following completion of the LEDS (equivalent to T6 for the patient group).

The results show that the SCRS has good internal reliability when completed by a clinical population. Similar to the results of Studies One, Two and Four, the VASQ and its subscales of the two-factor structure (Insecurity and Proximity-seeking) were found to have acceptable internal reliability, however, the Lack of Autonomy, Anxious-dependent and Ambivalent subscales of the four-factor structure were found to have very poor internal reliabilities. Therefore, a decision was made to use the scores from the overall VASQ and the two-factor structure consisting of Insecurity and Proximity-seeking styles when assessing the role of attachment in relation to problematic weight regulation in the present thesis.

Table 4.3: Cronbachs alpha for the SCRS and VASQ completed by patients at various stages of treatment and recovered participants during Study Three

Variable	Patients (*n* = 14)				Recovered
(Number of items)	**T1**	**T4**	**T5**	**T6***	**(*n* = 16)***
SCRS *(11)*	.94	.84	.92	.83	.87
VASQ					
Total score *(14)*	.74	.71	.85	.74	.67
VASQ: Two-factor structure					
Insecurity *(8)*	.77	.77	.82	.82	.76
Proximity-seeking *(6)*	.50	.62	.82	.55	.64
VASQ: Four-factor structure					
Lack of autonomy *(3)*	.61	.26	.50	.68	.78
Anxious-dependent *(3)*	.67	.54	.81	.78	.62
Ambivalent *(3)*	.10	.21	.55	.76	.79
Avoidant-dismissive *(5)*	.90	.89	.90	.90	.85

Note. *Denotes the point at which both groups completed the LEDS, SCRS and VASQ (Patient group completed the SCRS and VASQ for the last time during LEDS)

Table 4.4 displays the results of the reliability analysis for the EDE and its subscales which were used to assess eating pathology of the patients who took part in the trial at the Maudsley Hospital. The results of the reliability analysis for the EDE and its subscales suggest that this scale has good overall internal reliability.

Table 4.4: Cronbachs alpha for EDE and its subscales completed by AN patients (*n* = 14) during Study Three

Variable *(Number of items)*	T1 (Baseline)	T4 (End of treatment)	T5 (1-year follow-up)
EDE			
Total score *(22)*	.90	.95	.94
EDE: Subscales			
Dietary restraint *(5)*	.75	.88	.82
Eating concerns *(5)*	.67	.81	.70
Weight concerns *(5)*	.74	.80	.86
Shape concerns *(8)*	.89	.95	.92

The current chapter has provided an indepth overview of the constructs that will be examined in this research and tools that will be employed to measure them. The next four chapters present the empirical studies that have used these methods to answer the research questions set out in Chapter Three (*see Justification for the current research*).

Chapter 5: Study 1

The role of mood and affect systems on the trajectories of BMI, disordered eating and stress

Introduction

All of the evidence presented in earlier chapters is pertinent to this study. There is therefore no need to report it here. However, based on the evidence that has accumulated from the first three chapters, here is a summary of the most important points and how they led to the current research questions:

- Weight gain with age is normal
- Problematic weight regulation such as extreme fluctuations in weight and DE behaviours is associated with health problems and mortality
- History of weight loss is related to later weight regain
- Stress and life events contribute to changes in weight, specifically weight gain and although the evidence is less consistent, stressful life experiences are also related to weight regain following weight loss from obesity
- Affect systems (social rank and attachment) and processes (self-criticism/reassurance) have been implicated in the stress process and eating pathology but have, as yet, been largely unexplained in relation to changes in weight

Current study

Both Wardle et al.'s (2010) meta-analysis and other studies have demonstrated that stress relates to problematic weight regulation through physiological and behavioural changes (Dallman et al., 2003; Robert et al., 2007). However, many studies only measure stress at baseline as a predictor of weight change. Others, though longitudinal, have included only two time-points, which limits the ability to infer actual

change, rather than examining trajectories of change in stress and weight over multiple time-points. Wardle et al. (2010) also point out that the majority of the studies did not include a measure of food intake or what they refer to as "psychological eating behaviours" (e.g. binge eating, emotional eating, disinhibition). Stress has been associated with eating pathology (Ball & Lee, 2000; Bennett & Cooper, 1999) including disruption of dietary restraint behaviours resulting in overeating and weight gain (Wardle, Steptoe, Oliver & Lipsey, 2000). Since stress is a common factor that influences both BMI and DE, the present report focuses on those factors that are implicated in the stress process. Specifically, the role of affect systems (attachment and social rank), as well as affect regulation processes (self-criticism/reassurance) will be considered. Although attachment, self-criticism/reassurance and social rank have been related to experiences of stress and reported to influence the development of eating pathology, these affect regulatory systems have not been examined in relation to changes in weight. As the stress process has been found to contribute to weight change and eating pathology, it is important to assess how these affect regulatory systems and processes contribute to stress and its effect on weight and DE. The current study will also examine different aspects of the proposed model that was presented in Chapter Three (*see Figure 3.2*), including the relationship between stress, eating and weight, the role of affect regulatory systems and processes on stress and also, how these systems and processes may also contribute to eating and weight in a community-based sample of women.

Therefore, this study has been designed to answer the following questions which are split into broad and specific questions:

Broad research questions:

1) What are the trajectories of BMI, DE and stress and how do these trajectories influence each other in a community-based sample of women?
2) What is the impact of mood, affect regulatory systems (social rank, attachment) and processes (self-criticism/reassurance) and eating on changes in BMI, DE and

perceived stress?

Specific research questions:

1) What is the relationship between BMI, DE, stress, mood, affect regulatory systems and processes and eating in a sample of community-based women?
2) Do any of the variables of interest predict drop-out between T1 and T2?
3) Do BMI, DE and stress levels change over time when examined separately and are these changes dependent on the baseline level?
4) What are the predictors of BMI, DE and stress when assessed separately?
5) Do baseline levels and changes in BMI, DE and stress influence each other and what are the predictors of these levels when examined concurrently?

Methodology

Design

This was a longitudinal observational study, which consisted of four phases, all approximately six months apart. In addition to BMI (kg/m^2), scores on scales measuring DE, mood, stress, insecurity of attachment, social rank, self-criticism/reassurance and caloric intake were collected at each time point during the present study. Ethical approval for the current study was granted by the University of Hertfordshire School of Psychology Research Ethics Committee.

Participants

At baseline (T1), a total of 1157 female participants took part. The participants were a community-based sample of women with no known EDs. The number of participants who completed each stage of the study and the corresponding mean age and BMI are shown in *Figure 5.1*. From the 1157 participants, 820 females indicated that they would like to take part in future research and were sent the web link to the

remaining three phases of the study. Follow-ups took place within a mean of 2.9 months between T1 and T2, 6.4 months between T2 and T3 and 6.7 months between T3 and T4.

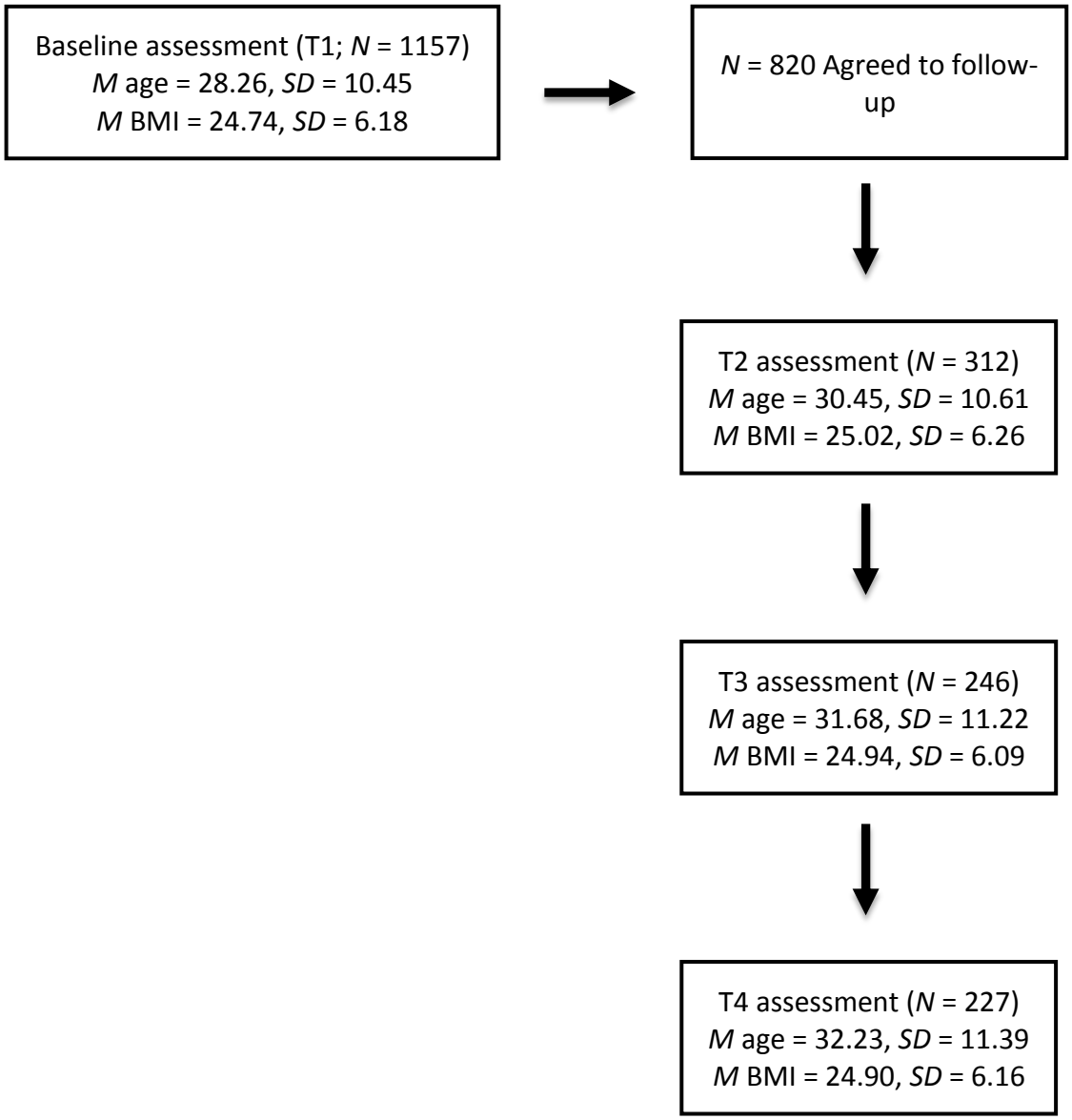


Figure 5.1: Flow diagram presenting the number of participants recruited at each time point and the corresponding mean age and BMI

Baseline demographic information is presented in *Table 5.1* which shows that the majority of the sample was British, single or in a relationship, had A Levels and/or a degree and was studying or in employment.

Table 5.1: Baseline demographic variables (*N* = 1157)

Variable	% (*n*)
Ethnicity	
British	63.7 (737)
Other European	11.5 (133)
Indian	4.0 (46)
Bangladeshi	.6 (7)
Pakistani	2.4 (28)
Caribbean	1.6 (19)
African	2.9 (34)
Chinese	1.4 (16)
Mixed ethnicity	3.3 (39)
Other	8.5 (39)
Marital status % (*n*)	
Single	37.4 (433)
Married/Cohabiting	33.2 (385)
In a relationship	25.5 (295)
Divorced/Widowed	3.8 (44)
Highest education % (*n*)	
GCSE	7.0 (81)
A Levels	41.1 (476)
Bachelors	31.4 (363)
Postgraduate	19.2 (222)
None	1.3 (15)
Employment % (*n*)	
Employed	40.6 (469)
Studying	50.0 (579)
Unemployed	9.4 (109)

Recruitment

Participants were recruited from the general population using a variety of opportunistic sampling methods. These included advertising the study on social networking sites such as Facebook and on forums promoting healthy living. Other recruitment methods included advertising the study in newspapers such as the London Metro and the Evening Standard and sending circular emails to students at the University of Hertfordshire and Kings College London/Institute of Psychiatry. During phase one of the study, participants were given the opportunity to take part in a longitudinal study and if participants indicated that they would like to be included, they were required to provide an email address in order to be sent further information regarding the longitudinal study. Those who indicated that they would like to be involved in the longitudinal study at phase one of the study were sent an email inviting them to take part in the remaining phases.

Measures and Procedure

Participants who were interested in taking part in the study were directed to the survey, which was created using the BOS facility (www.survey.bris.ac.uk). Participants were required to complete the following scales in addition to providing basic demographic information such as age, sex, height and weight measurements (to calculate BMI kg/m^2), ethnicity, marital status, highest education qualification and occupation. Participants were required to complete the following measures at each time point (*see Chapter Four for a full description of each of the measures used in this study*):

1. Caloric intake was measured using the FFQ (Cade & Margetts, 1988)
2. Disordered eating was assessed using the EDE-Q (Fairburn & Beglin, 1994)
3. Insecurity of attachment was measured using the VASQ (Bifulco et al., 2003)
4. Mood was measured using the SDHS (Joseph et al., 2004)
5. Self-criticism/reassurance was assessed using the FSCRS (Gilbert et al., 2004)
6. Social rank was measured using the SCRS (Allan & Gilbert, 1995)

7. Stress perceptions were assessed using the PSS-4 (Cohen & Williamson, 1988)

Statistical analysis

Several statistical analyses were conducted. Firstly, boxplots, descriptive statistics and correlational analyses were performed to determine the relationship between the variables of interest. Boxplots were generated to examine the dispersion and skewness of the data for each of the variables. This preliminary analysis revealed that caloric intake was positively skewed (skewness = 1.87) and highly kurtotic (kurtosis = 9.10), therefore, the data were log transformed. Secondly, correlations between the variables of interest were examined using Pearson's correlation coefficients.

Following correlational analysis, longitudinal data analysis was conducted as repeat observations collected during longitudinal studies like the present study enable the assessment of both between-subject and within-subject change over time. However, appropriate statistical analysis must be used in order to capture the change that occurs between individuals in a sample and within individuals in the same sample. Additionally, there are several features of longitudinal data that require advanced statistical analysis such as different sources of heterogeneity (within- and between-subject), non-independent within-subject responses, between-subject variation that may not be stable over time and the common occurrence of missing or incomplete data (Wang & Wang, 2012). Traditional methods such as Repeated Measures Analysis of Variance (RM-ANOVA) have well known limitations (Gueorguieva & Krystal, 2004). These limitations include observations must be collected at the same time for all individuals, omission of observations that contain missing values increasing the risk of sample bias and equal correlations between measures taken at different time points, for example, measurement correlation for T1 and T2 must be equal to measurement correlation between T1 and T3 (Gueorguieva & Krystal, 2004). Therefore, the current study will conduct Latent Growth Curve Modeling (LGCM) which is a common form of longitudinal data analysis used to get around limitations of RM-ANOVA (Gueorguieva & Krystal, 2004). LGCM is a variant of Latent Variable Modeling (LVM), that allow for both latent

(unobserved), and observed variables to be included in the analysis. LGCM analysis is a useful technique for analysing longitudinal data as it is an extension of linear regression but, in addition to providing two latent factors that assess the initial level (intercept), it also calculates the trajectory and the rate of change of the outcome variable over time (slope). In addition to the latent intercept and slope factors, LGCM also calculates variances which describes the individual variation around the overall means for the intercept and slope of each outcome variable. The association between the intercept and slope of the outcome variables can also be assessed using LGCM analysis in addition to the predictors of each of the outcome variables. LGCM can also be conducted to test nonlinear models such as a quadratic model, which describes a curve that is concave up or down over time. Additionally, LGCM can also be extended to run parallel process models (i.e., multivariate) which enable simultaneous modelling of multiple correlated outcome variables (i.e., BMI, EDE-Q and PSS-4 simultaneously).

LGCM will be conducted using Mplus version 6 (Muthén & Muthén, 2010) with analyses computed using the Full Information Maximum Likelihood estimator (FIML) as responses were approximately normally distributed. FIML is a useful method of estimating models as it uses every piece of information in the observed data for analysis. FIML is also a more efficient and less biased method for dealing with missing data, that assumes data are missing at random (MAR), than traditional approaches such as mean imputation methods, which assume data are missing completely at random (MCAR) (Wang & Wang, 2012). The MAR assumption means that missing data (e.g., due to drop-out) will not bias results as long as variables related to missingness are included in the model. The use of auxiliary variables is a strategy that can be adopted to extend the MAR assumption when using FIML. Auxiliary variables are variables that are not estimated as part of the model but are correlated with the variables and are allowed to influence variables estimated in the model and which can improve power and reduce bias (Graham, 2009). Auxiliary variables in the present study were determined by identifying predictors of those who went on to complete T2 compared to those who dropped out between T1 and T2.

Mplus (Muthén & Muthén, 2010) was then used to run several models to examine changes in BMI, EDE-Q and PSS-4 total scores over the four phases. Firstly, preliminary analysis (*see Preliminary analysis section under Results*) was conducted to ensure that the data were suitable for LGCM. Unconditional univariate (i.e., without covariates) LGCMs for BMI, EDE-Q and PSS-4 were conducted to assess the intercept and slope of each of the variables. Several conditional univariate (i.e., including covariates) LGCMs were conducted to examine the effect of psychological variables and caloric intake variables on the intercept and slope of BMI, EDE-Q and PSS-4 separately. This was conducted by regressing the intercept and slope of the outcome variables onto the predictor variables. All predictor variables were entered into the model simultaneously in order to examine the effect of each predictor while the effects of the other predictor variables were controlled for. The variables that were used as predictors for the conditional models predicting BMI were age, EDE-Q, SDHS, PSS-4, VASQ, SCRS, IS, RS, HS and caloric intake as measured at baseline. The models that followed the initial model for BMI were amended to firstly, include the EDE-Q sub-factors (dietary restraint, weight, shape and eating concerns) instead of the total score and secondly the higher-order factors derived from the VASQ (insecure and proximity-seeking). These models were conducted in order to determine which sub-types of EDE-Q and VASQ influence BMI. The model containing the EDE-Q sub-factors were also performed using the higher-order factors of the VASQ, separately. The conditional models predicting PSS-4 were similar to that of BMI but also including BMI as a predictor. The variables that were used as predictors for the conditional models predicting EDE-Q were age, BMI, SDHS, PSS-4, VASQ, SCRS, IS, RS, HS and caloric intake as measured at baseline. The model that followed the initial model for EDE-Q were amended to include the VASQ sub-factors (insecurity and proximity-seeking). As BMI, EDE-Q and PSS-4 are associated outcomes, unconditional and conditional multivariate LGCMs were also conducted with the observed variables for the four time points for each of these variables used to compute latent variables to represent the initial level and the changes in each of these variables.

Similar to the conditional univariate models of BMI and PSS-4, the VASQ total score was swapped for the sub-factors, insecurity and proximity-seeking styles.

As described in the EFA and CFA section of Chapter Four, several fit indices were examined to assess how well the proposed model fits the sample data. Firstly, a nonsignificant Chi^2 statistic was sought but once again due to the large sample size of the current study, a significant Chi^2 was expected based on standard statistical theory of how sample size, power and significance are associated (Cohen, 1992). Therefore, in addition to the Chi^2 statistic, several fit indices were also examined. In addition to the BIC, CFI, TLI and RMSEA described in Chapter Four, Akaike's Information Criterion (AIC) was also examined. Information criteria such as AIC is commonly used for model comparisons with the model with smaller values indicating better fit (Raftery, 1995). The RMSEA values are also provided with 90% confidence intervals (CI) and a well-fitting model would have a lower CI close to zero and an upper CI close to .08 (Hu & Bentler, 1999).

Results

Sample characteristics

Means, standard deviations and intercorrelations between the three outcome variables at each time point and the predictor variables at baseline are presented in *Table 5.2*. The variables of interest include BMI, EDE-Q and PSS-4 variables at all four time points (each time point is shown with a superscript both in the text and in *Table 5.2*). Baseline variables used as predictors of change were age, EDE-Q sub-factors (dietary restraint and eating, weight and shape concerns), SDHS, SCRS, VASQ and higher-order factors (insecure and proximity-seeking), FSCRS scales (IS, RS and HS) and caloric intake.

<u>Question 1:</u> What is the relationship between BMI, DE, stress, mood, affect regulatory systems and processes and eating in a sample of community-based women?

Several variables were found to significantly correlate with age including BMI[1-4], PSS-4[1], dietary restraint, HS and VASQ and its sub-factors, insecure and proximity-seeking. BMI[1-4] positively correlated with EDE-Q[1-4], however, PSS-4[1] positively correlated with BMI at time one, three and four. As expected, all of the EDE-Q sub-factors positively correlated with EDE-Q[1-4] but also with BMI[1-4] and PSS-4[1-4]. Both SDHS and SCRS negatively correlated with BMI[1-4], EDE-Q[1-4] and PSS-4[1-4] demonstrating that happier mood and higher social rank at baseline is related with lower BMI and EDE-Q and PSS-4 scores at each time point. VASQ and its insecure component were positively correlated with BMI[1], EDE-Q[1-4] and PSS-4[1-4]. However, the proximity-seeking component of VASQ was negatively correlated with BMI[2, 3] but positively correlated with EDE-Q[1] and PSS-4[1-4]. IS and HS as the two self-criticism components of the FSCRS were found to positively correlate with BMI[1-4], EDE-Q[1-4] and PSS-4[1-4] and RS was negatively correlated with BMI[1-4], EDE-Q[1-4] and PSS-4[1-4]. Lastly, caloric intake was negatively correlated with BMI[1] and EDE-Q[1, 3, 4] scores indicating that higher baseline caloric intake is associated with lower BMI and EDE-Q scores at these time points.

Table 5.2: Descriptive statistics and intercorrelations between the outcome variables at each time point and the predictor variables at baseline (*N* for each of the outcome variables presented on the diagonal)

Variable	*M*	SD	BMI[1]	BMI[2]	BMI[3]	BMI[4]	EDE-Q[1]	EDE-Q[2]	EDE-Q[3]	EDE-Q[4]	PSS-4[1]	PSS-4[2]	PSS-4[3]	PSS-4[4]
BMI[1]	24.74	6.18	1152											
BMI[2]	25.02	6.26	.98**	310										
BMI[3]	24.94	6.09	.97**	.96**	242									
BMI[4]	24.90	6.16	.96**	.98**	.99**	223								
EDE-Q[1]	2.13	1.46	.39**	.34**	.33**	.30**	1053							
EDE-Q[2]	2.08	1.45	.36**	.34**	.32**	.31**	.89**	285						
EDE-Q[3]	1.94	1.35	.29**	.30**	.31**	.30**	.80**	.81**	233					
EDE-Q[4]	1.90	1.42	.35**	.31**	.30**	.34**	.84**	.84**	.85**	223				
PSS-4[1]	11.33	3.34	.13**	.08	.19**	.21**	.41**	.36**	.44**	.44**	994			
PSS-4[2]	11.92	7.13	.05	.001	.01	.03	.27**	.28**	.18*	.27**	.43**	278		
PSS-4[3]	10.32	3.34	.03	.01	.02	.10	.33**	.29**	.35**	.39**	.52**	.28**	230	
PSS-4[4]	10.66	3.53	.11	.01	.11	.09	.41**	.40**	.34**	.42**	.54**	.20**	.65**	220
Age[1]	28.26	10.45	.26**	.27**	.23**	.27**	.03	-.01	.03	.03	-.07*	-.08	-.04	-.07
Restr[1]	1.77	1.60	.15**	.09	.14*	.09	.77**	.61**	.61**	.62**	.19**	.09	.17*	.22**
Eating[1]	1.26	1.42	.30**	.22**	.18**	.20**	.87**	.76**	.69**	.77**	.42**	.30**	.35**	.41**
Weight[1]	2.32	1.71	.44**	.40**	.39**	.35**	.95**	.84**	.76**	.80**	.40**	.26**	.31**	.39**

Shape[1]	2.81	1.83	.42**	.38**	.36**	.34**	.96**	.86**	.76**	.81**	.43**	.28**	.32**	.43**
SDHS[1]	17.67	4.17	-.20**	-.17**	-.23**	-.21**	-.49**	-.51**	-.43**	-.51**	-.71**	-.41**	-.47**	-.53**
SCRS[1]	59.04	18.02	-.21**	-.16**	-.22**	-.20**	-.42**	-.39**	-.37**	-.48**	-.52**	-.32**	-.46**	-.55**
VASQ[1]	38.46	7.80	.07*	-.06	.01	-.03	.35**	.25**	.23**	.28**	.52**	.38**	.42**	.43**
Insec[1]	20.43	5.79	.11*	.03	.12	.05	.36**	.32**	.26**	.30**	.46**	.35**	.37**	.43**
Prox[1]	18.03	4.03	-.02	-.14*	-.17*	-.12	.16**	-.01	.04	.08	.34**	.21**	.23**	.16*
IS[1]	18.29	6.39	.16**	.10	.20**	.24**	.49**	.44**	.43**	.52**	.57**	.33**	.40**	.51**
RS[1]	25.78	6.78	-.16**	-.15*	-.20**	-.21**	-.43**	-.44**	-.45**	-.51**	-.58**	-.39**	-.49**	-.51**
HS[1]	7.17	3.96	.22**	.13*	.14*	.13	.53**	.57**	.46**	.58**	.53**	.41**	.48**	.53**
Cal Int[1]	1478.54	687.73	-.10**	-.07	-.05	-.12	-.16**	-.07	-.23**	-.16*	-.02	.06	-.11	-.06

Note. * p <.05; ** p <.001; [1] Time 1; [2] Time 2; [3] Time 3; [4] Time 4;

<u>Question 2:</u> Do any of the variables of interest predict drop-out between T1 and T2?

Completers and non-completers at T2 differed on the following baseline variables (*see Table 5.3*): age, VASQ and the VASQ sub-factors, insecure and proximity-seeking. Those who were older, and reported less vulnerable attachment (including on both the insecure and proximity-seeking subscales) were more likely to take part at phase two. As age and VASQ were related to drop-out, to reduce the risk of bias, these variables were included in all of the models as a predictor or auxiliary variable.

Table 5.3: Independent sample *t*-test results comparing completers and non-completers at T2 on baseline variables

Variable	Completed T2	*N*	*M*	SD	Significance
Age	No	849	27.59	10.36	$t(1155) = -3.67$,
	Yes	308	30.12	10.48	$p < .001$
BMI	No	846	24.67	6.13	$t(1150) = -.64$,
	Yes	306	24.93	6.33	$p = .53$
Caloric intake (inc alcohol)	No	849	1469.41	687.82	$t(1155) = -.75$,
	Yes	308	1503.73	687.97	$p = .45$
EDE-Q (Total score)	No	756	2.16	1.48	$t(1051) = .96$
	Yes	297	2.07	1.41	$p = .34$
Dietary restraint	No	756	1.80	1.63	$t(1051) = 1.03$,
	Yes	297	1.69	1.53	$p = .30$
Eating concerns	No	756	1.29	1.42	$t(1051) = 1.23$,
	Yes	297	1.17	1.41	$p = .22$
Weight concerns	No	756	2.34	1.74	$t(1051) = .66$,
	Yes	297	2.26	1.65	$p = .51$
Shape concerns	No	756	2.84	1.84	$t(1051) = 1.03$,
	Yes	297	2.72	1.78	$p = .30$
SDHS	No	715	17.61	4.13	$t(1005) = -.74$,
	Yes	292	17.82	4.26	$p = .46$
SCRS	No	715	59.32	18.25	$t(1005) = .75$,
	Yes	292	58.37	17.45	$p = .45$
PSS-4	No	703	11.46	3.28	$t(992) = 1.94$,
	Yes	291	11.01	3.45	$p = .05$
VASQ (Total score)	No	692	39.05	7.84	$t(979) = 3.69$,
	Yes	289	37.05	7.52	$p < .001$
Insecurity	No	692	20.76	5.77	$t(979) = 2.71$,
	Yes	289	19.66	5.76	$p = .01$
Proximity-seeking	No	692	18.29	4.00	$t(979) = 3.20$,
	Yes	289	17.39	4.05	$p = .001$
Inadequate-Self	No	685	18.21	6.33	$t(972) = -.60$,
	Yes	289	18.48	6.55	$p = .55$
Reassured-Self	No	685	25.79	6.85	$t(972) = .06$,
	Yes	289	25.76	6.62	$p = .95$
Hated-Self	No	685	7.16	3.91	$t(972) = -.08$,
	Yes	289	7.18	4.08	$p = .94$

Preliminary analysis

Preliminary analysis was conducted to ensure that the data were suitable for conducting LGCM. To test for nonlinearity, LGCMs including a quadratic term were also run to test for a quadratic shape of BMI, EDE-Q and PSS-4, which revealed that a linear model was adequate for each outcome variable. Due to the variability in the intended and actual follow-up periods, sensitivity analysis was conducted. This involved comparing model estimates from unconditional univariate models where time since baseline assessment and each follow-up was either the intended number of months (i.e., 0, 6, 12 and 18 months) or the actual number of months from the baseline assessment and each follow-up. This revealed that the variability in follow-up did not impact on the results. Therefore, since it can be assumed that the data have balanced occasions where all the data were collected at the same time points (i.e., 0, 6, 12 and 18 months), intended follow-up periods were used in the analysis due to greater numerical simplicity in the analysis and to decrease the likelihood of experiencing difficulties with model estimation in the conditional multivariate models.

Latent Growth Curve Models (LGCMs)

Question 3: Do BMI, DE and stress levels change over time when examined separately and are these changes dependent on the baseline level?

The unconditional univariate model for BMI showed that the unstandardized estimated mean of the intercept was 24.74 (p <.001) with a slope of .01 (p = .01) indicating a slight but significant increase in BMI between each time point (6-month period). The intercept indicates a mean BMI score of 24.74 (close to the observed mean) at baseline and the slope indicates that BMI increased by .01 units per month indicating an increase in BMI of .18 over the 18 months follow-up. The covariance between the intercept and slope was statistically significant (b = -.08, p = .03) indicating that the rate of change in BMI was dependent on the initial level of BMI, with those reporting a higher BMI at baseline more likely to experience a reduction in BMI over time. The variance of both the intercept (b = 37.79, p <.001) and slope (b = .01, p <.001) were

statistically significant indicating that both baseline level and changes in BMI over time significantly varied across individuals in this sample. The model fitted the data well as seen by the model fit indices, χ^2 (6) = 5.34, *p* = .50, CFI = 1.00, TLI = 1.00, RMSEA <.001 [RMSEA 90% CI <.001, .04].

The unconditional univariate model for EDE-Q showed that the unstandardized estimated mean of the intercept was 2.15 (*p* <.001) with a slope of -.01 (*p* = .002) indicating a slight but significant decrease in EDE-Q scores between each time point which suggests that there was an improvement in DE behaviours over time. The intercept indicates a mean EDE-Q score of 2.15 (close to the observed mean) at baseline and the slope indicates that EDE-Q scores decreased by .01 units per month indicating a reduction in EDE-Q of .18 over the 18 months follow-up. The covariance between the intercept and slope was statistically significant (*b* = -.02, *p* <.001) indicating that the rate of change in EDE-Q scores was dependent on the initial level of EDE-Q and, similar to BMI, those who reported higher EDE-Q scores at baseline were more likely to experience a reduction in their EDE-Q scores over time. The variance of both the intercept (*b* = 2.05, *p* <.001) and the slope (*b* = .001, *p* <.001) were statistically significant demonstrating that both baseline and changes in EDE-Q over time significantly vary across individuals in this sample. The model fitted the data well as seen by the model fit indices, χ^2 (5) = 10.34, *p* = .07, CFI = .995, TLI = .994, RMSEA = .03 [RMSEA 90% CI <.001, .06].

The unconditional univariate model for PSS-4 showed that the unstandardized estimated mean of the intercept was 11.31 (*p* <.001) with a slope of -.04 (*p* = .002) indicating a slight but significant decrease in PSS-4 scores between each time point. The intercept indicates a mean PSS-4 score of 11.31 (close to the observed mean) at baseline and the slope indicates that PSS-4 scores decreased by .04 units per month indicating a reduction in PSS-4 scores of .72 over the 18 months follow-up. The covariance between the intercept and slope was not statistically significant (*b* = -.06, *p* = .61) indicating that the rate of change in PSS-4 scores is not dependent on the initial level of PSS-4. The variance of the intercept (*b* = 6.55, *p* <.001) was statistically significant indicating that

baseline PSS-4 scores vary across individuals in this sample but the slope (*b* = .01, *p* = .25) was nonsignificant demonstrating that the change over time did not vary across individuals in this sample. The model fitted the data adequately as seen by the model fit indices, χ^2 (5) = 20.75, *p*<.001, CFI = .919, TLI = .903, RMSEA = .06 [RMSEA 90% CI .03, .08].

Question 4: What are the predictors of BMI, DE and stress when assessed separately?

Conditional univariate models (i.e., including covariates) were conducted to examine the effect of psychological and caloric intake variables at baseline on the intercept and slope of BMI, EDE-Q and PSS-4, separately. The conditional models were examined in light of the results of the unconditional models which showed that changes in BMI, EDE-Q and PSS-4 were significant in this sample with BMI increasing but EDE-Q and PSS-4 scores decreasing over time. The model goodness-of-fit indices are presented in *Table 5.4* and similar to the unconditional LGCMs, the conditional models for BMI, EDE-Q and PSS-4 had good goodness-of-fit properties.

BMI

The conditional univariate models for BMI all revealed a similar pattern. The results of all four models indicated that age had a significant positive effect whereas SCRS had a significant negative effect on the initial level of BMI. This suggests that those who are older are more likely to have a higher BMI but those who report higher scores on the SCRS are more likely to have a lower BMI. Where EDE-Q total scores were used as predictors in the model (models one and three), EDE-Q scores had a significant positive effect on initial level of BMI. This suggests that those who reported higher EDE-Q scores were more likely to have a higher BMI. Where EDE-Q sub-factors were used as predictors in the model (models two and four), dietary restraint was found to have a significant negative effect, whereas weight concerns and shape concerns had a significant positive effect on the initial level of BMI. This suggests that those who report higher levels of dietary restraint are more likely to have a lower BMI but those who

report higher levels of weight and shape concerns are more likely to have higher BMI at baseline. Additionally, when the EDE-Q sub-factors were entered into the model, the RS component of the FSCRS had a significant positive effect on the BMI intercept indicating that those who report higher levels of RS report higher BMI as baseline. When the two higher-order factors of the VASQ were used instead of the total VASQ score, the results showed that proximity-seeking had a significant negative effect on BMI in model three indicating that higher scores on the proximity-seeking scale predicts lower BMI at the initial level. Finally, caloric intake was the only significant predictor of the changes in BMI. Thus, as the unconditional univariate model of BMI showed that the trajectory of BMI increased over time, this indicates that higher caloric intake at baseline predicted smaller increases in BMI over time or even decreases in weight over time.

EDE-Q

The univariate conditional models for EDE-Q also presented a consistent pattern. BMI, IS and HS all had a significant positive effect, whereas SDHS and caloric intake had a significant negative effect on the initial level of EDE-Q in all four models. This indicates that those who reported higher BMI and higher scores on the IS and HS scales at baseline were more likely to have a higher initial EDE-Q score. However, those who reported higher SDHS and caloric intake levels were more likely to report lower EDE-Q scores at baseline. In addition to these results, the first model showed that VASQ has a significant positive effect on the intercept of the EDE-Q but a significant negative effect on the slope of the EDE-Q. This indicates that higher scores on the VASQ predict higher scores on the EDE-Q at baseline but those who report higher VASQ scores at baseline are more likely to experience a decline in EDE-Q scores over time. When the two higher-order factors of the VASQ are used in model two instead of the total VASQ score, the results showed that the insecurity attachment component of the VASQ had a significant negative effect on EDE-Q scores over time. This suggests that higher insecurity of attachment corresponded to bigger decreases in EDE-Q scores over time.

PSS-4

Akin to the conditional univariate models for BMI and EDE-Q, the conditional univariate models for PSS-4 also showed a similar pattern. Common significant predictors amongst all four models were age, SDHS, IS and RS, with age, SDHS and RS significant negative predictors, whereas IS was a significant positive predictor of PSS-4 initial level. This indicates that those who are older, have higher SDHS and RS scores are more likely to report lower PSS-4 scores at baseline. However those who reported higher IS scores reported higher initial PSS-4 scores. The VASQ was found to be a significant positive predictor of PSS-4 intercept in models one and two and when the VASQ score was swapped for the two higher-order factors in model three, proximity-seeking was found to be a significant positive predictor of PSS-4 intercept. These results show that higher VASQ scores predict higher PSS-4 scores but within this scale it is specifically higher scores on the proximity-seeking (and not the insecure) scale that is predictive of higher PSS-4 scores at the initial level. Changes in PSS-4 were consistently predicted by SDHS, SCRS and HS with SDHS and HS having a positive effect and SCRS a significant negative effect on the changes in PSS-4 in all four models. This indicates that those who reported higher SDHS and HS scores were less likely to experience decreases in PSS-4 scores over time but those who reported higher SCRS scores at baseline were more likely to experience a decline in their PSS-4 scores over time.

Table 5.4: Model results for the conditional univariate LGCMs for BMI, EDE-Q and PSS-4

Model	Free parameter	-2LL	AIC	BIC	χ^2	DF	*p*-value	CFI	TLI	RMSEA	90% CI RMSEA
BMI											
1. Predictor model	28	-4269.25	8594.50	8731.12	25.36	26	.50	1.00	<.001	1.00	<.001, .03
2. Model 1 but swapping EDE-Q total score for the four sub-factors	34	-4211.41	8490.82	8656.72	30.82	32	.53	1.00	<.001	1.00	<.001, .02
3. Model 1 but swapping VASQ total score for the two higher-order factors	30	-4268.02	8596.03	8742.41	27.96	28	.47	1.00	<.001	1.00	<.001, .03
4. Model 3 but swapping EDE-Q total score for the four sub-factors	36	-4210.40	8492.81	8668.47	33.32	34	.50	1.00	<.001	1.00	<.001, .02
EDE-Q											
1. Predictor model	29	-2198.05	4454.09	4595.59	32.91	25	.13	.995	.02	.990	<.001, .03
2. Model 1 but swapping VASQ total score for the two higher-order factors	31	-2197.49	4456.97	4608.23	34.39	27	.15	.995	.02	.991	<.001, .03
PSS-4											
1. Predictor model	29	-3968.73	7995.47	8136.97	36.90	25	.06	.988	.02	.979	<.001, .04
2. Model 1 but swapping EDE-Q total score for the four sub-factors	35	-3966.06	8002.12	8172.90	37.22	31	.20	.994	.01	.989	<.001, .03
3. Model 1 but swapping VASQ total score for the two higher-order factors	31	-3956.22	7974.45	8125.71	38.89	27	.06	.989	.02	.979	<.001, .04
4. Model 3 but swapping EDE-Q total score for the four sub-factors	37	-3953.50	7981.00	8161.53	39.22	33	.21	.994	.01	.989	<.001, .03

Question 5: Do baseline levels and changes in BMI, DE and stress influence each other and what are the predictors of these levels when examined concurrently?

A multivariate (i.e., parallel process) model examined the effect of psychological and caloric intake variables on the intercept and slope of BMI, EDE-Q and PSS-4 and how the initial levels and changes in these three outcome variables influence each other. However, when running the parallel process model, BMI at T4 had little to no variance suggesting that there was no heterogeneity in this variable. Therefore, in order for the model to be interpreted the variance of this variable was fixed to zero.

Firstly, an unconditional (i.e., without covariates) multivariate model examined the relationship between the initial levels and the changes in the three outcome variables. The data were found to fit the model well as shown by the model fit indices, (χ^2 (52) = 89.13, p = .001, CFI = .991, TLI = .988, RMSEA = .03 [RMSEA 90% CI .02, .03]). The results revealed a significant positive correlation between the initial levels of BMI and EDE-Q (r = .39, p <.001) indicating that higher initial BMI is associated with higher EDE-Q scores at baseline. Similarly, a significant positive correlation between the changes in EDE-Q and BMI (r = .24, p = .01) was found, suggesting that smaller decreases in EDE-Q scores was associated with an increase in BMI (as the unconditional univariate models demonstrated that this sample experienced an increase in BMI but a decrease in EDE-Q scores over time). Initial EDE-Q level was a significant negative predictor of the changes in EDE-Q scores (β = -.55, p <.001) indicating that higher levels of EDE-Q scores at baseline predicted a bigger decrease in EDE-Q scores over time. Initial level of PSS-4 was positively associated with the initial level of BMI (r = .15, p <.001) and the initial level of EDE-Q scores (r = .53, p <.001) indicating that higher PSS-4 scores at baseline correspond to higher BMI and EDE-Q scores at baseline.

Conditional multivariate models were estimated to examine the relationship between BMI, EDE-Q and PSS-4 and the predictors of the initial levels and changes in these outcome variables. Model one consisted of the total scores for all of the baseline variables of interest predicting the initial levels and changes in BMI, EDE-Q and PSS-4. Model two was similar to model 1 but the total VASQ score was changed to represent the two sub-factors, insecurity and proximity-seeking styles of

attachment. Identical to the unconditional multivariate model, the conditional multivariate models showed that baseline EDE-Q level was a significant negative predictor of the changes in EDE-Q scores (β's = -.50, p <.001) indicating that higher levels of EDE-Q scores at baseline predicted a bigger decline in EDE-Q scores over time (*see Figure 5.2*).

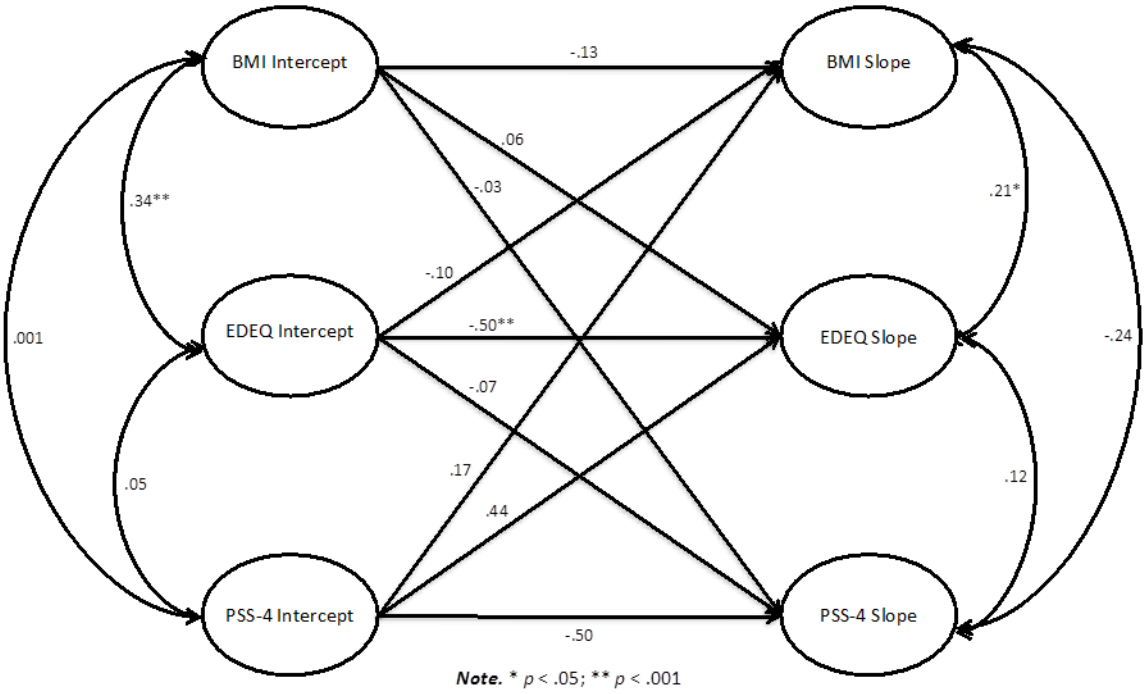


Figure 5.2: Path diagram of the multivariate model depicting the factor loadings and the correlations for the outcome latent variables

A significant positive correlation between the both the initial levels of EDE-Q and BMI (r = .34, p <.001) and changes in EDE-Q and BMI (r = .21, p = .03) was found. This suggests that higher BMI at baseline is associated with higher initial EDE-Q scores (or vice versa) and a smaller decrease in EDE-Q scores was associated with an increase in BMI. In addition to this, the model revealed a significant negative correlation between the changes in PSS-4 and BMI (r = -.24, p = .05) indicating that an increase in PSS-4 scores corresponded to a decrease in BMI over time. However, the correlation between the changes in EDE-Q and PSS-4 (r = .12, p = .39) was found to be non-significant indicating that the changes in EDE-Q and PSS-4 are independent. The parameter estimates for the covariates in the conditional

multivariate models are presented in *Table 5.5*. Both model one (χ^2 (100) = 133.03, *p* = .02, CFI = .993, TLI = .989, RMSEA = .02 [RMSEA 90% CI .01, .03]) and model two (χ^2 (106) = 140.10, *p* = .01, CFI= .993, TLI = .989, RMSEA = .02 [RMSEA 90% CI .01, .03]) were found to have excellent goodness-of-fit properties.

Table 5.5: Parameter estimates and standard errors (standardised values) for the conditional multivariate models

	BMI				**EDE-Q**				**PSS-4**			
	Intercept[a]		**Slope[b]**		**Intercept[a]**		**Slope[b]**		**Intercept[a]**		**Slope[b]**	
Variable	**Est.**	**SE**	**Est.**	**SE**	**Est.**	**SE**	**Est.**	**SE**	**Est.**	**SE**	**Est.**	**SE**
Model 1												
Age	.28**	.03	.04	.07	.07*	.03	.01	.08	-.08*	.03	.01	.10
VASQ	-.05	.04	-.11	.08	.05	.03	-.24*	.10	.16**	.03	.12	.13
SDHS	-.06	.05	.02	.16	-.18**	.04	.27	.21	-.58**	.05	.27	.39
SCRS	-.15*	.04	.05	.10	-.03	.04	-.16	.11	-.001	.04	-.34*	.14
Inadequate-Self	-.01	.05	.08	.10	.16**	.04	-.01	.12	.16**	.04	-.08	.16
Reassured-Self	.07	.05	.06	.11	-.02	.04	-.03	.12	-.11*	.04	.03	.16
Hated-Self	.18**	.05	-.02	.10	.28**	.04	-.04	.12	-.004	.04	.30*	.15
Caloric Intake	-.04	.03	-.14[†]	.07	-.12**	.03	-.06	.08	-.003	.03	-.06	.09
Model 2												
Age	.28**	.03	.04	.07	.07*	.03	.01	.08	-.07*	.03	.01	.10
Insecure	-.004	.04	-.12	.07	.05	.03	-.20*	.09	.02	.03	.16	.10
Proximity-seeking	-.06	.03	-.002	.08	.01	.03	-.09	.10	.19**	.03	-.03	.15
SDHS	-.05	.05	-.02	.18	-.17**	.04	.25	.24	-.63**	.05	.35	.47
SCRS	-.15*	.04	.05	.10	-.03	.04	-.16	.11	.01	.04	-.35*	.14
Inadequate-Self	-.01	.05	.08	.10	.17**	.04	-.01	.12	.15**	.04	-.09	.16
Reassured-Self	.07	.05	.05	.11	.02	.04	-.04	.12	-.11*	.04	.04	.16
Hated-Self	.18**	.05	-.01	.10	.28**	.04	-.04	.12	.01	.04	.29*	.14
Caloric Intake	-.04	.03	-.14[†]	.07	-.12**	.03	-.06	.08	-.01	.03	-.05	.09

Note. [†] *p* = .05; * *p* < .05; ** *p* < .001; [a] Initial level of outcome variable; [b] Rate of change of outcome variable over time; Standardised estimates reported; SE = Standard Error

As it can be seen in table, age, SCRS and the HS component of the FSCRS are all consistently significant predictors of the initial level of BMI indicating that those

who are older and report higher HS scores are more likely to report a higher BMI at baseline. However, those who report higher SCRS scores are more likely to report lower initial BMI. Once again, caloric intake was found to be a significant negative predictor of the changes in BMI indicating that higher caloric intake is predictive of decreases in BMI over time.

Akin to the conditional univariate models of EDE-Q, SDHS, HS and caloric intake at baseline were significant predictors of initial EDE-Q level. SDHS and caloric intake were found to have a negative effect and HS is shown to have a positive effect on initial EDE-Q scores suggesting that those who report higher SDHS scores and caloric intake are more likely to have lower EDE-Q scores whereas those who report higher HS scores are more likely to report higher EDE-Q scores. The multivariate model also revealed that age and IS component of the FSCRS are positive predictors of initial EDE-Q level in both models. These findings suggest that those who are older and report higher IS scores are more likely to report higher EDE-Q scores at baseline. Both models one and two displayed a similar pattern to that of the conditional univariate model of EDE-Q as it demonstrated that VASQ, specifically the insecure scale of the VASQ, has a significant negative effect on the changes in EDE-Q. This suggests that higher scores on the VASQ and more specifically, the insecure scale of the VASQ predict a bigger decline in EDE-Q scores over time.

Finally, the conditional multivariate models demonstrated that the PSS-4 intercept was significantly predicted by age, SDHS, IS and RS with age, SDHS and RS negative predictors, whereas IS was a positive predictor of initial PSS-4. This indicates that those who are older, report higher SDHS and RS scores are more likely to report lower PSS-4 scores but those who report higher IS scores are more likely to report higher PSS-4 scores at baseline. Similar to the unconditional univariate models, the VASQ (in model one) and the proximity-seeking component of the VASQ (in model two) were significant positive predictors of initial PSS-4. These results suggest that higher scores on the VASQ and proximity-seeking predict higher PSS-4 scores at baseline. Change in PSS-4 scores was significantly predicted by SCRS and HS scores with people reporting higher SCRS at baseline being more likely to report greater decreases in their PSS-4 scores over time. However, those who reported

higher HS scores initially are less likely to report a decline in their PSS-4 scores over time.

Discussion

The present study sought to examine the trajectories of weight, DE and stress over 18 months and how these changes influence each other. Additionally, the impact of age, mood and affect regulatory processes such as attachment, self-criticism/reassurance and social rank on baseline BMI, DE and stress levels and how these processes influenced the changes in BMI, DE and stress were also assessed. In order to achieve this, several models were estimated. Firstly, unconditional and conditional univariate models for BMI, DE and stress were conducted to examine the initial and change levels in BMI, DE and stress separately and then the processes that influence these individual levels. The univariate models were followed by unconditional and conditional parallel process models which explored initial and change levels of BMI, DE and stress and how these factors influenced each other and the processes that drive these trajectories. The conditional univariate and parallel process models revealed that the processes that influence BMI, DE and stress when these factors are assessed separately are similar to the processes that effect BMI, DE and stress when they are examined together within a multivariate parallel process model. Additionally, as the present study sought to identify how BMI, DE and stress changed over time, how these changes influenced each other and the processes behind these changes, the findings section that follows will provide a brief overview of the individual changes that were observed for BMI, DE and stress as presented by the unconditional univariate models but the remainder of the section will focus on the results that were observed from the multivariate models.

Findings

The unconditional models for BMI, DE and stress all revealed that this female sample experienced a significant change in BMI, DE and stress over the 18-month study period, with a slight increase in BMI but a slight decrease in DE and stress

levels. Consistent with previous research, BMI increased over time in the present sample, which supports previous findings that have revealed the prevalence of age-related weight gain (Bessesen, 2011). These findings are also in line with previous studies which have revealed that it is young women aged between 25 and 44 who are more likely to gain weight over time as the current sample had a mean age of 28 (Ball et al., 2003; Heitmann & Garby, 1999; Jackson et al., 2001; Van Lenthe et al., 2000; Williamson, 1993; Williamson et al., 1990). Previous research has also shown that, as women age, their disturbed eating behaviours decrease (Heatherton et al., 1995, 1997; Keel, Baxter, Heatherton & Joiner, 2007). In the current study, those with a higher BMI at baseline were more likely to experience a decrease in their BMI during the study, while those who reported higher levels of DE and stress at baseline were likely to experience a greater reduction in their DE and stress levels respectively over time.

The unconditional multivariate models indicated that the initial level and changes in BMI, DE and stress influence each other. Higher BMI at baseline was associated with higher levels of eating pathology at baseline which is consistent with previous research (Field et al., 2007). However, the relationship between changes in DE and BMI indicated that smaller decreases in eating pathology are associated with increases in BMI. More disturbed eating behaviours at baseline were associated with larger decreases in DE over time indicating that those who displayed greater eating pathology at the beginning of the study were more likely to reduce their DE behaviours during the study period. As expected, high stress levels at baseline were related to high BMI and DE at baseline (as described in Chapter Two). However, an increase in stress levels over time was associated with a reduction in BMI. Although one part of Chapter Two advocates the idea that stress induces overeating and weight gain, Stone and Brownell (1994) suggest that stress-induced overeating varies between and within individuals as some overeat to cope with stress-related emotions whilst others experience hypophagia which refers to eating less during stressful life experiences. However, it must be noted that these findings were correlational in nature.

The conditional multivariate models revealed that older adults are more likely to report a higher BMI and higher levels of DE but lower stress levels at

baseline. Once again these findings are in line with previous research which suggests that BMI increases with age (Bessesen, 2011). These findings are consistent with previous findings that have suggested that older adults report less stressful life events compared to their younger counterparts (Brown, Dobson, Bryson & Byles, 1999; Paykel, 1983a) but in contrast to the unconditional model of DE, these findings do not support previous research which has shown that women experience a decline in their disturbed eating behaviours as they age (Heatherton et al., 1995, 1997; Keel et al., 2007).

High caloric intake at baseline was predictive of low levels of DE at baseline and marginally predictive of decreases in BMI over time. These findings suggest that those who reported a higher caloric intake at baseline were more likely to experience a decrease in their BMI over time. As weight is on a continuum, with some people losing weight and others gaining weight over time, it is reasonable to expect that those at the extreme ends of the continuum would get closer to the average over time (referred to as regression to the mean: Hayes, 1988) although their caloric intake may remain relatively high. This also suggests that there may have been other factors (i.e., physical activity) that the model is not accounting for.

Positive affect at baseline was predictive of less eating pathology and lower stress levels which is in line with previous research that has shown that low mood is associated with DE behaviours (Crowther & Chernyk, 1986, Troop et al., 2001) and higher perceptions of stress (Hammen, 2005). Perceptions of higher social status at baseline were predictive of lower BMI at baseline and greater decreases in stress levels. Conversely, high baseline levels of self-criticism (specifically hated-self) predicted high BMI, higher eating pathology and less reduction in stress levels over time. Similarly, high levels of inadequate-self were predictive of high DE and high stress levels at baseline. These results expand previous cross-sectional findings which revealed that feelings of the self as inadequate predicted greater eating pathology in women (Kupeli et al., 2013b). High levels of self-reassurance were predictive of lower stress levels at baseline indicating that those who are able to act kindly towards themselves at times of failure perceive adverse experiences as less stressful compared to those who are not as reassuring towards themselves.

Vulnerable attachment styles were also predictive of DE and stress but the types of attachment style that were predictive of these differed. Insecure attachment in adulthood was predictive of decreases in DE over time which is in contrast to previous research which has advocated that insecurity of attachment is related to DE and even the development and maintenance of EDs (O'Kearney, 1996; O'Shaughnessy & Dallos, 2009; Ward et al., 2000; Zachrisson & Skårderud, 2010). However, those who reported higher levels of proximity-seeking reported high stress levels at baseline. These findings are in line with the notion that individuals who do not have secure attachment styles may not have the support networks and self-management skills to help them deal with the tension and anxiety that they may experience at times of stress (Sloman et al., 2003). These findings suggest that regulatory processes such as social rank and self-criticism are important for women's ability to regulate stress and stress related to changes in weight.

Strengths and limitations

The present study has a number of strengths. One notable aspect of the present study is the longitudinal nature of the research which enabled the assessment of the changes that occurred over the 18-month period. The present study is unique in what it set out to investigate as no previous study has attempted to examine the related trajectories of BMI, DE and stress and the affect regulatory processes that influence these factors. Further, the recruitment of a large, predominantly community-based sample provided good statistical power. Although the current study did experience a high attrition rate between baseline and the final follow-up, statistical methods designed to overcome problems associated with missing data were adopted to reduce potential bias.

The current study also has some limitations that must be addressed. Firstly, this study included the use of self-report measures which are susceptible to potential bias. The use of other methods to assess these factors such as employing the EDE interview (Fairburn & Cooper, 1987) to assess eating pathology could validate the findings of the current study. However, studies have found modest-to-good agreement and significant correlations between the investigator-based EDE interview (Fairburn & Cooper, 1987) and its self-report version, the EDE-Q (Fairburn

& Beglin, 1994) used here (Pretorius, Waller, Gowers & Schmidt, 2009; Wilfley, Schwartz, Spurrell & Fairburn, 1997). Therefore, it can be argued that the use of self-report measures did not influence the overall findings of the current study.

Secondly, the data were collected online and it is arguable that the use of traditional paper-and-pencil methods of data collection may have led to a different set of results. However, previous research has demonstrated that online responses are generally as valid and reliable as those collected offline (Hiskey, 2002).

Further, the present study may have missed the smaller fluctuations in BMI, DE and stress that may have occurred between measurements which were approximately six months apart. However, research has suggested that weight change is relatively stable but that, when changes in weight do occur they tend to increase rather than decrease (Hopman, Leroux, Berger, Joseph, Barr, Prior et al., & the CaMos Research Group, 2007; Katzmarzyk, Pérusse, Malina & Bouchard, 1999), thus emphasising the importance of assessing changes in weight over longer periods of time in order to better understand the obesity epidemic.

Another limitation of the current study is the lack of a male sample as men and women display different trajectories (Heatherton et al., 1995, 1997; Keel et al., 2007; Klesges, Klesges, Haddock & Eck, 1992; Williamson, 1993), hence it is not possible to generalize these findings to the male population. Therefore, future studies should recruit a large sample of men in order to examine if the same processes that influence BMI, eating pathology and stress in females also reveal a similar pattern for men. Also, the sample was predominantly white and young (mean age = 28). Thus, future research should aim to examine the processes driving BMI, eating pathology and stress in older adults who may display a different pattern of findings, and also need to be better placed to evaluate the role of ethnicity.

The study did not recruit a clinical sample and the average age of 28 years old for this sample is not representative of those who go on to develop EDs which is typically younger (Currin, Schmidt, Treasure & Jick, 2005; Polivy & Herman, 2002). Therefore, caution must be taken when using the findings such as the processes behind eating pathology to apply to a clinical population. Finally, another limitation of the present study is that it did not take other factors that may have influenced

weight change, DE and stress such as physical activity levels (Jakicic, 2011) and smoking status (Freedman, Ron, Ballard-Barbash, Doody & Linet, 2006).

Implications

Notwithstanding the above limitations, the findings of the present study have a number of important implications for theory, research and practice. From a theoretical point of view, the findings of the present study provide support for the link between stress and changes in weight although causality remains contentious. The current findings suggest that *increases* in stress may actually lead to a *reduction* in BMI as opposed to weight gain as previously suggested (e.g., Wardle et al., 2010). In addition, the most interesting findings come from the longitudinal analyses as unfavourable social comparison and hated-self predicted less decreases in stress levels over time. This longitudinal work has not been done before so it is important to have established the role of this aspect of affect regulation in the stress process.

The present findings also indicate that insecurity of attachment in adulthood predicted *decreases* in eating pathology over time, however a different pattern emerged when the different attachment patterns were examined. Attachment insecurity was no longer predictive of disturbed eating behaviours but the proximity-seeking aspect of attachment was found to predict higher stress levels at baseline.

Although this study did not employ a clinical sample, the findings can still have important implications for practice. For example, weight loss programmes, ED prevention programmes and stress management interventions should address the issues of perceived low social status, self-criticism and attachment. These may be either in terms of helping individuals to identify battles they can win or finding alternative contexts in which to achieve status (Sloman, 2008) or including therapeutic sessions with families to improve familial relations and reduce stress levels. Alternatively, interventions that render the effect of threats to social rank as less important may also be useful. For example, the ability to be self-soothing can tone down threat and self-criticism (Sloman, 2008). There is emerging evidence for the effectiveness of a number of approaches to increase self-compassion, of which the ability to self-soothe is a key component, including in EDs (Gale et al., 2012). These approaches include CFT (Gilbert, 2005, 2009), mindfulness based stress

reduction (Shapiro, Brown, & Biegel, 2007), experimental approaches (Kelly et al., 2010) and expressive writing (Imrie & Troop, 2012).

Finally, future research should aim to expand on this research and examine these processes in males and in older adults. This would enable us to identify if the same processes that influence changes in BMI, eating pathology and stress in females have the same effect on males and older ageing adults.

Conclusion

In conclusion, the current study has presented some valuable implications for theory, research and practice as the findings demonstrated that all three aspects of affect regulation play different roles in influencing weight, eating pathology and perceptions of stress. This study has also developed and expanded the research area by examining these vital psychological factors simultaneously using a medium-term longitudinal design.

However, the present study focused on examining these processes amongst individuals who had varying bodyweights and did not examine how these factors can influence weight change in those who were previously heavier. As described in Chapter Two of the current thesis, it is important to determine the factors that influence or even induce weight regain. Therefore, as relapse is relatively likely, it is important to examine and understand the processes that may drive the inability to maintain successful weight loss. Although many treatment programs have been developed to help those who are overweight or obese to lose weight, very few interventions have been designed to help successful weight losers to maintain the weight they have lost. Therefore, although it must be our goal to prevent weight gain, it is also important to help overweight and obese individuals to lose weight *and* to keep the weight off (Hill, Thompson & Wyatt, 2005).

Therefore, the next chapter will present a sub-sample of participants from the present study who were identified as weighing at least 3 BMI points less (upon entry into the study presented in this chapter) than their highest adult bodyweight. This sub-sample was recruited to examine more precisely the predictors of weight regain as following successful weight loss as these women are at greatest risk of weight gain.

Chapter 6: Study 2

Predictors of weight regain

Introduction

Maintaining a healthy balance between energy consumption and energy expenditure seems deceptively simple. Therefore, in theory, an imbalance in the equilibrium between energy consumption and expenditure, i.e., consisting of relatively lower caloric intake in relation to levels of physical activity, should lead to successful weight loss. So, why is weight regain following weight loss so prevalent? Although therapies aimed at treating obesity have improved compared to the 1970's (Brownell & Jeffery, 1987), obesity management methods such as decreasing caloric intake, increasing physical activity, psychosocial interventions, pharmacological treatments and surgical techniques have all been shown to be less than effective in 'managing' the obesity epidemic (Abu Dayyeh et al., 2011; Anderson et al., 1991; Christou et al., 2006; Davidson et al., 1999; Foreyt & Carlos Poston, 1998; Freire et al., 2012; Hsu et al., 1998, 2007; Kofman et al., 2010; Kramer et al., 1989; Livhitsm et al., 2011; Lowe et al., 2001; Magro et al., 2008; National Task Force on the Prevention and Treatment of Obesity, 1993; Odom et al., 2010; Sarzynski et al., 2011; Wadden et al., 1992, 1998, 2007). Nevertheless, the benefits of weight loss include the reduced risk of developing obesity-related conditions such as osteoarthritis, diabetes, cardiovascular diseases, depression and sleep apnea (Glenny, O'Meara, Melville, Sheldon & Wilson, 1997; Klem, Wing, Simkin-Silverman & Kuller, 1997; Stern et al., 1995; Sjöström, Lindroos, Peltonen, Torgerson, Bouchard, Carlsson et al., 2004; Will, Williamson, Ford, Calle & Thun, 2002). Therefore, it is essential to try to understand what factors may be involved in successful weight loss maintenance.

Weight loss maintenance versus weight regain

Foster and Kendall (1994) claim that weight regain following obesity treatment is inevitable even when financial rewards are offered (Kramer et al., 1986)

or following post-treatment support and guidance to prevent relapse (Perri et al., 1984). Failure to maintain successful weight loss has been associated with negative psychological consequences (Foreyt et al., 1995), a decline in health-related quality of life (Engel, Crosby, Kolotkin, Hartley, Williams, Wonderlich et al., 2003) and an increased risk of developing obesity-related diseases such as diabetes and hypertension (French, Jeffery, Folsom, McGovern & Williamson, 1996). Sadly, the number of people who are able to successfully shed excess weight and maintain some of the weight they have lost are very small, with statistics ranging from 2% to 19% (Crawford et al., 2000; Glenny et al., 1997; Johnson & Drenick, 1977; Stunkard & McLaren-Hume, 1959; Wadden, Stunkard & Liebschutz, 1988). Wing and Hill (2001) have suggested that successful long-term weight loss maintenance gets easier over time, i.e., is increased when weight loss has been sustained for 2-5 years (McGuire, Wing, Klem, Lang & Hill, 1999; Klem, Wing, Lang, McGuire & Hill, 2000; Weiss et al., 2007). Therefore, once the challenge of losing weight is achieved, how is weight loss successfully maintained and what factors predispose a person to relapse and regain the weight they have lost?

Factors for successful weight loss maintenance

There are many factors that can contribute to successful weight loss maintenance including healthy eating habits, higher levels of physical activity and self-monitoring strategies such as daily weighing and tracking caloric and fat intake which can enable one to detect the early stages of weight regain and employ strategies to avoid a relapse (Anderson, Konz, Frederich & Wood, 2001; Donnelly, Smith, Jacobsen, Kirk, DuBose, Hyder et al., 2004; Katahn, Pleas, Thackrey & Wallston, 1982; Kayman et al., 1990; Kruger, Blanck & Gillespie, 2006; Svetkey, Ard, Stevens, Loria, Young, Hollis et al., 2012; Wing & Hill, 2001). Weight loss maintenance can also be supplemented by joining another weight loss program following a 6-month weight management program (Katahn et al., 1982). However, behavioural factors are not the only processes that drive the ability to maintain prior weight losses. Even sex and age have been found to contribute to this ability as women have been reported to be more successful than men in maintaining weight they have lost during a weight reduction program (Forster & Jeffery, 1986; Jeffery,

Bjornson-Benson, Rosenthal, Kurth & Dunn, 1984a; Kramer et al., 1989). Additionally, children are better at maintaining weight loss compared to adults which emphasizes the importance of intervening earlier in life (Epstein, Valoski, Kalarchian & McCurley, 1995; Epstein, Valoski, Wing & McCurley, 1990, 1994). Interestingly, research has even suggested that life-changing experiences either in the form of a medical trigger or a major life event which spurs the initial motivation to lose weight (Epiphaniou & Ogden, 2010; Gorin, Phelan, Hill & Wing, 2004; Ogden & Hills, 2008; Roberts & Ashley, 1999; Wing & Phelan, 2005) and belief in one's ability to lose weight prior to starting a weight loss program can have a positive influence on weight loss maintenance (Hartigan, Baker-Strauch & Morris, 1982). Continued monitoring and support following weight loss programs are important for enhancing successful weight loss maintenance and preventing relapse (Glenny et al., 1997; Jeffery et al., 2000). The authors suggested that even small deviations from the rules and behaviours developed during weight loss interventions can result in adverse psychological reactions which can lead to weight regain (Jeffery et al., 2000). Following successful weight loss, cognitive processes such as high dietary restraint and avoiding emotional eating or disinhibited behaviours (McGuire et al., 1999; Sarlio-Lähteenkorva & Rissanen, 1998; Westerterp-Plantenga, Kempen & Saris, 1998) and self-generated positive feedback as a result of improved appearance and improved confidence (Roberts & Ashley, 1999) can enhance weight loss maintenance. Therefore, research has identified the factors that can aid and improve weight loss maintenance but what hinders the ability to maintain a bodyweight that is optimally healthy?

Risk factors for weight regain

The aetiology of weight regain is multifaceted with similar biological, behavioural and psychological factors that contribute to the initial weight gain driving the process (Brownell & Jeffery, 1987). Biological factors include change in Resting Metabolic Rate (RMR) during the weight loss (Wing & Hill, 2001) and having an obese parent (Pasman, Saris & Westerterp-Plantenga, 1999) can increase the likelihood of weight regain. Perceived lack of social support (Kayman et al., 1990), sleep disturbance (Marston & Criss, 1984) and a combination of low dietary restraint

and high disinhibition (Westerterp-Plantenga et al., 1998) have all been found to contribute to the failure to maintain weight losses. Other factors that have been found to predict weight regain include dieting (Mann et al., 2007), fluctuations in weight following weight loss attempts, more recent losses, maintenance of weight losses for fewer years and larger weight losses (Colditz et al., 1990; Jeffery, McGuire & French, 2002; McGuire et al., 1999; St Jeor et al., 1995). Additionally, reviews by Byrne (2002), Elfhag and Rössner (2005) and Westover and Lanyon (1990) highlight some of the psychological and behavioural factors that have been found to contribute to successful weight loss maintenance or the failure to maintain a stable, healthy bodyweight following weight loss. The factors that have been implicated in weight regain following successful weight loss include low self-efficacy (defined as the lack of confidence to refrain from emotion-driven eating), binge eating, low physical activity, lack of self-monitoring of weight and dietary intake (especially greater increases in the percentage of calories consumed from fat), high stress, adverse life events, limited social support and poor coping strategies, low mood and not reaching the self-determined goal weight set upon commencing weight loss (Byrne, 2002; Elfhag & Rössner, 2005; Westover & Lanyon, 1990). These reviews have illustrated that both stress and DE behaviours such as binge eating and eating in response to emotions are also important factors that can drive relapse following successful weight loss.

Current study

Despite some inconsistent findings, Chapter Two suggests stress (whether subjectively or objectively assessed) is related to weight regain. More conclusively, the role of stress and life events on eating pathology was confirmed by the review of studies in Chapter Two. All of these studies, however, have explored weight regain in people following a weight loss intervention. Processes involved in weight regain following weight loss may be different in a community sample rather than those recruited from weight loss interventions. The present study therefore addresses this issue. It must also be noted that the studies presented in Chapter Two did not include a physiological measure of stress as a contributing factor related to weight

regain. Therefore, the current study will also assess cortisol levels using a non-invasive method by collecting saliva samples.

Additionally, Chapter Three provided a basis for understanding how evolved affect regulatory systems such as attachment and social rank and processes such as self-criticism/reassurance can contribute to the way stress is regulated and hence may influence weight maintenance and eating behaviours. Chapter Five presented a longitudinal study which provided support for this concept, by demonstrating that these regulatory processes are important for regulating stress which in turn was related to changes in bodyweight. Although Study One revealed that an increase in stress is predictive of a reduction in BMI, which is at odds with a recent meta-analysis (Wardle et al., 2010), that study employed a community-based sample with varying bodyweights. Therefore, as suggested by the narrative review (Chapter Three), it is important to examine the role that affect regulation processes such as insecurity of attachment, low social comparison and self-critical feelings and behaviours play in regulating stress and weight regain. Based on the model presented in Chapter Three (*see Figure 3.2*), the current study will examine the relationship between stress, affect regulatory systems and processes, eating behaviours and weight and the role that affect regulatory systems and processes may have on changes in stress, eating and weight in a community-based sample of women who are at least 3kg/m^2 lighter than their heaviest adult weight.

Therefore, the current study has been designed to answer the following research questions:

1) Do stress and affect regulatory systems and processes contribute to changes in weight and eating pathology in a community-based sample of women who have previously lost weight?
2) Do affect regulatory systems and processes also contribute to changes in perceived and physiological levels of stress in this sample of women?

Methodology

Design

The current study used a repeated measures design, which consisted of two phases, approximately seven months apart. Ethical approval for the current study was granted by the NHS Cambridgeshire 1 Research Ethics Committee (REC).

Participants

At baseline (T1), 42 women from the community who were aged between 21–63 years took part. The sample had a mean age of 32.88 (*SD* = 10.60) and a mean BMI of 25.25kg/m^2 (*SD* = 5.86). Out of the 42 females who took part in the first phase of the study, a total of 38 were followed up at T2 who had a mean age of 33.84 (*SD* = 10.70) and a mean BMI of 25.77kg/m^2 (*SD* = 6.03), respectively. Three women indicated that they had previously suffered from an eating disorder (AN, BN and EDNOS) but they were recovered and had a normal BMI at the time of recruitment and throughout the study phase and so were retained.

Preliminary analysis comparing completers (*n* = 38) and non-completers (*n* = 4) on baseline variables suggested that non-completers did not differ from those who completed both phases of the study. However, fortunately for this study, the number of non-completers was too small for an adequate comparison. Demographic information for phase one of the study is presented in *Table 6.1* which shows that the majority of the sample was British, single, had completed a degree and was either studying or in employment.

Table 6.1: Baseline demographic variables (*N* = 42)

Variable	*N* (%)
Ethnicity	
British	25 (59.5)
Other European	6 (14.3)
Pakistani	2 (4.8)
Caribbean	1 (2.4)
African	2 (4.8)
Other	6 (14.3)
Marital status	
Single	16 (38.1)
Married/Cohabiting	15 (35.8)
In a relationship	4 (9.5)
Divorced/Widowed	7 (16.7)
Highest education	
GCSE	4 (9.5)
A Levels	7 (16.7)
Bachelors	19 (45.2)
Postgraduate	10 (23.8)
None	2 (4.8)
Employment	
Employed	20 (47.6)
Studying	17 (40.5)
Unemployed	5 (11.9)

Recruitment

All participants who took part in the present study were a subset of the larger longitudinal study in Chapter Five. As the current study required females who reported having previously lost weight, those female participants who provided current bodyweight, past highest adult bodyweight and contact information were screened. The operational criterion to determine whether women had experienced

significant weight loss was that current BMI was at least 3kg/m^2 less than highest adult BMI.

A total of 224 women were approached to take part in the current study. To enhance recruitment of participants, it was decided to offer each participant £20 worth of Amazon vouchers upon completion of both phases of the study. Of those who were contacted, 53 (24%) responded to take part and 42 returned the study pack for T1. Out of the 42 participants who took part in T1 of the study, 38 returned the study pack for T2.

Measures and procedure

Those who were eligible for participation were invited by email to take part in a study investigating the role of stress and other psychological factors in weight. Participants completed a demographics sheet that required information such as age, sex, height, current weight, past highest and lowest adult weight, ethnicity, marital status, highest education qualification and occupation. Participants were also asked if they have ever been diagnosed with an ED and to provide details of any medication they were currently taking. In addition to providing saliva samples, participants were also asked to complete the following self-report measures at two time points, seven months apart (*for more information regarding the self-report measures, please see Chapter Four*):

1. Caloric intake was measured using the FFQ (Cade & Margetts, 1988)
2. Disordered eating was assessed using the EDE-Q (Fairburn & Beglin, 1994)
3. Insecurity of attachment was measured using the VASQ (Bifulco et al., 2003)
4. Mood was measured using the SDHS (Joseph et al., 2004)
5. Self-criticism/reassurance was assessed using the FSCRS (Gilbert et al., 2004)
6. Social rank was measured using the SCRS (Allan & Gilbert, 1995)
7. Stress perceptions were assessed using the PSS-4 (Cohen & Williamson, 1988)

Participants also provided six saliva samples at both time points and participants were advised that they must collect saliva samples every two hours (e.g., 8am, 10am, 12pm etc.) over the course of a day. However, one participant

failed to provide the times of when she collected her saliva samples at T1 and four participants did not specify the times that they took the final saliva sample. Therefore, AUC was determined using the first five saliva samples for all the participants who completed both phases of the study.

Participants were recruited from all over the UK as well as internationally (mainly United States of America). Those who agreed to take part were sent a study pack containing the questionnaires and standard salivettes (Sarstedt, Leicester, UK) via Royal Mail for saliva collection (*see Chapter Four for more information regarding the procedure that participants followed to collect saliva samples*).

Statistical analyses

Several statistical analyses were conducted (*for more information regarding the calculation of AUC using cortisol levels, please refer to Chapter Four*). Firstly, descriptive statistics for all measures at baseline and follow-up were computed. Skewness and kurtosis values were examined as an indication of symmetry (or asymmetry) and peakedness (or flatness) of the distribution of the data, respectively. Secondly, correlational analyses using Pearson's correlation coefficients were performed to determine the relationship between the variable of interest.

Following the preliminary analysis of the data, change scores (Δ) for BMI, EDE-Q (and subscales: dietary restraint, weight, shape and eating concerns) SDHS, PSS-4, VASQ (and subscales: insecurity and proximity-seeking), SCRS, inadequate-self, reassured-self, hated-self, caloric intake and cortisol levels, were computed by subtracting T2 scores from T1 scores. The change scores for BMI, EDE-Q, PSS-4 and cortisol levels were used to create two groups of participants (by using median split) who remained stable or decreased their BMI, their EDE-Q, their PSS-4 scores and their cortisol levels in order to compare them to those who increased their BMI, their EDE-Q, their PSS-4 scores and their cortisol levels. For BMI, 18 women had a stable weight or experienced a decrease in their BMI (ΔM = -.59, SD = .85) and 19 women reported an increase (ΔM = 1.63, SD = 1.35) in their BMI over seven months. For EDE-Q scores over the two time points, 19 women remained stable or decreased (ΔM = -.66, SD = .47) and 18 women experienced an increase (ΔM = .60, SD = .46) in their eating pathology. For PSS-4, 21 women reported stable or a decrease (ΔM = -

2.29, *SD* = 1.93) in their stress scores and 17 women experienced an increase in their stress scores (Δ*M* = 2.47, *SD* = 1.62) over seven months. Finally, whilst 24 women's cortisol levels remained stable or decreased (Δ*M* = -12.31, *SD* = 8.27), 12 women experienced an increase (Δ*M* = 19.26, *SD* = 18.13) in their cortisol levels over time. Independent-samples *t*-test was performed to compare these groups on changes in BMI and change scores for measures of mood, stress (on both self-report and cortisol measures), DE behaviours, social comparison, insecurity of attachment, self-criticism/reassurance and caloric intake. Finally, effect sizes as measured by Cohen's *d* were reported with .20, .50 and .80 representing small, medium and large effects, respectively (Cohen, 1992).

Results

Sample characteristics

Means, standard deviations, skewness and kurtosis information for the variables of interest at T1 and follow-up are presented in *Table 6.2*. As this sample was selected on the basis of their current versus highest adult weight, this sample had a mean highest adult BMI of 30.42kg/m^2 (*SD* = 6.18). Paired samples *t*-test results revealed that this sample experienced an increase in their BMI over the seven months (*t*(36) = -2.11, *p* = .04) and although the means for T1 and T2 suggest that this sample reported a decrease in their EDE-Q and PSS-4 scores and cortisol levels, the reduction in these variables were non-significant (*t*(37) = .33, *p* = .75, *t*(37) = .39, *p* = .70 and *t*(35) = .55, *p* = .58, respectively). However, this sample did experience an increase in their RS scores, *t*(37) = -2.36, *p* = .02. Changes between T1 and T2 for the other variables were non-significant (*p*-values ranged between .09 to 1.00).

Table 6.2: Descriptive statistics of the variables at baseline and at follow-up

	T1 (*N* = 42)				T2 (*N* = 38)			
Variable	**Mean**	**SD**	**Skew**	**Kurtosis**	**Mean**	**SD**	**Skew**	**Kurtosis**
Age	32.88	10.60	1.08	.50	33.84	10.70	1.08	.55
BMI	25.25	5.86	2.59	9.57	25.77	6.03	2.52	9.05
Lowest adult BMI	21.15	4.24	3.30	15.98	-	-	-	-
Highest adult BMI	30.42	6.18	1.69	5.01	-	-	-	-
EDE-Q (Total score)	1.94	1.18	.37	-.56	1.80	1.11	.38	-.39
Dietary Restraint	1.88	1.41	.29	-.84	1.48	1.41	.93	.05
Eating Concerns	1.32	1.34	.97	-.31	1.14	1.24	1.27	.79
Weight Concerns	1.98	1.41	.47	-.59	1.98	1.32	.35	-.70
Shape Concerns	2.47	1.41	.09	-1.07	2.40	1.38	.01	-.84
PSS-4	12.24	3.49	-.08	-.56	12.08	3.18	-.03	-.66
SDHS	16.33	4.85	-.23	-1.06	16.71	4.78	-.35	-.79
SCRS	51.55	15.57	-.25	-.19	54.95	17.83	-.30	-1.00
VASQ (Total score)	40.29	8.50	-.24	-.004	40.21	8.23	-.40	.19
Insecurity	21.93	7.22	.28	-.77	22.39	5.63	-.01	-.24
Proximity-seeking	18.36	4.38	-.09	-.49	17.82	4.84	-.02	.38
Inadequate-self	21.17	6.00	-.46	-.54	21.08	6.72	-.69	-.60
Reassured-self	24.21	7.04	-.21	-.27	25.39	7.51	-.14	-.42
Hated-self	8.29	3.97	1.00	.38	8.24	4.54	.81	-.44
Caloric Intake	1384.73	502.15	.55	-.49	1282.29	456.56	.25	-.84
Cortisol	47.15	14.96	.79	1.74	45.71	24.62	1.07	1.53

The majority of the variables were normally distributed at both time points except for the BMI variables which were positively skewed and leptokurtic (refers to a distribution of data that has a high peak around the mean). The reason for this is that the screening criteria used to recruit women into the study required women to have previously been significantly heavier than their current weight. Therefore, the majority of the women recruited into the study were within the normal BMI range bracket (18.5 – 24.95kg/m^2).

Correlations

<u>Question 1:</u> What is the relationship between BMI, DE, stress, mood, affect regulatory systems and processes and eating in a sample of community-based women who are 3 BMI points lighter than their heaviest adult bodyweight?

Intercorrelations between the variables of interest are presented in *Table 6.3*. Cortisol and caloric intake did not correlate with any other variables except each other which was a negative relationship suggesting that higher cortisol levels are related to lower caloric intake. All three factors of the FSCRS consistently correlated with majority of the variables. Both self-criticism factors (IS and HS) correlated positively with EDE-Q and its subscales, PSS-4, VASQ and insecurity of attachment and negatively with SDHS, SCRS and the RS component of FSCRS. This suggests that high self-criticism is associated with more DE behaviours, stress levels and insecurity of attachment but lower levels of mood, SCRS and RS. IS was positively correlated to the proximity-seeking style of attachment indicating that high feelings of inadequate-self are related to high proximity-seeking behaviours. Both overall VASQ and insecurity of attachment positively correlated with the EDE-Q and its subscales (except dietary restraint) and PSS-4 and negatively correlated with SDHS and SCRS. This indicates that higher insecurity of attachment is related to more DE behaviours but not dietary restraint, low mood and feelings of unfavourable social comparison. SDHS and SCRS displayed similar relationships with other variables as both were positively correlated with each other but negatively correlated with PSS-4 and EDE-Q and the subscales. This shows that happier mood and favourable social comparison are associated with lower stress and DE scores. PSS-4 was positively related to eating and shape concerns but not overall EDE-Q and dietary restraint indicating that higher stress levels are related to more eating and shape concerns.

Table 6.3: Intercorrelations between the variables at baseline (T1) (*N* = 42)

Variable	Age	BMI	EDE-Q	Restr	Eating	Weight	Shape	PSS-4	SDHS	SCRS	VASQ	Insec	Prox	IS	RS	HS	Cal Int
BMI	.22																
EDE-Q	.06	-.07															
Dietary Restraint	.13	-.09	.81**														
Eating Concerns	.09	-.10	.85**	.61**													
Weight Concerns	.04	-.02	.92**	.63**	.71**												
Shape Concerns	-.03	-.06	.95**	.62**	.74**	.90**											
PSS-4	-.03	-.13	.26	.09	.32*	.16	.32*										
SDHS	-.25	-.03	-.48*	-.32*	-.55**	-.39*	-.47*	-.71**									
SCRS	-.11	-.01	-.51*	-.35*	-.51**	-.43*	-.51*	-.62**	.65**								
VASQ	.16	-.06	.43*	.28	.44*	.36*	.46*	.55**	-.59**	-.62**							
Insecurity	.08	-.01	.46*	.29	.55**	.37*	.48*	.46*	-.56**	-.61**	.86**						
Proximity-seeking	.18	-.10	.08	.06	-.04	.10	.10	.29	-.21	-.20	.53**	.02					
Inadequate-self	-.01	-.15	.57**	.44*	.53**	.48*	.56**	.64**	-.57**	-.76**	.65**	.56**	.35*				
Reassured-self	.03	.14	-.48*	-.31*	-.51**	-.39*	-.49*	-.78**	.74**	.85**	-.60**	-.57**	-.22	-.81**			
Hated-self	-.002	.01	.62**	.46*	.65**	.53**	.62**	.60**	-.64**	-.69**	.59**	.61**	.15	.76**	-.79**		
Caloric Intake	-.17	-.10	.002	-.18	.04	.01	.07	-.05	.13	.02	.04	.004	.08	.12	-.05	.03	
Cortisol	-.06	-.31	-.16	-.03	-.09	-.14	-.22	-.02	.16	.30	-.27	-.23	-.14	-.16	.18	-.29	-.34*

Note. * $p < .05$; ** $p < .001$

Independent samples t-test

Question 2: What are the predictors of the changes in BMI, DE and perceived and physiological stress?

The results of the independent samples *t*-test revealed that the two BMI groups (stable/decrease versus increase) did not differ on any of the variables (*p*-value ranged between .11 to .91). Similarly, those who reported a stable or experienced a decrease in their cortisol levels did not differ on any variables when compared to those who reported an increase in their cortisol levels (*p*-value ranged between .35 to .98). Therefore, *Table 6.4* presents the results of the independent samples *t*-test (and corresponding Cohen's *d* effect sizes) comparing the EDE-Q and PSS-4 groups only. The results revealed that those who reported stable/decreased EDE-Q scores reported an increase in their RS scores compared to those who experienced an increase in their EDE-Q scores, $t(35) = 2.33$, $p = .03$. The two PSS-4 groups were found to differ on SDHS, insecurity of attachment and SCRS scores and marginally differed on dietary restraint scores. Those who reported stable/decreased PSS-4 scores also reported an increase in SDHS ($t(31.04) = 4.72$, $p < .001$) and SCRS ($t(36) = 2.84$, $p = .01$) scores over time compared to those who reported an increase in their PSS-4 scores. This indicates that as perceptions of stress remained stable or decreased, women in this sample became happier and developed a more favourable social comparison. However, women who reported an increase in stress between baseline and follow-up reported greater increases in insecurity of attachment scores compared to those who experienced stable/decreased PSS-4 scores ($t(36) = -2.20$, $p = .03$).

Cohen's *d* was also computed as a measure of effect size revealing small to very large effects (Cohen, 1992). Very large effects were found for the significant differences between the two EDE-Q groups and the PSS-4 groups as discussed above. However, medium to large effects were also found for BMI and cortisol between those who reported an increase in their EDE-Q scores and those who were stable or experienced a decrease. Similarly, medium to large effects were found for dietary restraint when comparing those who experienced a decrease in their PSS-4 scores and those who reported an increase in their PSS-4 scores.

Table 6.4: Results of the independent-samples *t*-test comparing BMI, EDE-Q and PSS-4 groups on mean (*SD*) change scores

	EDE-Q			PSS-4		
Variable (Δ)	**Stable/Decrease (*n* = 19)**	**Increase (*n* = 18)**	**Effect size (*d*)**	**Stable/Decrease (*n* = 21)**	**Increase (*n* = 16)**	**Effect size (*d*)**
ΔBMI	.11 (1.43)	1.29 (2.36)	.60	.96 (2.32)	.42 (1.47)	.28
ΔEDE-Q	-.66 (.47)	.60 (.46)**	-2.71	.03 (.81)	-.15 (.76)	.23
ΔRestraint	-1.03 (.78)	.57 (.92)**	-1.88	.05 (1.19)	-.66 (1.00)	.65
ΔEating Concerns	-.34 (.58)	.09 (.63)*	-.71	-.14 (.72)	-.11 (.51)	-.05
ΔWeight Concerns	-.59 (.78)	.88 (.65)**	-.2.05	.23 (1.15)	-.05 (.82)	.28
ΔShape Concerns	-.70 (.59)	.82 (1.03)	-.14	-.03 (1.56)	.13 (1.08)	-.12
ΔSDHS	-.11 (3.09)	1.00 (3.61)	-.33	2.10 (3.40)	-1.94 (1.75)**	1.49
ΔPSS-4	.21 (2.62)	-.67 (3.38)	.29	-2.29 (1.93)	2.47 (1.62)**	-2.67
ΔVASQ	-.53 (5.14)	-.72 (5.19)	.04	-1.19 (5.68)	.59 (4.54)	-.35
ΔInsecurity	.21 (3.07)	.33 (3.88)	-.03	-.67 (3.50)	1.71 (3.06)*	-.72
ΔProximity-seeking	-.74 (3.30)	-1.06 (4.33)	.08	-.53 (4.23)	-1.12 (3.26)	.16
ΔSCRS	5.05 (10.82)	1.78 (11.20)	.30	7.33 (10.31)	-2.00 (9.76)*	.93
ΔInadequate-self	-.53 (3.58)	.33 (2.93)	-.26	-.19 (3.70)	.24 (2.80)	-.13
ΔHated-self	.32 (2.60)	-.11 (2.85)	.16	-.29 (3.15)	.59 (1.87)	-.34
ΔReassured-self	2.05 (2.68)	-.06 (2.84)*	.76	1.48 (3.01)	.71 (2.91)	.26
ΔCaloric Intake	-100.78 (317.75)	-114.22 (297.47)	.04	-95.65 (278.68)	-110.59 (335.69)	.05
ΔCortisol	4.18 (21.62)	-7.69 (15.67)	.63	-.26 (22.43)	-3.92 (14.59)	.19

Note. Asterisks indicate that mean differences are significant at: * $p < .05$; ** $p < .001$;

Discussion

The present study examined the role of stress and affect regulatory processes on weight change and eating pathology in women who have previously lost weight and at baseline were at least 3 BMI points lighter than their highest adult weight. In addition, the impact of insecurity of attachment, perceived low social comparison and self-criticism/reassurance on perceptions of stress in this sample of women was examined.

Findings

As this subsample of women was selected based on the difference between their current weight (at baseline) and previous highest adult weight, it was expected that these women would report an increase in their weight over the study period. Consistent with previous research suggesting that weight gain is a common occurrence amongst those who have lost weight (Foster & Kendall, 1994), this sample of women experienced a small but significant increase in BMI over seven months. This is consistent with the findings of the longitudinal study (*see Chapter Five*). However, stress and affect regulatory processes such as attachment and perceived low social rank did not differ between those who experienced an increase in weight compared to those who remained stable or reported a decrease in BMI over seven months.

The secondary aim of this study was to explore if changes in BMI and affect regulatory processes such as attachment, social comparison and self-criticism/reassurance also contribute to perceived levels of stress and eating pathology in this sample of women. Those who reported stable or reduced DE behaviours reported an increase in their self-reassurance. Although the route of causality between BMI, DE and self-reassurance cannot be inferred from this two-part study, one might suggest that those who were able to focus on their own positive features (and not their bodyweight) and be more supportive of themselves at times of difficulty experienced a reduction in their disturbed eating behaviours

(which includes fasting and restriction) and hence gained weight during the study period.

In addition, those who reported an increase in their stress levels also reported an increase in their insecure attachment style but a decrease in mood and perceived social rank. There was a marginal (though non-significant) reduction in dietary restraint in those reporting an increase in stress. Though not consistent with some studies (e.g., Wardle et al., 2010), this may be due to the sample size. The relationship between stress and affect regulatory processes such as attachment and social rank are in line with the findings of the longitudinal study (*see Chapter Five*) presented in this thesis which found that more favourable social comparison at baseline was predictive of greater decreases in stress levels and higher levels of attachment insecurity was related to higher levels of stress. Importantly, here the finding relates to *changes* in affect regulatory processes, not merely baseline predictors. Therefore, overall these findings are consistent with the notion that affect regulatory processes such as attachment and social rank are important for regulating stress.

Strengths and limitations

The current study has some notable features, for example, employing a community-based sample. Nevertheless, the sample was predominantly white and young (mean age = 33), hence the findings may not be generalisable to older adults. However, the present study collected both self-report perceptions of stress and salivary cortisol as a physiological measure of stress which is a great strength of the current research. This is an improvement on previous studies which has used only self-report measures of stress to examine the effects of stress on weight regain (as described in Chapter Two), though curiously these were uncorrelated here.

The current study also has some important limitations that must be addressed. Firstly, as in Study One, the present study mainly used self-report measures which are susceptible to bias but can provide a reasonable basis to explore the relationship between psychological constructs. Hence, direct measurement of weight and height at both time points in order to calculate BMI instead of collecting self-report information or employing the EDE (Fairburn & Cooper, 1987) interview as

a measure of eating pathology could validate the current findings as discussed earlier.

Another possible limitation is that the current study only assessed weight at two time points, approximately seven months apart. Although the present study is exploratory and examines whether stress and affect regulatory processes can influence weight regain, a longitudinal design with at least 3 time points spanning a longer period of time would be required to provide a more detailed assessment of the role of stress and affect regulation in relation to fluctuations in weight.

A fourth limitation of the current study is the recruitment of a small sample of women. Although from the longitudinal study (*see Chapter Five*) we identified a large number of women who were eligible for this study, recruitment was problematic. Therefore, future studies should identify ways to recruit a larger sample of women. The study did not recruit a clinical sample and the average age of 33 years for this sample is not representative of those who go onto develop EDs which is typically younger (Currin et al., 2005; Polivy & Herman, 2002). Therefore, the findings relating to eating pathology may not be generalisable to a clinical population.

The present study also did not assess how (whether weight was lost naturally or following participation in a weight loss programme), why and when weight was lost as research has shown that weight loss maintenance is better sustained following a major life event like a medical trigger (Epiphaniou & Ogden, 2010; Gorin et al., 2004; Ogden & Hills, 2008; Roberts & Ashley, 1999; Wing & Phelan, 2005) and maintenance of any weight that is lost becomes easier after 2-5 years (McGuire et al., 1999; Klem et al., 2000; Weiss et al., 2007). These factors may have predicted who gained weight or maintained weight loss in the present study.

Implications

The findings of the current study have a number of important implications for theory, research and practice. From a theoretical point of view, the findings of the present study provide further support for previous research which has demonstrated that weight gain is a common occurrence for those who have successfully lost weight. Additionally, similar to the findings of the longitudinal study presented in

Chapter Five, the current study also demonstrates that there is a link between stress and affect regulatory processes. However, no link was found between stress and weight gain. The current research extends the findings of the longitudinal study by showing that changes in affect regulation (rather than baseline predictors as described in Study One), especially perceived low social rank and insecurity of attachment, play an important role in changes in stress amongst those who were formerly heavier than their weight on entry into the study. Therefore, some of the implications that were described in Study One (Chapter Five) also apply to the findings of the current study. Specifically, addressing issues of perceived low social comparison and attachment insecurities in stress management programmes and interventions that are designed to teach individuals to focus on the positive aspects of the self can divert negative feelings away from unhealthy eating behaviours that may contribute to problematic weight regulation. Additionally, as described in the previous study, these findings are consistent with recent research that has shown the effectiveness of increasing self-compassion on ED symptoms during CFT (Goss & Allan, 2010; Gale et al., 2012).

Conclusion

The present study presents evidence that affect regulatory processes such as insecurity of attachment and perceived low social rank are important for stress regulation and eating pathology for those who have lost weight but that these factors do not influence weight regain. Therefore, the next study will examine the role of life events and difficulties on AN by comparing those who have recovered from AN to those who either remit, maintain their symptoms or relapse following treatment for AN.

Chapter 7: Study 3

The role of life events and difficulties in predicting recovery and relapse from AN

Introduction

So far this thesis has examined the role of stress on changes in bodyweight in both those from a normal/general population and those who were at least 3 BMI points lighter than their highest adult weight by using self-report and a physiological measure of stress. On the opposite end of the problematic weight regulation spectrum, AN has been characterized as the refusal or inability to maintain normal body weight for age and height (less than 85%; BMI less than 17.5kg/m^2), with the fear of gaining weight or becoming fat (APA, 2000). Diagnostic criteria also include distorted perceptions of shape and weight and experiencing amenorrhea (lack of menstruation).

Research has been conducted to identify key risk factors of disturbed eating pathology and many psychological factors have been found to contribute to the development and maintenance of eating pathology. Relapse in AN is also a matter of concern as approximately one in three women with anorexia relapse following treatment (Herzog et al., 1999). Therefore, it is crucial to understand the factors that can predict recovery, symptom maintenance and relapse from AN.

As described in Chapter Two of the current thesis, stress in the form of life events and difficulties can be a precipitating factor for AN (Berge et al., 2012; Blasse & Elklit, 2001; Fornari et al., 1994; Karwautz et al., 2001, 2011; McFarlane et al., 1988; Nygaard, 1990; Pike et al., 2006, 2008; Råstam & Gillberg, 1992; Schmidt et al., 1997). Additionally, Chapter Three provided a narrative review of affect regulatory systems and processes which have been implicated in EDs. In summary, both clinical and non-clinical samples who report dysfunctional eating behaviours also report insecure styles of attachment in adulthood, unfavourable social comparison and critical self-evaluations (Bellew et al., 2006; Connan et al., 2007; Feinson & Meir,

2012; Fennig et al., 2008; O'Kearney, 1996; O'Shaughnessy & Dallos, 2009; Pinto-Gouveia et al., 2012; Schmidt & Treasure, 2006; Stormer & Thompson, 1996; Troop et al., 2003, 2013; Troop & Baker, 2008; Thompson et al., 1999; Ward et al., 2000; Williams et al., 1993; Zachrisson & Skårderud, 2010).

Therefore, this chapter will firstly describe the Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978) which will then be followed by a review of the literature which has used the LEDS to examine the relationship between stressful life experiences and psychopathology such as depression and anxiety. This chapter will then be followed by a brief summary of the literature examining the role of life events and difficulties on AN as described in Chapter Two.

Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978)

The LEDS was developed to overcome the problems associated with using traditional checklist instruments to obtain information about life events (Brown, 1972). Gorman (1993) showed that checklist methods of assessing life events are susceptible to over-reporting. Brown (1972) also suggested that checklist methods of assessing life events have low reliability as the same event can have different meanings for different people and measures must focus on specific meaning rather than a general meaning of adversity. Checklist methods of assessing life events examine a class of life events as having one general meaning without taking into account the context in which the event occurs (Brown, 1989). An example of a self-report measure of life events is the Social Readjustment Rating Scale (SRRS; Holmes & Rahe, 1967). When completing the SRRS (Holmes & Rahe, 1967), individuals were provided with a checklist of 43 life events ranging from the death of a spouse to going on holiday and were instructed to indicate which of these events they had experienced over a specified period of time. Each life event was assigned a predetermined rating score. Therefore, Holmes and Rahe (1967) used a subjective approach to determine and quantify the role of a limited number of life events on illness. No attempt was made to estimate the importance of life events to individuals or the context in which specific life events occurred. Later studies did attempt to address the issue of importance of particular life events to individuals by including a self-report rating of how positive or negative each life event was but these measures

still failed to assess the context in which these life events were experienced (Life Experiences Survey [LES]; Sarason et al., 1978). Therefore, in order to determine the meaning of life events for individuals, it is imperative to obtain information about the context that surrounded the event (Averill, 1973).

The LEDS is a semi-structured interview designed to systematically elicit information regarding events and difficulties that occurred over a specified time period. A wide range of areas are covered in the interview such as education, work, reproduction, housing, money/possessions, legal, health, martial/partner relationships, other relations (e.g., child, parent), and other events (including bereavement). This wide range of life domains enables the researcher to determine the types of events that are most likely to be related to the disorder under examination. For example, Chapter Two revealed that the most frequently reported events that precede the onset of ED are interpersonal difficulties such as relationship problems with family and friends (Karwautz et al., 2011; Rojo et al., 2006; Schmidt et al., 1997; Troop & Treasure, 1997). During the interview, individuals are also requested to include events that have occurred to them and to others who are close to them ("close ties"). Close ties include spouse, partner, parent, sibling, child or confidant (a confidant is a close friend who the interviewee can confide in with complete trust). The LEDS is a detailed guide that includes a lengthy list of probing questions to elicit contextual information regarding the circumstances that surrounded each event. The aim of obtaining this level of detail is to ensure that the interviewer can rate each event and difficulty on both reported and contextual threat. Threat refers to the degree to which the event or difficulty produces emotional disturbance or poses problems for the person's security, relationships, future or other important aspects of life. The reported rating is determined by using the information on how the individual reacted to the experience. However, as this method of assessing life events is subjective, Brown and Harris (1978) introduced contextual ratings to supplement reported ratings. The process of applying contextual ratings to each event is to use the idea of an average person's likely response to an event if they were in the same set of circumstances. The researcher must use their judgement and training to determine the meaning of each event and difficulty given the circumstances that surround the experience whilst ignoring what

the interviewee has said about their reaction or knowledge of the interviewee's symptoms.

The contextual information provided by the interviewee is used to rate the severity of the event. Each event is rated in terms of threat or unpleasantness on a 4-point scale (1: 'marked', 2: 'moderate', 3: 'some' and 4: 'little/none'). The short and long-term severity of each event is rated, with short-term referring to the threat/unpleasantness of the event during the first few days and long-term severity referring to the threat/unpleasantness for 10-14 days after the event to capture long-term implications of severe events. A severe event refers to an experience that is rated as 'marked' or 'moderate' on the long-term severity. Problems lasting four weeks or more are classified as difficulties, which are rated on a 7-point scale and marked difficulties are those rated on the top three scale points for at least two months (1: 'high marked', 2: 'low marked' and 3: 'high moderate'). A marked difficulty found to be lasting two or more years is labelled as a major difficulty. A difficulty can also consist of events, for example, the illness, hospitalisation and death of a partner or close tie will be a difficulty but also three separate events. These three events would be identified to capture the severity and impact of hospitalisation, death and funeral of the partner on the individual under study.

In addition to the reported and contextual threat ratings, each life event and difficulty is dated. Assigning a date to events and difficulties is an important aspect of life events and illness research as it can be used to determine if the event is related to onset of an illness (as those that occurred prior to onset) or possibly related to recovery or relapse from an illness. Changes to difficulties are also dated. For example, if an individual is repaying a financial debt to a family member over a period of time but then accrues more debt with credit facilities, the date that the threat/unpleasantness rating increases for their financial difficulty would be recorded. Each event and difficulty is also given a focus rating which involves classifying the event or difficulty as focused on the participant, joint-focused with another person or solely focused on another person. Focus ratings are used to determine if an event or difficulty should be classified as a severe event or major/marked difficulty as Brown and Harris (1978) defined severely threatening events as those that are focused on the individual. However, events that involve

others may also be included as a severe event but only if it has a substantial impact on the individual under study, for example, if one's partner has been made redundant which is likely to produce financial difficulties for the family.

The LEDS is a valuable tool when assessing life events and difficulties as it does not rely on subjective reporting but determines severity of events based on contextual information whilst overcoming any influence of investigator bias. This allows researchers to account for life circumstances and the goals and commitments of the respondent. Validity is further increased by the use of comprehensive manuals with precise criteria specified as well as extensive examples of previously collected material gathered over several thousand interviews. Consensus meetings are also held during which other trained raters, blind to diagnosis, discuss and agree on ratings. Members of the consensus meeting are provided with the contextual information rather than the individual's subjective response to an event in order to reach an objective rating.

LEDS and psychological disorders

The LEDS is a reliable and well-recognised research tool amongst researchers examining the role of life events and difficulties on psychopathology including depression, anxiety and psychosis. Brown and Harris (1986) reviewed studies which used the LEDS to examine the relationship between stressful life events and depression and found that a higher proportion of women (76%) who developed depression reported stressful life events prior to onset compared with women who did not go onto develop depression (30%). Also, compared to women without an onset of depression, more women who were depressed reported interpersonal crises and loss events such as death of someone close or unemployment as occurring in the six months prior to onset (Brown et al., 1987). Early work using the LEDS was focused on examining the role of life events on depression. In a review of studies using the LEDS, Brown, Bifulco and Harris (1987) concluded that 83% of women reported some sort of depression triggering agent prior to onset of depression. Similarly, Bebbington, Wilkins, Jones, Foerster, Murray, Toone et al. (1993) also found that those with an onset of psychosis reported significantly more severe life events during the six months prior to onset.

The usefulness of the LEDS has also been shown by studies which have demonstrated that it can be specific types of events that can trigger psychopathology. Brown et al. (1995) found that it is stressful life events that induce a perceived loss of rank or feelings of entrapment that are more influential in triggering depression compared to events that involve loss alone. These events include situations where the individual is rejected or humiliated in some way by a close tie or trapped in an unhappy marriage or job. Similarly, Farmer and McGuffin (2003) also found that events that signify loss or humiliation are associated with depression.

However, the experience of adversity is not just related to onset but also to poor outcome following treatment for depression. Murphy (1983) suggested that the experience of stressful life events in the year following therapy is associated with poor recovery. Mildly threatening life events and difficulties can also play an important role in producing psychological disturbances. Studies have shown that although severely stressful life events can provoke the onset of depression, it is actually mildly threatening life events that are pronounced in predicting relapse in depression (Brilman & Ormel, 2001; Ormel, Oldehinkel & Brilman, 2001). Similarly, Brown (1993) suggested that whilst negative life events are influential in triggering depression, positive life events such as those that indicate a fresh start or instill hope, are important for recovery from depression. In comparison, recovery from anxiety-related disorders was associated with events that bring about an increase in security such as re-employment following a period of unemployment (Brown, Lemyre & Bifulco, 1992).

The LEDS is a versatile tool as it has been used to label events and difficulties for many different meanings. Although Brown and Harris (1978) devised a comprehensive guide for classifying events and difficulties, they did not declare this to be a definitive list. For example, Malkoff-Schwartz, Frank, Anderson, Hlastala, Luther, Sherrill et al. (2000) used the LEDS to investigate the role of severe life events and difficulties on various types of affective disorders. This study set out to examine if events that interfere with an individual's sleep or daily routine such as timing of eating and exercise, can provoke the onset of a mood disorder. Malkoff-Schwartz et al. (2000) labelled these events as social rhythm disruption (SRD) and in

line with their hypothesis, the authors found that those who experienced a manic episode reported SRD-related events during the 8- and 20-week period prior to onset. Other studies have also developed various LEDS dimensions including pudicity events in EDs (Schmidt et al., 1997), goal frustration events in gastrointestinal disorders (Craig & Brown, 1984) and events involving conflict over speaking out are associated with difficulty in voice production (known as functional dysphonia; House & Andrews, 1988). Schmidt et al. (1997) described pudicity events as those that are of a sexual nature which are embarrassing or shameful for the individual. Craig and Brown (1984) defined goal frustration as life events which hinder or stop an individual from attempting to achieve their aims and ambitions and conflict over speaking out was developed to describe stressful events which stop an individual from freely expressing their thoughts as it may result in more conflict (House & Andrews, 1988).

In conclusion, this brief review has demonstrated that life events and difficulties do play an important role in both the aetiology and maintenance of psychopathologies such as depression and psychosis. These studies have all demonstrated the invaluable utility of the LEDS. Although the LEDS is labour intensive and time-consuming, it has also been shown to significantly reduce response errors associated with checklist methods (Kessler, 1997; Monroe, 2008). The studies described in this chapter and in Chapter Two have illustrated that the LEDS is adaptable to specific research needs, useful for eliciting contextually meaningful information and suitable for determining the timing and types of events that are related to onset, recovery and relapse from physical and psychological disorders. The systematic review presented earlier in this thesis has already indicated that stressful experiences can contribute to the development and maintenance of AN (Blaase & Elklit, 2001, Grilo et al., 2012; Karwautz et al., 2001, 2011; Mitchell et al., 1985; Pike et al., 2006; Raffi et al., 2000; Rojo et al., 2006; Schmidt et al., 1997; Sohlberg, 1990; Troop & Treasure, 1997; Welch et al., 1997). Several of these studies used the LEDS in relation to the onset of EDs (Rojo et al., 2006; Schmidt et al., 1997; Troop & Treasure, 1999). However, it must be highlighted that only one study (Sohlberg, 1990) examined the role that stressful life events on recovery or relapse from AN and found that negative life experiences predicted

worse outcome at follow-up. However, these findings must be taken with caution as a small sample of AN patients (*n* = 8) were recruited. Therefore, this highlights the need for methodologically strong studies examining the role of negative life events on relapse and recovery from AN.

Current study

This chapter will examine the role of life events and difficulties and affect regulation in relation to recovery and relapse from AN. In order to do this, the LEDS will be used to interview two groups of participants, one who have been recovered from AN for at least two years and a second group who recently attended therapy for AN. In addition, it is well established that those with AN report insecure attachment styles in adulthood (O'Kearney, 1996; O'Shaughnessy & Dallos, 2009; Ward et al., 2000; Zachrisson & Skårderud, 2010). Recent studies have also begun to demonstrate that unfavourable social comparison and self-critical thoughts and feelings are an important feature of AN (Bellew et al., 2006; Connan et al., 2007; Feinson & Meir, 2012; Fennig et al., 2008; Pinto-Gouveia et al., 2012; Schmidt & Treasure, 2006; Stormer & Thompson, 1996; Troop et al., 2003, 2013; Troop & Baker, 2008; Thompson et al., 1999; Williams et al., 1993). Based on the model presented in Chapter Three (*see Figure 3.2*), the current study will examine the role of stressful life experiences on eating behaviours and weight and the role that affect regulatory systems and processes play in recovery and relapse in a sample of women who have recovered following AN and women who have recently undergone treatment for AN.

Therefore, the current study will answer the following broad and specific research questions:

Broad research questions:

1) What is the role of stressful life changes on recovery and maintenance of ED symptoms following treatment for AN?
2) Do affect regulatory systems and processes contribute to recovery and maintenance of ED symptoms following treatment for AN?

Specific research questions:

1) How many patients were in remission, relapsed or maintained ED symptoms during and following treatment? Do these groups differ on BMI, DE behaviours and affect regulatory systems during and following treatment? Do these groups also differ on these variables compared to the recovered group?
2) Do more patients report severe and non-severe life events and difficulties compared to the recovered group? Do these groups differ when life events and difficulties are examined separately?
3) What are the types of life events and difficulties are reported by patients and those who have recovered?
4) Do those who have recovered and those who have recently undergone treatment for AN who report experiencing severe life events and difficulties over the two-year period report more unfavourable social comparison and attachment insecurity compared to those who do not report any adverse life events and difficulties?

Methodology

Design

This is a retrospective study employing a semi-structured interview to examine the life events and difficulties experienced over the two-year period prior to interview by recovered AN participants (as defined using the criteria described in the *Recruitment* section – see below) and AN patients who recently underwent treatment.

Participants

A total of 30 women aged between 19 and 60 years took part in the current study, which consisted of 16 women who had recovered from AN and 14 women who had recently undergone treatment for AN. The recovered group (n = 16) had a mean age of 30.4 years (SD = 11.5) and a mean BMI of 21.5kg/m^2 (SD = 3.0). From a potential pool of 48 trial patients who were eligible to take part in the current study,

14 agreed and were interviewed using the LEDS. The patient group had a mean age of 31.9 years (*SD* = 10.8) and a mean BMI (at time of interview) of 18.4kg/m^2 (*SD* = 1.6). The patients who completed the current study did not differ on baseline characteristics (upon entry to the trial) such as age, BMI or eating pathology to those who did not take part in the study (*p*-values ranged from .08 to .79). Preliminary analysis comparing the recovered group to the patient group revealed that these two groups did not differ on age, ($t(28) = -.36$, $p = -.72$) but, as expected, the patients reported a significantly lower BMI compared to the recovered participants, $t(22.34) = 3.48$, $p = .002$.

Demographic information for the study is presented in *Table 7.1* which shows that majority of the sample was British, single and had completed a degree. The recovered and patient groups did not differ on any of the demographic variables.

Table 7.1: Baseline demographic variables

Variable	Group		Significance
	Recovered (*n* = 16)	Patients (*n* = 14)	
Ethnicity *n* (%)			
British	13 (81.3)	13 (92.9)	χ^2 (df=3) = 2.77, *p* = .43
Other European	2 (12.5)	0	
Bangladeshi	1 (6.3)	0	
Other (Guam)	0	1 (7.1)	
Marital status *n* (%)			
Single	9 (56.3)	10 (71.4)	χ^2 (df=4) = 1.46, *p* = .83
Married/Cohabiting	5 (31.3)	3 (21.4)	
In a relationship	1 (6.3)	1 (7.1)	
Divorced	1 (6.3)	0	
Highest education *n* (%)			
A Levels	3 (18.8)	5 (35.7)	χ^2 (df=3) = 2.77, *p* = .43
Bachelors	9 (56.3)	8 (57.1)	
Postgraduate	4 (25)	1 (7.1	

Recruitment

The current study recruited two groups of participants. The first set of participants were those who were had been recovered from AN for at least two years. Recovered participants were recruited using the b-eat (beating eating disorders) website (www.b-eat.co.uk). This charity supports and promotes research examining EDs. Therefore, b-eat currently offers to advertise research on the website and allow researchers to contact willing participants through their database. The recovered participants were invited to take part in the study if they fulfilled the following criteria:

Inclusion criteria

- Females over the age of 18
- Diagnosed with AN by a mental health professional in the past

- Have received treatment for AN in the past
- Not currently undergoing treatment for AN or any other eating disorder
- Have been recovered for 24 months and have had periods for the last 12 months

Exclusion criteria

- Respondents whose level of English is insufficient to be able to comprehend the interview questions
- Currently have an eating disorder

The second set of participants were patients who had received treatment for AN as part of a randomised control trial. The trial was conducted at the Eating Disorders Outpatient Service of the South London and Maudsley NHS Foundation Trust and patients were randomised to one of two types of therapy known as the Specialist Supportive Clinical Management (SSCM) and the Maudsley Model for Treatment of Adults with AN (MANTRA). Both groups of patients attended weekly sessions for 20 weeks followed by monthly follow-up sessions for four months. The researcher was blind to the patient therapy group assignment to ensure knowledge of this did not influence the current study. These two therapies have been described briefly in Chapter One but, as the current study is not an assessment of therapy effectiveness in treating AN, no more information regarding the effectiveness of these two therapies will be provided.

Measures and procedure

Participants who were recruited using the b-eat research volunteer facility were sent an email inviting them to take part. Those who expressed an interest contacted the researcher for further information regarding the study, followed by arrangement of an interview date. Similarly, at one-year post-intervention, patients who had taken part in the Maudsley intervention were approached by the trial researchers and invited to take part in the current study. Those who were interested were advised to contact the researcher in order to arrange an interview date.

All participants were interviewed using the LEDS which, as described in the introduction, is a semi-structured interview designed to elicit information regarding events and difficulties that occurred over a specified time period. In addition to the LEDS interview guide, items were developed to ask about self-critical and self-reassuring thoughts and feelings in relation to people's emotional responses. These items were asked in relation to specific events and difficulties (rather than general trait-like responses) but because too few participants indicated any self-criticism/reassurance, this was not analysed and will not be discussed further.

For the purpose of the current study, both groups of participants were interviewed regarding the two years prior to the interview date. To aid the participant's memory for dates, the researcher placed a timeline covering the two years in front of the interviewee. For telephone and Skype interviews, the interviewer advised the participant that she was creating a timeline of their life for the previous two years and in order to do this she would need them to try and provide a date or a timeframe during which each event and difficulty occurred. For patients who completed the trial, this covered the year of the intervention and the year following therapy. The majority of participants attended a face-to-face interview (*n* = 23) but those who were unable to meet at a suitable location were interviewed over the telephone (*n* = 6) or via Skype (*n* = 1).

Participants were required to complete the following scales in addition to providing basic demographic information such as age, height and weight measurements (to calculate BMI kg/m^2), ethnicity, marital status, highest education qualification and occupation (*for more information regarding these measures, please refer to Chapter four*):

1. Insecurity of attachment was measured VASQ (Bifulco et al., 2003)
2. Social rank was measured using the SCRS (Allan & Gilbert, 1995)
3. Levels of eating pathology for each time point (except T6) for clinical patients were determined using the EDE interview (Fairburn et al., 2008)

The recovered group completed the self-report measures on one occasion following completion of the LEDS. The patient group completed the self-report measures at

several points but, for the purpose of the current study, the data collected at baseline (pre-treatment), T4 (end of treatment), T5 (1-year follow-up) and T6 (following completion of the LEDS) will be presented. The patients were contacted by the trial researchers on two further occasions during the therapy but this was either a short telephone review in which patients did not complete self-report measures (T2) or six months into treatment (T3). These assessments are not included here.

Statistical analysis

Several statistical analyses were conducted. Firstly, descriptive statistics for all measures at baseline and follow-ups were computed. This was followed by independent samples *t*-test comparing the patient group to the recovered group on BMI, SCRS and VASQ (including subscales) scores taken at T6. The next stage of the data analyses involved grouping patients into those who were in remission immediately following treatment (at T4) and at 1-year follow-up (T5) and those who maintained their AN symptoms (this is described in more detail below). Upon determining remitters and "still ill" groups, repeated measures ANOVAs were conducted to compare these two groups on various variables between baseline (T1) and end of treatment (T4) and then between end of treatment (T4) and 1-year follow-up (T5). The between-subjects variable was the *group* that the participants were assigned (remitters versus still ill) and the within-subjects variables were *time* (the first set of analyses compared baseline (pre-treatment) versus end of treatment (T4) scores; the second set of analyses compared end of treatment (T4) to 1-year follow-up (T5) scores) and *measures* (BMI, EDE (including subscales), SCRS and VASQ (including subscales)). A oneway ANOVA was then conducted to compare the two patient groups (remitters and still ill) and the recovered group on T6 BMI, SCRS and VASQ (including subscales) scores.

The next phase of the data analyses involved examining the data collected during the LEDS interviews. The data analyses were based on severe life events and marked difficulties unless otherwise stated. The first group of analyses compared the overall patient group to the recovered group. Firstly, the proportion of patients (overall group) who experienced life events and difficulties was compared to the

number of recovered participants who experienced stressful life changes over the two-year period. When examining frequencies within a 2x2 table, the Fishers Exact test was reported but when one or more cells contained fewer than 5 observations, Yates Continuity Correction was applied (Yates, 1934). For tables larger than 2x2, the Chi^2 statistic was reported.

Further analyses were conducted to compare the number of patients and recovered women who did or did not experience life events and difficulties during the first year (year of treatment for the patient group) and the second year (follow-up period for the patient group), separately. Independent samples *t*-test was then used to assess if patients experienced a greater mean number of severe and non-severe life events and difficulties over the two-year period compared to the recovered group. A group of two-way ANOVAs were conducted to compare recovered participants and AN patients who experienced stressful life events over the two-year period to those who didn't report any stressful life changes on T6 SCRS, VASQ and VASQ subscales scores. The between-subjects variables were group (recovered versus patients) by those who experienced a life event versus those who didn't report any stressful life events. The within-subjects variables were SCRS and VASQ (including subscales).

Additional Chi^2 analyses was then conducted to compare the recovered participants to the patient subgroups (remitters and still ill) on the number of individuals in each group who experienced life events and difficulties over the two-year period. This analysis was conducted to examine if a higher proportion of patients who were still ill reported life events and/or difficulties compared to the number of recovered participants and patients who were in remission who reported life events and/or difficulties.

Finally, where appropriate, effect sizes as measured by partial eta-squared (η^2) and Cohen's *d* were reported. For partial eta-squared, values of .01, .06 and .16 represented small, medium and large effect sizes, respectively (Cohen, 1977) and for Cohen's *d*, values of .20, .50 and .80 represented small, medium and large effect sizes, respectively (Cohen, 1992).

Results

Sample characteristics

Means and standard deviations for the variables of interest at T1, T4, T5 and T6 for the patient group and for the recovered group are presented in *Table 7.2*. Comparisons between the patient group and the recovered group at T6 revealed that these two groups differ on BMI, $t(28) = 3.07$, $p = .01$ and the SCRS, with patients reporting more unfavourable social comparison compared to the recovered group, $t(28) = 2.32$, $p = .03$. However, these two groups did not differ on total VASQ scores, $t(28) = -.74$, $p = .47$ or scores from the two subscales, insecurity, $t(28) = -.99$, $p = .33$, and proximity-seeking, $t(28) = .02$, $p = .99$.

Table 7.2: Means (*SD*) for the patient and recovered groups

	Patient Group (*n* = 14)				**Recovered**
Variable	**T1**	**T4**	**T5**	**T6***	**(*n* = 16)***
EDE (Total score)	2.83 (1.32)	2.27 (1.70)	1.92 (1.61)	-	-
Dietary restraint	3.69 (1.72)	2.53 (2.20)	2.33 (2.02)	-	-
Eating concerns	2.46 (1.51)	1.94 (1.78)	1.40 (1.43)	-	-
Weight concerns	2.66 (1.57)	2.06 (1.59)	1.61 (1.78)	-	-
Shape concerns	2.57 (1.83)	2.42 (2.11)	2.19 (1.94)	-	-
BMI	16.82 (.91)	18.21 (1.34)	18.64 (1.84)	18.64 (1.86)	21.49 (.77)
SCRS	41.21 (18.13)	50.83 (13.50)	48.77 (15.66)	49.21 (12.09)	61.38 (15.98)
VASQ (Total score)	42.79 (7.75)	42.32 (6.94)	39.85 (8.35)	39.50 (6.26)	37.63 (7.54)
Insecurity	22.93 (5.77)	22.21 (5.79)	21.62 (5.71)	21.21 (5.09)	19.31 (5.42)
Proximity-seeking	19.86 (3.70)	20.11 (3.44)	18.23 (4.21)	18.29 (3.45)	18.31 (4.17)

Note. *Denotes the point at which both groups completed the LEDS, SCRS and VASQ (Patient group completed the SCRS and VASQ for the last time during the LEDS)

Defining recovery in the patient group

Question 1: How many patients were in remission, relapsed or maintained ED symptoms during and following treatment? Do these groups differ on BMI, DE

behaviours and affect regulatory systems during and following treatment? Do these groups also differ on these variables compared to the recovered group?

The patient group was divided into two groups to reflect those who were in remission immediately following treatment (T4) and at 1-year follow-up (T5) and those who had maintained their ED symptoms at the end of treatment (T4) and at 1-year follow-up (T5). These were labeled as "in remission" and "still ill". Total EDE scores less than one standard deviation above community mean (i.e. below 1.74) were used to group the patients into those who were in remission and those who were still ill. This criterion has been used by Fairburn et al. (2009) and Schmidt et al. (2012) and has been shown to reflect clinically meaningful change (Kendall, Marrs-Garcia, Nath & Sheldrick, 1999). Using this criterion, it was found that at T4 (end of treatment), 6 patients had an overall EDE score below 1.74 and these patients were labelled as being in remission. The same 6 patients were found to continue to be in remission at 1-year follow-up (T5) as their EDE scores were still below 1.74. The 8 patients who were still ill at T4 were also still ill at T5. These two groups were compared on various baseline measures (*see Table 7.3*) and the results revealed that those who maintained their ED symptoms had been ill for longer compared to those who were in remission.

Table 7.3: Comparing patients who were in remission to those who were still ill on various baseline variables

Variable	Remission (*n* = 6)	Still ill (*n* = 8)	Significance
Age	26.67 (10.25)	31.75 (11.00)	$t(12) = -.88$, $p = .40$
Age of onset	23.80 (12.70)	14.00 (3.79)	$t(4.51) = 1.67$, $p = .16$
Illness duration (years)	4.50 (5.22)	17.50 (13.07)	$t(9.90) = -2.51$, $p = .03$

The two patient groups were compared on various psychological measures over the year of treatment and follow-up period. Results of the repeated measures ANOVAs comparing patients who were in remission and those who were still ill on various variables taken at both T1 (baseline) and T4 (end of treatment) are shown in *Table 7.4*. There was an effect of group on the EDE and its subscales with those who were still ill scoring higher compared to those who were in remission. There was an

effect of time on EDE, dietary restraint and SCRS scores with higher EDE and dietary restraint but low SCRS scores reported at baseline (T1) compared to end of treatment (T4). The interaction effects revealed that the patients who were in remission reported a greater decrease in their overall EDE and dietary restraint scores compared to those who were still ill between baseline (T1) and end of treatment (T4). The interaction effects on SCRS scores showed that it was those who were still ill who reported a greater increase in SCRS scores compared to those who were in remission. However, it must also be highlighted that those who were still ill reported lower SCRS mean scores at baseline compared to those who later remitted. Although significant interaction effects were not found for any other variables of interest, partial eta-squared values revealed that there are still medium to large effects especially for BMI and weight and shape concerns.

Table 7.4: Results of the repeated measures ANOVAs comparing patients who were in remission (*n* = 6) and those who were still ill (*n* = 8) on mean (*SD*) baseline (T1) and end of treatment (T4) scores

	T1 scores		T4 scores		Group	Time	Interaction	
Variable	**Remission**	**Still ill**	**Remission**	**Still ill**	***F*(1, 12)**	***F*(1, 12)**	***F*(1, 12)**	**η^2**
BMI	16.17	17.15	18.78	17.73	.004,	8.01,	3.23,	.23
	(.72)	(.63)	(2.11)	(1.79)	*p* = .95	*p* = .02	*p* = .10	
EDE	1.73	3.65	.51	3.60	45.81,	10.08,	8.33,	.41
(Total score)	(1.10)	(.73)	(.65)	(.62)	*p* <.001	*p* = .01	*p* = .01	
Dietary	2.53	4.55	.37	4.15	26.07,	11.39,	5.40,	.31
restraint	(1.65)	(1.24)	(.65)	(1.31)	*p* .<.001	*p* = .01	*p* = .04	
Eating	1.40	3.25	.53	3.00	13.19,	2.86,	.87,	.07
concerns	(1.53)	(.94)	(.67)	(1.60)	*p* = .003	*p* = .12	*p* = .37	
Weight	2.10	3.08	.73	3.05	7.26,	3.25,	3.02,	.11
concerns	(1.73)	(1.40)	(.99)	(1.16)	*p* = .02	*p* = .10	*p* = .11	
Shape	1.10	3.67	.41	3.93	24.11,	.76,	3.61,	.23
concerns	(1.10)	(1.46)	(.57)	(1.41)	*p* <.001	*p* = .40	*p* = .08	
SCRS	45.50	35.50	50.50	51.17	.26,	19.43,	5.18,	.34
	(10.15)	(23.96)	(10.37)	(17.13)	*p* = .62	*p* = .001	*p* = .05	
VASQ	44.67	41.38	45.58	39.88	1.52,	.04,	.76,	.06
(Total score)	(3.50)	(9.87)	(3.88)	(7.92)	*p* = .24	*p* = .84	*p* = .40	
Insecurity	24.50	21.75	24.67	20.38	1.49,	.32,	.52,	.04
	(4.23)	(6.73)	(3.88)	(6.52)	*p* = .25	*p* = .58	*p* = .49	
Proximity-	20.17	19.63	20.92	19.50	.28,	.19,	.37,	.03
seeking	(2.40)	(4.60)	(3.14)	(3.74)	*p* = .61	*p* = .67	*p* = .55	

Table 7.5 shows how the two patient groups compared on scores between T4 (end of treatment) and T5 (1-year follow-up). The repeated measures ANOVAs revealed that there was an effect of group on the EDE and its subscales' scores with those who remained in remission at 1-year follow-up reporting significantly lower scores compared to those who were still ill. There was a significant effect of time on VASQ and proximity-seeking scores with higher VASQ scores (specifically the

proximity-seeking style of attachment) reported at the end of treatment (T4) compared to at 1-year follow-up (T5) scores. There was an interaction effect on BMI, VASQ and insecurity subscale scores. Those who were still ill reported an increase in BMI between T4 and T5 while those in remission reported a greater decrease in VASQ (and the insecurity subscale) than those who were still ill. In fact, those who were still ill reported an increase in VASQ scores (and the insecurity subscale). Effects sizes using partial eta-squared revealed that there were small effects for the SCRS and the proximity-seeking subscale of the VASQ and medium to large effects for BMI, VASQ and the insecurity subscale of the VASQ.

Table 7.5: Results of the repeated measures ANOVAs comparing patients who were in remission (*n* = 6) and those who were still ill (*n* = 8) on mean (*SD*) end of treatment (T4) and 1-year follow-up (T5) scores

	T4 scores		T5 scores		Group	Time	Interaction	
Variable	**Remission**	**Still ill**	**Remission**	**Still ill**	***F*(1, 12)**	***F*(1, 12)**	***F*(1, 12)**	**η^2**
BMI	18.78	17.73	18.70	18.53	.32,	3.18,	4.86,	.31
	(2.11)	(1.79)	(2.12)	(1.88)	*p* = .56	*p* = .10	*p* = .05	
EDE	.51	3.60	.36	3.09	84.34,	1.97,	.57,	.05
(Total score)	(.65)	(.62)	(.42)	(1.00)	*p* .<.001	*p* = .19	*p* = .47	
Dietary	.37	4.15	.47	3.73	38.68,	.50,	1.31,	.10
restraint	(.65)	(1.31)	(.60)	(1.45)	*p* <.001	*p* = .49	*p* = .28	
Eating	.53	3.00	.17	2.33	27.18,	1.52,	.13,	.01
concerns	(.67)	(1.60)	(.32)	(1.21)	*p* .<.001	*p* = .24	*p* = .72	
Weight	.73	3.05	.33	2.58	15.20,	1.59,	.01,	.001
concerns	(.99)	(1.16)	(.41)	(1.81)	*p* = .002	*p* = .23	*p* = .92	
Shape	.42	3.93	.38	3.55	38.65,	.59,	.38,	.03
concerns	(.57)	(1.41)	(.47)	(1.37)	*p* <.001	*p* = .46	*p* = .55	
SCRS	50.50	52.40	52.83	43.60	.20,	.39,	1.17,	.12
	(10.37)	(18.85)	(18.19)	(16.20)	*p* = .67	*p* = .55	*p* = .31	
VASQ	45.58	38.71	39.50	40.14	.58,	5.17,	13.47,	.55
(Total score)	(3.88)	(7.78)	(5.50)	(10.67)	*p* = 46	*p* = .04	*p* = .004	
Insecurity	24.67	19.43	21.17	22.00	.53,	.26,	10.99,	.50
	(3.88)	(6.43)	(3.49)	(7.39)	*p* = .48	*p* = .62	*p* = .01	
Proximity-	20.92	19.29	18.33	18.14	.18,	11.73,	1.75,	.14
seeking	(3.14)	(3.99)	(3.88)	(4.78)	*p* = .68	*p* = .01	*p* = .21	

The two patient groups were also compared to the recovered group on T6 BMI, SCRS and VASQ scores (*see Table 7.6*). The results show that these three groups differed on BMI and SCRS. Post-hoc comparisons using the Bonferroni adjustment revealed that the 3 groups did not differ significantly from each other on BMI but the patient group who were still ill scored significantly lower on the SCRS compared to the recovered group (mean difference = -16.13, *p* = .04). However, patients who

were in remission did not differ on SCRS from patients who were still ill (mean difference = 9.25, p = .71) or those who had recovered (mean difference = -6.88, p = .96). The recovered group had SCRS scores that are comparable to healthy student controls (Troop et al., 2003). Large effects for group differences for BMI and SCRS scores were found (*see table 7.6*).

Table 7.6: Oneway ANOVA comparing the two patient groups to the recovered group on T6 scores

Variable	Patient Group		Recovered	Significance	
	Remission (n = 6)	Still ill (n = 8)	(n = 16)	$F(2, 29)$	η^2
BMI	18.50 (2.11)	18.74 (1.79)	21.49 (2.98)	4.54, p = .02	.26
SCRS	54.50 (8.57)	45.25 (13.31)	61.38 (15.98)	3.47, p = .05	.20
VASQ (Total score)	41.17 (2.86)	38.25 (7.92)	37.63 (7.54)	.56, p = .58	.04
Insecurity	22.67 (2.88)	20.13 (6.24)	19.31 (5.42)	.88, p = .43	.06
Proximity-seeking	18.50 (1.76)	18.13 (4.45)	18.31 (4.17)	.02, p = .98	.001

LEDS

Question 2: Do more patients report severe and non-severe life events and difficulties compared to the recovered group? Do these groups differ when life events and difficulties are examined separately?

Interviews lasted on average approximately one hour and 45 minutes and there was no significant difference in the length of the interviews between the recovered and patient groups (t(28) = -.05, p = .96). Preliminary analysis revealed that 56% of recovered participants and 35% of patients reported at least one life event and/or difficulty, but these proportions did not differ significantly (Fishers exact test; p = .30). Similarly, these two groups also did not differ when they were compared on life events and difficulties separately (Fishers exact test: p = .72 and Continuity correction = .08, p = .78, respectively).

Differences in the number of recovered, remitted and still ill women who experienced at least one severe life event or marked difficulty during the first year (during treatment for the patient group) and the second year (during follow-up for patients) were also examined separately. During the first year, 31% of the recovered

participants and 14% of the patient group reported experiencing life events and/or difficulties and these proportions were not significantly different (Continuity correction = .44, *p* = .51). Similarly, the number of patients (29%) and recovered participants (44%) who reported experiencing life events and/or difficulties during the second year of assessment did not significantly differ (Continuity correction = .23, *p* = .63). Indeed, the number of severe events reported by recovered women is actually greater than that reported by patients.

Although similar proportions of patients and recovered participants experienced at least one life event or difficulty over the two years of interview (and during the first and second years separately), the data were analysed to examine if the patient group differed on the mean *number* of severe and non-severe life events and difficulties experienced over the two-year period (*see Table 7.7*) when compared to the recovered group. Independent samples *t*-test revealed that these two groups did not differ on the number of severe or non-severe events and difficulties experienced over the two years. However, a medium effect size (as measured by Cohen's *d*) was found for non-severe life events.

Table 7.7: Independent samples *t*-tests comparing recovered and patients on the mean number of severe and non-severe events and difficulties experienced over the two-year period

	Group			
	Recovered (*n* = 16)	**Patient (*n* = 14)**	**Significance *t*(28)**	**Effect size *(d)***
Severe events/difficulties				
Events	.63 (.89)	.71 (1.38)	-.21, *p* = .83	-.07
Difficulties	.25 (.45)	.21 (.58)	.19, *p* = .85	.08
Non-severe events/difficulties				
Events	12.69 (7.04)	10.00 (6.24)	1.10, *p* = .28	.40
Difficulties	3.31 (1.82)	3.71 (2.23)	.27, *p* = .59	-.20

Question 3: What are the types of life events and difficulties are reported by patients and those who have recovered?

Both recovered participants and patients reported a total of 20 severe life events (recovered = 10 and patients = 10) and 7 marked difficulties (recovered = 4 and patients = 3) over the two-year period between them. The types of severe life events that were reported by the recovered group included crisis at work, relationship breakdown and health-related events. The recovered group also reported marked difficulties resulting from work-, relationship- and money-related problems. Similarly, patients reported severe life events such as breakdown of a relationship, physical illness involving attending hospital, bereavement and quitting studying or work. The types of marked difficulties reported by patients included relationship problems and bereavement. Overall, both groups indicated that 45% of the severe life events and 29% of the marked difficulties that they experienced were as a result of interpersonal problems.

Question 4: Do those who have recovered and those who have recently undergone treatment for AN who report experiencing severe life events and difficulties over the two-year period report more unfavourable social comparison and attachment insecurity compared to those who do not report any adverse life events and difficulties?

A set of two-way ANOVAs were computed to compare recovered and AN patient participants who experienced stressful life events over the two-year period to those who did not report any stressful life experiences on SCRS, VASQ and VASQ subscale scores completed at T6. The results indicate that there was an effect of group on SCRS scores with the recovered group scoring higher compared to the patient group. However, there was no effect of experiencing a life event of difficulty on SCRS, VASQ and VASQ subscale scores. Similarly, there were no interaction effects and small effect sizes were found (*see Table 7.8*).

Table 7.8: Results of the two-way ANOVAs comparing patients and recovered participants who experienced life events or difficulties and those who didn't report any stressful life changes on T6 measures

	Recovered Group		Patient Group					
	Report event/difficulty		Report event/difficulty		Event/Diff	Group	Interaction	
Variable	**Yes (*n* = 9)**	**No (*n* = 7)**	**Yes (*n* = 5)**	**No (*n* = 9)**	***F*(1, 26)**	***F*(1, 26)**	***F*(1, 26)**	**η^2**
SCRS	61.33	61.43	53.20	47.00	.31,	4.18,	.33,	.01
	(19.49)	(11.50)	(11.92)	(12.29)	*p* = .59	*p* = .05	*p* = .57	
VASQ	38.67	36.29	41.80	38.22	1.26,	.91,	.05,	.02
(Total score)	(7.97)	(7.32)	(3.27)	(7.29)	*p* = .27	*p* = .35	*p* = .82	
Insecurity	19.67	18.86	22.80	20.33	.65,	1.29,	.17,	.01
	(5.79)	(5.34)	(2.77)	(5.98)	*p* = .43	*p* = .27	*p* = .69	
Proximity-	19.00	17.43	19.00	17.89	.82,	.02,	.02,	.001
seeking	(4.18)	(4.31)	(2.00)	(4.11)	*p* = .37	*p* = .88	*p* = .88	

Note. Event/Diff = Reported event/difficulty grouping

Additional statistical analysis was conducted to examine differences in the rates of severe life events/difficulties between patients who were still ill and a combined group of recovered and remitted women. More recovered participants and remitted patients (59%) experienced severe life events and difficulties compared to the patients who were still ill (13%) but this result was only marginally significant, (Continuity correction = 3.42, *p* = .07).

Discussion

The present study examined the role of stressful life changes on recovery and maintenance of ED symptoms following treatment for AN. In addition, the impact of insecurity of attachment and perceived low social status (affect regulatory systems) were examined in relation to recovery and maintenance of ED symptoms.

Findings

Data collected from the LEDS (Brown & Harris, 1978) revealed that just under half (45%) of the severe life events reported by both recovered participants and the patient group, were as a result of interpersonal problems, consistent with previous research (Rojo et al., 2006; Schmidt et al., 1997; Troop & Treasure, 1997). However, in contrast to the studies reviewed in Chapter Two, the current study did not find a significant relationship between stressful life changes and current AN. The results of this study posit that stressful life changes may not have an enduring influence on the maintenance of ED symptoms as previously suggested by Smyth et al. (2008). These findings are similar to that of Barraclough, Pinder, Cruddas, Osmond, Taylor and Perry (1992) who found that the experience of severe life events did not have psychogenic properties in relation to relapse from breast cancer. To the extent that where there were differences, those in remission and recovery reported *more* severe life events and difficulties compared to those who were still ill (although this result was only marginally significant).

Those who had been recovered from AN for at least two years reported a more favourable social comparison than did those who had recently undergone treatment. These findings are in line with previous research that has found that unfavourable social comparison is associated with ED symptoms (Connan et al., 2007; Pinto-Gouveia et al., 2012; Schmidt & Treasure, 2006; Troop et al., 2003, 2013; Troop & Baker, 2008).

During treatment, patients who remitted as well as those who remained ill reported an increase in their social comparison suggesting that attending therapy assisted in improving how anorexic patients compare themselves with others, regardless of recovery. However, when the two patient groups were compared on their final assessment scores to the recovered group, it was found that those who had been recovered for at least two years reported more favourable social comparison compared to those who were still ill but did not differ significantly from those who were in remission. These findings provide even further support that an improvement in social comparison evaluations is associated with an improvement in AN symptoms which is similar to the results presented by Connan et al. (2007) who found that those in recovery reported social comparison scores that were

intermediate between those who were still ill and healthy adults. Interestingly, in this study, women who had recovered had similar mean SCRS scores to healthy populations (Troop et al., 2003). When the two patient groups were compared on changes in their scores between end of treatment and 1-year follow-up, it was found that those who maintained recovery over the follow-up period also reported a decrease in their insecurity of attachment, whilst those who remained ill reported an increase. These findings suggest that attachment-related behaviours change with recovery. However, it is not clear whether it is remission that leads to improvement in attachment security or whether improved attachment security helps to maintain remission.

Strengths and limitations

The current study also has a number of strengths including the use of an extensive and objective measure of life events and difficulties (LEDS; Brown & Harris, 1978) which overcomes problems associated with self-report measures of stress and subjective measures of life events. Secondly, this study recruited a clinical sample of AN patients and those with a history of AN. Thirdly, the patients who took part in the therapeutic intervention were assessed using an investigator-based measure of eating pathology (EDE; Fairburn et al., 2008) at various time points. However, dysfunctional eating behaviours of those who were in the recovered group were not assessed and although these participants were recruited on the basis that they had been recovered for at least two years, they may still present some dysfunctional eating patterns. Therefore, future studies should assess DE behaviours of comparison groups with a history of EDs, which can easily be achieved using self-report measures such as the EDE-Q (Fairburn & Beglin, 1994).

Other limitations of the current study include the use of self-report measures of social comparison and adult attachment styles which are susceptible to bias but can provide a reasonable basis to explore the relationship between psychological constructs. A second limitation of the current study is the small sample which was partly due to a slow uptake of patients onto the randomised control trial for the treatment of AN during the early stages of recruitment. Only 30% of the eligible patients who completed therapy agreed to take part in the current study. Therefore,

future studies should recruit a larger sample of ED patients and recovered participants. Although the current study used the LEDS as a method of eliciting detailed information about life events and difficulties, there is always the possibility of error due to human memory when data are based on retrospective recall. However, the LEDS (Brown & Harris, 1978) is a thorough and comprehensive guide for overcoming any potential investigator bias and issues surrounding self-reported and predetermined rating scales (Brown, 1989) and is the best available tool for answering the research questions of the current study.

Another limitation of the present study is related to the method of assessing self-critical and reassuring evaluations. Self-critical and reassuring questions were added to the LEDS and asked in relation to any life events and difficulties nominated (rather than use the FSCRS as in the other studies presented in this thesis). However, no self-critical/reassuring thoughts or feelings were reported. Therefore, these findings may have differed if a general measure of self-criticism and reassurance such as the FSCRS (Gilbert et al., 2004) had been employed. However, this approach has been successfully applied in relation to crisis support (Brown & Harris, 1986) and coping (Bifulco & Brown, 1996), including in relation to EDs (Troop & Treasure, 1997; Troop et al., 1998) and so the failure to elicit this information here may have been due to a flaw in the measure that was developed. A final limitation (as discussed previously) is the self-report method of assessing height and weight of recovered participants.

Implications

The theoretical implications of the current findings demonstrate that unfavourable social comparison and insecurity of attachment in adulthood are related recovery from EDs. These findings add to the literature on problematic weight regulation as the results of the current study and those of Study Two (Chapter Six) suggest that ineffective affect regulatory systems are associated with problematic weight regulation in both those with AN and those who were formerly heavier than their weight on entry into the study. Therefore, these findings have similar implications to that of Study Two, specifically, interventions designed to treat

AN should also address issues of unfavourable social comparison and attachment insecurities.

Finally, although the current study did not find an association between stressful life experiences and affect regulation systems, it must be emphasised that these null findings should be taken with caution as the current study did employ a very small sample hence limiting the statistical power of the analyses. Nevertheless, rates of life events/difficulties were higher in those who were recovered or in remission than those who were still ill. Clearly, it is not the occurrence of life stresses per se that adversely affect recovery from AN. Further implications will be discussed in the final Discussion Chapter.

Conclusion

The present study presents evidence that affect regulatory processes such as insecurity of attachment and perceived low social rank are important for eating pathology in a clinical sample of ED patients. Therefore, taking together all the findings presented throughout this thesis so far, the next study examines the impact of an intervention thought to influence affect regulation and changes in BMI, DE and stress.

Chapter 8: Study 4

The impact of an emotionally expressive writing intervention on stress, bodyweight, and eating pathology

Introduction

The current thesis has presented a synthesis of the research that has examined the relationship between stress and problematic weight regulation followed by how affect regulatory systems (social rank and attachment) and processes (self-criticism and reassurance) can contribute to this relationship. In summary, the studies described so far examined the change trajectories of BMI, DE and stress, how these changes influence each other and the predictors of these changes, predictors of weight regain and the role of life events and affect regulation systems in recovery and symptom maintenance in AN.

The first study presented in this thesis revealed the importance of the affect regulatory systems and processes in changes in weight, eating pathology and stress. Importantly, this longitudinal study showed that contrary to previous research, increases in stress are related to reductions to BMI. Also, perceptions of higher social status are predictive of a decrease in stress levels over time, whereas high levels of proximity-seeking and self-criticism (in the form of inadequate thoughts and feelings) were related to higher stress levels at baseline and less decreases in stress levels over time, respectively. Independently of this, more favourable social comparison was related to lower BMI at baseline.

The second study examined the changes in BMI, DE and stress levels in women who had previously been overweight. Those who reported an increase in their stress levels over six months also reported a decrease in their social status compared to those who reported stable or decreased stress perceptions. Again, however, stress was not related to weight gain in these women. Similarly, affect regulatory systems also relate to recovery from AN following treatment (Study Three; Chapter Seven) but life events did not (if anything, stress was higher in those

who were in recovery or remission than those who were still ill). In summary, Studies One and Two highlight the role of affect regulatory systems and processes in the perception of stress, which has an impact on changes in weight as shown by Study One. In addition, Study Three has emphasised the influence that affect regulation systems have on eating pathology. However, the studies presented so far have been naturalistic and an experimental study using an affect regulation manipulation would provide a suitable method for examining the role of affect regulation in influencing changes in stress, eating and bodyweight during a stressful period.

Expressive writing

Disclosure in the form of expressive writing is a powerful therapeutic method that may aid the healing process following a traumatic experience and can bring about a general reduction in biological indicators of stress and stress-related illness (Pennebaker, 1985, 1997). Pennebaker and Beall (1986) proposed that inhibiting thoughts and/or feelings following a traumatic event requires physiological effort which then increases the risk of developing stress-related diseases (Selye, 1976). Their seminal study launched the expressive writing paradigm, which involves writing for 15-20 minutes for 3-5 consecutive days either about a stressful experience or about a neutral topic. Since this pioneering paradigm was introduced, numerous research studies using this form of disclosure have followed. It is beyond the scope of this chapter to review all of the studies that have examined the role of expressive writing on a variety of outcomes. However, several systematic reviews and meta-analyses have shown that expressive writing has a beneficial effect on a range of social, behavioural, psychological and health outcomes (Baikie & Wilhelm, 2005; Frattarolli, 2006; Frisina, Borod & Lepore, 2004; Harris, 2006; Lowe, 2006; Meads, Lyons & Carroll, 2003; Sloan & Marx, 2004b; Smyth, 1998; Wright & Chung, 2001). Improved social and behavioural benefits of expressive disclosure include reduced absenteeism from work, quicker re-employment after job loss, improved working memory, reduced weight gain after smoking cessation, improved sporting performance, achieving better grades at University and a reduction in disturbed eating behaviours. Benefits to health include improved immune function, lower levels of depression, fewer visits to health centres and reduced blood pressure, pain

and medication use (Ames, Ames, Stevens, Patten, Werch & Schroeder, 2008; Baikie & Wilhelm, 2005; East, Startup, Roberts & Schmidt, 2010; Frattarolli, 2006; Frisina et al., 2004; Harris, 2006; Meads et al., 2003; Sloan & Marx, 2004b; Smyth, 1998; Wright & Chung, 2001). Meta-analyses reveal *r*-effect sizes ranging from .08 to .23 (Frattarolli, 2006; Frisina et al., 2004; Smyth, 1998) and Hedges's *g* effect sizes ranging between .06 and .21 (Harris, 2006) which correspond to small to medium effects (Cohen, 1992). However, whilst Frisina et al. (2004) found that expressive writing is most effective for physical health outcomes, Smyth's (1998) findings suggested that the effect of expressive writing is strongest for psychological well-being. Despite these inconsistent findings, Lowe (2006) refers to expressive writing as being "mightier than the pill" (p. 62) and Spiegel (1999) suggests that, if pharmacological interventions had similar effect sizes as those produced by disclosure, it would be a significant advance in medicine.

Adaptations of expressive writing

Since the launch of expressive writing, nearly three decades ago, there have been many adaptations to this interesting paradigm. Adaptions to the original expressive writing task have revealed that writing for 15 minutes three times during a single hour can produce similar health benefits as the original method of writing for 15 minutes a day over three consecutive days (Chung & Pennebaker, 2008). Interestingly, even the act of writing for just two minutes on each of two days is related to fewer health-related complaints (Burton & King, 2008). Disclosing in the form of talking rather than writing is also effective (Pennebaker, 1985, 1997). Most studies have been carried out in laboratories but carrying out disclosure in the comfort of one's own home is just as effective (van Middendorp, Sorbi, van Doornen, Bijlsma & Geenen, 2007).

Furthermore, although the majority of the earlier studies were conducted with students, later research found that disclosure in the form of expressive writing is also beneficial for an array of different people including maximum-security prisoners, distressed crime victims, trauma survivors, those diagnosed with medical conditions such as cancer, arthritis and chronic pain, those who were made redundant from their jobs, women who had recently given birth to their first child

(Baikie & Wilhelm, 2005; Pennebaker & Seagal, 1999; Sloan & Marx, 2004a) and couples who have experienced infidelity (Gordon, Baucom & Snyder, 2004). These findings illustrate that the benefits of expressive writing can still be achieved without committing too much time or effort to complete the task, by self-administering at home and by many types of people. This opens many doors for the use of expressive writing as a cost-effective and easy to administer intervention for managing the effects of stress.

While most studies find that writing about trauma has an effect on stress and health, some recent studies have found the same effect using positive writing tasks but without the short-term negative consequences for mood (Burton & King, 2004, 2008, 2009; Danner, Snowdon & Friesen, 2001; King, 2001a; King & Miner, 2000; Low, Stanton & Danoff-Burg, 2006; Marlo & Wagner, 1999; Stanton, Danoff-Burg, Sworowski, Collins, Branstetter, Rodriguez-Hanley et al., 2002; Troop, Chilcot, Hutchings & Varnaite, 2013; Wing, Schutte & Byrne, 2006).

Positive expressive writing

There have been several studies which have examined the utility of writing about positive experiences. Marlo and Wagner (1999) compared the effects of writing about trauma to writing about an extremely positive experience on health. The authors assigned 156 students to one of three groups who were instructed to write either about a traumatic event, an extremely happy experience or a neutral topic like their schedule for the day. There was a beneficial effect of writing on psychological health but not on physical health, and the effect was significantly more profound for those who wrote about a positive experience compared to those who wrote about a stressful event or a neutral topic. Not only did writing about positive experiences induce happier emotions immediately post-intervention but it also surpassed the longer-term effects of traumatic disclosure.

Adapting the original expressive disclosure paradigm to writing about positive emotions has also been found to produce beneficial effects for those diagnosed with breast cancer. Stanton et al. (2002) randomly allocated 60 breast cancer patients to either write about their thoughts and feelings surrounding their illness, positive emotions about their diagnosis or the facts about their breast cancer

experience. Those who wrote about their thoughts and feelings or the positive thoughts and emotions regarding their breast cancer attended fewer appointments for cancer-related illnesses over the three months post-intervention. Once again, this study suggests that disclosing positive emotions, even regarding what is normally considered a catastrophic experience can result in benefits to health.

Interestingly, writing about the perceived benefits of a previously experienced trauma has also been shown to be effective in improving health. King and Miner (2000) assigned 118 student participants to four conditions which required participants to write either about trauma, a trauma and the perceived benefits of the traumatic experience, just the positive aspects of a traumatic experience or a control tropic such as their plans for the following day. The results showed that at three months post-intervention, those who wrote about trauma or the perceived benefits from a traumatic experience made fewer health centre visits compared to the control group.

Similarly, Burton and King (2004) compared the effects of writing about an intensely positive experience (IPE) to writing about a control topic on the health of 90 students. Similar to the original study by Pennebaker and Beall (1986), this study also found that those who wrote about IPEs made fewer visits to the health centre during the three months post-intervention. However, unlike the original expressive writing paradigm, writing about IPEs were related to an increase in positive mood immediately post-intervention (Burton & King, 2004). Burton and King also replicated these findings in 2008 and demonstrated that writing about an IPE for just two minutes each day for two consecutive days can result in a decline in health complaints 4-6 weeks post-intervention (Burton & King, 2008).

However, it is not just writing about previously experienced positive events that can result in health benefits. Writing about the future with a positive outlook can also induce similar effects. King (2001a) assigned 81 student participants to write either about a traumatic life event, their best possible future self, a combination of writing about both trauma and best possible future self or a control topic for 20 minutes a day over four consecutive days. At five months post-intervention, those who wrote about trauma or their best possible future self or both reported a decrease in illness-related outcomes compared to those who wrote about an

unemotional topic, although this effect was stronger for those who wrote about trauma or their best possible future self alone (i.e., not in combination). However, writing about positive emotions was related to less negative emotion arousal immediately post-writing but produced similar health benefits to writing about trauma in the long-term. Similarly, a more recent study also found that writing about life goals (as opposed to a control writing task) resulted in a reduction in self-criticism measured two-weeks post-intervention (Troop et al., 2013).

However, it is not just taking part in positive expressive writing tasks that have been related to an improvement in health. Danner et al. (2001) examined the autobiographies of 180 Catholic nuns and found that there was a strong association between writing about positive emotions and a decrease in the risk of mortality. This demonstrates that even the act of writing about positive emotions without specific instructions can have beneficial effects. The use of writing about positive experiences has been adapted further to include an element of emotion regulation (Wing et al., 2006). Wing et al. (2006) assigned 175 adult participants into three conditions which required individuals to write about an IPE with an added emotion regulation component, an IPE or a control topic. The instruction for the IPE task used in this study was composed by Burton and King (2004) but the additional emotion regulation element encouraged individuals to re-experience the happy potent emotions related to the IPE to enhance well-being. The findings suggested that writing about a happy experience and being encouraged to think about and evaluate how to re-experience positive emotions can result in enhanced life satisfaction immediately post-intervention and at 12-week follow-up.

The studies which have examined the effects of positive writing have shown that this type of writing can lead to improved health, a decreased risk of mortality, an increase in life satisfaction and a reduction in self-critical thoughts and feelings without experiencing the immediate negative emotions triggered by writing about trauma. However, what are the mechanisms that drive the powerful effects of expressive writing?

Theoretical reasoning of expressive writing

Despite the multitude of studies that have examined the role of disclosure, the jury is still out with regards to determining the exact mechanisms behind this simple yet effective method that promotes health. Sloan and Marx (2004b) have provided a thorough evaluation of several theories that have been offered as an explanation of the processes that underlie expressive writing in its ability to facilitate health improvements. The effects of expressive writing have been explained using the notion of emotional and exposure processing, emotion inhibition, cognitive adaptation and emotion regulation.

The assumptions of exposure processing are rooted in learning theory which suggests that inhibition is a form of avoidance behaviour and disclosure provides exposure to the inhibited thoughts and feelings surrounding the trauma (Mowrer, 1960). Therefore, repeatedly exposing the individual to thoughts and feelings that were previously avoided can allow for the extinction of the aversive responses such as intrusive thoughts and avoidance-related behaviours (Sloan & Marx, 2004b). However, inconsistent findings have demonstrated that written disclosure does not always promote a reduction in inhibitory behaviours (which refers to the conscious or unconscious restraint of certain behaviours) or rumination (Kloss & Lisman, 2002; Sloan & Marx, 2004b).

Another theory that has received attention in the light of the expressive writing phenomenon is emotion inhibition. The idea that inhibition of emotion can result in ill-health dates back to the Freudian era (Breuer & Freud, 1957/1895) when inhibition of trauma-related emotions were related to psychopathology but a release (i.e., by disclosing these thoughts and feelings) could lead to a reduction in stress and an improvement in health. However, the theory of emotion inhibition failed to explain the finding that writing about previously disclosed traumas or even the thoughts and feelings associated with an imaginary traumatic experience can also have health benefits (Greenberg & Stone, 1992; Greenberg, Wortman & Stone, 1996).

Both exposure processing and the notion of emotion inhibition fail to provide a comprehensive account of how expressive writing functions to improve health. A third theory that has been proposed is cognitive adaptation which employ's Janoff-

Bulman's (1992) perspective of restoring assumptions about the self and the world. This view posits that a traumatic experience can alter three global assumptions, that we are decent and moral people, the world is a good place and that our experiences in the world are meaningful. Therefore, to manage or rectify the shift in these assumptions, one must process the thoughts, feelings and events surrounding the traumatic experience. Disclosure can aid this process as it enables the individual to compose a coherent narrative of the trauma (Pennebaker, 1997).

However, a single theory on its own has not been sufficient in explaining the phenomena of emotional disclosure. James Pennebaker (1985) proposed a psychosomatic theory of inhibition which combines the notion of emotion inhibition with an element cognitive processing. Initial thoughts by Pennebaker (1985) suggested that the act of inhibiting thoughts and feelings surrounding a traumatic experience require physiological effort. Therefore, the physiological energy used to store and conceal personal trauma can result in rumination about the event and in the accumulation of stress leading to a higher incidence of diseases (Pennebaker, 1985; Pennebaker & Beall, 1986). However, Pennebaker and colleagues (Pennebaker, 1985, 1997, 2004; Pennebaker & Beall, 1986; Pennebaker & Seagal, 1999) posit that the act of disclosing thoughts and feelings regarding previously experienced trauma works as a way of reorganising the event into a coherent narrative and confronting the emotions associated with the experience, which reduces the physiological exertion caused by inhibition. Evidence for the idea that disclosure can result in cognitive integration of the thoughts and feelings associated with trauma and lead to a reduction in stress comes from studies which have conducted text analysis of written disclosure essays (Pennebaker, 1997; Pennebaker, Mehl & Niederhoffer, 2003; Pennebaker & Seagal, 1999). Interestingly, these studies have revealed that in addition to composing a coherent story of the trauma, certain types of words used to express the emotionally potent thoughts and feelings are also the key to reaping the benefits of written disclosure.

The Linguistic Inquiry and Word Count (LIWC; Pennebaker, Booth & Francis, 2007) is a software program developed to use an internal dictionary to search for approximately 4,000 words and word stems in a given text. In addition to providing standard linguistic measures such as word count, number of pronouns and articles,

this innovative facility examines various language dimensions to group words into grammatical (e.g., numbers, pronouns), psychological (e.g., emotions, cognitive) and personal (e.g., achievement, death) categories. Emotion categories refer to positive and negative emotion words (i.e., *happy*, *sad*) and cognitive mechanism words group into categories which refer to causation (i.e., *because*, *depend*), insight (i.e., *know*, *explain*), discrepancy (i.e., *should*, *could*), inhibition (i.e., *block*, *stop*), tentativeness (i.e., *perhaps*, *might*) and certainty (i.e., *always*, *never*). These categories have been validated by independent judges and are reliable predictors of health and psychological outcomes (Pennebaker & King, 1999; Pennebaker, Mayne & Francis, 1997; Pennebaker et al., 2003).

Early work using the LIWC (Pennebaker et al., 2007) revealed that improved health is associated with the use of emotion (i.e., positive, negative) and cognitive mechanism words which are classified as being causal (i.e., *because*, *cause*) or insightful (i.e., *think*, *consider*). Combining a greater use of positive emotion words with a moderate number of negative emotion words and increasing use of cognitive words over the writing period has been suggested to provide the ideal prose for promoting health and well-being (Pennebaker, 1997; Pennebaker et al., 2003).

However, this account of expressive disclosure relies on recounting and reappraising relatively inhibited trauma whilst research has revealed that sharing trauma-related thoughts and feelings that have been previously disclosed or even the emotions surrounding an imaginary event can also have health benefits (Greenberg & Stone, 1992; Greenberg et al., 1996).

An emotion regulation view of expressive writing has been offered to account for the role that mastery plays in managing the emotions, physiological responses and behaviours associated with the trauma (Greenberg et al., 1996; Lepore, Greenberg, Bruno & Smyth, 2002; Lowe, 2006). This interpretation suggests that the therapeutic element of emotional arousal following expressive disclosure itself is the important component rather than the stimulus that produced the response (Greenberg et al., 1996; Lepore et al., 2002). Therefore, expressive disclosure functions as an affect regulation process as the act of confronting thoughts and feelings associated with trauma can improve perceptions of control and self-efficacy over the negative emotions experienced as a result of adversity

(Cameron & Jago, 2008). Expressive disclosure provides a vital opportunity to focus on the emotional reactions to trauma and develop a self-soothing and accepting approach towards one's thoughts and feelings (Greenberg et al., 1996; Lepore et al., 2002). The ability to empathise with one's trauma-associated emotions can aid the process of cognitive restructuring and relieve the stress-related responses such as intrusive thoughts and avoidance behaviours (Lepore et al., 2002; Lepore & Greenberg, 2002; Lowe, 2006). Expressive disclosure can enhance resilience to the effects of stress (Greenberg et al., 1996) and equip individuals with the ability to manage stress and traumatic experiences in a productive way (Lepore, 1997). Therefore, expressive disclosure may not reduce the perception of stress per se, but the impact that the experience of stress has on emotions and behaviour (Lepore, 1997).

The emotion regulation interpretation of the processes driving expressive writing can also be applied to the promising effects of writing about the positive aspects of a negative event, previously experienced positive events and writing about the future with an optimistic outlook. For example, writing about the perceived benefits of a traumatic experience provide the writer with the opportunity to enhance their ability to deal with the emotions by allowing them to focus on the positive aspects of the event without the need to relive the negative episode (King & Miner, 2000). The function of positive expressive writing guides people through a process of structuring their thoughts and feelings to gain a better understanding of the emotions associated with the experience. Similarly, writing about IPEs or about a best possible self in the future provide an opportunity to enhance self-regulatory processes which not only induces positive affect but also reinforces the ability to arrive at a better understanding of one's emotions and needs (Burton & King, 2004). King (2001) suggested that the act of writing about life goals enhances self-regulation by encouraging individuals to think about their future priorities and develop a more clear and optimistic outlook of where they are heading in life. Troop et al. (2013) supported these findings by demonstrating that those who benefitted (reported a reduction in self-criticism) used fewer discrepancy words (i.e., words that indicate doubt or the possibility of failure), which also demonstrates that positive expressive writing is beneficial for enhancing affect regulatory processes,

specifically self-criticism. Overall, these studies highlight the versatility of the expressive writing paradigm and ways of achieving beneficial outcomes as found with the original expressive disclosure paradigm but without stimulating negative feelings.

The use of expressive writing has also been modified to tap directly into affect regulatory processes. Leary, Tate, Adams, Allen and Hancock (2007) adapted the expressive writing paradigm to induce self-compassion in participants. The procedure that was used in this study involved writing about a negative event that resulted in feelings of humiliation or rejection and then answering three questions that were designed to elicit self-compassion. These questions included writing about how other people may also experience similar negative events, expressing self-kindness to themselves as if they were expressing it to a friend who may have experienced the event and to describe the emotions they experienced as a result of the event but writing about feelings in an objective fashion. These three questions were designed elicit the three self-compassion components described by Neff (2003a,b), common humanity, self-kindness and mindfulness, respectively. Leary et al. (2007) found that those who completed the self-compassion expressive task reported significantly less negative affect post-intervention compared to those who were assigned to disclose their deepest emotions about the negative event.

Imrie and Troop (2012) adapted this approach in a small group of hospice patients and found an enhancement in people's ability to self-soothe two weeks later. Those who wrote about stress from a self-compassionate perspective used more cognition words than those who wrote about stress only. This study provides support for therapies such as Compassion Focused Therapy (Gilbert, 2005, 2009) which have been developed to train individuals to reduce self-critical evaluations and instead, rejuvenate their attachment system in order to develop a self-soothing method of alleviating feelings of stress.

While the three studies in this thesis found no effect of stress on weight gain, weight regain or relapse from AN, evidence from previous research leaves the question open about the link between stress and weight. Therefore, as psychological factors in eating pathology and weight may include stress and the ability to regulate emotion it is proposed that an intervention which has been demonstrated to have a

positive impact on stress-related health outcomes via its effect on emotion regulation should have an effect on stress-related eating behaviour and DE. To test this hypothesis, an intervention study will explore the role of stress and affect systems (social rank and insecurity of attachment) and processes (self-criticism and self-reassurance) on problematic weight regulation and eating at times of stress. This study will take advantage of a naturally occurring stressor (students undertaking exams) and deliver an affect regulation intervention to *reduce* the impact of stress and measure its effect on weight change and eating. Additionally, the study will use the LIWC (Pennebaker et al., 2007) software program to examine if certain groups of words are predictive of changes in stress, eating and weight.

The LIWC (Pennebaker et al., 2007) has been used to examine language use across many methods of writing from traditional paper-and-pencil diary entries to modern day digital records of instant messaging, blogging and electronic mailing (Chung, 2009; Pennebaker et al., 2003; Slatcher & Pennebaker, 2006; Robinson & Serfaty, 2008). Chung (2009) found that successful dieters who use an online blogging community to track and share their dieting progress use more positive emotion words compared to those who are not as successful at achieving their weight loss goals. Interestingly, sharing personal events with other bloggers was more effective in achieving weight loss than keeping an online food diary (Chung, 2009). These findings highlight the benefits of writing and participating in online social communities to promote weight loss behaviours. However, online social communities have also been found to provide an arena for the promotion of ED behaviours, which are known as pro-anorexia and pro-bulimia (or pro-ana and pro-mia for short, respectively) websites. Text analyses of pro-anorexia sites have found that users of these communities make fewer self-references but use more positive emotion and fewer cognitive mechanism words (Lyons, Mehl & Pennebaker, 2006). Similarly, language use between pro-ED blogs differed to blogs about recovery from EDs (Wolf, Theis & Kordy, 2013). Wolf et al. (2013) revealed that pro-ED bloggers wrote in a close-minded fashion, were less emotionally expressive and featured more eating-related material compared to recovery bloggers. However, writing within a therapeutic environment can aid recovery from EDs (East et al., 2010; Schmidt, Bone, Hems, Lessem & Treasure, 2002; Robinson & Serfaty, 2008). Text

analysis of email records from ED patients to their therapists showed that there was a positive correlation between the number of words written and symptom improvement in those diagnosed with BN and BED (Robinson & Serfaty, 2008). These findings suggest that those who make use of a therapeutic writing task within a supported environment achieve better outcomes. However, expressive disclosure is not only beneficial for use in a clinical setting but has also been found to buffer the effects of stress on eating pathology in a student sample (Arigo & Smyth, 2012) and improve perceptions of body image in a sample of undergraduate students (Lafont, 2011).

Current study

In conclusion, research suggests that expressive writing is an effective method of regulating emotions at times of stress. Therefore, as previous research suggests that maybe stress can influence the ability to regulate bodyweight and eating behaviours, it is important to examine the effects of expressive writing on stress, bodyweight and eating behaviours during a stressful period (students undertaking exams). Based on the model presented in Chapter Three (*see Figure 3.2*), the current study has been designed to determine if writing about IPEs as a method of regulating emotions will have a positive influence on eating behaviours and weight in a student sample of women during a period of stress (unseen exams).

Therefore, based on previous research, the current study has been designed to answer the following broad and more specific empirical questions:

Broad research questions:

1) Will writing about IPEs influence changes in stress, bodyweight, eating behaviours and affect systems and processes during a stressful period?
2) Will specific types of language used by expressive writers determine the changes in stress, bodyweight, DE and affect regulatory systems and processes?

Specific research questions:

1) What are the immediate effects of expressive writing on mood and do expressive writers rate their entries as more meaningful and personal compared to those who wrote about a control topic?
2) Will the act of writing about IPEs influence changes in BMI, eating, stress, mood and affect regulatory systems (social rank and attachment) and processes (self-criticism/reassurance) over time?
3) What types of IPEs do the expressive writers write about?
4) Contingent on the results of the enduring effects of writing about IPEs, do those who improve write differently over the three days compared to those who do not improve?

Methodology

Design

The current study used a repeated measures design with the addition of an intervention involving a writing task. Each participant completed self-report measures of DE, mood, stress, social rank, insecurity of attachment, self-criticism/reassurance, caloric intake and physical activity at two time points. In addition to self-report measures, participants were also required to provide saliva samples and information on bodyweight as measured by BMI. Between these two time points, participants were also randomly assigned to the expressive writing condition or the control writing condition.

Participants

Out of the 225 female students who registered an interest in taking part in the study, 79 participants completed the T1 measures of the study (*see Figure 8.1*). The figure below provides details of the number of students who booked to attend the assessment day at T1 to those who completed all phases of the study. The final sample consisted of 27 expressive writers and 30 participants in the control group.

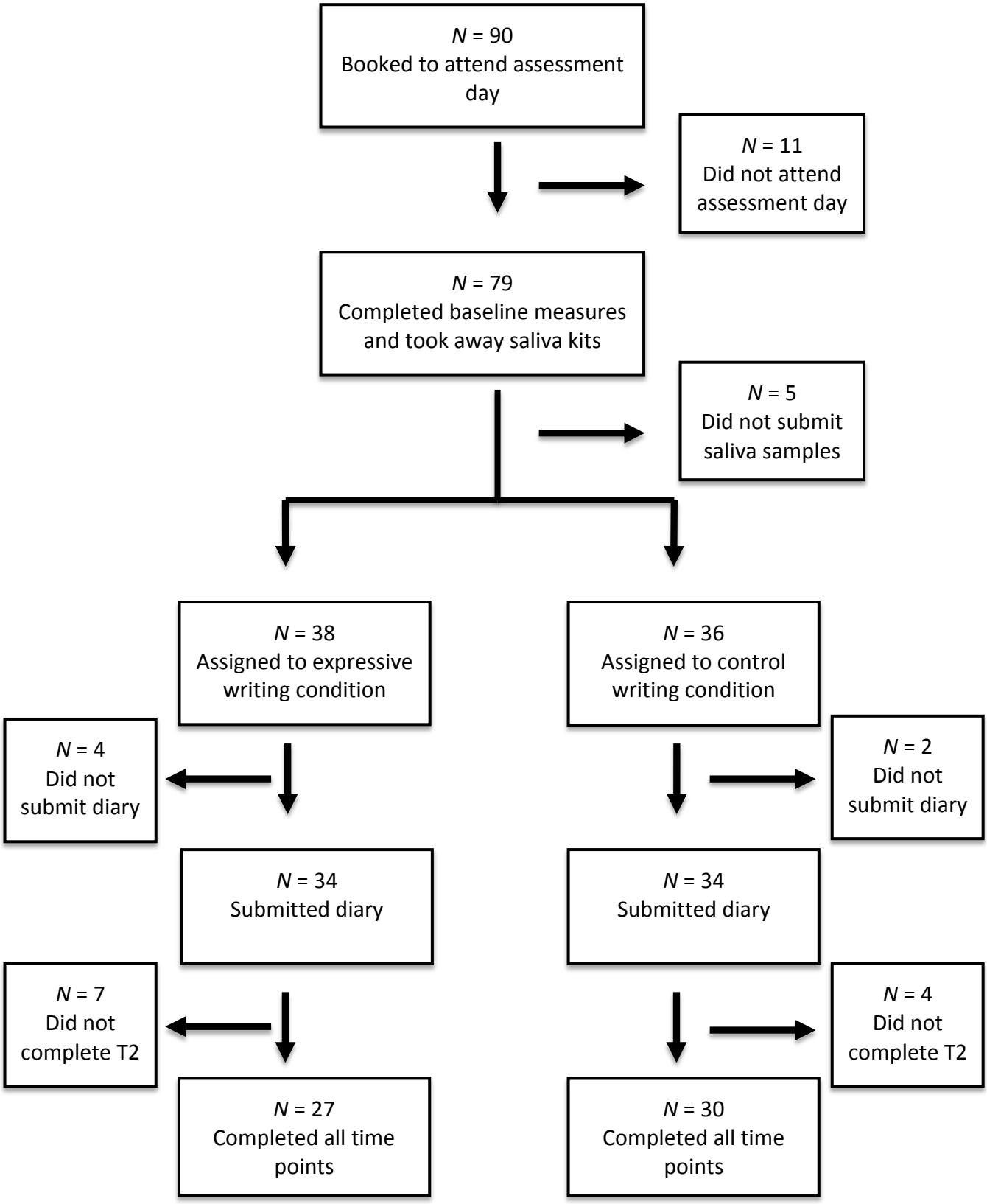


Figure 8.1: Flow chart for the number of participants who completed each phase of the study

The data from two expressive-writing participants were removed, one because she did not complete the diary for three consecutive days and forgot to label the T1 saliva samples with the times that she took them and another because she was pregnant by the time phase two was rolled out. A control participant was also removed as she did not follow the saliva collection instructions. Finally, one participant returned her saliva samples for both time points, but she did not complete the online survey for the second phase of the study so the final sample at T2 consisted of 27 in the expressive writing group and 30 in the control group. Completers (n = 57) and non-completers (n = 19) did not differ on any baseline variables (p-values between .06 and .88). Demographic information for control and expressive writing groups for T1 of the study are presented in *Table 8.1* which shows that majority of the sample were British and single. The two groups did not differ on any of the baseline demographic variables.

Table 8.1: Baseline demographic variables

Variable	Total (*N* = 57)	Expressive (*n* = 27)	Control (*n* = 30)	Significance
Age *M* (*SD*)	20.38 (3.92)	20.33 (4.18)	20.30 (2.71)	$t(55) = -.04$, $p = .97$
Ethnicity *n* (%)				
British	38 (66.7)	19 (33.3)	19 (33.3)	
Other European	1 (1.8)	1 (1.8)	0	
Indian	3 (5.3)	0	3 (5.3)	
Pakistani	1 (1.8)	0	1 (1.8)	
Caribbean	1 (1.8)	0	1 (1.8)	χ^2 (df=9) = 10.20 $p = .33$
African	4 (7.0)	1 (1.8)	3 (5.3)	
Mixed ethnicity	2 (3.6)	1 (1.8)	1 (1.8)	
Chinese	3 (5.3)	2 (3.5)	1 (1.8)	
Other	4 (7.0)	3 (5.3)	1 (1.8)	
Marital status *n* (%)				
Single	30 (52.6)	10 (17.5)	20 (35.1)	
Married/Cohabiting	8 (14.0)	4 (7.0)	4 (7.0)	χ^2 (df=4) = 7.34 $p = .12$
In a relationship	18 (31.6)	12 (21.1)	6 (10.5)	
Widowed	1 (1.8)	1 (1.8)	0	
Highest education *n* (%)				
A Levels	54 (94.7)	26 (45.6)	28 (49.1)	Fishers exact test*:*
Bachelors	3 (5.3)	1 (1.8)	2 (3.5)	$p = .62$

Measures and procedure

Following recruitment, which took place during undergraduate Psychology lectures at the University of Hertfordshire, participants were required to attend an assessment day. During the assessment day, participants provided basic demographic information such as age, gender, height and weight measurements (which were measured by the researcher in order to calculate BMI kg/m^2), ethnicity, marital status, highest education qualification and occupation. Participants also completed the following measures using the online BOS facility (*see Chapter Four for more information regarding the self-report measures*):

1. Caloric intake was measured using the FFQ (Cade & Margetts, 1988)
2. Disordered eating was assessed using the EDE-Q (Fairburn & Beglin, 1994)
3. Insecurity of attachment was measured using the VASQ (Bifulco et al., 2003)
4. Mood was measured using the SDHS (Joseph et al., 2004)
5. Self-criticism/reassurance was assessed using the FSCRS (Gilbert et al., 2004)
6. Social rank was measured using the SCRS (Allan & Gilbert, 1995)
7. Stress perceptions were assessed using the PSS-4 (Cohen & Williamson, 1988)

In addition to the above self-report measures, participants also provided saliva samples. Similar to the instructions which were given to participants in Study Two (*see Chapter Six*), participants were instructed to collect 4 saliva samples at 2-hour intervals during a normal day (i.e., 8am to 2pm). Participants also completed a food diary to record what they ate and drank on the day they collected their saliva samples. This was performed to ensure that participants did not eat or drink any fluids 30 minutes prior to taking a saliva sample. Participants were also provided with re-sealable specimen bags, a permanent marker pen to record the time the saliva sample was taken and return them to the researcher upon completion.

After returning their saliva samples, participants were randomly assigned to one of two conditions, expressive writing task or the control writing task. The randomisation process was based on when participants submitted their saliva samples, so the researcher recorded which and how many participants were being assigned to each condition to ensure that approximately equal number of participants were assigned to both tasks. However, if participants arrived in groups, they were all assigned to the same writing condition in an attempt to blind each group to the other condition (although participants were advised not to discuss the study with others until the study had been completed, there was no way of ensuring that this was adhered to). All participants were advised to complete the writing task and return it to the researcher within 6 weeks. Both groups of participants were provided with a notebook and asked either to write about an IPE or about a control topic for 15 minutes each day 3 consecutive days. Participants were given the following instructions:

1. Expressive writing condition (IPE; Burton & King, 2004):

"Think of the most wonderful experience in your life, happiest moments, ecstatic moments, moments of rapture, perhaps from being in love, or from listening to music, or suddenly "being hit" by a book or painting or from some great creative moment. Choose one such experience or moment. Try to imagine yourself at that moment, including all the feelings and emotions associated with the experience. Now write about the experience in as much detail as possible trying to include the feelings, thoughts, and emotions that were present at the time. Please try your best to re-experience the emotions involved. All of your writing will be completely confidential. Don't worry about spelling, sentence structure, or grammar. The only rule is that once you begin writing, continue to do so until your time is up."

On the second and third days of writing, these instructions also included the following sentence *"You may either write about the same experience as yesterday, or you may choose a new one."*

2. Control writing condition (Troop et al., 2013):

"In as much detail as possible, write a review of a film or book that you have recently seen or read. Please be as objective as possible, avoiding any emotions, personal opinions or beliefs when writing so that someone who reads your review can make up their own mind about whether to read the book or see the film. All of your writing will be completely confidential. Don't worry about spelling, sentence structure, or grammar. The only rule is that once you begin writing, continue to do so until your time is up."

On the second and third days of writing, these instructions also included the following sentence *"You may either write about the same film or book as yesterday, or you may choose a new one."*

Following each diary entry, all participants were also required to complete a 3-item scale measuring how personal they felt their diary entry was and how meaningful they felt the entry was. Both items were measured using a 7-point Likert scale (not very personal/not very meaningful (-3) to (+3) very personal/very meaningful). The last item was a mood check for which participants were required to rate how they felt on a 7-point scale (very sad (-3) to (+3) very happy). High scores on these measures indicated that the diary entry was meaningful and personal and that they felt very happy immediately post-intervention. The expressive writing diaries were typed up and the text was analysed using the LIWC (Pennebaker et al., 2007).

For the T2 of the study, participants were provided with salivettes and a food diary to take away with them over the Christmas break. Participants were advised that they would be contacted via text message to inform them of when they would need to take the saliva samples (which took place 8 weeks after baseline). Concurrently, the researcher also sent out an email directing participants to the link on BOS for completion of the online self-report measures. A return addressed envelope was also provided for participants to send the saliva samples and the food diary back to the researcher upon completion. Participants were then required to attend a final weigh-in and to collect their £15 Amazon voucher shortly after their exams had finished. All participants were provided with a debriefing sheet and an information leaflet about specialist services set up to assist individuals who suffer from mental health problems such as depression or EDs.

Statistical analysis

Several statistical analyses were conducted (*please refer to Chapter Four for more information regarding the calculation of AUC using cortisol levels*). Firstly, descriptive statistics for all measures at baseline and follow-up were computed. This was followed by a series of independent samples *t*-tests which examined the immediate effects of writing on mood over the three days and if those who wrote about IPE's differed in their ratings on how personal and meaningful they felt their diary entry was over the three days in comparison to those who wrote about a neutral topic.

The enduring effects of expressive writing were examined using repeated measures ANOVA. The between-subjects variable was the writing task condition that the participants were assigned to (control versus IPE) and the within-subjects variables were *time* (baseline [pre-intervention] versus follow-up) and *measure*. These tests were conducted to examine the effect of the expressive writing task on changes in the outcome variables. For outcome variables such as BMI, SDHS, SCRS, PSS-4, cortisol, caloric intake and the total scores for the EDE-Q and VASQ, repeated measures ANOVA with one between- (condition) and one within-subjects (time) were conducted. As the EDE-Q (dietary restraint, eating, weight and shape concerns), VASQ (insecurity and proximity-seeking) and FSCRS (inadequate-self, hated-self and reassured-self) have subscales, repeated measured ANOVA with one between- (condition) and two within-subjects (time and subscale) were computed. For any significant effects found, pairwise comparisons using the Bonferroni adjustment were computed to determine where these differences were present.

Finally, the LIWC (Pennebaker et al., 2007) program was used to assess the expressive writers' language use and the changes in the types of words used over the course of the writing task. The entries were assessed for general measures such as word count and linguistic dimensions such as prepositions and pronouns. The entries were also assessed for the presence of psychological processes such as positive and negative emotion words, cognitive processes such as causal reasoning and insight and social processes such as references to family and friends. Contingent on the results of the repeated measures ANOVAs conducted to assess the enduring effects of expressive writing on various outcome measures, where a significant improvement is found, language use between those who improved were compared with those who did not improve. In order to assess this, Univariate ANOVAs were conducted comparing improvers and non-improvers on the various LIWC measures.

Finally, where appropriate, effect sizes were examined using partial eta-squared (η^2) with values of .01, .06 and .16 representing small, medium and large effect sizes, respectively (Cohen, 1977)

Results

Sample characteristics

Means, standard deviations, skewness and kurtosis information for the variables of interest at T1 are presented in *Table 8.2*. All variables were normally distributed.

Table 8.2: Descriptive statistics of the variables at baseline as a function of condition

	Control (*n* = 30)				Expressive (*n* = 27)			
Variable	**Mean**	**SD**	**Skew**	**Kurtosis**	**Mean**	**SD**	**Skew**	**Kurtosis**
BMI	24.21	4.19	.88	.14	24.04	6.33	1.59	2.18
EDE-Q	1.54	1.39	1.26	.52	1.79	1.16	.24	-.75
Dietary restraint	1.05	1.55	1.73	1.92	1.50	1.30	.34	-1.33
Eating concerns	1.07	1.27	1.79	2.91	.97	1.01	2.25	6.04
Weight concerns	1.61	1.55	1.18	.38	2.08	1.54	.07	-1.08
Shape concerns	2.22	1.65	1.02	.37	2.42	1.44	.39	-.91
PSS-4	11.47	3.31	-.36	-.48	11.37	2.57	.26	.62
SDHS	18.67	3.85	-.98	.74	18.78	3.92	-1.12	1.74
SCRS	59.17	16.71	.40	.35	56.15	17.19	-.03	-.47
VASQ	38.33	7.74	.20	-.05	38.22	8.48	.60	-.41
Insecurity	19.23	4.86	.28	-.43	19.26	6.58	.39	-.54
Proximity-seeking	19.10	3.82	-.38	-.04	18.96	3.89	.87	1.46
Inadequate-self	16.57	6.48	.04	-.97	17.89	7.03	.14	-1.36
Reassured-self	27.03	7.18	-.37	-.31	26.78	6.82	-.28	-.67
Hated-self	6.20	3.02	1.64	1.75	5.81	2.77	1.43	.70
Caloric intake	1668.13	675.34	.45	-.61	1574.13	402.09	-.21	.45
Cortisol	44.50	20.09	1.13	1.15	40.23	18.19	.87	.34

Immediate effects of expressive writing

Question 1: What are the immediate effects of expressive writing on mood and do expressive writers rate their entries as more meaningful and personal compared to those who wrote about a control topic?

Independent samples *t*-tests were conducted to examine mean mood levels immediately after each writing session over the three days and how personal and meaningful the two groups felt that their diary entries were as averaged out across the three days. Expressive writers reported higher mood levels following each writing session and indicated that they felt that their diary entries were more personal and meaningful compared to the control group (*see Table 8.3*).

Table 8.3: Results of the Independent samples *t*-test comparing controls and expressive writers on mean (*SD*) scores for mood and how personal and meaningful their diary entries were immediately post-intervention over the three days

	Control (*n* = 30)	**Expressive (*n* = 27)**	**Significance *t*(55)**
Mood rating	.63 (.93)	1.62 (.76)	-4.38, p <.001
Personal rating	2.94 (1.15)	5.09 (1.19)	-6.89, p <.001
Meaningful rating	2.91 (1.14)	5.51 (.94)	-9.31, p < .001

Note. * p <.05; ** p <.001

Follow-up effects of expressive writing

Question 2: Will the act of writing about IPEs influence changes in BMI, eating, stress, mood and affect regulatory systems (social rank and attachment) and processes (self-criticism/reassurance) over time?

Repeated measures ANOVA were conducted to examine the effect of the expressive writing task on changes in the variables of interest (*see Table 8.4*). Results revealed that there was no effect of the intervention on changes in BMI, stress (as measured by either self-report or salivary cortisol), SCRS, VASQ and its subscales, FSCRS scales and overall caloric intake. There was a marginally significant effect of the intervention on mood with those who completed the expressive writing task reporting an increase in their mood (indicating more happiness).

Table 8.4: Results of the repeated measures ANOVA's comparing control and experimental groups on mean (*SD*) pre- and post-intervention scores for the variables of interest

	Control (*n* = 30)		**Expressive (n = 27)**		**Time**	**Group**	**Interaction**	
Variable	**T1**	**T2**	**T1**	**T2**	***F***	***F***	***F***	**η^2**
BMI	24.21 (4.19)	24.12 (4.16)	24.04 (6.33)	23.84 (6.37)	*F*(1, 55) = .68, *p* = .42	*F*(1, 55) = .03, *p* = .87	*F*(1, 55) = .11, *p* = .74	.002
EDE-Q (Total score)	1.54 (1.39)	1.53 (1.49)	1.79 (1.16)	1.61 (1.20)	*F*(1, 55) = .47, *p* = .50	*F*(1, 55) = .26, *p* = .62	*F*(1, 55) = .36, *p* = .55	.01
EDE-Q subscales								
Dietary restraint	1.05 (1.55)	1.04 (1.60)	1.50 (1.30)	.73 (.89)	*F*(1, 55) = .52, *p* = .48	*F*(3, 53) = 2.05, *p* = .12	*F*(3, 53) = 3.41, *p* = .02	.34
Eating concerns	1.07 (1.27)	1.17 (1.43)	.97 (1.01)	.86 (.81)				
Weight concerns	1.61 (1.55)	1.70 (1.60)	2.08 (1.54)	2.09 (1.79)				
Shape concerns	2.22 (1.65)	2.05 (1.68)	2.42 (1.44)	2.47 (1.85)				
PSS-4	11.47 (3.30)	11.47 (4.01)	11.37 (2.57)	12.30 (3.24)	*F*(1, 55) = 1.71, *p* = .20	*F*(1, 55) = .20, *p* = .65	*F*(1, 55) = 1.17, *p* = .20	.03
SDHS	18.67 (3.85)	17.97 (4.01)	18.78 (3.92)	19.22 (3.72)	*F*(1, 55) = .15, *p* = .70	*F*(1, 55) = .49, *p* = .49	*F*(1, 55) = 2.97, *p* = .09	.05
SCRS	59.17 (16.71)	59.57 (16.10)	56.15 (17.19)	59.44 (18.82)	*F*(1, 55) = 1.89, *p* = .18	*F*(1, 55) = .13, *p* = .72	*F*(1, 55) = 1.16, *p* = .29	.02
VASQ (Total score)	38.33 (7.74)	40.47 (8.22)	38.22 (8.48)	40.26 (8.60)	*F*(1, 55) = 10.10,	*F*(1, 55) = .01,	*F*(1, 55) = .01,	<.00 1

					p = .002	*p* = .94	*p* = .94	
VASQ subscales								
Insecurity	19.23 (4.86)	21.20 (5.74)	19.26 (6.58)	21.04 (6.75)	*F*(1, 55) = <.001, *p* = .99	*F*(1, 55) = .01, *p* = .94	*F*(1, 55) = .05, *p* = .82	.001
Proximity-seeking	19.10 (3.82)	19.27 (3.94)	18.96 (3.89)	19.22 (4.08)				
FSCRS scales								
Reassured -self	27.03 (7.18)	26.60 (7.15)	26.78 (6.82)	26.93 (6.76)	*F*(2, 54) = .99, *p* = .38	*F*(1, 55) = .07, *p* = .79	*F*(2, 54) = .44, *p* = .65	.02
Inadequate -self	16.57 (6.48)	16.63 (6.75)	17.89 (7.03)	17.15 (6.67)				
Hated-self	6.20 (3.02)	6.80 (3.79)	5.81 (2.77)	6.22 (2.76)				
Cortisol	44.50 (20.09)	36.74 (21.16)	40.23 (18.19)	37.88 (21.00)	*F*(1, 55) = 1.68, *p* = .20	*F*(1, 55) = .07, *p* = .79	*F*(1, 55) = .83, *p* = .37	.02
Caloric intake	1668.13 (675.34)	1584.24 (629.21)	1587.19 (402.09)	1334.25 (371.35)	*F*(1, 55) = 7.78, *p* = .01	*F*(1, 55) = 1.70, *p* = .20	*F*(1, 55) = 1.81, *p* = .18	.03

Although there was no significant effect of expressive writing on overall EDE-Q scores, the intervention was found to have an effect on the EDE-Q subscales. In addition, effect size analysis also found a large effect of intervention on EDE-Q subscale scores. However, post-hoc analysis using a Bonferroni adjustment revealed that this interaction was only present for dietary restraint (*p* = .01) and not the other EDE-Q subscales. *Figure 8.2* shows that there was a time by condition interaction for dietary restraint with expressive writers reducing their dietary restraint behaviours compared to controls.

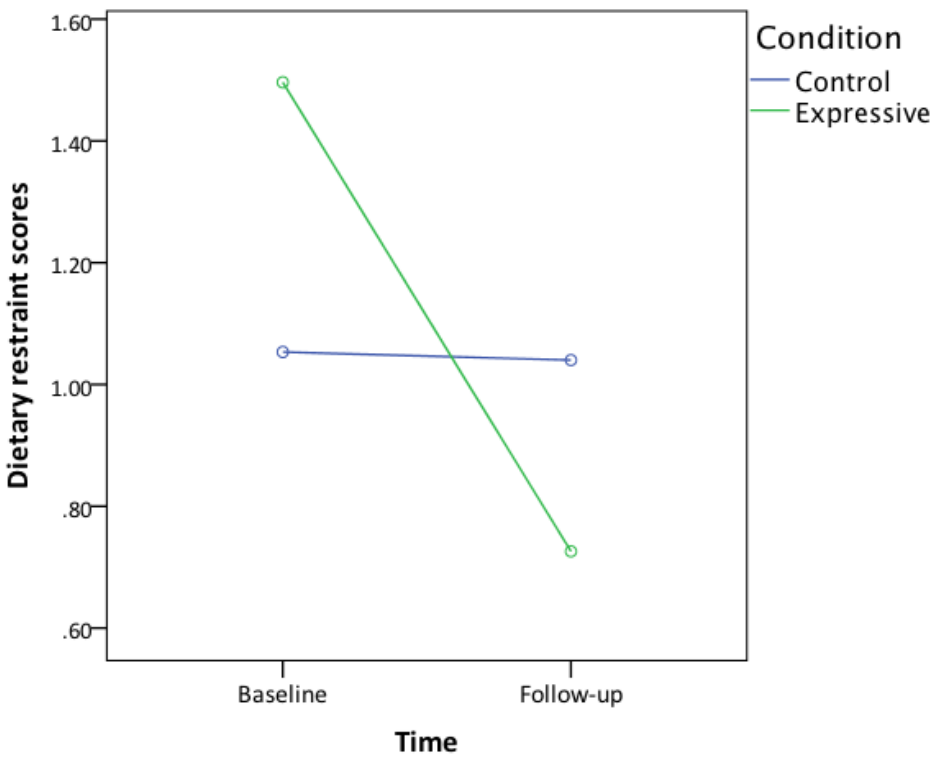


Figure 8.2: Time x Condition interaction for dietary restraint as a function of condition

Content and text analysis – Expressive writers

Question 3: What types of IPEs do the expressive writers write about?

Prior to conducting text analysis of the expressive writers diary entries, each entry was coded to determine the types of themes that expressive writers disclosed. Some of the themes that were derived from the diary entries included going on holiday (25.9%), interpersonal events (18.5%), finishing exams or receiving exam results for entry to University (11.1%), celebrations such as birthdays and Christmases (9.9%), attending a music concert or festival (7.4%), spending time with family and friends (6.2%), birth of a child including own child or sibling (4.9%), listening to a favourite song or watching a favourite film (3.7%), and other events such as getting married, starting a first job and taking part in a sky dive (12.4%).

<u>Question 4:</u> Contingent on the results of the enduring effects of writing about IPEs, do those who improve write differently over the three days compared to those who do not improve?

The LIWC (Pennebaker et al., 2001) was used to conduct text analysis of the expressive writers entries. As expressive writing was found to have a positive impact on dietary restraint levels, the expressive writers were grouped (using a median split) into those who improved their restraint (*n* = 18) and non-improvers (*n* = 9). *Table 8.5* shows that the two dietary restraint groups differed on the number of words they wrote with improvers writing significantly more words over the writing period compared to the non-improvers. There were no other significant differences and quite small effect sizes were found as shown by the partial eta-squared values (*see Table 8.5*).

Table 8.5: Results of the Univariate ANOVAs comparing mean (*SD*) language use between dietary restraint improvers and non-improvers

Variable	Non-improvers (*n* = 9)	Improvers (*n* = 18)	Significance *F*(1, 79)	η^2
Word count	270.27 (65.87)	336.48 (147.53)	5.83, *p* = .02	.07
Total pronouns	19.04 (3.19)	18.02 (2.84)	2.27, *p* = .14	.03
1st person	9.60 (3.33)	8.54 (3.56)	1.82, *p* = .18	.02
2nd person	.38 (1.07)	.42 (.95)	.03, *p* = .86	<.001
Past tense	8.27 (3.66)	9.08 (2.82)	1.25, *p* = .27	.02
Present tense	5.72 (3.70)	4.40 (2.81)	3.36, *p* = .07	.04
Future tense	.76 (.75)	.55 (.52)	2.29, *p* = .13	.03
Social processes	9.12 (4.53)	9.06 (3.83)	.004, *p* = .95	<.001
Family	.88 (1.39)	1.11 (1.45)	.52, *p* = .47	.01
Friends	.43 (.47)	.58 (.63)	1.40, *p* = .24	.02
Humans	.64 (.64)	.68 (.76)	.07, *p* = .79	.001
Affective processes	6.50 (2.06)	6.76 (2.18)	.29, *p* = .59	.004
Positive emotion	5.26 (2.07)	5.52 (2.33)	.27, *p* = .61	.003
Negative emotion	1.22 (.66)	1.19 (1.00)	.02, *p* = .89	<.001
Cognitive processes	19.83 (3.93)	19.17 (3.82)	.58 *p* = .45	.01
Insight	3.71 (2.03)	3.01 (1.65)	2.91, *p* = .09	.04
Causation	1.45 (.97)	1.48 (1.29)	.01, *p* = .92	<.001
Discrepancy	1.48 (.97)	1.45 (.86)	.02, *p* = .88	<.001
Tentative	2.22 (1.09)	1.79 (1.13)	2.94, *p* = .09	.04
Certainty	1.73 (1.20)	1.93 (.76)	.83, *p* = .36	.01
Inhibition	.52 (.56)	.48 (.42)	.08, *p* = .77	.001
Work	1.42 (1.58)	.86 (1.58)	2.48, *p* = .12	.03
Achievement	1.65 (1.00)	1.57 (1.51)	.06, *p* = .81	.001
Leisure	1.89 (1.81)	1.85 (1.68)	.01, *p* = .91	<.001
Home	.81 (.98)	.63 (.77)	.86, *p* = .36	.01
Money	.47 (.65)	.43 (.61)	.05, *p* = .83	.001
Religion	.20 (.37)	.38 (.80)	1.45, *p* = .23	.02
Death	.02 (.08)	.01 (.04)	.73, *p* = .40	.01

Discussion

In the present study the expressive writing paradigm (specifically writing about intensely positive experiences) was used to explore the role of stress and affect systems (social rank and insecurity of attachment) and processes (self-criticism and self-reassurance) on problematic weight regulation and eating behaviours at times of stress in a group of female students undertaking exams.

Findings

Positive expressive writing promoted marginal improvements in mood. More importantly, it led to significant reductions in disturbed eating behaviours, specifically dietary restraint. These findings are in line with previous research which has suggested that expressive writing is a beneficial method of alleviating low mood (Baikie & Wilhelm, 2005; Pennebaker, 2004; Pennebaker & Seagal, 1999; Smyth, 1998) and disturbed eating behaviours (East et al., 2010; Schmidt et al., 2002). It is possible to suggest that it was stress that resulted in a reduction of dietary restraint behaviours, for example, previous research has found that stress can result in hyperphagia which refers to eating more during stressful experiences (e.g., Greeno & Wing, 1994; Levine & Marcus, 1997; Oliver & Wardle, 1999; Stone & Brownell, 1994; Wardle et al., 2000). However, the control group who went through the same stressful experience did not report similar reductions. Therefore, these changes are most highly attributable to writing about IPEs.

Text analysis of improvers and non-improvers revealed that writing more words over the disclosure period is related to an improvement in restrictive eating behaviours. These results support findings by Robinson and Serfaty (2008) who found that making the most of an expressive writing task is related to an improvement in ED symptoms reported by those diagnosed with BN and BED. However, the current study did not find a significant effect of expressive writing on changes in stress and bodyweight. Previous research has also reported null findings when examining the effects of expressive writing on self-report perceptions of stress and physiological measures of stress (Frattaroli, 2006; Imrie & Troop, 2012; Troop et

al., 2013). Finally, it should also be noted that Burton and King (2004) and King (2001) argued that on the basis of previous studies, when there was a difference, there was always a benefit of expressive writing. Therefore, when these authors reported a significant positive effect of expressive writing on health centre visits, these results were based on one-tailed tests (p = .04). When one-tailed tests are performed for the current study, a significant effect of expressive writing on mood emerges though this was nonsignificant when a two-tailed test was used. Therefore, this suggests that potentially, there is an effect but it is much weaker than suggested by some meta-analyses (Frattarolli, 2006; Frisina et al., 2004; Harris, 2006; Smyth, 1998). Furthermore, given the number of tests carried out, it is difficult to argue that the effect is robust.

Strengths and limitations

Firstly, the current study has some important features, for example, the design included using a natural stressor. Students undertaking exams provided the optimal opportunity for exploring the effects of expressive writing on stress-related changes in bodyweight and eating behaviours. Secondly, although several of the psychological constructs were assessed using self-report measures, BMI at both time points were calculated using height and weight measurements taken by the author. In addition to collecting self-report perceptions of stress, the current study also included the collection of salivary samples to assess cortisol as a physiological measure of stress. However, since even the control group did not experience an increase in stress (either in the self-report or the cortisol measures) in the lead up to exams, it appears the exam period may not have been particularly stressful. This may be due to the recruitment of mainly first and second year students for whom exams may not appear as important as they are to final year students.

The current study also has several limitations that must be mentioned. The same limitations as the previous studies in this thesis that arise as a result of using self-report measures apply to the current study. Although the same arguments that mitigate against this being a limitation also apply (i.e., the use of stringent psychometric evaluation to validate and develop the FSCRS [Gilbert et al., 2004] and VASQ [Bifulco et al., 2003] which are used throughout this thesis).

Another limitation of the current study is the final sample size which was reduced due to an attrition rate of 27% between pre- and post-intervention. Therefore, future studies should recruit a larger sample of women. Additionally, the present study did not employ a clinical sample of ED patients so the findings relating to restrictive eating behaviours may not be generalisable to a clinical population.

Finally, although the current study has high ecological validity as participants completed the writing task at home, it also provides participants the optimal setting to misinterpret or ignore writing task instructions. However, studies have shown that disclosure can be successfully self-administered and home-based writing sessions produce larger effects (Frattaroli, 2006; van Middendorp et al., 2007).

Implications

The findings of the current study also have a number of important implications for theory, research and practice. While an affect regulation task led to changes in dietary restraint, it is not certain that this was due to changes in affect regulation, either in terms of systems (social rank and attachment) or processes (self-criticism and self-reassurance). This could be because the measures used in this study are not sensitive to the change caused by expressive writing or due to the type of expressive writing task that was used in this study. Troop et al. (2013) also measured self-criticism/reassurance using the FSCRS (Gilbert et al., 2004) in their expressive writing study and found that writing about life goals resulted in a reduction in self-criticism. Therefore, writing about life goals may be a more effective way of influencing a change in affect regulation as it may instill confidence in the individual to achieve goals which may seem out of their reach.

As the current study did not find a direct effect of expressive writing on perceptions of stress or physiological measures of stress, this suggests that this type of expressive writing, if it works at all, works by regulating emotions (Greenberg et al., 1996; Lepore et al., 2002). Therefore, it can be suggested that expressive writers in this sample were better equipped to manage the stressful experience (Lepore, 1997) of the exam period. Performing expressive writing provided the opportunity to write about a life event that had given the individual special pleasure at the time. Therefore, recreating those emotions not only induced positive emotions but may

also have reinforced the ability to develop a better understanding of one's emotions (Burton & King, 2004).

The findings of the current study also have implications for research. Failing to find an effect of expressive writing on all of the psychological outcomes corresponds with two recent meta-analyses which reviewed a combined total of 91 studies but concluded that disclosure in the form of expressive writing does not produce momentous positive health effects on most physical and psychological outcomes (Meads & Nouwen, 2005; Mogk, Otte, Reinhold-Hurley & Kröner-Herwig, 2006). However, expressive disclosure may have also had an impact on other outcomes that were not measured in the current study, such as health centre visits, immune function and exam grade results as previously shown (Pennebaker, 1997, 2004; Smyth, 1998).

Furthermore, two meta-analyses have revealed that expressive writing is more effective on physical health outcomes rather than on psychological well-being (Frattaroli, 2006; Frisina et al., 2004). Frattaroli (2006) demonstrated that those who reported high stress levels experienced greater overall health benefits but not psychological well-being. Despite finding that stress moderates the relationship between expressive writing and health outcomes, this meta-analysis also indicated that expressive writing may not have an effect on stress-related measures such as cortisol and disturbed eating behaviours suggesting that it is not a beneficial technique for alleviating stress directly or improving eating-related problems (Frattaroli, 2006). Our findings suggest that expressive writing is not a therapeutic method of buffering the effects of stress on weight change but it does appear to have a direct effect on eating pathology, particularly in reducing dietary restraint behaviours.

However, other reasons why the current study did not find a significant effect of expressive disclosure on stress-related outcomes as previously suggested (Kloss & Lisman, 2002; Lowe, 2006) may be because the majority of the participants who took part in this study were first and second year undergraduate students. Therefore, this sample of students may not have been particularly stressed about their course and exams in order for the intervention to have a beneficial effect on their ability to manage their stress. Also, the current study only recruited female participants

whereas, compared to women, men have been found to show greater benefits from expressive writing (Smyth, 1998).

Finally, it is possible to put the lack of expressive disclosure benefits in this study down to modern day society. The Western world has changed greatly since the introduction of the expressive writing paradigm, nearly three decades ago. The 21st century saw the birth of virtual social networking. On a daily basis, individuals disclose and share many aspects of their life for all to view and comment within an online domain. Therefore, as suggested by Smyth and Pennebaker (2008), the question is whether a time has come when we can discuss our emotions freely without the fear of stigma or judgement rather than inhibiting our responses? If so, the traditional writing paradigm may no longer be as effective as it was in a culture where expressing thoughts and feelings about stress and emotions was not a common occurrence. However, as no longitudinal research has explored time trends in the effects of the expressive writing paradigm, currently this point is rather speculative. Therefore, this is a question that should be explored by future research or by meta-analysis looking at year of publication as a factor contributing to the beneficial effects of expressive writing.

Although the current study did not employ a clinical sample, the findings still have some implications for practice. An important finding of the present study is that writing about positive experiences can alleviate restrictive eating behaviours at times of stress. Dietary restraint often leads to greater likelihood of overeating, so to find that expressive writing reduces dietary restraint has important preventive implications. These findings support previous recommendations that expressive disclosure within a therapeutic environment could be an effective adjunct to psychological and medical therapy for treating pathological eating patterns (Robinson & Serfaty, 2008; Schmidt et al., 2002) and as a self-help tool rather than as a therapeutic method on its own (Baikie & Wilhelm, 2005; Lowe, 2006; Marlo & Wagner, 1999; Wright & Chung, 2001).

Conclusion

The current study presents evidence that expressive disclosure in the form of positive writing is an effective method of reducing dietary restraint at times of

stress. The final chapter that follows will provide a general discussion of the research findings that have been presented in this thesis and how they fit together to form a bigger picture of the role of stress and affect regulation on problematic weight regulation.

Chapter 9: General Discussion

Discussion overview

The empirical chapters presented in this thesis have included a comprehensive discussion of each of the studies conducted as part of this research programme. Therefore, the aim of this chapter is to provide a final overview of all of the research findings, a summary of the strengths and limitations of the studies conducted during this programme, the implications that these findings have for theory, research and practice and the next step that future researchers in this area should take. This final chapter will be divided into four sections. The first section will provide a summary of the empirical findings in light of the objectives set out in Chapter Three. The second section will present the strengths and limitations of the studies that were conducted. The third section will highlight the overall implications that the research findings have for theory, research and practice. The final section will present recommendations for the next stage of research studies in this field.

Findings

This section of the general discussion will provide a summary of the empirical findings derived from each study conducted as part of this research programme.

Study One

The role of mood and affect systems on the trajectories of BMI, disordered eating and stress (Chapter Four)

The overarching question that was set for this study was how do longitudinal changes in stress, bodyweight and DE behaviours impact on each other and do affect regulatory systems (social rank and attachment) and processes (self-criticism and self-reassurance) contribute to these changes? The findings of this study suggest that whilst there appear to be cross-sectional associations between stress, DE and bodyweight, the longitudinal findings are less consistent. The cross-sectional findings

showed that, as expected, high levels of stress predict higher BMI and greater eating pathology. However, the longitudinal findings revealed that an increase in stress predicted a *decrease* in bodyweight over time. Although the direction of these findings was not as expected, there are some plausible explanations for this relationship. Firstly, previous studies (e.g., Stone and Brownell, 1994) have revealed that whilst some people eat more at times of difficulty (referred to as hyperphagia), others experience a loss of appetite (known as hypophagia). Therefore, it is possible to suggest that when a study examines the role of stress on changes in weight and eating, the reason for inconclusive findings from the studies reviewed in Chapter Two is because some people report eating more and gaining weight whilst others report eating less and hence experiencing weight loss at times of difficulty. To complicate matters even more, a recent study showed that those who eat more at times of adversity eat less when they are content but those who experience hypophagia actually eat *more* following a positive experience (Sproesser, Schupp & Renner, 2013). Therefore, without taking between- and within-person variability into account, it would be difficult to conclusively determine the exact relationship between stress and changes in bodyweight.

Secondly, although Chapter Two suggested that stress can disrupt healthy eating behaviours and even contribute to the development of EDs, Study One found that whilst high stress levels are predictive of greater DE behaviours cross-sectionally, this relationship is not present when studied longitudinally. These findings add to the literature as only one study has previously examined the effects of perceived stress on eating pathology over a long period of time (Striegel-Moore et al., 2007). However, life events research in EDs (specifically AN) show this also (as described in Chapter Two).

The secondary aim for this study was to examine how affect systems and affect regulation processes may contribute to the changes in stress, bodyweight and DE. Interestingly, the findings revealed that favourable social comparison is predictive of low BMI at baseline and greater decreases in stress levels over time. This result indicates that perceiving oneself to be similar or higher in social comparison in relation to others is related to healthy bodyweight but, importantly, it is important for the regulation of stress. However, thoughts and feelings of hostility

(in the form of hated-self from the FSCRS [Gilbert et al., 2004]) towards oneself predicted high BMI, greater levels of disturbed eating behaviours and less reduction in stress levels over time. Similarly, feelings of inadequacy (as measured by the inadequate-self component of self-criticism from the FSCRS [Gilbert et al., 2004]) predicted greater DE behaviours and high stress levels. In contrast, acting kindly towards oneself at times of failure was predictive of lower stress levels at baseline indicating that adopting a self-reassuring manner can help in regulating stress perceptions at times of adversity. The other biosocial goal that drives human behaviour is attachment which is also an important predictor of stress and problematic weight regulation. In contrast to previous research which has established a firm relationship between insecurity of attachment and eating pathology (O'Kearney, 1996; O'Shaughnessy & Dallos, 2009; Ward et al., 2000; Zachrisson & Skårderud, 2010), the current study found that insecure attachment at baseline was related to decreases in DE over time. However, in line with previous research, higher levels of proximity-seeking patterns of attachment were related to greater stress levels at baseline (Ditzen et al., 2008; Kidd et al., 2011).

Study Two

Predictors of weight regain (Chapter five)

The second study was designed to examine the role of stress and affect regulation on weight change and eating pathology in women who have previously lost weight and at baseline were at least 3 BMI points lighter than their highest adult weight. The findings of this study were in line with previous research which has found that those who have lost weight commonly regain the weight they have lost (Foster & Kendall, 1994) as this group of women, on average, experienced weight gain over the 7-month period. However, this could be age-related weight gain rather than weight *regain*, per se. Although Chapter Two suggested that stress may play an important role in regaining weight that is lost, Study Two did not find stress to be predictive of weight gain in this sample of women. However, affect processes were associated with changes in DE. The current study revealed that those who reported a stable or a reduction in their DE behaviours also reported an increase in their self-reassurance scores. Although causality remains contentious, it can be argued that

having the ability to self-reassure at times of difficulty can help individuals to focus on their positive characteristics rather than the features that they are unhappy about resulting in a decrease in unhealthy eating habits.

Although social rank and adult attachment styles did not differentiate between those who gained weight and those who maintained a stable bodyweight, these affect regulatory systems were important for stress regulation. Those who reported an increase in stress levels over the study period also reported an increasingly unfavourable social comparison and an increase in attachment insecurity. These results are in line with the findings of Study One which showed that whilst greater levels of insecurity of attachment was predictive of higher stress perceptions at baseline, favourable social comparison predicted a decrease in stress levels over time. Therefore, both Studies One and Two provide further support for Gilbert's (2005) positive affect systems which encourage the activation and immobilization of the threat-defence system at times of stress. At times of adversity, a securely developed attachment system is activated and the ability to self-soothe immobilizes the threat-defence system. In contrast, the social rank system can activate the threat-defence system when efforts to be valued by others are rendered ineffective and a perception of low social status ensues. However, causal direction is, again, not proven. Furthermore, these results were not obtained using the cortisol measure of stress.

Study Three

The role of life events and difficulties in predicting relapse and recovery from AN (Chapter six)

The third study was designed to determine the role of stress (as measured in the form of life events and difficulties) and affect regulation in relapse and recovery from AN. As this study did not find anyone who relapsed following treatment, these factors were examined in relation to remission and symptom maintenance in AN. Adopting the definition of recovery used by Fairburn et al. (2009) and Schmidt et al. (2013), six (43%) of the patients who took part in the treatment trial at the Maudsley Hospital and in this study were found to be in remission at the end of treatment and 1-year follow-up post-treatment, whilst eight patients maintained their ED

symptoms. These findings are comparable to the recovery rate of 47% in EDs reported by Steinhausen (2002).

Although Chapter Two proposed the idea that stressful life events can contribute to the development, maintenance and even result in relapse following remission from AN, the findings of the current study do not provide support for this. Indeed, if anything, more life events and difficulties were reported by those in remission or full recovery than those who were still ill. Nevertheless, a large proportion of the life events reported by both patients and recovered individuals were due to interpersonal problems, which is in line with previous research (Rojo et al., 2006; Schmidt et al., 1997; Troop & Treasure, 1997). Although the current study was unable to confirm the relationship between stressful life events and AN symptoms, the results did suggest that it may be affect regulatory systems that are more important for recovery in AN.

Similar to Studies One and Two, this study showed that those who had been recovered from AN for at least two years reported more favourable social comparison compared to those who had recently undergone treatment for AN and that this was in the normative range for healthy, non-clinical samples. Both patients who maintained their ED symptoms and patients who were in recovery at the end of treatment reported an improvement in their social comparison perceptions between pre-treatment and end of treatment. However, when the these two patient groups were compared to those who had been recovered for at least 2 years, those who maintained their ED symptoms reported significantly more unfavourable social comparison compared to those who were recovered whereas those who were in remission did not differ from either the recovered group or those who were still ill. Insecurity of attachment in adulthood also differed between the two patient groups. Those who maintained their ED symptoms reported an increase in their feelings of relationship insecurity between the end of treatment and 1-year follow-up, whereas those who were in remission reported a reduction in feelings of insecurity in their relationships during the post-treatment year. These findings suggest that affect regulatory systems have important implications for problematic weight regulation, specifically AN.

Study Four

The impact of an emotionally expressive writing intervention on stress, bodyweight, and eating pathology (Chapter seven)

The final empirical chapter in this thesis was developed to examine if an intervention addressing the regulation of emotions can alleviate the effects of stress on changes in bodyweight and eating behaviours during a stressful period. This experimental study examined the effects of expressive writing on stress, bodyweight, eating behaviours and affect regulation during a stressful period in a group of female students who were coming up to an exam period. This study found that writing about positive experiences is effective in reducing dietary restraint during a stressful period. Those who reported an improvement in dietary restraint wrote more words during the expressive writing condition compared to those who did not experience a reduction in dietary restraint. This suggests that those who benefited were more engaged in the process.

However, the current study did not find an effect of expressive writing on changes in stress, bodyweight and the measures of affect regulation used here. When comparing these findings to other studies which have used a positive expressive writing task rather than the original paradigm of disclosing thoughts and feelings associated with negative life events, the findings of this study do not seem so different. For example, King and colleagues (Burton & King, 2004; King, 2001) tested the prediction that when expressive writing has an impact on outcome measures, it always has a beneficial influence. These authors did not consider that expressive writing may not be effective in alleviating psychological and physical outcomes. Based on their one-tailed prediction, King and colleagues (Burton & King, 2004; King, 2001) reported that expressive writers made fewer visits to the health centre compared to those who wrote about control topics. However, these findings would be nonsignificant if reported using two-tailed tests. Therefore, although extensive research has demonstrated that expressive writing is beneficial for health, these effects may not be as strong as previously thought (Frattarolli, 2006; Frisina et al., 2004; Harris, 2006; Smyth, 1998). In addition, no mediators were identified, for example in terms of changes in mood or processes identified through text analysis.

Overall findings

Bringing together all of the findings from the four empirical studies revealed that the relationship between stress, affect regulation, bodyweight and eating behaviours differed when examined cross-sectionally and over time.

Cross-sectional findings from Study One showed that:

- High stress levels are associated with higher levels of BMI, greater DE behaviours and lower self-reassurance
- Favourable social comparison is related to low BMI
- More proximity-seeking behaviours are related to higher stress levels
- Higher levels of self-criticism are related to higher BMI and greater levels of stress and DE

These findings are consistent with previous research, but were not replicated when examined over time.

Longitudinal/retrospective findings show that:

- Increases in stress are either unrelated to changes in bodyweight (Study Two and Four) or related to decreases in bodyweight (Study One) or even a greater likelihood of remission or recovery from AN (Study Three)
- Increases in stress related to developing a more unfavourable social comparison and a more insecure style of attachment (Study Two)
- Favourable social comparison is related to greater decreases in stress (Study One) and remission or recovery from AN (Study Three)
- A reduction in attachment insecurity associated with remission from AN (Study Three)
- Higher levels of self-criticism (as measured by the hated-self component of the FSCRS [Gilbert et al., 2004]) are predictive of less reductions in stress (Study One)
- Increases in self-reassurance related to stable or decreasing eating pathology (Study Two)

- Expressive writing as a method of enhancing affect regulation is unrelated to changes in stress or bodyweight but predictive of reductions in dysfunctional eating behaviours, specifically dietary restraint (Study Four)

Altogether these findings suggest that positive affect systems (social rank and attachment) and affect regulation processes (self-criticism and self-reassurance) have important implications for regulating stress and eating behaviours over and above the impact of stress on changes in bodyweight and eating behaviours. Although Chapter Two posited that stress plays an important role in problematic weight regulation, specifically weight regain following successful weight loss and development/maintenance of anorexic symptoms, the current study did not find support for this. Although concurrent findings suggest that stress is related to bodyweight and eating pathology in a community-based sample, when stress is examined in relation to weight regain following weight loss and recovery and symptom maintenance in AN, stress levels, as measured by self-report, cortisol levels and stressful life changes, do not appear to be as important as affect regulation. In conclusion, despite very obvious physical differences between people who regain weight following a period of weight loss and those with a history or a current diagnosis of AN, it seems that the impact of affect regulation, especially social comparison, is a clear similarity between them.

The current programme of research set out to test different aspects of the following model as presented in Chapter Three (*see Figure 9.1*). Studies One and Two (*see Chapters Five and Six, respectively*) examined the relationship between stress, eating and weight, the role of affect regulatory systems and processes on stress and also, how these systems and processes may also contribute to eating and weight in a community-based sample of women with varying bodyweights and in a community-based sample of women who are at least 3kg/m^2 lighter than their heaviest adult weight, respectively. Study Three (*see Chapter Seven*) examined the role of stressful life experiences on eating behaviours and weight and the role that affect regulatory systems and processes play in recovery and relapse in a sample of women who have recovered and women who have recently undergone treatment for AN. Finally Study Four (*see Chapter Eight*) was designed to determine if writing about IPEs as a

method of regulating emotions at times of stress would have a positive influence on eating behaviours and weight in a student sample of women who undergone an exam period.

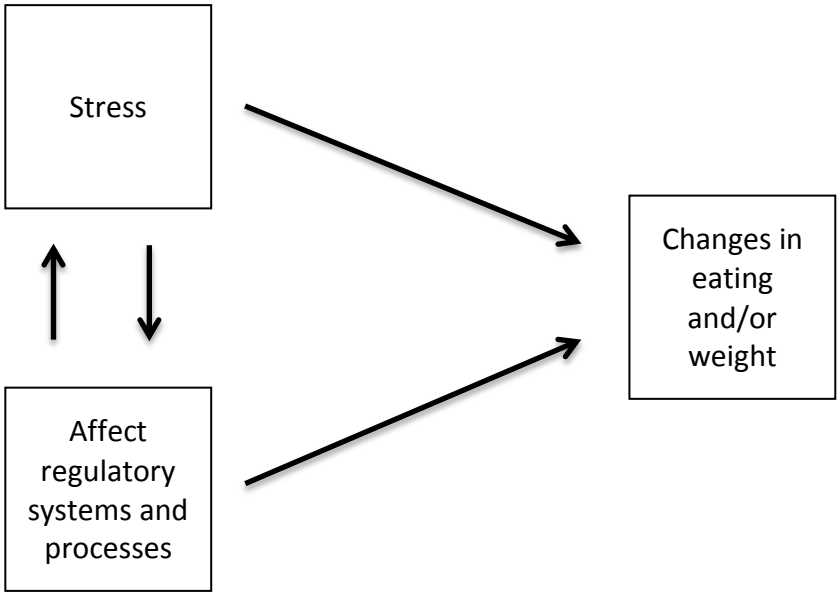


Figure 9.1: Proposed model (*presented in Chapter Three; Figure 3.2*)

However, based on the overall findings as presented in this chapter, it is suggested that stress may not be as important in influencing changes in eating and weight as once thought. Rather, it is the the way in which we regulate our emotions at times of stress that has important implications for how we regulate the way we eat and changes to the weight process. Therefore, the figure below (*see Figure 9.2*) displays the amended model based on the empirical findings.

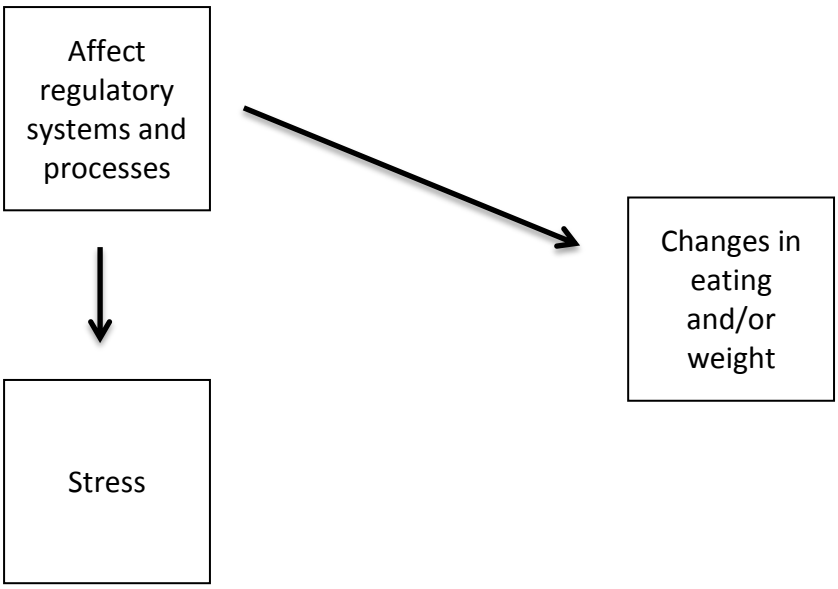


Figure 9.2: Adaptation to the original proposed model based on overall findings

Strengths and limitations

The strengths and limitations of each of the studies have been discussed as part of the individual discussion sections. However, this section will discuss the main strengths and limitations of the studies designed to answer each of the research questions.

The current research programme has some noteworthy merits. For example, the first study presented in this thesis had a longitudinal design which included a large sample of participants. This design of the study enabled the use of sophisticated statistical modeling to examine how changes in stress, BMI and DE can influence each other and the predictors of these changes. In addition, the current research programme examined predictors of problematic weight regulation in two very different groups of women. Firstly by recruiting a subsample of women who had lost weight since their past highest adult weight and, secondly, a sample of clinical patients who had recently completed treatment for AN and women who had fully recovered. Therefore, despite very obvious differences between these groups of women, the current study demonstrated that affect regulation is an important mechanism in how these women regulate stressful experiences and DE behaviours.

These findings would not have been achieved if these two very different groups of women had not been studied as part of one research agenda, which is one of the ways that this thesis makes an original contribution.

Also, unlike other studies that have examined the role of stress on problematic weight regulation such as weight regain and DE, the current research programme employed three different methods of assessing stress. Out of the four studies, three included a self-report measure whilst two of these supplemented the self-report measure with a physiological measure of stress and one study examined stress using a semi-structured interview to measure life events and difficulties. Therefore, although these empirical studies included self-report measures, where possible, other methods of assessing psychological constructs were included.

However, the current research also has several limitations that must be addressed. A primary limitation of the current research is the lack of male samples. Although this thesis argued that the factors associated with problematic weight regulation may be more salient for women, in this day and age when obesity and DE behaviours are both on the rise (e.g., Craig & Hirani, 2010; Darby et al., 2009), it would be important to determine if affect regulation is an important process in managing stress and weight in men as it is in women.

Secondly, the majority of the studies conducted as part of this research programme measured various psychological constructs using self-report measures. However, as part of the research programme, advanced statistical methods were used to further develop and validate two of the measures, FSCRS (Gilbert et al., 2004) and VASQ (Bifulco et al., 2003) that have not undergone extensive evaluation previously. Therefore, the findings presented in this thesis are based on newly developed and validated versions of these two instruments. However, DE was assessed using the interviewer-based EDE (Fairburn et al., 2008) in one study whilst the other three studies employed the self-report version, the EDE-Q (Fairburn & Beglin, 1994). Although the interview-based assessment would be a more reliable and valid option, the EDE-Q has been shown to have high correlations with the EDE (Pretorius et al., 2009; Wilfley et al., 1997).

Another limitation of the methods adopted in these studies is the assessment of bodyweight and height. Due to the nature of the studies presented in this thesis,

actual bodyweight and height could not always be measured, therefore, we had to rely on participants' self-report measurements. However, where it was feasible to collect actual measurements, these were used instead of self-report measures. Nevertheless, Rowland (1990) has suggested that there is a high intercorrelation (r = .95) between self-reported and actual measures of height and bodyweight.

Another main limitation of the research presented in this thesis is that despite maintaining a certain level of consistency in the instruments that were used throughout the four empirical studies, there were some differences between these studies. For example, Study Three attempted to examine self-criticism and self-reassurance in response to life events and difficulties but this method resulted in a floor effect. When the interviewer identified a severe event or difficulty during the LEDS (Brown & Harris, 1978), additional questions regarding how the individual felt about the way she had responded were asked. These questions were developed for the study and were designed to gain an understanding if those with a current diagnosis of AN were more self-critical/less self-reassuring regarding the way they responded to severe life events and difficulties compared to those who were in remission or had been recovered for at least two years. However, when the interviewer used these questions, participants stated that they had not felt very self-critical or self-reassuring about the way they responded to the stressful event. Therefore, due to the lack of data generated from these questions, affect regulation processes were not examined in relation to AN. However, it must also be highlighted that the assessment of self-criticism and self-reassurance in Study Three was in relation to specific experiences rather than a general measure of these constructs as examined in the other studies. In retrospect, it might have been informative to examine if the currently ill patients reported greater levels of self-critical thoughts and/or less reassuring thoughts and feelings compared to those who had recovered or who were found to be in remission. This was not possible here however as the patients in the clinical trial, from which participants were recruited, already completed a large battery of measures for the clinical trial.

Implications

Notwithstanding the above limitations, the current research findings have important implications for theory, research and practice.

Firstly, from a theoretical perspective, the current study has supplemented the body of literature that has examined the predictors of stress and problematic weight regulation. Affect regulation systems (social rank and attachment) and affect regulation processes (self-criticism and self-reassurance) appear to play a significant role in managing stress and DE behaviours. Furthermore, their role in problematic weight regulation, especially disturbed eating patterns, seems more important than does the role of stress/life events.

There are also important implications for research as the current study found that a self-report measure of stress (PSS-4; Cohen & Williamson, 1988) and a physiological measure of stress (cortisol) were uncorrelated. Recent research has suggested that there is a discord between self-report and physiological measures of stress. For example, Faresjö, Theodorsson, Charziarzenis, Sapouna, Claesson, Koppner et al. (2013) found that a sample of individuals had low cortisol levels at the same time as scoring high on the PSS (Cohen et al., 1983). Similarly, O'Brien, Tronick and Moore (2012) demonstrated that whilst individual self-report measures of stress like the PSS (Cohen & Williamson, 1988) did not correlate with a biological measure of stress, a composite measure combining several self-report measures of stress did. Therefore, these findings emphasise the importance of developing a consistent approach to the measurement of stress in order to ensure that findings are not due to measurement error. A uniform approach to measuring stress, especially in relation to bodyweight, will iron out the inconsistencies that are still present in this field. Therefore, based on these findings, it is recommended that a battery of self-report measures that have been developed to examine stress should be used to create a global assessment of stress. The battery of measures should include a general perception of stress such as the PSS (Cohen et al., 1983; Cohen Williamson, 1988) and measures that target specific stressors such as those experienced as a result of occupation (i.e., Job Content Questionnaire [JCQ]; Karasek, Brisson,

Kawakami, Houtman, Bongers & Amick, 1998) and those that are specific to gender (i.e., FGRS; Gillespie & Eider, 1992). In addition, the type of stress measure that is used also depends on the design of the study. For example, if a retrospective design were used to examine the impact of life events on illness onset or relapse following recovery, then the LEDS (Brown & Harris, 1978) would be a suitable tool to use to elicit information regarding stressful experiences prior to onset or relapse. However, if a prospective design were required to examine the changes in stress on various outcomes then physiological measures such as salivary cortisol and/or a battery of measures as recommended above should be used. Interestingly, although the current study collected saliva samples over the course of a day, awakening cortisol response (ACR) which requires the collection of one saliva sample within 35-45 minutes of awakening and cortisol in hair have both been shown to be reliable indicators of stress (e.g., Clow, Thorn, Evans & Hucklebridge, 2004; O'Brien et al., 2012; Russell, Koren, Rieder & Van Uum, 2012).

Another implication for research is the finding that favourable social comparison and a reduction in attachment insecurity is related to remission from AN. However, these findings cannot be applied to the recovery processes in other EDs and future research should examine if these positive affect systems are implicated in remission or recovery from BN, BED and EDNOS.

Although not all of the findings in this research are based on clinical samples, the findings can still have important implications for practice. The current research findings are important for developing and evaluating interventions for people with psychopathology and for preventing people from developing stress-related problems. Firstly, the current research revealed that affect systems and affect regulatory processes are crucial for women's ability to regulate stress-related experiences. Therefore, this has important implications for other conditions that are related to stress (i.e., some physical conditions such as immune function and gastrointestinal disorders and psychological disorders including depression, PTSD). Researchers developing interventions for conditions such as EDs, depression and PTSD should evaluate the impact of methods to enhance/improve regulation systems and processes. For example, interventions aimed at improving pathological eating behaviours or chronic low mood could include tasks or activities designed to

improve one's perceived social status. An example of how this can be achieved is by teaching individuals to avoid engaging in upward comparisons which will discourage perceived feelings of low rank. Another method of inducing positive affect regulation is by promoting more self-reassuring thoughts and feelings in response to adversity.

A way that this can be done is by using an adapted version of the expressive writing paradigm (Pennebaker & Beall, 1986) to directly manipulate affect regulatory systems and processes. For example, Leary et al. (2007) found that inducing self-compassionate thoughts and feelings in response to a traumatic event reduces the response triggered by the stressful experience. Similarly, Imrie and Troop (2012) used a similar approach and found that the ability to self-soothe can be developed with the use of a self-compassionate writing task. Therefore, to take the current research forward, future studies should apply this approach to examine if it is effective in influencing healthy changes in bodyweight and eating behaviours at times of stress. In addition, as Troop et al. (2013) revealed that writing about life goals is an effective approach to reducing self-critical thoughts and feelings (as measured by the FSCRS [Gilbert et al., 2004]), it may be that it is writing about future aspirations that is important for affect regulation. Hence, future researchers who want to enhance affect regulatory processes should use this expressive writing adaptation.

Also, weight loss programmes, ED prevention programmes and stress management interventions should address the issues of perceived low social status, self-criticism and attachment insecurities. These may be either in terms of helping individuals to identify battles they can win or to find alternative contexts in which to achieve status (Sloman, 2008) or to include therapeutic sessions with families to improve familial relations. Alternatively, interventions that render the effect of threats to social rank (i.e., how we compare ourselves to others) as less stressful may also be useful. For example, the ability to be self-soothing can tone down threat and self-criticism (Sloman, 2008). There is emerging evidence for the effectiveness of a number of approaches to increase self-compassion, of which the ability to self-soothe is a key component, including in EDs (Gale et al., 2012; Goss & Allan, 2010). These approaches include CFT (Gilbert, 2005, 2009), mindfulness based stress reduction (Shapiro et al., 2007), experimental approaches (Kelly et al., 2010) and

expressive writing (Imrie & Troop, 2012; Troop et al., 2013). The beneficial effects of writing about positive experiences as part of an expressive writing intervention on dietary restraint presented in this thesis suggests that including a writing task within a therapeutic environment can potentially alleviate eating pathology, specifically dietary restraint. The degree to which this benefits people with an ED or reduce the risk of subsequent DE, however, is an empirical question and requires further research.

The findings have also highlighted the issue surrounding conventional crisis management programmes as a method of managing stress-related problems, obesity and pathological eating patterns. Others have proposed that positive affect systems are shaped early on in childhood (Gilbert, 2005). Therefore, programmes should be developed to educate and train people from a younger age on the detrimental effects that maladaptive regulatory systems and processes can have. For example, although an infant cannot avoid being reared in a hostile environment, the adverse effects on the ability to regulate emotions can be relieved with preventative measures aimed at younger groups.

Future research

Although this research programme has added to the body of literature in various ways, it has also stimulated more research questions that need to be answered. On the basis of the current study findings, this research should be taken forward to evaluate how and which of the two positive affect systems (social rank and attachment) may be activated to promote health and well-being. Gilbert (2005) proposed that the positive affect systems are activated by internal and external stimuli. The social rank system is a resource/achievement-focused system which can be activated by internal thoughts of goal attainment and social comparisons triggering the need for achievement and social status. In comparison, the attachment system is focused on affiliation and can be stimulated by recalling memories of warm and affectionate caregivers which induces self-soothing and reassuring thoughts and feelings. Importantly for research and clinical practice,

these positive affect systems can also be manipulated externally. Several methods of triggering the processes of the social rank and attachment systems can be suggested for future research.

Visualisation techniques designed to stimulate a soothing and reassuring image suggested to activate the attachment system have been shown to be effective in enhancing self-compassion in self-critical individuals (Gilbert & Irons, 2004), reducing self-reported shame and skin complaints in chronic acne sufferers (Kelly, Zuroff & Shapira, 2009), the regulation of negative emotions triggered by stressful experiences (Lincoln, Hohenhaus & Hartmann, 2013; Selcuk, Zayas, Günaydin, Hazan & Kross, 2012) and even decreasing the number of cigarettes smoked (Kelly et al., 2010). In addition to stimulating the affiliation-focused processes of the attachment system, Kelly et al. (2010) also developed a visualisation technique to stimulate the achievement-focused processes of the social rank system in relation to smoking cessation. Instructing individuals to generate a mental representation of an image that would provide energetic words of encouragement when the urge to smoke is experienced was proposed to activate the processes of the social rank system. This was suggested to stimulate feelings that are commonly associated with experiences of striving and achieving a personal goal. Similar to other studies (Gilbert & Irons, 2004, Kelly et al., 2009), the affiliation-focused system was stimulated by visualising an image that would provide warmth and soothing at times of smoking urges. In this study, activation of the achievement- and affiliation-focused processes were both effective in reducing the number of cigarettes smoked (Kelly et al., 2010). This study provides an intervention model that can be used in future research to explore the utility of these positive affect systems in enhancing health and well-being further.

Although experimental studies using visualisation have demonstrated that mental imagery is useful for manipulating the processes of the positive affect systems, there are also other methods of stimulating achievement- and affiliation-focused orientations. For example, the expressive paradigm can be adapted to directly manipulate the processes of the affect regulatory systems. This can be done by instructing individuals to write about their accomplishments in life or the positive aspects of the relationships that they have or have had. Also, as suggested above in the "*Implications*" section, writing about future goals may be important for affect

regulation. Therefore, future research can use writing about life goals from an achievement- or relationship-focused perspective to activate the positive affect systems. It is proposed that writing about their past or future achievements will trigger the social rank system and give them pleasure in remembering what they have accomplished in life or trigger the need for achieving personal goals. In comparison, writing about the positive relationships they had or the goals they have for their relationships will stimulate feelings of safeness and soothing. Therefore, whether an expressive writing task which activates the achievement- or affiliation-focused system by recalling positive memories or planning future goals, future research should determine which of the two positive affect systems are more effective in regulating emotions and thus, produce health benefits.

Using the expressive writing paradigm will also facilitate the examination of the linguistic markers that may influence the benefits to health. There are some inconsistencies in the types of words that have been suggested to relate to improved outcomes. For example, earlier studies have suggested that increasing use of cognitive mechanism words (i.e., *because*, *consider*) are related to improved health (Pennebaker, 1997; Pennebaker et al., 2003), however, a more recent study showed that those who reported a decrease in self-critical thoughts and feelings wrote *fewer* cognitive mechanism words, specifically discrepancy words (i.e., *should*, *could*) compared with those who did not improve (Troop et al., 2013). Therefore, whilst it is important to determine if activating the social rank or the attachment system will result in improved outcomes, future research should also examine any mediators such as the types of words that are used that may be associated with greater improvement.

The notion of activating the two positive affect systems by adopting visualisation techniques or using the expressive writing paradigm can also be taken further. As the present study found that favourable social comparison is related to remission or recovery from AN, future research should examine if activating the achievement-focused processes lead to a reduction in pathological eating patterns and promote recovery in those with AN and other EDs such as BN, BED and EDNOS. Similarly, as the current study revealed that increases in stress are related to unfavourable social comparison, an intervention that stimulates the social rank

system should be used to examine if triggering achievement-related processes are beneficial for regulating stress in those diagnosed with stress-related disorders such as PTSD and depression. On a broader note, future research should use these techniques to determine if interventions developed to cultivate the achievement-focused processes would be better placed for improving symptoms associated with other disorders such as anxiety and psychosis compared with processes that promote affiliation.

Future studies which employ visualisation techniques or expressive writing tasks to stimulate affect regulatory systems can also be used to address other issues in this area. Firstly, future studies should be designed to examine if computer-assisted interventions that stimulate the processes of the affect regulation systems produce better results compared with when exercises are delivered by interventionists or therapists. Although, both computer-assisted (Kelly et al., 2009, 2010) and person-delivered methods (Gilbert & Irons, 2004; Lincoln et al., 2013) of stimulating affect regulatory processes with visualisation techniques have demonstrated promising results, these two different methods of delivering the intervention have not been directly compared.

Secondly, studies should determine the optimal number of times the intervention exercises must be actively executed in order to produce beneficial results on health and well-being. For example, studies have demonstrated that engaging in expressive writing for 15 minutes three times during a single hour can produce similar health benefits as the original method of writing for 15-20 minutes a day over three consecutive days (Chung & Pennebaker, 2008). Similarly, studies have demonstrated that performing visualisation techniques to stimulate the affect regulatory systems three times a day over two weeks (Kelly et al., 2009) and every time a smoking urge is experienced over a period of three weeks (Kelly et al., 2010) have both produced beneficial results. Finally, future research should explore the length of time that the effects of stimulating positive affect regulatory systems last and if activating the two positive affect systems produce similar effects or if activation of one system produces longer lasting effects compared to the other.

Another objective for future research is to examine various rank-related constructs in relation to stress and problematic weight regulation. The current study

included one type of rank-related construct (social comparison) which has been shown to be an important factor in predicting greater levels of disinhibition in those enrolled into a weight management programme (Matos et al., In Press) and for eating pathology (Bellew et al., 2006; Connan et al., 2007; Stormer & Thompson, 1996; Thompson et al., 1999; Troop et al., 2003, 2013; Troop & Baker, 2008). However, previous research has demonstrated that other rank-related constructs such as shame and submissiveness are related to eating pathology (e.g., Pinto-Gouveia et al., 2012; Troop et al., 2013; Troop & Baker, 2008), whilst social defeat has been associated with other conditions such as PTSD (Troop & Hiskey, 2013). Therefore, as these rank-related constructs have not been examined altogether in relation to stress and problematic weight regulation, future research should examine the role of other rank-related characteristics on stress, BMI and eating pathology.

Finally, the reviews on stress and problematic weight regulation presented in Chapter Two were found to examine the relationship between stress and problematic weight regulation mainly in women. Although, this was not an issue for the current research which purposefully recruited solely women (because of apparent differences in the rates or mechanisms of weight regulation and DE in men and women), this finding has highlighted the need for more studies to examine the relationship between stress and problematic weight regulation in men. The urgent need for more studies in this area is also emphasised by Wardle et al.'s (2010) meta-analysis which found that although there is an effect of stress on weight gain in both men and women, this effect is stronger for men. In addition, the current study did find that affect regulation is important for the way women manage stress and eating behaviours but are these positive affect systems and regulatory processes also important for men? Therefore, as men and women differ in the way that they appraise stress and in their trajectories of bodyweight and eating behaviours (Green & Pope, 1999; Heatherton et al., 1995, 1997; Keel et al., 2007; Koopmans & Lamers, 2007; Matud, 2004), it is important for future research to examine if affect regulation influences the ability to regulate stress-related experiences and factors related to problematic weight regulation in men.

Concluding comments

The current study indicated that a shift in the research agenda examining the risk factors for problematic weight regulation is needed. Future research should move away from stress towards affect regulation as the current research suggests that affect regulation is a crucial part of women's ability to manage stress and an optimally healthy relationship with food.

References

Abbott, D. H., Keverne, E. B., Bercovitch, F. B., Shively, C. A., Mendoza, S. P., Saltzman, W., Snowdon, C. T., Ziegler, T. E., Banjevic, M., Garland Jr, T., & Sapolsky, R. M. (2003). Are subordinates always stressed? A comparative analysis of rank differences in cortisol levels among primates. *Hormones and Behavior*, *43*(1), 67-82

Abu Dayyeh, B. K., Lautz, D. B., & Thompson, C. C. (2011). Gastrojejunal stoma diameter predicts weight regain after Roux-en-Y gastric bypass. *Clinical Gastroenterology and Hepatology, 9,* 228-233

Adam, T. C., & Epel, E. S. (2007). Stress, eating and the reward system. *Physiology and Behavior*, *91*(4), 449-458

Adams, T., & Rini, A. (2007). Predicting 1-year change in body mass index among college students. *Journal of American College Health*, *55*(6), 361-366

Adler, N. E., Epel, E. S., Castellazzo, G., & Ickovics, J. R. (2000). Relationship of subjective and objective social status with psychological and physiological functioning: Preliminary data in healthy, White women. *Health Psychology*, *19*(6), 586-592

Aderka, I. M., Weisman, O., Shahar, G., & Gilboa-Schechtman, E. (2009). The roles of the social rank and attachment systems in social anxiety. *Personality and Individual Differences*, *47*(4), 284-288

Ainsworth, M. D. S. (1964). Patterns of attachment behavior shown by the infant in interaction with his mother. *Merrill-Palmer Quarterly of Behavior and Development*, *10*(1), 51-58

Ainsworth, M. D. S. (1985). Attachments across the life span. *Bulletin of the New York Academy of Medicine*, *61*(9), 792-813

Ainsworth, M. D. S., & Bell, S. M. (1970). Attachment, exploration, and separation: Illustrated by the behavior of one-year-olds in a strange situation. *Child Development, 41*(1), 49-67

Ainsworth, M. D. S., & Wittig, B. A. (1969). Attachment and exploratory behavior of one-year-olds in a strange situation. In B. M. Foss (Ed.), *Determinants of infant behaviour IV*. (pp. 111-136) London: Methuen

Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the strange situation*. Lawrence Erlbaum

Akinnusi, M. E., Saliba, R., Porhomayon, J., & El-Solh, A. A. (2012). Sleep disorders in morbid obesity. *European Journal of Internal Medicine, 23,* 219-226

Allan, S. & Gilbert, P. (1995). A social comparison scale: Psychometric properties and relationship to psychopathology. *Personality and Individual Differences, 19,* 293-299

Allan, S., & Gilbert, P. (1997). Submissive behaviour and psychopathology. *British Journal of Clinical Psychology*, *36*(4), 467-488

Allan, S., & Gilbert, P. (2002). Anger and anger expression in relation to perceptions of social rank, entrapment and depressive symptoms. *Personality and Individual Differences*, *32*(3), 551-565

Allen, N. B., & Badcock, P. B. (2003). The social risk hypothesis of depressed mood: Evolutionary, psychosocial, and neurobiological perspectives. *Psychological Bulletin*, *129*(6), 887-913

Allen, J. G., Coyne, L., & Huntoon, J. (1998). Complex Posttraumatic Stress Disorder in women from a psychometric perspective. *Journal of Personality Assessment, 70,* 277-298

Allison, D. B., & Casey, D. E. (2001). Antipsychotic-induced weight gain: A review of the literature. *Journal of Clinical Psychiatry, 62*(7), 22-31

American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders (4th Ed. Revised)* (DSM-IV) Washington, DC: American Psychological Association

American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders (5th Ed.)* (DSM-V) Arlington, VA: American Psychiatric Publishing

Ames, S. C., Ames, G. E., Stevens, S. R., Patten, C. A., Werch, C. E., & Schroeder, D. R. (2008). Effect of expressive writing as a treatment adjunct for reducing smoking cessation related weight gain in young adult smokers. *Substance Use and Misuse*, *43*(10), 1315-1325

Anderson, D. A. & Wadden, T. A. (1999). Treating the obese patient: Suggestions for Primary Care Practice. *Archives of Family Medicine, 8,* 156-167

Anderson, J. W., Hamilton, C. C., Crown-Weber, E., Riddlemoser, M., & Gustafson, N. J. (1991). Safety and effectiveness of a multidisciplinary very-low-calorie diet program for selected obese individuals. *Journal of the American Dietetic Association, 91*(12), 1582-1584

Anderson, J. W., Konz, E. C., Frederich, R. C. & Wood, C. L. (2001). Long-term weight-loss maintenance: A meta-analysis of US studies. *American Journal of Clinical Nutrition, 74,* 579-584

Arigo, D., & Smyth, J. M. (2012). The benefits of expressive writing on sleep difficulty and appearance concerns for college women. *Psychology and Health*, *27*(2), 210-226

Arrindell, W. A., Steptoe, A., & Wardle, J. (2003). Higher levels of state depression in masculine than in feminine nations. *Behaviour Research and Therapy*, *41*(7), 809-817

Arsenian, J. M. (1943). Young children in an insecure situation. *The Journal of Abnormal and Social Psychology*, *38*(2), 225-229

Astrup, A., Hill, J. O., & Rössner, S. (2004). The cause of obesity: Are we barking up the wrong tree? *Obesity Reviews, 5,* 125-127

Atkinson, R. L. (2008). Could viruses contribute to the worldwide epidemic of obesity? *International Journal of Pediatric Obesity, 3,* 37-43

Avena, N. M., & Bocarsly, M. E. (2011). Dysregulation of brain reward systems in eating disorders: Neurochemical information from animal models of binge eating, bulimia nervosa, and anorexia nervosa. *Neuropharmacology, 63*(1), 87-96

Averill, J. R. (1973). Personal control over aversive stimuli and its relationship to stress. *Psychological Bulletin*, *80*(4), 286-303

Bacaltchuk, J., & Hay, P. P. J. (2003). Antidepressants versus placebo for people with bulimia nervosa. *Cochrane Database of Systematic Reviews, 4,* Art. No.: CD003391

Baikie, K. A., & Wilhelm, K. (2005). Emotional and physical health benefits of expressive writing. *Advances in Psychiatric Treatment*, *11*(5), 338-346

Bailer, U. F., Price, J. C., Meltzer, C. C., Mathis, C. A., Frank, G. K., Weissfeld, L., McConaha, C. W., Henry, S. E., Brooks-Achenbach, S., Barbarich, N. C., & Kaye, W.

H. (2004). Altered 5-HT2A receptor binding after recovery from bulimia-type anorexia nervosa: Relationships to harm avoidance and drive for thinness. *Neuropsychopharmacology*, *29*(6), 1143-1155

Ball, K., Brown, W., & Crawford, D. (2002). Who does not gain weight? Prevalence and predictors of weight maintenance in young women. *International Journal of Obesity*, *26*(12), 1570-1578

Ball, K. & Crawford, D. (2005). Socioeconomic status and weight change in adult: A review. *Social Science and Medicine, 60,* 1987-2010

Ball, K., Crawford, D., Ireland, P., & Hodge, A. (2003). Patterns and demographic predictors of 5-year weight change in a multi-ethnic cohort of men and women in Australia. *Public Health Nutrition*, *6*(3), 269-280

Ball, K., & Lee, C. (2000). Relationships between psychological stress, coping and disordered eating: A review. *Psychology and Health*, *14*(6), 1007-1035

Ball, K., & Lee, C. (2002). Psychological stress, coping, and symptoms of disordered eating in a community sample of young Australian women. *International Journal of Eating Disorders*, *31*(1), 71-81

Ball, K., Lee, C., & Brown, W. (1999). Psychological stress and disordered eating: An exploratory study with young Australian women. *Women and Health*, *29*(2), 1-15

Bamford, B. H., & Mountford, V. A. (2012). Cognitive behavioural therapy for individuals with longstanding anorexia nervosa: Adaptations, clinician survival and system issues. *European Eating Disorders Review*, *20*(1), 49-59

Barraclough, J., Pinder, P., Cruddas, M., Osmond, C., Taylor, I., & Perry, M. (1992). Life events and breast cancer prognosis. *BMJ: British Medical Journal*, *304*(6834), 1078-1081

Barrington, W. E., Ceballos, R. M., Bishop, S. K., McGregor, B. A., & Beresford, S. A. (2012). Perceived stress, behavior, and Body Mass Index among adults participating in a Worksite Obesity Prevention Program, Seattle, 2005–2007. *Preventing Chronic Disease*, *9*-18

Bartholomew, K. & Horowitz, L. M. (1991). Attachment styles among young adults: A test of a four-category model. *Journal of Personality and Social Psychology, 61*(2), 226-244

Beccuti, G., & Pannain, S. (2011). Sleep and obesity. *Current Opinion in Clinical Nutrition and Metabolic Care, 14,* 402-412

Becker, A. E., Burwell, R. A., Herzog, D. B., Hamburg, P., & Gilman, S. E. (2002). Eating behaviours and attitudes following prolonged exposure to television among ethnic Fijian adolescent girls. *The British Journal of Psychiatry*, *180*(6), 509-514

Bekker, M. H., & Boselie, K. A. (2002). Gender and stress: is gender role stress? A re-examination of the relationship between feminine gender role stress and eating disorders. *Stress and Health*, *18*(3), 141-149

Bellew, R., Gilbert, P., Mills, A., McEwan, K., & Gale, C. (2006). Eating attitudes and striving to avoid inferiority. *Eating Disorders*, *14*(4), 313-322

Bellocco, R., Jia, C., Ye, W., & Lagerros, Y. T. (2010). Effects of physical activity, body mass index, waist-to-hip ratio and waist circumference on total mortality risk in the Swedish National March Cohort. *European Journal of Epidemiology, 25,* 777-788

Bell, S. M. (1970). The development of the concept of object as related to infant-mother attachment. *Child Development*, 291-311

Bell, S. M., & Ainsworth, M. D. S. (1972). Infant crying and maternal responsiveness. *Child Development*, 1171-1190

Bennett, D. A., & Cooper, C. L. (1999). Eating disturbance as a manifestation of the stress process: A review of the literature. *Stress and Health*, *15*(3), 167-182

Benn, L., Harvey, J. E., Gilbert, P., & Irons, C. (2005). Social rank, interpersonal trust and recall of parental rearing in relation to homesickness. *Personality and Individual Differences*, *38*(8), 1813-1822

Berge, J. M., Loth, K., Hanson, C., Croll-Lampert, J., & Neumark-Sztainer, D. (2012). Family life cycle transitions and the onset of eating disorders: A retrospective grounded theory approach. *Journal of Clinical Nursing*, *21*(9-10), 1355-1363

Bergh, C., Brodin, U., Lindberg, G., & Södersten, P. (2002). Randomized controlled trial of a treatment for anorexia and bulimia nervosa. *Proceedings of the National Academy of Sciences*, *99*(14), 9486-9491

Bergin, J. E., Neale, M. C., Eaves, L. J., Martin, N. G., Heath, A. C., & Maes, H. H. (2012). Genetic and environmental transmission of Body Mass Index fluctuation. *Behavior Genetics*, *42*(6), 867-874

Berg, K. C., Frazier, P., & Sherr, L. (2009). Change in eating disorder attitudes and behavior in college women: Prevalence and predictors. *Eating Behaviors*, *10*(3), 137-142

Beglin, S. J., & Fairburn, C. G. (1992). Evaluation of a new instrument for the detection of eating disorders in community samples. *Psychiatry Research, 44*, 191-201

Berset, M., Semmer, N. K., Elfering, A., Jacobshagen, N., & Meier, L. L. (2011). Does stress at work make you gain weight? A two-year longitudinal study. *Scandinavian Journal of Work, Environment and Health*, *37*(1), 45-53

Bessesen, D. H. (2011). Regulation of body weight: What is the regulated parameter? *Physiology and Behavior*, *104*(4), 599-607

Beukes, M., Walker, S., & Esterhuyse, K. (2010). The role of coping responses in the relationship between perceived stress and disordered eating in a cross-cultural sample of female university students. *Stress and Health*, *26*(4), 280-291

Bifulco, A., & Brown, G. W. (1996). Cognitive coping response to crises and onset of depression. *Social Psychiatry and Psychiatric Epidemiology*, *31*(3-4), 163-172

Bifulco, A., Kwon, J., Jacobs, C., Moran, P. M., Bunn, A., & Beer, N. (2006). Adult attachment style as mediator between childhood neglect/abuse and adult depression and anxiety. *Social Psychiatry and Psychiatric Epidemiology*, *41*(10), 796-805

Bifulco, A., Mahon, J., Kwon, J. H., Moran, P. M. & Jacobs, C. (2003). The Vulnerable Attachment Style Questionnaire (VASQ): An interview-based measure of attachment styles that predict depressive disorder. *Psychological Medicine, 33,* 1099-1110

Birchwood, M., Meaden, A., Trower, P., Gilbert, P., & Plaistow, J. (2000). The power and omnipotence of voices: Subordination and entrapment by voices and significant others. *Psychological Medicine*, *30*(02), 337-344

Birmingham, C. L., Muller, J. L., Palepu, A., Spinelli, J. J., & Anis, A. H. (1999). The cost of obesity in Canada. *Canadian Medical Association Journal, 160*(4), 483-488

Birmingham, C. L., Su, J., Hlynsky, J. A., Goldner, E. M., & Gao, M. (2005). The mortality rate from anorexia nervosa. *International Journal of Eating Disorders, 38*(2), 143-146

Birmingham, C. L., Touyz, S. & Harbottle, J. (2008). Are anorexia nervosa and bulimia nervosa separate disorders? Challenging the 'transdiagnostic'theory of eating disorders. *European Eating Disorders Review*, *17*(1), 2-13

Blackburn, G. L., Wilson, G. T., Kanders, B. S., Stein, L. J., Lavin, P. T., Adler, J., & Brownell, K. D. (1989). Weight cycling: The experience of human dieters. *American Journal of Clinical Nutrition, 49,* 1105-1109

Blaase, H., & Elklit, A. (2001). Psychological characteristics of women with eating disorders: Permanent or transient features? *Scandinavian Journal of Psychology*, *42*(5), 467-478

Blatt, S. J., & Homann, E. (1992). Parent-child interaction in the etiology of dependent and self-critical depression. *Clinical Psychology Review, 12*, 47-91

Blatt, S. J., & Zuroff, D. C. (1992). Interpersonal relatedness and self-definition: Two prototypes for depression. *Clinical Psychology Review*, *12*(5), 527-562

Blehar, M. C., Lieberman, A. F., & Ainsworth, M. D. S. (1977). Early face-to-face interaction and its relation to later infant-mother attachment. *Child Development, 48*(1), 182-194

Block, J. P., He, Y., Zaslavsky, A. M., Ding, L., & Ayanian, J. Z. (2009). Psychosocial stress and change in weight among US adults. *American Journal of Epidemiology*, *170*(2), 181-192

Bodell, L. P., Smith, A. R., Holm-Denoma, J. M., Gordon, K. H., & Joiner, T. E. (2011). The impact of perceived social support and negative life events on bulimic symptoms. *Eating Behaviors*, *12*(1), 44-48

Bond, D. S., Phelan, S., Leahey, L. H., Hill, J. O., & Wing, R. R. (2009). Weight loss maintenance in successful weight losers: Surgical vs. non-surgical methods. *International Journal of Obesity, 33*(1), 173-180

Bouquegneau, A., Dubois, B. E., Krzesinski, J. M., & Delanaye, P. (2012). Anorexia Nervosa and the kidney. *American Journal of Kidney Diseases*, 60(2), 299-307

Bowlby, J. (1958). A child's tie to his mother. *International Journal of Psychoanalysis*, 39, 350-373

Bowlby, J. (1960). Separation anxiety. *International Journal of Psychoanalysis*, 41, 89-113

Bowlby, J. (1969). *Attachment and loss. Vol. 1: Attachment*. London: Hogarth Press

Bowlby, J. (1973). *Attachment and loss. Vol. 2: Separation*. London: Hogarth Press

Bowlby, J. (1980). *Attachment and loss. Vol. 3: Loss*. London: Hogarth Press

Bowlby, J. (1977a). The making and breaking of affectional bonds. I. Aetiology and psychopathology in the light of attachment theory. An expanded version of the Fiftieth Maudsley Lecture, delivered before the Royal College of Psychiatrists, 19 November 1976. *The British Journal of Psychiatry*, *130*(3), 201-210

Bowlby, J. (1977b). The making and breaking of affectional bonds: Aetiology and psychopathology in the light of attachment theory. *British Journal of Psychiatry, 130,* 201-210

Bradford, E., & Lyddon, W. J. (1994). Assessing adolescent and adult attachment: An update. *Journal of Counseling and Development*, *73*(2), 215-219

Bray, G. A. (2008). Lifestyle and pharmacological approaches to weight loss: Efficacy and safety. *Journal of Clinical Endocrinology Metabolism, 93,* S81-S88

Bray, G. A., York, B., & DeLany, J. (1992). A survey of the opinions of obesity experts on the causes and treatment of obesity. *American Journal of Clinical Nutrition, 55*, 151S-154S

Breuer, J., & Freud, S. (1957). *Studies on hysteria* (J. Strachey, Trans.). New York: Basic Books. (Original work published 1895)

Brewin, C. R., & Furnham, A. (1986). Attributional versus preattributional variables in self-esteem and depression: A comparison and test of learned helplessness theory. *Journal of Personality and Social Psychology*, *50*(5), 1013-1020

Brown, G. W. (1972). Life-events and psychiatric illness: Some thoughts on methodology and causality. *Journal of Psychosomatic Research*, *16*(5), 311-320

Brown, G. W. (1989). Life events and measurement. In G. W. Brown & T. Harris, (Ed.). (1989). *Life events and illness* (pp. 3-45). London: Guilford Press

Brown, G. W. (1993). Life events and affective disorder: Replications and limitations. *Psychosomatic Medicine*, *55*(3), 248-259

Brown, G. W., Bifulco, A., & Harris, H. O. (1987). Life events, vulnerability and onset of depression: Some refinements. *British Journal of Psychiatry, 150*, 30-42

Brown, G. W. & Harris, T. O. (1978). *Social origins of depression: A study of psychiatric disorder in women.* London: Tavistock

Brown, G. W. & Harris, T. O. (1986) Stressor, vulnerability and depression: A question of replication. *Psychological Medicine*, 16, 739-744

Brown, G. W., Harris, T. O., & Hepworth, C. (1995). Loss, humiliation and entrapment among women developing depression: A patient and non-patient comparison. *Psychological Medicine*, *25*(1), 7-22

Brown, W. J., Dobson, A. J., Bryson, L., & Byles, J. E. (1999). Women's health Australia: On the progress of the main cohort studies. *Journal of Women's Health and Gender-Based Medicine*, *8*(5), 681-688

Brownell, K. D. (1991). Personal responsibility and control over our bodies: When expectation exceeds reality. *Health Psychology*, *10*(5), 303-310

Brownell, K. D., Greenwood, M. R. C., Stellar, E., & Shrager, E. E. (1986). The effects of repeated cycles of weight loss and regain in rats. *Physiology and Behavior*, *38*(4), 459-464

Brownell, K. D., & Jeffery, R. W. (1987). Improving long-term weight loss: Pushing the limits of treatment. *Behavior Therapy*, *18*(4), 353-374

Brownell, K. D., Marlatt, G. A., Lichtenstein, E., & Wilson, G. T. (1986). Understanding and preventing relapse. *American Psychologist, 41*(7), 765-782

Brownell, K. D., & Rodin, J. (1994). The dieting maelstrom: Is it possible and advisable to lose weight? *American Psychologist*, *49*(9), 781-791

Brugha, T., Bebbington, P., Tennant, C., & Hurry, J., (1985). The List of Threatening Experiences: A subset of 12 life event categories with considerable long-term contextual threat. *Psychological Medicine, 15*, 189-194

Buchwald, H., Avidor, Y., Braunwald, E., Jensen, M. D., Pories, W., Fahrbach, K., & Schoelles, K. (2004). Bariatric surgery: A systematic review and meta-analysis. *Journal of American Medical Association, 292,* 1724-1737

Buchwald, H., & Oien, D. M. (2009) Metabolic/bariatric surgery worldwide 2008. *Obesity Surgery, 19*, 1605-1611

Buchwald, H., & Williams, S. E. (2004). Bariatric surgery worldwide 2003. *Obesity Surgery, 14,* 1157-64

Bugental, D. B. (2000). Acquisition of the algorithms of social life: a domain-based approach. *Psychological Bulletin*, *126*(2), 187-219

Bulik, C. M., Berkman, N. D., Brownley, K. A., Sedway, J. A., & Lohr, K. N. (2007). Anorexia nervosa treatment: A systematic review of randomized controlled trials. *International Journal of Eating Disorders, 40,* 310-320

Burke, L. E., & Wang, J. (2011). Treatment strategies for overweight and obesity. *Journal of Nursing Scholarship, 43*(4), 368-375

Burton, C. M. & King, L. A. (2004). The health benefits of writing about intensely positive experiences. *Journal of Research in Personality, 38,* 150-163

Burton, C. M., & King, L. A. (2008). Effects of (very) brief writing on health: The two-minute miracle. *British Journal of Health Psychology*, *13*(1), 9-14

Burton, C. M., & King, L. A. (2009). The health benefits of writing about positive experiences: The role of broadened cognition. *Psychology and Health*, *24*(8), 867-879

Buss, D. M. (1991). Evolutionary personality psychology. *Annual Review of Psychology*, *42*(1), 459-491

Butland, B., Jebb, S., Kopelman, P., McPherson, K., Thomas, S., Mardell, J., & Parry, V. (2007). Tackling Obesities: Future Choices – Project Report. *Foresight Tackling Obesities: Future Choices*. URL: http://www.foresight.gov.uk

Butow, P. N., Beumont, P. J. V., & Touyz, S. W. (1993). Cognitive processes in dieting disorders. *International Journal of Eating Disorders, 14*(3), 319-329

Butryn, M. L., Webb, V., & Wadden, T. A. (2011). Behavioral treatment of obesity. *Psychiatric Clinics of North America, 34,* 841-859

Buunk, A. P., Peíró, J. M., Rodríguez, I., & Bravo, M. J. (2007). A loss of status and a sense of defeat: An evolutionary perspective on professional burnout. *European Journal of Personality*, *21*(4), 471-485

Buunk, B. P., Collins, R. L., Taylor, S. E., VanYperen, N. W., & Dakof, G. A. (1990). The affective consequences of social comparison: Either direction has its ups and downs. *Journal of Personality and Social Psychology*, *59*(6), 1238-1249

Buunk, B. P., & Ybema, J. F. (1997). Social comparison and occupational stress: the identification-contrast model. In B. P. Buunk & F. X Gibbons, (Eds), Social Comparison, Health and Coping, (pp. 359-388). Mahwah: NJ, Erlbaum

Byrne, B. M. (1998). *Structural equation modelling with Lisrel, Prelis, and Simplis: Basic concepts, applications, and programming*. Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Byrne, S. M. (2002). Psychological aspects of weight maintenance and relapse in obesity. *Journal of Psychosomatic Research, 53,* 1029-1036

Byrne, S. M., Cooper, Z. & Fairburn, C. G. (2003). Weight maintenance and relapse in obesity: A qualitative study. *International Journal of Obesity, 27,* 955-962

Byrne, S. M., Cooper, Z. & Fairburn, C. G. (2004). Psychological predictors of weight regain in obesity. *Behaviour Research and Therapy, 42,* 1341-1356

Byrne, S. M., Fursland, A., Allen, K. L., & Watson, H. (2011). The effectiveness of enhanced cognitive behavioural therapy for eating disorders: An open trial. *Behaviour Research and Therapy*, *49*(4), 219-226

Cachelin, F. M. Striegel-Moore, R. H. & Brownell, K. D. (1998). Beliefs about weight gain and attitudes toward relapse in a sample of women and men with obesity. *Obesity Research, 6*(3), 231-237

Cacioppo, J. T., Berntson, G. G., Sheridan, J. F., & McClintock, M. K. (2000). Multilevel integrative analyses of human behavior: Social neuroscience and the complementing nature of social and biological approaches. *Psychological Bulletin*, *126*(6), 829-843

Cade, J. E. & Margetts, B. M. (1988). Nutrient sources in the English diet: quantitative data from three English towns. *International Journal of Epidemiology, 17,* 844-848

Calle, E. E., Thun, M. J., Petrelli, J. M., Rodriguez, C., & Heath, C. W. (1999). Body-mass index and mortality in a prospective cohort of U.S. adults. *New England Journal of Medicine, 341,* 1097-1105

Cameron, L. D., & Jago, L. (2008). Emotion regulation interventions: A common-sense model approach. *British Journal of Health Psychology*, *13*(2), 215-221

Capasso, A., Petrella, C., & Milano, W. (2009). Recent clinical aspects of eating disorders. *Reviews on Recent Clinical Trials*, *4*(1), 63-69

Carnelley, K. B., Pietromonaco, P. R., & Jaffe, K. (1994). Depression, working models of others, and relationship functioning. *Journal of Personality and Social Psychology, 66,* 127-140

Cattanach, L., Phil, M., Malley, R. and Rodin, J. (1988) Psychologic and physiologic reactivity to stressors in eating disordered individuals. *Psychosomatic Medicine, 50,* 591-599

Carter, F. A., Jordan, J., McIntosh, V. V., Luty, S. E., McKenzie, J. M., Frampton, C. M. A., Bulik, C. M., & Joyce, P. R. (2011). The long-term efficacy of three psychotherapies for anorexia nervosa: A randomized, controlled trial. *International Journal of Eating Disorders*, *44*(7), 647-654

Carter, J. C., Blackmore, E., Sutandar-Pinnock, K., & Woodside, D. B. (2004). Relapse in anorexia nervosa: A survival analysis. *Psychological Medicine*, *34*(04), 671-679

Carter, J. C., Mercer-Lynn, K. B., Norwood, S. J., Bewell-Weiss, C. V., Crosby, R. D., Woodside, D. B., & Olmsted, M. P. (2012). A prospective study of predictors of relapse in anorexia nervosa: Implications for relapse prevention. *Psychiatry Research, 200,* 518-523

Chang, M. W., Brown, R., & Nitzke, S. (2009). Participant recruitment and retention in a pilot program to prevent weight gain in low-income overweight and obese mothers. *BMC Public Health*, *9*(1), 424-434

Chen, J., Wang, Z., Guo, B., Arcelus, J., Zhang, H., Jia, X., Xu, Y., Qiu, J., Xiao, Z., & Yang, M. (2012). Negative affect mediates effects of psychological stress on disordered eating in young Chinese women. *PloS ONE*, *7*(10), e46878

Chiriboga, D. E., Ma, Y., Li, W., Olendzki, B. C., Pagoto, S. L., Merriam, P. A., Matthews, C. E., Hebert, J. R., & Ockene, I. S. (2008). Gender differences in predictors of body weight and body weight change in healthy adults. *Obesity*, *16*(1), 137-145

Christou, N. V., Look, D., & MacLean, L. D. (2006). Weight gain after short- and long-limb gastric bypass in patients followed for longer than 10 years. *Annals of Surgery, 244*(5), 734-740

Christou, N. V., Sampalis, J. S., Liberman, M., Look, D., Auger, S., McLean, A. P. H., & MacLean, L. D. (2004). Surgery decreases long-term mortality, morbidity, and health care use in morbidly obese patients. *Annals of Surgery, 240*(3), 416-424

Chung, C. K. (2009). *Predicting weight loss in blogs using computerized text analysis.* (Doctoral dissertation, Texas State University)

Chung, C. K., & Pennebaker, J. W. (2008). Variations in the spacing of expressive writing sessions. *British Journal of Health Psychology, 13*, 15-21

Cizza, G., Requena, M., Galli, G., & de Jonge, L. (2011). Chronic sleep deprivation and seasonality: Implications for the obesity epidemic. *Journal of Endocrinological Investigation, 34*(10), 793-800

Cizza, G., & Rother, K. I. (2012). Beyond fast food and slow motion: Weighty contributors to the obesity epidemic. *Journal of Endocrinological Investigation, 35*(2), 236-242

Clow, A., Thorn, L., Evans, P., & Hucklebridge, F. (2004). The awakening cortisol response: methodological issues and significance. *Stress: The International Journal on the Biology of Stress*, *7*(1), 29-37

Cnattingius, S., Hultman, C. M., Dahl, M., & Sparen, P. (1999). Very preterm birth, birth trauma, and the risk of anorexia nervosa among girls. *Archives of General Psychiatry*, *56*(7), 634-638

Coakley, E. H., Rimm, E. B., Colditz, G., Kawachi, I., & Willett, W. (1998). Predictors of weight change in men: Results from the Health Professionals follow-up study. *International Journal of Obesity*, *22*(2), 89-96

Cohen, D. L., & Petrie, T. A. (2005). An examination of psychosocial correlates of disordered eating among undergraduate women. *Sex Roles*, *52*(1-2), 29-42

Cohen, J. (1977). *Statistical power analysis for the behavioral sciences*. New York: Academic Press

Cohen, J. (1992). Power primer. *Psychological Bulletin, 112,* 155-159

Cohen, S., Janicki-Deverts, D., & Miller, D. E. (2007). Psychological stress and disease. *Journal of American Medical Association, 298*(14), 1685-1687

Cohen, S., Kamarck, T. & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*(4), 385-396

Cohen, S. & Williamson, G. (1988). Perceived stress in a probability sample of the United States. In S. Spacapam & S. Oskamp, (Ed.) *The Social Psychology of Health: Claremont Symposium on Applied Social Psychology.* Newbury Park, CA: Sage

Colditz, G. A., Willett, W. C., Stampfer, M. J., London, S. J., Segal, M. R., & Speizer, F. E. (1990). Patterns of weight change and their relation to diet in a cohort of healthy women. *The American Journal of Clinical Nutrition*, *51*(6), 1100-1105

Colvin, R. H. & Olson, S. B. (1983). A descriptive analysis of men and women who have lost significant weight and are highly successful at maintaining the loss. *Addictive Behaviors, 8*, 287-295

Connan, F., Troop, N., Landau, S., Campbell, I. C., & Treasure, J. (2007). Poor social comparison and the tendency to submissive behavior in anorexia nervosa. *International Journal of Eating Disorders*, *40*(8), 733-739

Cooley, E., Toray, T., Valdez, N., & Tee, M. (2007). Risk factors for maladaptive eating patterns in college women. *Eating and Weight Disorders*, *12*(3), 132-139

Cooper, M. L., Shaver, P. R., & Collins, N. L. (1998). Attachment styles, emotion regulation, and adjustment in adolescence. *Journal of Personality and Social Psychology, 74,* 1380-1397

Cooper, Z., & Fairburn, C. (1987). The eating disorder examination: A semi-structured interview for the assessment of the specific psychopathology of eating disorders. *International Journal of Eating Disorders*, *6*(1), 1-8

Cooper, Z., Cooper, P. J., & Fairburn, C. G. (1989). The validity of the Eating Disorder Examination and its subscales. *British Journal of Psychiatry, 154*, 807-812

Corning, A. F., Krumm, A. J., & Smitham, L. A. (2006). Differential social comparison processes in women with and without eating disorder symptoms. *Journal of Counseling Psychology, 53*(3), 338-349

Costa, J. L., Brennen, M. B., & Hochgeschwender, U. (2002). The human genetics of eating disorders lessons from the leptin/melanocortin system. *Child Adolescent Psychiatric Clinics of North America*, *11*(2), 387-397

Costarelli, V., & Patsai, A. (2012). Academic examination stress increases disordered eating symptomatology in female university students. *Eating and Weight Disorders*, *17*(3), e164-e169

Craig, R. & Hirani, V. (2010). Vol 1: Health and Lifestyle. *NHS Information Centre Health Survey for England.* URL: www.ic.nhs.uk/pubs/hse09report

Craig, T. K. J., & Brown, G. W. (1984). Goal frustration and life events in the aetiology of painful gastrointestinal disorder. *Journal of Psychosomatic Research*, *28*(5), 411-421

Crawford, D., Jeffery, R. W., & French, S. A. (2000). Can anyone successfully control their weight? Findings of a three year community-based study of men and women. *International Journal of Obesity*, *24*(9), 1107-1110

Crowell, J. A., & Treboux, D. (1995). A review of adult attachment measures: Implications for theory and research. *Social Development*, *4*(3), 294-327

Crowther, J. H., & Chernyk, B. (1986). Bulimia and binge eating in adolescent females: A comparison. *Addictive Behaviors*, *11*(4), 415-424

Cruddas, S., Gilbert, P., & McEwan, K. (2012). The relationship between self-concealment and disclosure, early experiences, attachment, and social comparison. *International Journal of Cognitive Therapy*, *5*(1), 28-37

Cunningham, S. A., Vaquera, E., Maturo, C. C., & Narayan, K. M. V. (2012). Is there evidence that friends influence body weight? A systematic review of empirical research. *Social Science and Medicine, 75,* 1175-1183

Currin, L., Schmidt, U., Treasure, J., & Jick, H. (2005). Time trends in eating disorder incidence. *The British Journal of Psychiatry*, *186*(2), 132-135

Dagnan, D., & Sandhu, S. (1999). Social comparison, self-esteem and depression in people with intellectual disability. *Journal of Intellectual Disability Research*, *43*(5), 372-379

Dallman, M. F., Pecoraro, N., Akana, S. F., la Fleur, S. E., Gomez, F., Houshya, H., Bell, M. E., Bhatnagar, S., Laugero, K. D. & Manalo, S. (2003). Chronic stress and obesity: A new view of "comfort food". *Proceedings of the National Academy of Sciences, 100*(20), 11696-11701

Dallman, M. F., la Fleur, S. E., Pecoraro, N., Gomez, F., Houshyar, H. & Akana, S. F. (2004). Minireview: Glucocorticoids – Food intake, abdominal obesity and wealthy nations in 2004. *Endocrinology, 145*(6), 2633-2638

Dancyger, I. F., & Fornari, V. M. (2005). A review of eating disorders and suicide risk in adolescence. *TheScientificWorldJOURNAL*, *5*, 803-811

Danner, D. D., Snowdon, D. A., & Friesen, W. V. (2001). Positive emotions in early life and longevity: Findings from the nun study. *Journal of Personality and Social Psychology*, *80*(5), 804-813

Darby, A., Hay, P., Mond, J., Quirk, F., Buttner, P., & Kennedy, L. (2009). The rising prevalence of comorbid obesity and eating disorder behaviors from 1995 to 2005. *International Journal of Eating Disorders*, *42*(2), 104-108

Davidson, M. H., Hauptman, J., DiGirolamo, M., Foreyt, J. P., Halsted, C. H., Heber, D., Heimburger, D. C., Lucas, C. P., Robbins, D. C., Chung, J., & Heymsfield, S. B. (1999). Weight control and risk factor reduction in obese subjects treated for 2 years with orlistat. *Journal of American Medical Association, 281*(3), 235-242

DeJong, H., Broadbent, H., & Schmidt, U. (2011). A systematic review of dropout from treatment in outpatients with anorexia nervosa. *International Journal of Eating Disorders*, *45*(5), 635-647

Delahanty, L. M., Meigs, J. B., Hayden, D., Williamson, D. A., & Nathan, D. M. (2002). Psychological and behavioral correlates of baseline BMI in the diabetes prevention program (DPP). *Diabetes Care*, *25*(11), 1992-1998

DePue, J. D., Clark, M. M., Ruggiero, L., Medeiros, M. L. & Pera, V. (1995). Maintenance of weight loss: A needs assessment. *Obesity Research, 3*(3), 241-248

Deter, H. C., & Herzog, W. (1994). Anorexia nervosa in a long-term perspective: Results of the Heidelberg-Mannheim study. *Psychosomatic Medicine*, *56*(1), 20-27

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*(3), 355-391

Ding, D. & Gebel, K. (2012). Built environment, physical activity, and obesity: What have we learned from reviewing the literature? *Health and Place, 18*, 100-105

Dise-Lewis, J. E. (1988). The life events and coping inventory: An assessment of stress in children. *Psychosomatic Medicine*, *50*(5), 484-499

Ditzen, B., Schmidt, S., Strauss, B., Nater, U. M., Ehlert, U., & Heinrichs, M. (2008). Adult attachment and social support interact to reduce psychological but not cortisol responses to stress. *Journal of Psychosomatic Research, 64,* 479-486

Dohm, F., Beattie, J. A., Aibel, C. & Striegel-Moore, R. H. (2001). Factors differentiating women and men who successfully maintain weight loss from women and men who do not. *Journal of Clinical Psychology, 57*(1), 105-117

Dohrenwend, B. S., Askenasy, A. R., Krasnoff, L., & Dohrenwend, B. P. (1978). Exemplification of a method for scaling life events: The PERI Life Events Scale. *Journal of Health and Social Behavior, 19*(2) 205-229

Donnelly, J. E., Smith, B., Jacobsen, D. J., Kirk, E., DuBose, K., Hyder, M., Bailey, B., & Washburn, R. (2004). The role of exercise for weight loss and maintenance. *Best Practice and Research Clinical Gastroenterology*, *18*(6), 1009-1029

Douketis, J. D., Macie, C., Thabane, L., & Williamson, D. F. (2005). Systematic review of long-term weight loss studies in obese adults: Clinical significance and applicability to clinical practice. *International Journal of Obesity, 29,* 1153-1167

Drapeau, V., Provencher, V., Lemieux, S., Despres, J. P., Bouchard, C., & Tremblay, A. (2003). Do 6-y changes in eating behaviors predict changes in body weight? Results from the Quebec Family Study. *International Journal of Obesity*, *27*(7), 808-814

Driscoll, R. (1989). Self-condemnation: A comprehensive framework for assessment and treatment. *Psychotherapy: Theory, Research, Practice, Training*, *26*(1), 104-111

Dubbert, P. M. & Wilson, G.T. (1984). Goal-setting and spouse involvement in the treatment of obesity. *Behaviour Research and Therapy, 22*(3), 227-242

Dunkley, D. M., & Grilo, C. M. (2007). Self-criticism, low self-esteem, depressive symptoms, and over-evaluation of shape and weight in binge eating disorder patients. *Behaviour Research and Therapy*, *45*(1), 139-149

Dunkley, D. M., Sanislow, C. A., Grilo, C. M., & McGlashan, T. H. (2009). Self-criticism versus neuroticism in predicting depression and psychosocial impairment for 4 years in a clinical sample. *Comprehensive Psychiatry*, *50*(4), 335-346

Durand, C. P., Andalib, M., Dunton, G. F., Wolch, J., & Pentz, M. A. (2011). A systematic review of the built environment factors related to physical activity and obesity risk: Implications for smart growth urban planning. *Obesity Reviews, 12,* e173-e182

East, P., Startup, H., Roberts, C., & Schmidt, U. (2010). Expressive writing and eating disorder features: A preliminary trial in a student sample of the impact of three writing tasks on eating disorder symptoms and associated cognitive, affective and interpersonal factors. *European Eating Disorders Review*, *18*(3), 180-196

Eckert, E. D., Halmi, K. A., Marchi, P., Grove, W., & Crosby, R. (1995). Ten-year follow-up of anorexia nervosa: Clinical course and outcome. *Psychological Medicine*, *25*(1), 143-156

Eisler, I., Dare, C., Russell, G. F., Szmukler, G., le Grange, D., & Dodge, E. (1997). Family and individual therapy in anorexia nervosa: A 5-year follow-up. *Archives of General Psychiatry*, *54*(11), 1025-1030

Elfhag, K. & Rössner, S. (2005). Who succeeds in maintaining weight loss: A conceptual review of factors associated with weight loss maintenance and weight regain. *Obesity Reviews, 6,* 67-85

Engeln–Maddox, R. (2005). Cognitive responses to idealized media images of women: The relationship of social comparison and critical processing to body image disturbance in college women. *Journal of Social and Clinical Psychology*, *24*(8), 1114-1138

Engel, S. G., Crosby, R. D., Kolotkin, R. L., Hartley, G. G., Williams, G. R., Wonderlich, S. A., & Mitchell, J. E. (2003). Impact of weight loss and regain on quality of life: Mirror image or differential effect? *Obesity Research*, *11*(10), 1207-1213

Eng, W., Heimberg, R. G., Hart, T. A., Schneier, F. R., & Liebowitz, M. R. (2001). Attachment in individuals with social anxiety disorder: The relationship among adult attachment styles, social anxiety and depression. *Emotion, 4,* 365-380

Epel, E., Jimenez, S., Brownell, K., Stroud, L., Stoney, C., & Niaura, R. (2004). Are stress eaters at risk for the metabolic syndrome? *Annals of the New York Academy of Sciences*, *1032*, 208-210

Epel, E., Lapidus, R., McEwen, B. & Brownell, K. (2001). Stress may add to appetite in women: A laboratory study of stress-induced cortisol and eating behavior. *Psychoneuroendocrinology, 26,* 37-49

Epiphaniou, E., & Ogden, J. (2010). Evaluating the role of life events and sustaining conditions in weight loss maintenance. *Journal of Obesity, Art ID: 859413*

Epstein, L. H., Jankowiak, N., Nederkoorn, C., Raynor, H. A., French, S. A., & Finkelstein, E. (2012). Experimental research on the relation between food price changes and food-purchasing patterns: A targeted review. *American Journal of Clinical Nutrition, 95,* 789-809

Epstein, L. H., Valoski, A. M., Kalarchian, M. A., & McCurley, J. (1995). Do children lose and maintain weight easier than adults: A comparison of child and parent weight changes from six months to ten years. *Obesity Research*, *3*(5), 411-417

Epstein, L. H., Valoski, A., Wing, R. R., & McCurley, J. (1990). Ten-year follow-up of behavioral, family-based treatment for obese children. *The Journal of the American Medical Association*, *264*(19), 2519-2523

European Medicines Agency. (2008). URL: http://www.emea.europa.eu/humandocs/PDFs/EPAR/acomplia/53777708en.pdf (accessed January 2012)

European Medicines Agency. (2010). URL: http://www.ema.europa.eu/docs/en_GB/document_library/Referrals_document/Sibutramine_107/WC500094238.pdf (accessed January 2012)

Everitt, B. S. (1975). Multivariate analysis: The need for data, and other problems. *The British Journal of Psychiatry*, *126*(3), 237-240

Expert panel on the identification, evaluation, and treatment of overweight and obesity in adults. (1998). Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: Executive summary. *American Journal of Clinical Nutrition, 68,* 899-917

Fabrigar, L. R., Wegener, D. T., MacCallum, R. C., & Strahan, E. J. (1999). Evaluating the use of exploratory factor analysis in psychological research. *Psychological Methods*, *4*(3), 272-299

Fairburn, C. G. (1997). Eating disorders. In D. M. Clark & C. G. Fairburn (Ed.), *Science and Practice of Cognitive Behaviour Therapy.* Oxford, England: Oxford University Press

Fairburn, C. G. & Beglin, S. J. (1994). Assessment of eating disorders: Interview or self-report questionnaire. *International Journal of Eating Disorders, 16,* 363-370

Fairburn, C. G., Cooper, Z., Doll, H. A., O'Connor, M. E., Bohn, K., Hawker, D. M., Wales, J. A., & Palmer, R. L. (2009). Transdiagnostic cognitive-behavioral therapy for patients with eating disorders: A two-site trial with 60-week follow-up. *The American Journal of Psychiatry*, *166*(3), 311-319

Fairburn, C. G., Cooper, Z., Doll, H. A., O'Connor, M. E., Palmer, R. L., & Dalle Grave, R. (2013). Enhanced cognitive behaviour therapy for adults with anorexia nervosa: A UK–Italy study. *Behaviour Research and Therapy, 51*(1), R2-R8

Fairburn, C. G., Cooper, Z., Doll, H. A., & Welch, S. L. (1999). Risk factors for anorexia nervosa: Three integrated case-control comparisons. *Archives of General Psychiatry, 56*, 468-476

Fairburn, C. G., Cooper, Z., & O'Connor, M. (2008). Eating Disorder Examination (Edition 16.0D). In C. G. Fairburn (Ed.), *Cognitive Behavior Therapy and Eating Disorders* (pp. 265-308). New York: Guildford Press

Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: A "transdiagnostic" theory and treatment. *Behaviour Research and Therapy*, *41*(5), 509-528

Fairburn, C. G., Doll, H. A., Welch, S. L., Hay, P. J., Davies, B. A., & O'Connor, M. E., (1998). Risk factors for binge eating disorder: A community-based, case-control study. *Archives of General Psychiatry, 155*, 425-432

Fairburn, C. G., & Harrison, P. J. (2003). Eating disorders. *Lancet, 361,* 407-416

Fairburn, C. G., Shafran, R., & Cooper, Z. (1999). A cognitive behavioural theory of anorexia nervosa. *Behaviour Research and Therapy*, *37*(1), 1-13

Fairburn, C. G., Welch, S. L., Doll, H. A., Davies, B. A., & O'Connor, M. E. (1997). Risk factors for bulimia nervosa: A community-based case-control study. *Archives of General Psychiatry*, *54*(6), 509-517

Faresjö, Å., Theodorsson, E., Chatziarzenis, M., Sapouna, V., Claesson, H., Koppner, J., & Faresjö, T. (2013). Higher perceived stress but lower cortisol levels found among young Greek adults living in a stressful social environment in comparison with Swedish young adults. *PLoS ONE 8*(9), e73828

Farmer, A. E., & McGuffin, P. (2003). Humiliation, loss and other types of life events and difficulties: A comparison of depressed subjects, healthy controls and their siblings. *Psychological Medicine*, *33*(07), 1169-1175

Fazaa, N., & Page, S. (2003). Dependency and self-criticism as predictors of suicidal behavior. *Suicide and Life-Threatening Behavior*, *33*(2), 172-185

Feingold, A. (1992). Good-looking people are not what we think. *Psychological Bulletin*, *111*(2), 304-341

Feinson, M. C., & Meir, A. (2012). Disordered eating and complexities of cultural origin: A focus on Jews from Muslim countries. *Eating Behaviors*, *13*(2), 135-138

Fennig, S., Hadas, A., Itzhaky, L., Roe, D., Apter, A., & Shahar, G. (2008). Self-criticism is a key predictor of eating disorder dimensions among inpatient adolescent females. *International Journal of Eating Disorders*, *41*(8), 762-765

Ferreira, C., Pinto-Gouveia, J., & Duarte, C. (2013a). Drive for thinness as a women's strategy to avoid inferiority. *International Journal of Psychology and Psychological Therapy*, *13*(1), 15-30

Ferreira, C., Pinto-Gouveia, J., & Duarte, C. (2013b). Physical appearance as a measure of social ranking: The role of a new scale to understand the relationship between weight and dieting. *Clinical Psychology and Psychotherapy*, *20*(1), 55-66

Festinger, L. (1954). A theory of social comparison processes. *Human Relations*, *7*(2), 117-140

Field, A. E., Aneja, P., Austin, S. B., Shrier, L. A., Moor, C., & Gordon-Larsen, P. (2007). Race and gender differences in the association of dieting and gains in BMI among young adults. *Obesity*, *15*(2), 456-464

Field, A. E., Manson, J. E., Taylor, C. B., Willett, W. C., & Colditz, G. A. (2004). Association of weight change, weight control practices, and weight cycling among women in the Nurses' Health Study II. *International Journal of Obesity*, *28*(9), 1134-1142

Field, T. M. (1998). Touch therapy effects on development. *International Journal of Behavioral Development*, 22, 779-797

Fitzgibbon, M. L., & Kirschenbaum, D. S. (1990). Heterogeneity of clinical presentation among obese individuals seeking treatment. *Addictive Behaviors, 15*(3), 291-295

Flora, D. B., & Curran, P. J. (2004). An empirical evaluation of alternative methods of estimation for confirmatory factor analysis with ordinal data. *Psychological Methods*, *9*(4), 466-491

Fogel, C. I. (1993). Hard T: the stressful nature of incarceration for women. *Issues in Mental Health Nursing*, *14*(4), 367-377

Fogelholm, M., & Kukkonen-Harjula, K. (2001). Does physical activity prevent weight gain – A systematic review. *Obesity Reviews, 1,* 95-111

Folkman, S., & Lazarus, R .S. (1985). If it changes it must be a process: Study of emotion and coping during three stages of a college examination. *Journal of Personality and Social Psychology, 48*, 150-170

Foreyt, J. P., Brunner, R. L., Goodrick, G. K., Cutter, G., Brownell, K. D., & St Jeor, S. T. (1995). Psychological correlates of weight fluctuation. *International Journal of Eating Disorders*, *17*(3), 263-275

Foreyt, J. P., & Carlos Poston, W. S. (1998). What is the role of Cognitive-Behavior Therapy in patient management? *Obesity Research, 6*(1), 18S-22S

Foreyt, J. P., Carlos Poston, W. S., & Goodrick, G. K. (1996). Future directions in obesity and eating disorders. *Addictive Behaviors*, *21*(6), 767-778

Foreyt, J. P. & Goodrick, G. K. (1994). Attributes of successful approaches to weight loss and control. *Applied and Preventive Psychology, 3,* 209-215

Fornari, V., Kent, J., Kabo, L., & Goodman, B. (1994). Anorexia nervosa:"Thirty something". *Journal of Substance Abuse Treatment*, *11*(1), 45-54

Forster, J. L. & Jeffery, R. W. (1986). Gender differences related to weight history, eating patterns, efficacy expectations, self-esteem, and weight loss among participants in a weight reduction program. *Addictive Behaviors, 11,* 141-147

Foster, G. D., & Kendall, P. C. (1994). The realistic treatment of obesity: Changing the scales of success. *Clinical Psychology Review*, *14*(8), 701-736

Foster, G. D., Wadden, T. A., Kendall, P. C., Stunkard, A. J. & Vogt, R. A. (1996). Psychological effects of weight loss and regain: A prospective evaluation. *Journal of Consulting and Clinical Psychology, 64*(4), 752-757

Fowler-Brown, A. G., Bennett, G. G., Goodman, M. S., Wee, C. C., Corbie-Smith, G. M., & James, S. A. (2009). Psychosocial stress and 13-year BMI change among Blacks: The Pitt County Study. *Obesity*, *17*(11), 2106-2109

Frattaroli, J. (2006). Experimental disclosure and its moderators: A meta-analysis. *Psychological Bulletin, 132*(6), 823-865

Freedman, D. M., Ron, E., Ballard-Barbash, R., Doody, M. M., & Linet, M. S. (2006). Body mass index and all-cause mortality in a nationwide US cohort. *International Journal of Obesity*, *30*(5), 822-829

Freire, R. H., Borges, M. C., Alvarez, J. I., & Correia, M. I. T. D. (2012). Food quality, physical activity, and nutritional follow-up as determinant of weight regain after Roux-en-Y gastric bypass. *Nutrition, 28,* 53-58

French, S. A., & Jeffery, R. W. (1994). Consequences of dieting to lose weight: Effects on physical and mental health. *Health Psychology*, *13*(3), 195-212

French, S. A., Jeffery, R. W., Folsom, A. R., McGovern, P. & Williamson, D. F. (1996). Weight loss maintenance in young adulthood: Prevalence and correlations with health behavior and disease in a population-based sample of women aged 55-69 years. *International Journal of Obesity, 20,* 303-310

French, S. A., Jeffery, R. W., & Forster, J. L. (1994). Dieting status and its relationship to weight, dietary intake, and physical activity changes over two years in a working population. *Obesity Research*, *2*(2), 135-144

French, S. A., Jeffery, R. W., Forster, J. L., McGovern, P. G., Kelder, S. H., & Baxter, J. E. (1994). Predictors of weight change over two years among a population of working adults: The Healthy Worker Project. *International Journal of Obesity*, *18*(3), 145-154

Frisina, P. G., Borod, J. C. & Lepore, S. J. (2004). A meta-analysis of the effects of written emotional disclosure on the health outcomes of clinical populations. *The Journal of Nervous and Mental Disease, 192*(9), 629-634

Fryer, S., Waller, G., & Kroese, B. S. (1997). Stress, coping, and disturbed eating attitudes in teenage girls. *International Journal of Eating Disorders*, *22*(4), 427-436

Gale, C., Gilbert, P., Read, N., & Goss, K. (2012). An evaluation of the impact of introducing compassion focused therapy to a standard treatment programme for people with eating disorders. *Clinical Psychology and Psychotherapy*

Garnefski, N., Van Den Kommer, T., Kraaij, V., Teerds, J., Legerstee, J., & Onstein, E. (2002). The relationship between cognitive emotion regulation strategies and emotional problems: Comparison between a clinical and a non-clinical sample. *European Journal of Personality*, *16*(5), 403-420

Garner, D. (1991). *Eating Disorder Inventory-2.* Odessa, FL: Psychological Assessment Resources

Garner, D. (2004). *Eating Disorder Inventory-3. Professional manual.* Lutz, FL: Psychological Assessment Resources

Garner, D. M., & Garfinkel, P. E. (1979). The Eating Attitudes Test: An index of the symptoms of anorexia nervosa. *Psychological Medicine*, *9*(2), 273-279

Garner, D. M., Olmsted, M. P., Bohr, Y., & Garfinkel, P. E. (1982). The eating attitudes test: Psychometric features and clinical correlates. *Psychological Medicine*, *12*(4), 871-878

Garner, D. M., Olmstead, M. P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders, 2*, 15-34

Garrow, J. S., & Webster, J. (1985). Quetlet's index (W/H^2) as a measure of fatness. *International Journal of Obesity, 9,* 147-153

George, C., Kaplan, N., & Main, M. (1984). *Attachment interview for adults*. Unpublished manuscript, University of California, Berkeley

Gerace, T. A., & George, V. A. (1996). Predictors of weight increases over 7 years in fire fighters and paramedics. *Preventive Medicine*, *25*(5), 593-600

Gilbert, P. (1993). Defence and safety: Their function in social behaviour and psychopathology. *British Journal of Clinical Psychology*, *32*(2), 131-153

Gilbert, P. (1995). Biopsychosocial approaches and evolutionary theory as aids to integration in clinical psychology and psychotherapy. *Clinical Psychology and Psychotherapy*, *2*(3), 135-156

Gilbert, P. (1997). The evolution of social attractiveness and its role in shame, humiliation, guilt and therapy. *British Journal of Medical Psychology*, *70*(2), 113-147

Gilbert, P. (2000). The relationship of shame, social anxiety and depression: The role of the evaluation of social rank. *Clinical Psychology and Psychotherapy*, *7*(3), 174-189

Gilbert, P. (2001). Depression and stress: A biopsychosocial exploration of evolved functions and mechanisms. *Stress: The International Journal on the Biology of Stress*, *4*(2), 121-135

Gilbert, P. (2001). Evolution and social anxiety: The role of attraction, social competition, and social hierarchies. *Psychiatric Clinics of North America, 24*(4), 723-751

Gilbert, P. (2001). Evolutionary approaches to psychopathology: The role of natural defences. *Australian and New Zealand Journal of Psychiatry*, *35*(1), 17-27

Gilbert, P. (2004). Depression: A biopsychosocial, integrative and evolutionary approach. In M. Power (Ed.), *Mood disorders: A handbook of Science and Practice* (pp. 99-142). Chichester: Wiley

Gilbert, P. (2005). Compassion and cruelty: A biopsychosocial approach. In P. Gilbert (Ed.), *Compassion: Conceptualisations, research and use in psychotherapy* (pp. 9-74). London: Routledge

Gilbert, P. (2009). Introducing compassion-focused therapy. *Advances in Psychiatric Treatment*, *15*(3), 199-208

Gilbert, P., & Allan, S. (1994). Assertiveness, submissive behaviour and social comparison. *British Journal of Clinical Psychology*, *33*(3), 295-306

Gilbert, P., & Allan, S. (1998). The role of defeat and entrapment (arrested flight) in depression: An exploration of an evolutionary view. *Psychological Medicine*, *28*(3), 585-598

Gilbert, P., Allan, S., Brough, S., Melley, S., & Miles, J. N. V. (2002). Relationship of anhedonia and anxiety to social rank, defeat and entrapment. *Journal of Affective Disorders*, *71*(1), 141-151

Gilbert, P., Allan, S., & Goss, K. (1996). Parental representations, shame, interpersonal problems, and vulnerability to psychopathology. *Clinical Psychology and Psychotherapy*, *3*(1), 23-34

Gilbert, P., Allan, S., & Trent, D. R. (1995). Involuntary subordination or dependency as key dimensions of depressive vulnerability? *Journal of Clinical Psychology*, *51*(6), 740-752

Gilbert, P. Baldwin, M.W., Irons, C., Baccus, J.R. & Palmer, M. (2006). Self-criticism and self-warmth: An imagery study exploring their relation to depression. *Journal of Cognitive Psychotherapy, 20,* 183-200

Gilbert, P., Boxall, M., Cheung, M., & Irons, C. (2005). The relation of paranoid ideation and social anxiety in a mixed clinical population. *Clinical Psychology and Psychotherapy*, *12*(2), 124-133

Gilbert, P., Broomhead, C., Irons, C., McEwan, K., Bellew, R., Mills, A., Gale, C., & Knibb, R. (2007). Development of a striving to avoid inferiority scale. *British Journal of Social Psychology*, *46*(3), 633-648

Gilbert, P., Clarke, M., Hempel, S., Miles, J. N. V., & Irons, C. (2004). Criticizing and reassuring oneself: An exploration of forms, styles and reasons in female students. *British Journal of Clinical Psychology, 43*, 31-50

Gilbert, P., Cheung, M., Grandfield, T., Campey, F., & Irons, C. (2003). Recall of threat and submissiveness in childhood: Development of a new scale and its relationship with depression, social comparison and shame. *Clinical Psychology and Psychotherapy*, *10*(2), 108-115

Gilbert, P., & Irons, C. (2004). A pilot exploration of the use of compassionate images in a group of self-critical people. *Memory*, *12*(4), 507-516

Gilbert, P., Irons, C., Olsen, K., Gilbert, J., & McEwan, K. (2006). Interpersonal sensitivities: Their links to mood, anger and gender. *Psychology and Psychotherapy: Theory, Research and Practice*, *79*(1), 37-51

Gilbert, P., McEwan, K., Bellew, R., Mills, A., & Gale, C. (2009). The dark side of competition: How competitive behaviour and striving to avoid inferiority are linked to depression, anxiety, stress and self-harm. *Psychology and Psychotherapy: Theory, Research and Practice*, *82*(2), 123-136

Gilbert, P., McEwan, K., Irons, C., Bhundia, R., Christie, R., Broomhead, C., & Rockliff, H. (2010). Self-harm in a mixed clinical population: The roles of self-criticism, shame, and social rank. *British Journal of Clinical Psychology*, *49*(4), 563-576

Gilbert, P., McEwan, K., Matos, M., & Rivis, A. (2011). Fears of compassion: Development of self-report measures. *Psychology and Psychotherapy: Theory, Research and Practice, 84*, 239-255

Gilbert, P., & McGuire, M. T. (1998). Shame, social roles and status: The psychobiological continuum from monley to human. In P. Gilbert & B. Andrews (Ed.), *Shame: Interpersonal behavior, psychopathology and culture* (pp. 99-125). New York: Oxford University Press

Gilbert, P., & Miles, J. N. (2000a). Evolution, genes, development and psychopathology. *Clinical Psychology and Psychotherapy*, *7*(4), 246-255

Gilbert, P., & Miles, J. N. (2000b). Sensitivity to social put-down: It's relationship to perceptions of social rank, shame, social anxiety, depression, anger and self-other blame. *Personality and Individual Differences*, *29*(4), 757-774

Gilbert, P., Price, J., & Allan, S. (1995). Social comparison, social attractiveness and evolution: How might they be related? *New Ideas in Psychology*, *13*(2), 149-165

Gilbert, P., & Procter, S. (2006). Compassionate mind training for people with high shame and self-criticism: Overview and pilot study of a group therapy approach. *Clinical Psychology and Psychotherapy, 13,* 353-379

Gillespie, B. L., & Eider, R. M. (1992). Development of the Feminine Gender Role Stress scale: A cognitive/behavioral measure of stress, appraisal, and coping for women. *Behavior Modification, 16*, 426-438

Giskes, K., Avendano, M., & Kunst, A. E. (2010). A systematic review of studies on socioeconomic inequalities in dietary intakes associated with weight gain and overweight/obesity conducted among European adults. *Obesity Reviews, 11,* 413-429

Giskes, K., Kamphuis, C. B. M., van Lenthe, F. J., Kremers, S., Droomers, M., & Brug, J. (2007). A systematic review of associations between environmental factors, energy and fat intakes among adults: Is there evidence for environments that encourage obesogenic dietary intakes? *Public Health Nutrition, 10*(10), 1005-1017

Giskes, K., van Lenthe, F., Avendano-Pabon, M., & Brug, J. (2011). A systematic review of environmental factors and obesogenic dietary intakes among adults: Are we getting closer to understanding obesogenic environments? *Obesity Reviews, 12,* e95-e106

Glanz, K., Baderm, M. D. M., & Iyer, S. (2012). Retail grocery store marketing strategies and obesity: An integrative review. *American Journal of Preventive Medicine, 42*(5), 503-512

Glenny, A. M., O'Meara, S., Melville, A., Sheldon, T. A., & Wilson, C. (1997). The treatment and prevention of obesity: A systematic review of the literature. *International Journal of Obesity*, *21*(9), 715-737

Goodyer, I. M. (1990). *Life experiences, development and childhood psychopathology*. Chichester: Wiley

Gordon, K. C., Baucom, D. H., & Snyder, D. K. (2004). An integrative intervention for promoting recovery from extramarital affairs. *Journal of Marital and Family Therapy*, *30*(2), 213-231

Gore, S. A., Brown, D. M., & Smith West, D. (2003). The role of postpartum weight retention in obesity among women: A review of the evidence. *Annals of Behavioral Medicine, 26*(2), 149-159

Gorin, A. A., Phelan, S., Hill, J. O., & Wing, R. R. (2004). Medical triggers are associated with better short-and long-term weight loss outcomes. *Preventive Medicine*, *39*(3), 612-616

Gormally, J., & Rardin, D. (1981). Weight loss and maintenance and changes in diet and exercise for behavioral counseling and nutrition education. *Journal of Counseling Psychology*, *28*(4), 295-304

Gormally, J., Rardin, D., & Black, S. (1980). Correlates of successful response to a behavioral weight control clinic. *Journal of Clinical Psychology, 27*(2), 179-191

Gorman, D. M. (1993). A review of studies comparing checklist and interview methods of data collection in life event research. *Behavioral Medicine*, *19*(2), 66-73

Goss, K., & Allan, S. (2010). Compassion focused therapy for eating disorders. *International Journal of Cognitive Therapy, 3*(2), 141-158

Goss, K., Gilbert, P., & Allan, S. (1994). An exploration of shame measures: I: The `other as shamer scale'. *Personality and Individual Differences, 17*, 713-717

Graham, J. W. (2009). Missing data analysis: Making it work in the real world. *Annual Review of Psychology*, *60*, 549-576

Grave, R. D. (2011). Eating disorders: Progress and challenges. *European Journal of Internal Medicine*, *22*(2), 153-160

Gray, L. J., Cooper, N., Dunkley, A., Warren, F. C., Ara, R., Abrams, K., Davies, A. J., Khunti, K., & Sutton, A. (2012). A systematic review and mixed treatment comparison of pharmacological interventions for the treatment of obesity. *Obesity Reviews, 13*, 483-498

Greenberg, M. A., & Stone, A. A. (1992). Emotional disclosure about traumas and its relation to health: Effects of previous disclosure and trauma severity. *Journal of Personality and Social Psychology*, *63*(1), 75-84

Greenberg, M. A., Wortman, C. B., & Stone, A. A. (1996). Emotional expression and physical heath: Revising traumatic memories or fostering self-regulation? *Journal of Personality and Social Psychology*, *71*(3), 588-602

Green, C. A., & Pope, C. R. (1999). Gender, psychosocial factors and the use of medical services: A longitudinal analysis. *Social Science and Medicine, 48*, 1363-1372

Greeno, C. G., & Wing, R. R. (1994). Stress-induced eating. *Psychological Bulletin, 115*(3), 444-464

Grief, S. N., & Miranda, R. L. F. (2010). Weight loss maintenance. *American Family Physician, 82*(6), 630-634

Grilo, C. M., Pagano, M. E., Stout, R. L., Markowitz, J. C., Ansell, E. B., Pinto, A., Zanarini, M. C., Yen, S., & Skodol, A. E. (2012). Stressful life events predict eating disorder relapse following remission: Six-year prospective outcomes. *International Journal of Eating Disorders*, *45*(2), 185-192

Groesz, L. M., McCoy, S., Carl, J., Saslow, L., Stewart, J., Adler, N., Laraia, B., & Epel, E. (2012). What is eating you? Stress and the drive to eat. *Appetite*, *58*(2), 717-721

Gruen, R. J., Silva, R., Ehrlich, J., Schweitzer, J. W., & Friedhoff, A. J. (1997). Vulnerability to stress: Self-criticism and stress-induced changes in biochemistry. *Journal of Personality*, *65*(1), 33-47

Gueorguieva, R., & Krystal, J. H. (2004). Move over ANOVA: progress in analyzing repeated-measures data and its reflection in papers published in the Archives of General Psychiatry. *Archives of General Psychiatry*, *61*(3), 310-317

Gustafson, T. B., & Sarwer, D. B. (2004). Childhood sexual abuse and obesity. *Obesity Reviews, 5,* 129-135

Haaga, D. A. F., Yarmus, M., Hubbard, S., Brody, C., Solomon, A., Kirk, L., & Chamberlain, J. (2002). Mood dependency of self-rated attachment style. *Cognitive Therapy and Research, 26,* 57-71

Habhab, S., Sheldon, J. P., & Loeb, R. C. (2009). The relationship between stress, dietary restraint, and food preferences in women. *Appetite*, *52*(2), 437-444

Hainer, V., Kunesova, M., Bellisle, F., Parizkova, J., Braunerova, R., Wagenknecht, M., Lajka, J., Hill, M., & Stunkard, A. (2006). The Eating Inventory, body adiposity and

prevalence of diseases in a quota sample of Czech adults. *International Journal of Obesity*, *30*(5), 830-836

Haller, E. (1992). Eating disorders. A review and update. *Western Journal of Medicine*, *157*(6), 658-662

Halliwell, E., & Harvey, M., (2006). Examination of a sociocultural model of disordered eating among male and female adolescents. *British Journal of Health Psychology, 11*, 235-248

Hammen, C. (2005). Stress and depression. *Annual Reviews of Clinical Psychology*, *1*, 293-319

Harlow, H. F., Rowland, G. L., & Griffin, G. A. (1964). The effect of total social deprivation on the development of monkey behavior. *Psychiatric Research Reports*, *19*, 116-135

Harris, A. H. S. (2006). Does expressive writing reduce health care utilization? A meta-analysis of randomized trials. *Journal of Consulting and Clinical Psychology, 74*(2), 243-252

Hartigan, K. J., Baker-Strauch, D., & Morris, G. W. (1982). Perceptions of the causes of obesity and responsiveness to treatment. *Journal of Clinical Psychology, 29*(5), 478-485

Hartz, A. J., Kalkhoff, R. K., Rimm, A. A., & McCall, R. J. (1979). A study of factors associated with the ability to maintain weight loss. *Preventive Medicine, 8,* 471-483

Hayes, R. J. (1988). Methods for assessing whether change depends on initial value. *Statistics in Medicine*, *7*(9), 915-927

Hays, N. P., Bathalon, G. P., McCrory, M. A., Roubenoff, R., Lipman, R., & Roberts, S. B. (2002). Eating behavior correlates of adult weight gain and obesity in healthy women aged 55–65 y. *The American Journal of Clinical Nutrition*, *75*(3), 476-483

Hazan, C., & Shaver, P. (1987). Romantic love conceptualized as an attachment process. *Journal of Personality and Social Psychology*, *52*(3), 511-524

Hazan, C., & Shaver, P. (1990). Love and work: An attachment-theoretical perspective. *Journal of Personality and Social Psychology, 59,* 270-280

Heatherton, T. F., Mahamedi, F., Striepe, M., Field, A. E., & Keel, P. (1997). A 10-year longitudinal study of body weight, dieting, and eating disorder symptoms. *Journal of Abnormal Psychology*, *106*(1), 117-125

Heatherton, T. F., Nichols, P., Mahamedi, F., & Keel, P. (1995). Body weight, dieting, and eating disorder symptoms among college students, 1982 to 1992. *American Journal of Psychiatry*, *152*(11), 1623-1629

Heatherton, T. F., Polivy, J., & Herman, C. P. (1991). Restraint, weight loss, and variability of body weight. *Journal of Abnormal Psychology*, *100*(1), 78-83

Heitmann, B. L., & Garby, L. (1999). Patterns of long-term weight changes in overweight developing Danish men and women aged between 30 and 60 years. *International Journal of Obesity*, *23*(10), 1074-1078

Henry, J. P. (1982). The relation of social to biological processes in disease. *Social Science and Medicine*, *16*(4), 369-380

Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of Personality*, *43*(4), 647-660

Herzog, D. B., Dorer, D. J., Keel, P. K., Selwyn, S. E., Ekeblad, E. R., Flores, A. T., Greenwood, D. N., Burwell, R. A., & Keller, M. B. (1999). Recovery and relapse in anorexia and bulimia nervosa: A 7.5-year follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *38*(7), 829-837

Herzog, D. B., Sacks, N. R., Keller, M. B., & Lavori, P. W. (1993). Patterns and predictors of recovery in anorexia nervosa and bulimia nervosa. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*, 835-842

Herzog, W., Schellberg, D., & Deter, H. C. (1997). First recovery in anorexia nervosa patients in the long-term course: a discrete-time survival analysis. *Journal of Consulting and Clinical Psychology, 65*(1), 169-177

Hill, J. O., Thompson, H., & Wyatt, H. (2005). Weight maintenance: What's missing? *Journal of the American Dietetic Association*, *105*(5), 63-66

Hiskey, S. (2002). *Intrusive experiences and psychological adjustment following trauma*. Unpublished doctoral dissertation, University of Essex, Essex, United Kingdom

Hoek, H. W., & Van Hoeken, D. (2003). Review of the prevalence and incidence of eating disorders. *International Journal of Eating Disorders*, *34*(4), 383-396

Holmes, T. H., & Rahe, R. H. (1967). The social readjustment rating scale. *Journal of Psychosomatic Research*, *11*(2), 213-218

Hopman, W. M., Leroux, C., Berger, C., Joseph, L., Barr, S. I., Prior, J. C., Harrison, M., Poliquin, S., Towheed, T., Anastassiades, T., Goltzman, D., & the CaMos Research Group. (2007). Changes in body mass index in Canadians over a five-year period: Results of a prospective, population-based study. *BMC Public Health*, *7*(1), 150-160

Horikawa, C., Kodama, S., Yachi, Y., Heianza, Y., Hirasawa, R., Ibe, Y., Saito, K., Shimano, H., Yamada, N., & Hirohito, S. (2011). Skipping breakfast and prevalence of overweight and obesity in Asian and Pacific regions: A meta-analysis. *Preventive Medicine, 53,* 260-267

Horne, J. (2011). Obesity and short sleep: Unlikely bedfellows? *Obesity Reviews, 12,* e84-e94

Horowitz, L. M., & Vitkus, J. (1986). The interpersonal basis of psychiatric symptoms. *Clinical Psychology Review*, *6*(5), 443-469

House, A. O., & Andrews, H. B. (1988). Life events and difficulties preceding the onset of functional dysphonia. *Journal of Psychosomatic Research, 32*, 311-319

Hsu, L. G. (1997). Can dieting cause an eating disorder? *Psychological Medicine*, *27*(03), 509-513

Hsu, L. K. G., Benotti, P. N., Dwyer, J., Roberts, S. B., Saltzman, E., Shikora, S., Rolls, B. J., & Rand, W. (1998). Nonsurgical factors that influence the outcome of bariatric surgery: A review. *Psychosomatic Medicine, 60,* 338-346

Hsu, L. K. G., Sullivan, S. P., & Benotti, P. N. (2007). Eating disturbances and outcome of gastric bypass surgery: A pilot study. *Journal of Eating Disorders, 21*(4), 385-390

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling, 6,* 1-55

Imrie, S., & Troop, N. A. (2012). The effectiveness and feasibility of compassion-focused expressive writing in Day Hospice patients. *Palliative and Supportive Care, 10*, 115-122

Irons, C., & Gilbert, P. (2005). Evolved mechanisms in adolescent anxiety and depression symptoms: The role of the attachment and social rank systems. *Journal of Adolescence*, *28*(3), 325-341

Isner, J. M., Sours, H. E., Paris, A. L., Ferrans, V. J., & Roberts, W. C. (1979). Sudden, unexpected death in avid dieters using the liquid-protein-modified-fast diet. Observations in 17 patients and the role of the prolonged QT interval. *Circulation, 60,* 1401-1412

Iversen, L. B., Strandberg-Larsen, K., Prescott, E., Schnohr, P., & Rod, N. H. (2012). Psychosocial risk factors, weight changes and risk of obesity: The Copenhagen City Heart Study. *European Journal of Epidemiology, 27*(2), 119-130

Jackson, M., Ball, K., & Crawford, D. (2001). Beliefs about the causes of weight change in the Australian population. *International Journal of Obesity, 25*(10), 1512-1516

Jakicic, J. M. (2011). The effect of physical activity on body weight. *Obesity, 17*(S3), S34-S38

Jakicic, J. M., & Davis, K. K. (2011). Obesity and physical activity. *Psychiatric Clinics of North America, 34,* 829-840

James, W. P. T., Caterson, I. D., Coutinho, W., Finer, N., Van Gaal, L. F., Maggioni, A. P., Torp-Pedersen, C., Sharma, A. M., Shepherd, G. M., Rode, R. A., & Renz, C. L. (2010). Effect of sibutramine on cardiovascular outcomes in overweight and obese subjects. *New England Journal of Medicine, 363*(10), 905-917

Janoff-Bulman, R. (1992). *Shattered assumptions: Towards a new psychology of trauma*. New York: Free Press

Jáuregui-Garrido, B., & Jáuregui-Lobera, I. (2012). Sudden death in eating disorders. *Vascular Health and Risk Management*, *8*, 91-98

Jeffery, R. W., Bjornson-Benson, W. M., Rosenthal, B. S., Kurth, C. L., & Dunn, M. M. (1984a). Effectiveness of monetary contracts with two repayment schedules on weight reduction in men and women from self-referred and population samples. *Behavior Therapy, 15,* 273-279

Jeffery, R. W., Bjornson-Benson, W. M., Rosenthal, B. S., Lindquist, R. A., Kurth, C. L., & Johnson, S. L. (1984b). Correlates of weight loss and its maintenance over two years follow-up among middle-aged men. *Preventive Medicine, 13,* 155-168

Jeffery, R. W., Drewnowski, A., Epstein, L. H., Stunkard, A. J., Wilson, G. T., Wing, R. R., & Hill, D. R. (2000). Long-term maintenance of weight loss: Current status. *Health Psychology*, *19*(1S), 5-16

Jeffery, R. W., McGuire, M. T., & French, S. A. (2002). Prevalence and correlates of large weight gains and losses. *International Journal of Obesity*, *26*(7), 969-972

Jeffery, R. W., Wing, R. R., Sherwood, N. E., & Tate, D. F. (2003). Physical activity and weight loss: Does prescribing higher physical activity goals improve outcome? *American Journal of Clinical Nutrition, 78,* 684-689

Jeffery, R. W., Wing, R. R., Thorson, C., & Burton, L. R. (1998). Use of personal trainers and financial incentives to increase exercise in a behavioral weight-loss program. *Journal of Consulting and Clinical Psychology, 66*(5), 777-783

Jéquier, E., & Tappy, L. (1999). Regulation of body weight in humans. *Physiological Reviews*, *79*(2), 451-480

Johnson, D., & Drenick, E. J. (1977). Therapeutic fasting in morbid obesity: Long-term follow-up. *Archives of Internal Medicine*, *137*(10), 1381-1382

Johnson, F., Pratt, M., & Wardle, J. (2012). Dietary restraint and self-regulation in eating behaviour. *International Journal of Obesity, 36,* 665-674

Johnson, S. L., Leedom, L. J., & Muhtadie, L. (2012). The dominance behavioral system and psychopathology: Evidence from self-report, observational, and biological studies. *Psychological Bulletin*, *138*(4), 692-743

Jones, D. C. (2001). Social comparison and body image: Attractiveness comparisons to models and peers among adolescent girls and boys. *Sex Roles*, *45*(9-10), 645-664

Jones, L. R., Wilson, C. I., & Wadden, T. A. (2007). Lifestyle modification in the treatment of obesity: An educational challenge and opportunity. *Nature, 81*(5), 776-779

Joseph, S., Linley, S. J., Harwood, J., Lewis, C. A., & McCollam, P. (2004). Rapid assessment well-being: The Short Depression-Happiness Scale (SDHS). *Psychology and Psychotherapy: Theory, Research and Practice, 77,* 463-478

Kalarchian, M. A., & Marcus, M. D. (2003). Management of the bariatric surgery patient: Is there a role for the Cognitive Behavior Therapist? *Cognitive and Behavioral Practice, 10,* 112-119

Kalarchian, M. A., Marcus, M. D., Courcoulas, A. P., Cheng, Y., Levine, M. D., & Josbeno, D. (2012). Optimizing long-term weight control after bariatric surgery: A pilot study. *Surgery for Obesity and Related Diseases, 8,* 710-716

Kanazawa, S., & Kovar, J. L. (2004). Why beautiful people are more intelligent. *Intelligence*, *32*(3), 227-243

Kaplan, A. S., Walsh, B. T., Olmsted, M., Attia, E., Carter, J. C., Devlin, M. J., Pike, K. M., Woodside, D. B., Rockert, W., Roberto, C. A., & Parides, M. (2009). The slippery slope: Prediction of successful weight maintenance in anorexia nervosa. *Psychological Medicine*, *39*(6), 1037-1045

Karasek, R., Brisson, C., Kawakami, N., Houtman, I., Bongers, P., & Amick, B. (1998). The Job Content Questionnaire (JCQ): An instrument for internationally comparative assessments of psychosocial job characteristics. *Journal of Occupational Health Psychology*, *3*(4), 322-355

Karwautz, A. F. K., Rabe-Hesketh, S., Hu, X., Zhao, J., Sham, P., Collier, D. A., & Treasure, J. L. (2001). Individual-specific risk factors for anorexia nervosa: A pilot study using a discordant sister-pair design. *Psychological Medicine*, *31*(02), 317-329

Karwautz, A. F. K., Wagner, G., Waldherr, K., Nader, I. W., Fernandez-Aranda, F., Estivill, X., Holiday, J., Collier, D. A., & Treasure, J. L. (2011). Gene-environment interaction in anorexia nervosa: Relevance of non-shared environment and the serotonin transporter gene. *Molecular Psychiatry*, *16*(6), 590-592

Katahn, M., Pleas, J., Thackrey, M., & Wallston, K. A. (1982). Relationship of eating and activity self-reports to follow-up weight maintenance in the massively obese. *Behavior Therapy, 13,* 521-528

Katzmarzyk, P. T., Pérusse, L., Malina, R. M., & Bouchard, C. (1999). Seven-year stability of indicators of obesity and adipose tissue distribution in the Canadian population. *The American Journal of Clinical Nutrition*, *69*(6), 1123-1129

Kaye, W. H., Nagata, T., Weltzin, T. E., Hsu, L. G., Sokol, M. S., McConaha, C., Plotnicov, K. H., Weise, J., & Deep, D. (2001). Double-blind placebo-controlled administration of fluoxetine in restricting-and restricting-purging-type anorexia nervosa. *Biological Psychiatry*, *49*(7), 644-652

Kaye, W. H., Weltzin, T. E., Hsu, L. K., & Bulik, C. M. (1991). An open trial of fluoxetine in patients with anorexia nervosa. *Journal of Clinical Psychiatry, 52,* 464-471

Kayman, S., Bruvold, W., & Stern, J. S. (1990). Maintenance and relapse after weight loss: Behavioral aspects. *American Journal of Clinical Nutrition, 52,* 800-807

Keel, P. K., Baxter, M. G., Heatherton, T. F., & Joiner, T. E. (2007). A 20-year longitudinal study of body weight, dieting, and eating disorder symptoms. *Journal of Abnormal Psychology, 116(2)*, 422-432

Keel, P. K., Dorer, D. J., Franko, D. L., Jackson, S. C., & Herzog, D. B. (2005). Postremission predictors of relapse in women with eating disorders. *American Journal of Psychiatry*, *162*(12), 2263-2268

Kelly, A. C., Carter, J. C., Zuroff, D. C., & Borairi, S. (2013). Self-compassion and fear of self-compassion interact to predict response to eating disorders treatment: A preliminary investigation. *Psychotherapy Research*, *23*(3), 252-264

Kelly, A. C., Carter, J. C., & Borairi, S. (2013). Are improvements in shame and self-compassion early in eating disorders treatment associated with better patient outcomes? *International Journal of Eating Disorders*

Kelly, A. C., Zuroff, D. C., Foa, C. L., & Gilbert, P. (2010). Who benefits from training in self-compassionate self-regulation? A study of smoking reduction. *Journal of Social and Clinical Psychology*, *29*(7), 727-755

Kelly, A. C., Zuroff, D. C., & Shapira, L. B. (2009). Soothing oneself and resisting self-attacks: The treatment of two intrapersonal deficits in depression vulnerability. *Cognitive Therapy and Research*, *33*(3), 301-313

Kendall, P. C., Marrs-Garcia, A., Nath, S. R., & Sheldrick, R. C. (1999). Normative comparisons for the evaluation of clinical significance. *Journal of Consulting and Clinical Psychology*, *67*(3), 285-299

Keys, A., Fidanza, F., Karvonen, M. J., Kimura, N., & Taylor, H. L. (1972). Indices of relative weight and obesity. *Journal of Chronic Diseases*, *25*(6), 329-343

Kidd, T., Hamer, M., & Steptoe, A. (2011). Examining the association between adult attachment style and cortisol responses to acute stress. *Psychoneuroendocrinology, 36,* 771-779

Kiefer, I., Rathmanner, T., & Kunze, M. (2005). Eating and dieting differences in men and women. *The Journal of Men's Health and Gender*, *2*(2), 194-201

King, K. A., Vidourek, R., & Schwiebert, M. (2009). Disordered eating and job stress among nurses. *Journal of Nursing Management*, *17*(7), 861-869

King, L. A. (2001). The health benefits of writing about life goals. *Personality and Social Psychology Bulletin, 27*(7), 798-807

King, L. A., & Milner, K. N. (2000). Writing about the perceived benefits of traumatic events: Implications for physical health. *Personality and Social Psychology Bulletin, 26,* 220-230

Kissane, N. A., & Pratt, J. S. A. (2011). Medical and surgical treatments of obesity. *Best Practice and Research Clinical Anaesthesiology, 25,* 11-25

Kivimäki, M., Head, J., Ferrie, J. E., Shipley, M. J., Brunner, E., Vahtera, J., & Marmot, M. G. (2006). Work stress, weight gain and weight loss: Evidence for bidirectional effects of job strain on body mass index in the Whitehall II study. *International Journal of Obesity*, *30*(6), 982-987

Kjelsås, E., Bjørnstrøm, C., & Götestam, K. G. (2004). Prevalence of eating disorders in female and male adolescents (14-15 years). *Eating Behaviors, 5,* 13-25

Klemchuk, H. P., Hutchinson, C. B., & Frank, R. I. (1990). Body dissatisfaction and eating-related problems on the college campus: Usefulness of the Eating Disorder Inventory with a nonclinical population. *Journal of Counseling Psychology, 37*(3), 297-305

Klem, M. L., Wing, R. R., Lang, W., McGuire, M. T., & Hill, J. O. (2000). Does weight loss maintenance become easier over time? *Obesity Research, 8*(6), 438-444

Klem, M. L., Wing, R. R., Simkin-Silverman, L., & Kuller, L. H. (1997). The psychological consequences of weight gain prevention in healthy, premenopausal women. *International Journal of Eating Disorders*, *21*(2), 167-174

Klesges, R. C., Isbell, T. R., & Klesges, L. M. (1992). Relationship between dietary restraint, energy intake, physical activity, and body weight: A prospective analysis. *Journal of Abnormal Psychology*, *101*(4), 668-674

Klesges, R. C., Klesges, L. M., Haddock, C. K., & Eck, L. H. (1992). A longitudinal analysis of the impact of dietary intake and physical activity on weight change in adults. *The American Journal of Clinical Nutrition*, *55*(4), 818-822

Klingenberg, L., Sjödin, A. M., Holmbäck, U., Astrup, A., & Chaput, J. P. (2012). Short sleep duration and its association with energy metabolism. *Obesity Reviews, 13,* 565-577

Kloss, J. D., & Lisman, S. A. (2002). An exposure-based examination of the effects of written emotional disclosure. *British Journal of Health Psychology*, *7*(1), 31-46

Klump, K. L., McGue, M., & Iacono, W. G. (2000). Age differences in genetic and environmental influences on eating attitudes and behaviors in preadolescent and adolescent female twins. *Journal of Abnormal Psychology*, *109*(2), 239-251

Knutson, K. L. (2012). Does inadequate sleep play a role in vulnerability to obesity? *American Journal of Human Biology, 24,* 361-371

Knutson, K. L., & Van Cauter, E. (2008). Associations between sleep loss and increased risk of obesity and diabetes. *Annals of the New York Academy of Sciences, 1129,* 287-304

Kobak, R. R., & Sceery, A. (1988). Attachment in late adolescence: Working models, affect regulation, and representations of self and others. *Child Development, 59*(1), 135-146

Kobak, R. R., Sudler, N., & Gamble, W. (1991). Attachment and depressive symptoms during adolescence: A developmental pathways analysis. *Development and Psychopathology, 3*(4), 461-474

Kofman, M. D., Lent, M. R., & Swencionis, C. (2010). Maladaptive eating patterns, quality of life, and weight outcomes following gastric bypass: Results of an internet survey. *Obesity, 18,* 1938-1943

Kohn, P. M., Lafreniere, K., & Gurevhich, M. (1990). The inventory of college student's recent life experiences: A decontaminated hassles scale for a special population. *Journal of Behavioral Medicine, 13*, 619-630

Koopmans, G. T., & Lamers, L. M. (2007). Gender and health care utilization: the role of mental distress and help-seeking propensity. *Social Science and Medicine, 64*(6), 1216-1230

Kopelman, P. (2007). *Health Risks Associated with Overweight and Obesity*. Short Science Review. Foresight Tackling Obesities: Future Choices. *Obesity Reviews, 8*(s1), 13-17. URL: http://www.foresight.gov.uk

Korkeila, M., Kaprio, J., Rissanen, A., Koskenvuo, M., & Sörensen, T. I. A. (1998). Predictors of major weight gain in adult Finns: Stress, life satisfaction and personality traits. *International Journal of Obesity, 22,* 949-957

Korkeila, M., Rissanen, A., Kaprio, J., Sørensen, T. I., & Koskenvuo, M. (1999). Weight-loss attempts and risk of major weight gain: A prospective study in Finnish adults. *The American Journal of Clinical Nutrition*, *70*(6), 965-975

Koskenvuo, M., Langinvainio, H., Kaprio, J., Rantasalo, I., & Sarna, S. (1979). *The Finnish Twin Registry: Baseline characteristics, Section III – Occupational and Psychosocial Factors.* Helsinki, Finland: University Press

Kouvonen, A., Kivimäki, M., Cox, S. J., Cox, T., & Vahtera, J. (2005). Relationship between work stress and body mass index among 45,810 female and male employees. *Psychosomatic Medicine*, *67*(4), 577-583

Kraemer, H. C., Berkowitz, R. I., & Hammer, L. D. (1990). Methodological difficulties in studies in obesity: I. Measurement issues. *Annals of Behavioral Medicine, 12*, 112-118

Kramer, F. M., Jeffery, R. W., Forster, J. L., & Snell, M. K. (1989). Long-term follow-up of behavioral treatment for obesity: Patterns of weight regain among men and women. *International Journal of Obesity, 13,* 123-136

Kramer, F. M., Jeffery, R. W., Snell, M. K., & Forster, J. L. (1986). Maintenance of successful weight loss over 1 year: Effects of financial contracts for weight maintenance or participation in skills training. *Behavior Therapy*, *17*(3), 295-301

Kroke, A., Liese, A. D., Schulz, M., Bergmann, M. M., Klipstein-Grobusch, K., Hoffmann, K., & Boeing, H. (2002). Recent weight changes and weight cycling as predictors of subsequent two year weight change in a middle-aged cohort. *International Journal of Obesity, 26*(3), 403-409

Kruger, J., Blanck, H. M., & Gillespie, C. (2006). Dietary and physical activity behaviors among adults successful at weight loss maintenance. *International Journal of Behavioral Nutrition and Physical Activity*, *3*(1), 17-26

Kuczmarski, R. J. (1992). Prevalence of overweight and weight gain in the United States. *The American Journal of Clinical Nutrition*, *55*(2), 495S-502S

Kupeli, N., Chilcot, J., Schmidt, U. H., Campbell, I. C., & Troop, N. A. (2013a). A Confirmatory Factor Analysis and Validation of the Forms of Self-Criticism/Reassurance Scale, *British Journal of Clinical Psychology, 52,* 12-25

Kupeli, N., Chilcot, J., Platts, E., & Troop, N. A. (2013b). Stress, mood and eating pathology: The role of self-criticism, self-reassurance and self-esteem

Kupeli, N., Norton, S., Chilcot, J., Campbell, I. C., Schmidt, U. H., & Troop, N. A. (2013c). Predicting the trajectory of body mass index, disordered eating and stress: The role of mood and affect systems

Kupeli, N., Norton, S., Chilcot, J., Schmidt, U. H., Campbell, I. C., & Troop, N. A. (2013d). A Confirmatory Factor Analysis and validation of the Vulnerable Attachment Style Questionnaire

Kyrou, I., & Tsigos, C. (2009). Stress hormones: Physiological stress and regulation of metabolism. *Current Opinion in Pharmacology, 9*(6), 787-793

Lachat, C., Nago, E., Verstraeten, R., Roberfroid, D., Van Camp, J., & Kolsteren, P. (2012). Eating out of home and its association with dietary intake: A systematic review of the evidence. *Obesity Reviews, 13,* 329-346

Lachowycz, K., & Jones, A. P. (2011). Greenspace and obesity: A systematic review of the evidence. *Obesity Reviews, 12,* e183-e189

Lafont, J. (2011). *A comparison of expressive writing effects on body image: Symptomatic vs. asymptomatic Women* (Masters dissertation, Texas State University)

Laithwaite, H., O'Hanlon, M., Collins, P., Doyle, P., Abraham, L., Porter, S., & Gumley, A. (2009). Recovery after psychosis (RAP): A compassion focused programme for individuals residing in high security settings. *Behavioural and Cognitive Psychotherapy*, *37*(5), 511-526

Lane, R. C. (2002). Anorexia, masochism, self-mutilation, and autoerotism: the spider mother. *Psychoanalytic review*, *89*(1), 101-123

Lang, T., Hauser, R., Buddeberg, C., & Klaghofer, R. (2002). Impact of gastric banding on eating behavior and weight. *Obesity Surgery, 12,* 100-107

Latzer, Y., & Hochdorf, Z. (2005). Dying to be thin: Attachment to death in Anorexia Nervosa. *TheScientificWorldJOURNAL*, *5*, 820-827

Laurino Neto, R. M., Herbella, F. A. M., Tauil, R. M., Silva, F. S., & de Lima Jr, S. E. (2012). Comorbidities remission after Roux-en-Y gastric bypass for morbid obesity is sustained in a long-term follow-up and correlates with weight regain. *Obesity Surgery, 22,* 1580-1585

Leary, M. R., Tate, E. B., Adams, C. E., Allen, A. B., & Hancock, J. (2007). Self-compassion and reactions to unpleasant self-relevant events: The implications of treating oneself kindly. *Journal of Personality and Social Psychology, 92*, 887-904

Lee, D. A., (2005). The perfect nurturer: A model to develop a compassionate mind within the context of cognitive therapy. In Gilbert, P. (Ed.), *Compassion: Conceptualisations, Research and Use in Psychotherapy.* (pp. 326-351). London: Brunner Routledge

Lee, S., Lee, A. M., Leung, T., & Yu, H. (1997). Psychometric properties of the eating disorders inventory (EDI-1) in a nonclinical Chinese population in Hong Kong. *International Journal of Eating Disorders*, *21*(2), 187-194

Lemon, S. C., Rosal, M. C., Zapka, J., Borg, A., & Andersen, V. (2009). Contributions of weight perceptions to weight loss attempts: Differences by body mass index and gender. *Body Image*, *6*(2), 90-96

Leon, G. R., Fulkerson, J. A., Perry, C. L., & Cudeck, R. (1993). Personality and behavioral vulnerabilities associated with risk status for eating disorders in adolescent girls. *Journal of Abnormal Psychology, 102(3),* 438-444

Leon, G. R., Fulkerson, J. A., Perry, C. L., & Early-Zald, M. B. (1995). Prospective analysis of personality and behavioral vulnerabilities and gender influences in the later development of disordered eating. *Journal of Abnormal Psychology, 104(1),* 140-149

Lepore, S. J. (1997). Expressive writing moderates the relation between intrusive thoughts and depressive symptoms. *Journal of Personality and Social Psychology*, *73*(5), 1030-1037

Lepore, S. J., & Greenberg, M. A. (2002). Mending broken hearts: Effects of expressive writing on mood, cognitive processing, social adjustment and health following a relationship breakup. *Psychology and Health*, *17*(5), 547-560

Lepore, S. J., Greenberg, M. A., Bruno, M., & Smyth, J. M. (2002). Expressive writing and health: Self-regulation of emotion-related experience, physiology, and

behavior. In S. J. Lepore & J. M. Smyth (Eds.), *The writing cure: How expressive writing promotes health and emotional well-being* (pp. 99-117). Washington DC, US: American Psychological Association

Leproult, R., & Van Cauter, E. (2010). Role of sleep and sleep loss in hormonal release and metabolism. *Endocrine Development, 17,* 11-21

Levine, M. D., & Marcus, M. D. (1997). Eating behavior following stress in women with and without bulimic symptoms. *Annals of Behavioral Medicine*, *19*(2), 132-138

Levine, M. P., & Murnen, S. K. (2009). "Everybody knows that mass media are/are not [pick one] a cause of eating disorders": A critical review of evidence for a causal link between media, negative body image, and disordered eating in females. *Journal of Social and Clinical Psychology*, *28*(1), 9-42

Lewinsohn, P. M., Seeley, J. R., Moerk, K. C., & Striegel-Moore, R. H. (2002). Gender differences in eating disorder symptoms in young adults. *International Journal of Eating Disorders, 32(4),* 426-440

Liao, K. L. (2000). Cognitive-behavioural approaches and weight management: An overview. *The Journal of the Royal Society for the Promotion of Health, 120*(1), 27-30

Lincoln, T. M., Hohenhaus, F., & Hartmann, M. (2013). Can paranoid thoughts be reduced by targeting negative emotions and self-esteem? An experimental investigation of a brief compassion-focused intervention. *Cognitive Therapy and Research, 37*, 1-13

Lindroos, A. K., Lissner, L., Mathiassen, M. E., Karlsson, J., Sullivan, M., Bengtsson, C., & Sjöström, L. (1997). Dietary intake in relation to restrained eating, disinhibition, and hunger in obese and nonobese Swedish women. *Obesity Research*, *5*(3), 175-182

Liotti, G., & Gilbert, P. (2011). Mentalizing, motivation, and social mentalities: Theoretical considerations and implications for psychotherapy. *Psychology and Psychotherapy: Theory, Research and Practice*, *84*(1), 9-25

Livhitsm, M., Mercado, C., Yermilov, I., Parikh, J. A., Dutson, E., Mehran, A., Ko, C. Y., & Gibbons, M. M. (2011). Patient behaviors associated with weight regain after laparoscopic gastric bypass. *Obesity Research and Clinical Practice, 5,* e258-e265

Longe, O., Maratos, F. A., Gilbert, P., Evans, G., Volker, F., Rockliff, H., & Rippon, G. (2010). Having a word with yourself: Neural correlates of self-criticism and self-reassurance. *Neuroimage*, *49*(2), 1849-1856

Loos, R. J. F. (2012). Genetic determinants of common obesity and their value in prediction. *Best Practice and Research Clinical Endocrinology and Metabolism, 26,* 211-226

Lorenz, K. (1981). The foundations of ethology. New York: Springer-Verlag

Lo Russo, L., Campisi, G., Di Fede, O., Di Liberto, C., Panzarella, V., & Lo Muzio, L. (2008). Oral manifestations of eating disorders: A critical review. *Oral Diseases*, *14*(6), 479-484

Loth, K., van den Berg, P., Eisenberg, M. E., & Neumark-Sztainer, D. (2008). Stressful life events and disordered eating behaviors: Findings from Project EAT. *Journal of Adolescent Health*, *43*(5), 514-516

Lovasi, G. S., Hutson, M. A., Guerra, M., & Neckerman, K. M. (2009). Built environments and obesity in disadvantaged populations. *Epidemiologic Reviews, 31*, 7-20

Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories*. Behaviour Research and Therapy, 33*(3), 335-343

Low, C. A., Stanton, A. L., & Danoff-Burg, S. (2006). Expressive disclosure and benefit finding among breast cancer patients: Mechanisms for positive health effects. *Health Psychology, 25*, 181-189

Lowe, G. (2006). Health-related effects of creative and expressive writing. *Health Education*, *106*(1), 60-70

Lowe, M. R. (1993). The effects of dieting on eating behavior: A three-factor model. *Psychological Bulletin*, *114*, 100-121

Lowe, M. R., Annunziato, R. A., Markowitz, J. T., Didie, E., Bellace, D. L., Riddell, L., Maille, C., McKinney, S., & Stice, E. (2006). Multiple types of dieting prospectively predict weight gain during the freshman year of college. *Appetite, 47*, 83-90

Lowe, M. R., Miller-Kovach, K., & Phelan, S. (2001). Weight-loss maintenance in overweight individuals one to five years following successful completion of a

commercial weight loss program. *International Journal of Obesity Related Metabolic Disorders, 25,* 325-331

Lucassen, E. A., Rother, K. I., & Cizza, G. l. (2012). Interacting epidemics? Sleep curtailment, insulin resistance, and obesity. *Annals of the New York Academy of Sciences, 1264,* 110-134

Lyddon, W. J., Bradford, E., & Nelson, J. P. (1993). Assessing adolescent and adult attachment: A review of current self-report measures. *Journal of Counseling and Development*, *71*(4), 390-395

Lyons, E. J., Mehl, M. R., & Pennebaker, J. W. (2006). Pro-anorexics and recovering anorexics differ in their linguistic Internet self-presentation. *Journal of Psychosomatic Research, 60*, 253-256

Machado, P. P. P., Machado, B. C., Gonçlaves, S., & Hoek, H. W. (2007). The prevalence of Eating Disorders Not Otherwise Specified. *International Journal of Eating Disorders, 40*(3), 212-217

Macht, M., & Simons, G. (2000). Emotions and eating in everyday life. *Appetite, 35*(1), 65-71

Maes, H. H. M., Neale, M. C., & Eaves, L. J. (1997). Genetic and environmental factors in relative body weight and human adiposity. *Behavior Genetics, 27*(4), 325-351

Maggard, M. A., Shugarman, L. R., Suttorp, M., Maglione, M., Sugarman, H. J., Livingston, E. H., Nguyen, N. T., Li, Z., Mojlca, W. A., Hilton, L., Rhodes, S., Morton, S. C., & Shekelle, P. G. (2005). Meta-analysis: Surgical treatment of obesity. *Annals of Internal Medicine, 142,* 547–559

Magro, D. O., Geloneze, B., Delfini, R., Pareja, B. C., Callejas, F., & Pareja, J. C. (2008). Long-term weight regain after gastric bypass: A 5-year prospective study. *Obesity Surgery, 18,* 648-651

Main, M., Kaplan, N., & Cassidy, J. (1985). Security in infancy, childhood, and adulthood: A move to the level of representation. *Monographs of the Society for Research in Child Development, 50*(1/2), 66-104

Makoundou, V., Bobbioni-Harsch, E., Gachoud, J. P., Habicht, F., Pataky, Z., & Golay, A. (2010). A 2-year multifactor approach of weight loss maintenance. *Eating and Weight Disorders*, *15*(1-2), e9-e14

Malkoff-Schwartz, S., Frank, E., Anderson, B. P., Hlastala, S. A., Luther, J. F., Sherrill, J. T., Houck, P. R., & Kupfer, D. J. (2000). Social rhythm disruption and stressful life events in the onset of bipolar and unipolar episodes. *Psychological Medicine*, *30*(05), 1005-1016

Mann, T., Tomiyama, A. J., Westling, E., Lew, A. M., Samuels, B., & Chatman, J. (2007). Medicare's search for effective obesity treatments. *American Psychologist*, *62*(3), 220-233

Manson, J. E., & VanItallie, T. B. (1995). America's obesity epidemic and women's health. *Journal of Women's Health, 5*(4), 329-334

Marcos, A. (1997). Nutrition, immunology, neuroscience, and behaviour: Part VII. *Nutrition, 13*(10), 853-862

Marlo, H., & Wagner, M. K. (1999). Expression of negative and positive events through writing: Implications for psychotherapy and health. *Psychology and Health, 14*, 193-215

Marshall, N. S., Glozier, N., & Grunstein, R. R. (2008). Is sleep duration related to obesity? A critical review of the epidemiological evidence. *Sleep Medicine Reviews, 12,* 289-298

Marston, A. R., & Criss, J. (1984). Maintenance of successful weight loss: Incidence and prediction. *International Journal of Obesity, 8,* 435-439

Martí, A., Marcos, A., & Martínez, J. A. (2001). Obesity and immune function relationships. *Obesity Reviews, 2,* 131-140

Martz, D. M., Handley, K. B., & Eisler, R. M. (1995). The relationship between feminine gender role stress, body image, and eating disorders. *Psychology of Women Quarterly*, *19*(4), 493-508

Matos, M., Duarte, C., Stubbs, J., Gilbert, P., & Morris, L. (In press). Struggling to lose weight: The impact of shame, self-criticism and social rank on eating psychopathology in overweight and obese people.

Matud, M. P. (2004). Gender differences in stress and coping styles. *Personality and Individual Differences, 37*(7), 1401-1415

Mauri, M. C., Rudelli, R., Somaschini, E., Roncoroni, L., Papa, R., Mantero, M., Longhini, M., & Penati, G. (1996). Neurobiological and psychopharmacological

basis in the therapy of bulimia and anorexia. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *20*(2), 207-240

Mavanji, V., Billington, C. J., Kotz, C. M., & Teske, J. A. (2012). Sleep and obesity: A focus on animal models. *Neuroscience and Biobehavioral Reviews, 36,* 1015-1029

McCarthy, M. (1990). The thin ideal, depression and eating disorders in women. *Behaviour Research and Therapy*, *28*(3), 205-214

McCormick, B., & Stone, I. (2007). *Economic Costs of Obesity and the Case for Government Intervention*. Short Science Review. Foresight Tackling Obesities: Future Choices. *Obesity Reviews, 8*(s1), 161-164*.* URL: http://www.foresight.gov.uk

McFarlane, A. C., McFarlane, C. M., & Gilchrist, P. N. (1988). Posttraumatic bulimia and anorexia nervosa. *International Journal of Eating Disorders*, *7*(5), 705-708

McGuire, M. T., Wing, R. R., Klem, M. L., Lang, W., & Hill, J O. (1999). What predicts weight regain in a group of successful weight losers? *Journal of Consulting and Clinical Psychology, 67*(2), 177-185

McIntosh, V. V., Jordan, J., Carter, F. A., Luty, S. E., McKenzie, J. M., Bulik, C. M., Frampton, C. M. A., & Joyce, P. R. (2005). Three psychotherapies for anorexia nervosa: A randomized, controlled trial. *American Journal of Psychiatry*, *162*(4), 741-747

McIntosh, V. V., Jordan, J., Luty, S. E., Carter, F. A., McKenzie, J. M., Bulik, C. M., & Joyce, P. R. (2006). Specialist supportive clinical management for anorexia nervosa. *International Journal of Eating Disorders*, *39*(8), 625-632

McShane, K. E., & Cunningham, J. A. (2003). The role of relevancy in normative feedback for university students' drinking patterns. *Addictive Behaviors*, *28*(8), 1523-1528

Meads, C., Lyons, A., & Carroll, D. (2003). *The impact of the emotional disclosure intervention on physical and psychological health: A systematic review* (West Midlands Health Technology Assessment Collaboration Report). Birmingham, England: University of Birmingham

Meads, C., & Nouwen, A. (2005). Does emotional disclosure have any effects? A systematic review of the literature with meta-analyses. *International Journal of Technology Assessment in Health Care, 21*, 153-164

Meltzer, A. A., & Everhart, J. E. (1995). Self-reported substantial 1-year weight change among men and women in the United States. *Obesity Research*, *3*(S2), 123s-134s

Meredith, P., Strong, J., & Feeney, J. A. (2006). Adult attachment, anxiety, and pain self-efficacy as predictors of pain intensity and disability. *Pain, 123,* 146-154

Metalsky, G. I., & Joiner, T. E., Jr. (1992). Vulnerability to depressive symptomatology: A prospective test of the diathesis-stress and causal mediation components of the hopelessness theory of depression. *Journal of Personality and Social Psychology, 63*(4), 667-675

Micali, N., Hagberg, K. W., Petersen, I., & Treasure, J. L. (2013). The incidence of eating disorders in the UK in 2000–2009: Findings from the General Practice Research Database. *BMJ Open*, *3*(5), e002646

Mickelson, K. D., Kessler, R. C., & Shaver, P. R. (1997). Adult attachment in a nationally representative sample. *Journal of Personality and Social Psychology, 73,* 1092-1106

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, *133*(1), 25-45

Miller, W. I. (1997). The anatomy of disgust. Cambridge, USA; Harvard University Press

Miller, W. R., & Seligman, M. E. (1975). Depression and learned helplessness in man. *Journal of Abnormal Psychology*, *84*(3), 228-238

Mikulincer, M., Birnbaum, G., Woddis, D., & Nachmias, O. (2000). Stress and accessibility of proximity-related thoughts: Exploring the normative and intraindividual components of attachment theory. *Journal of Personality and Social Psychology*, *78*(3), 509-523

Mikulincer, M., Florian, V., & Tolmacz, R. (1990). Attachment styles and fear of personal death: A case study of affect regulation. *Journal of Personality and Social Psychology*, *58*(2), 273-280

Mintz, L. B., O'Halloran, M. S., Mulholland, A. M., & Schneider, P. A. (1997). Questionnaire for eating disorder diagnoses: Reliability and validity of

operationalizing DSM-IV criteria into a self-report format. *Journal of Counseling Psychology, 44*(1), 63-79

Misra, M. (2008). Long-term skeletal effects of eating disorders with onset in adolescence. *Annals of the New York Academy of Sciences*, *1135*(1), 212-218

Mitchell, J. E., Davis, L., & Goff, G. (1985). The process of relapse in patients with bulimia. *International Journal of Eating Disorders*, *4*(4), 457-463

Mogk, C., Otte, S., Reinhold-Hurley, B., & Kröner-Herwig, B. (2006). Health effects of expressive writing on stressful or traumatic experiences: A meta-analysis. *GMS Psycho-Social Medicine*, *3*, Doc06

Moldovan, A. R., & David, D. (2011). Effect of obesity treatment on eating behaviour: Psychosocial interventions versus surgical interventions. A systematic review. *Eating Behaviors, 12,* 161-167

Monroe, S. M. (2008). Modern approaches to conceptualizing and measuring human life stress. *Annual Review of Clinical Psychology*, *4*, 33-52

Moore, C. J., & Cunningham, S. A. (2012). Social position, psychological stress, and obesity: A systematic review. *Journal of the Academy of Nutrition and Dietetics, 112,* 518-526

Morylowska-Topolska, J., & Kośląk, A. (2010). Bioelements and eating disorders-aspects of the quality of life. *Journal of Elementology*, *15*(2), 405-412

Mowrer, O. H. (1960). *Learning theory and behavior*. New York: Wiley.

Mozaffarian, D., Hao, T., Rimm, E. B., Willett, W. C., & Hu, F. B. (2011). Changes in diet and lifestyle and long-term weight gain in women and men. *The New England Journal of Medicine, 364,* 2392-2404

Murphy, B., & Bates, G. W. (1997). Adult attachment style and vulnerability to depression. *Personality and Individual Differences, 22,* 835-844

Murphy, E. (1983). The prognosis of depression in old age. *The British Journal of Psychiatry*, *142*(2), 111-119

Mussap, A. J. (2007). The relationship between feminine gender role stress and disordered eating symptomatology in women. *Stress and Health*, *23*(5), 343-348

Muthén, L. K., & Muthén, B. O. (2010). *Mplus Version 6.* Los Angeles, CA: Muthén & Muthén

National Institute for Clinical Excellence (NICE). (2004). Eating disorders: Core interventions in the treatment and management of anorexia nervosa, bulimia nervosa and related eating disorders. NICE Clinical Guideline no 9. London: NICE. URL: http://www.nice.org.uk/nicemedia/pdf/CG9FullGuideline.pdf (accessed January, 2013)

National Institutes of Health Consensus Development Panel. (1991). Gastrointestinal surgery for severe obesity. *Annals of Internal Medicine, 115,* 956-961

National Task Force on the Prevention and Treatment of Obesity. (1993). Very low-calorie diets. *Journal of American Medical Association, 270,* 967-974

Needham, B. L., Epel, E. S., Adler, N. E., & Kiefe, C. (2010). Trajectories of change in obesity and symptoms of depression: The CARDIA study. *American Journal of Public Health*, *100*(6), 1040-1046

Neely, M. E., Schallert, D. L., Mohammed, S. S., Roberts, R. M., & Chen, Y. J. (2009). Self-kindness when facing stress: The role of self-compassion, goal regulation, and support in college students' well-being. *Motivation and Emotion*, *33*(1), 88-97

Neovius, K., Johansson, K., Kark, M., & Neovius, M. (2009). Obesity status and sick leave: A systematic review. *Obesity Reviews, 10,* 17-27

Neziroglu, F., Khemlani-Patel, S., & Veale, D. (2008). Social learning theory and cognitive behavioral models of body dysmorphic disorder. *Body Image*, *5*(1), 28-38

Nielsen, L. S., Danielsen, K. V., & Sørensen, T. I. A. (2011). Short sleep duration as a possible cause of obesity: Critical analysis of the epidemiological evidence. *Obesity Reviews, 12,* 78-92

Nielsen, S. (2001). Epidemiology and mortality of eating disorders. *Psychiatric Clinics of North America*, *24*(2), 201-214

Novak, K. B., & Crawford, L. A. (2001). Perceived drinking norms, attention to social comparison information, and alcohol use among college students. *Journal of Alcohol and Drug Education*, *46*(3), 18-33

Nygaard, J. A. (1990). Anorexia nervosa. Treatment and triggering factors. *Acta Psychiatrica Scandinavica*, *82*(s361), 44-49

O'Brien, K. M., & Vincent, N. K. (2003). Psychiatric comorbidity in anorexia and bulimia nervosa: nature, prevalence, and causal relationships. *Clinical Psychology Review*, *23*(1), 57-74

O'Connor, L. E., Berry, J. W., Weiss, J., & Gilbert, P. (2002). Guilt, fear, submission, and empathy in depression. *Journal of Affective Disorders*, *71*(1), 19-27

Odom, J., Zalesin, K. C., Washington, T. L., Miller, W. W., Hakmeh, B., Zaremba, D. L., Altattan, M., Balasubramaniam, M., Gibbs, D. S., Krause, K. R., Chengelis, D. L., Franklin, B. A., & McCullough, P. A. (2010). Behavioral predictors of weight regain after bariatric surgery. *Obesity Surgery, 20,* 349-356

Ogden, J., & Hills, L. (2008). Understanding sustained behavior change: The role of life crises and the process of reinvention. *Health: An International Journal, 12,* 419-437

Ogden, J., Stavrinaki, M., & Stubbs, J. (2009). Understanding the role of life events in weight loss and weight gain. *Psychology, Health and Medicine, 14*(2), 239-249

O'Kearney, R. (1996). Attachment disruption in Anorexia Nervosa and Bulimia Nervosa: A review of theory and empirical research. *International Journal of Eating Disorders, 20,* 115-127

Oliver, G., & Wardle, J. (1999). Perceived effects of stress on food choice. *Physiology and Behavior*, *66*(3), 511-515

O'Neil, C., Dietz, W., DiGirolamo, M., Kicklighter, J., Lahmayer, R., Pi-Sunyer, F. X., & Williamson, D. (1991). Panel discussion: Factors associated with maintenance of weight loss: Prevention of relapse. *Nutrition, 7*(4), 302-306

O'Shaughnessy, R., & Dallos, R. (2009). Attachment research and eating disorders: A review of the literature. *Clinical Child Psychology and Psychiatry, 14,* 559-574

Otagaki, Y., Yonezawa, H., Shiwa, S., Saito, H., & Nakamura, K. (2005). Self-esteem in eating disorders. *Japanese Journal of Psychosomatic Medicine, 45*(3), 225-231

Padwal, R. S., & Majumdar, S. R. (2007). Drug treatments for obesity: Orlistat, sibutramine, and rimonabant. *Lancet, 369,* 71-77

Pagano, M. E., Skodol, A. E., Stout, R. L., Shea, M. T., Yen, S., Grilo, C. M., Sanislow, C. A., Bender, D. S., McGlashan, T. H., Zanarini, M. C., & Gunderson, J. G. (2004). Stressful life events as predictors of functioning: Findings from the Collaborative

Longitudinal Personality Disorders Study. *Acta Psychiatrica Scandinavica, 110,* 421-429

Panossian, L. A., & Veasey, S. C. (2012). Daytime sleepiness in obesity: Mechanisms beyond Obstructive Sleep Apnea – A review. *Sleep, 35*(5), 605-615

Parker, G. A. (1974). Assessment strategy and the evolution of fighting behaviour. *Journal of Theoretical Biology*, 47, 223-243

Parham, E. S. (1990). The interaction of eating restraint and stressful life events on women's weight and weight changes. *Nutrition Research*, *10*(8), 831-835

Pasman, W. J., Saris, W. H. M., & Westerterp-Plantenga, M. S. (1999). Predictors of weight maintenance. *Obesity Research, 7*(1), 43-50

Patel, D. R., Phillips, E. L., & Pratt, H. D. (1998). Eating disorders. *Indian Journal of Pediatrics, 65,* 487-494

Patel, S. R., & Hu, F. B. (2008). Short sleep duration and weight gain: A systematic review. *Obesity, 16*(3), 643-653

Paykel, E. S. (1983a). Methodological aspects of life events research. *Journal of Psychosomatic Research*, *27*(5), 341-352

Paykel, E. S. (1983b). The classification of depression. *British Journal of Clinical Pharmacology*, *15*(S2), 155S-159S

Paykel, E. S. (1997). The interview for recent life events. *Psychological Medicine*, *27*(02), 301-310

Pearson, N., & Biddle, S. J. H. (2011). Sedentary behavior and dietary intake in children, adolescents, and adults: A systematic review. *American Journal of Preventive Medicine, 41*(2), 178-188

Pederson, K. J., Roerig, J. L., & Mitchell, J. E. (2003). Towards the pharmacotherapy of eating disorders. *Expert Opinion on Pharmacotherapy*, *4*(10), 1659-1678

Pekkarinen, T., Koskela, K., Huikuri, K., & Mustajoki, P. (1994). Long-term results of gastroplasty for morbid obesity: Binge-eating as a predictor of poor outcome. *Obesity Surgery, 4,* 248-255

Pennebaker, J. W. (1985). Traumatic experience and psychosomatic disease: Exploring the roles of behavioural inhibition, obsession, and confiding. *Canadian Psychology*, *26*(2), 82-95

Pennebaker, J. W. (1993). Putting stress into words: Health, linguistic, and therapeutic implications. *Behaviour Research and Therapy*, *31*(6), 539-548

Pennebaker, J. W. (1997). Writing about emotional experiences as a therapeutic process. *Psychological Science, 8,* 162-166

Pennebaker, J. W. (2002). Writing, social processes, and psychotherapy: From past to future. In S. Lepore & J. M. Smyth (Ed.), *The writing cure: how expressive writing promotes health and emotional well-being*. Washington, DC: American Psychological Association

Pennebaker, J. W. (2004). Theories, therapies, and taxpayers: On the complexities of the expressive writing paradigm. *Clinical Psychology: Science and Practice*, *11*(2), 138-142

Pennebaker, J. W., & Beall, S. K. (1986). Confronting a traumatic event: Toward an understanding of inhibition and disease. *Journal of Abnormal Psychology, 95*(3), 274-281

Pennebaker, J. W., Booth, R. J., & Francis, M. E. (2007). *Linguistic Inquiry and Word Count* (LIWC): LIWC 2007. Austin, Texas: liwc.net

Pennebaker, J. W., & King, L. A. (1999). Linguistic styles: Language use as an individual difference. *Journal of Personality and Social Psychology, 77*, 1296-1312

Pennebaker, J. W., Mayne, T. J., & Francis, M. E. (1997). Linguistic predictors of adaptive bereavement. *Journal of Personality and Social Psychology, 72*, 863-871

Pennebaker, J. W., Mehl, M. R., & Niederhoffer, K. G. (2003). Psychological aspects of natural language use: Our words, our selves. *Annual Review of Psychology*, *54*(1), 547-577

Pennebaker, J. W., & Seagal, J. D. (1999). Forming a story: The health benefits of narrative. *Journal of Clinical Psychology*, *55*(10), 1243-1254

Perri, M. G. (1998). The maintenance of treatment effects in the long-term management of obesity. *Clinical Psychology: Science and Practice, 5*(4), 526-543

Perri, M. G., Shapiro, R. M., Ludwig, W. W., Twentyman, C. T., & McAdoo, W. G. (1984). Maintenance strategies for the treatment of obesity: An evaluation of relapse prevention training and posttreatment contact by mail and telephone. *Journal of Consulting and Clinical Psychology*, *52*(3), 404-413

Perry, B. D., Pollard, R. A., Blakley, T. L., Baker, W. L., & Vigilante, D. (1995). Childhood trauma, the neurobiology of adaptation, and "Use-dependent" development of the brain: How "states" become "traits". *Infant Mental Health Journal*, *16*(4), 271-291

Peterson, C. B., Crosby, R. D., Wonderlich, S. A., Joiner, T., Crow, S. J., Mitchell, J. E., Bardone-Cone, A. M., Klein, M., & le Grange, D. (2007). Psychometric properties of the eating disorder examination-questionnaire: Factor structure and internal consistency. *International Journal of Eating Disorders, 40*(4), 386-389

Pettem, O., West, M., Mahoney, A., & Keller, A. (1993). Depression and attachment problems. *Journal of Psychiatry and Neuroscience, 18,* 78-81

Phelan, S., & Wadden, T. A. (2002). Combining behavioral and pharmacological treatments for obesity. *Obesity Research, 10,* 560-574

Pike, K. M. (1998). Long-term course of anorexia nervosa: Response, relapse, remission, and recovery. *Clinical Psychology Review*, *18*(4), 447-475

Pike, K. M., Hilbert, A., Wilfley, D. E., Fairburn, C. G., Dohm, F. A., Walsh, B. T., & Striegel-Moore, R. H. (2008). Toward an understanding of risk factors for Anorexia Nervosa: A case-control study. *Psychological Medicine*, *38*(10), 1443-1453

Pike, K. M., Walsh, B. T., Vitousek, K., Wilson, G. T., & Bauer, J. (2003). Cognitive behavior therapy in the posthospitalization treatment of anorexia nervosa. *American Journal of Psychiatry*, *160*(11), 2046-2049

Pike, K. M., Wilfley, D., Hilbert, A., Fairburn, C. G., Dohm, F. A., & Striegel-Moore, R. H. (2006). Antecedent life events of binge-eating disorder. *Psychiatry Research*, *142*(1), 19-29

Pinto-Gouveia, J., Ferreira, C., & Duarte, C. (2012). Thinness in the pursuit for social safeness: An integrative model of social rank mentality to explain eating psychopathology. *Clinical Psychology and Psychotherapy*

Polivy, J., & Herman, C. P. (1985). Dieting and binging: A causal analysis. *American Psychologist*, *40*(2), 193-201

Polivy, J., & Herman, C. P. (1992). Undieting: A program to help people stop dieting. *International Journal of Eating Disorders*, *11*(3), 261-268

Polivy, J., & Herman, C. P. (2002). Causes of eating disorders. *Annual Review of Psychology*, *53*(1), 187-213

Preti, A., Rocchi, M. B. L., Sisti, D., Camboni, M. V., & Miotto, P. (2011). A comprehensive meta-analysis of the risk of suicide in eating disorders. *Acta Psychiatrica Scandinavica*, *124*(1), 6-17

Pretorius, N., Waller, G., Gowers, S., & Schmidt, U. (2009). Validity of the Eating Disorders Examination-Questionnaire when used with adolescents with bulimia nervosa and atypical bulimia nervosa. *Eating and Weight Disorders*, *14*(4), e243-e248

Price, J. S., & Sloman, L. (1987). Depression as yielding behavior: An animal model based on Schjelderup-Ebbe's pecking order. *Ethology and Sociobiology*, *8*, 85-98

Price, J., S., Sloman, L., Gardner, R., Gilbert, P., & Rohde, P. (1994). The social competition hypothesis of depression. *The British Journal of Psychiatry*, *164*(3), 309-315

Pronk, N. P., & Wing, R. R. (1994). Physical activity and long-term maintenance of weight loss. *Obesity Research, 2,* 587-599

Provencher, V., Drapeau, V., Tremblay, A., Després, J. P., & Lemieux, S. (2003). Eating behaviors and indexes of body composition in men and women from the Quebec family study. *Obesity Research*, *11*(6), 783-792

Provencher, V., Polivy, J., Wintre, M. G., Pratt, M. W., Pancer, S. M., Birnie-Lefcovitch, S., & Adams, G. R. (2009). Who gains or who loses weight? Psychosocial factors among first-year university students. *Physiology and Behavior*, *96*(1), 135-141

Raffi, A. R., Rondini, M., Grandi, S., & Fava, G. A. (2000). Life events and prodromal symptoms in bulimia nervosa. *Psychological Medicine*, *30*(03), 727-731

Raftery, A. E. (1995). Bayesian model selection in Social Research. *Sociological Methodology, 25,* 111-163

Råstam, M., & Gillberg, C. (1992). Background factors in anorexia nervosa: A controlled study of 51 teenage cases including a population sample. *European Child and Adolescent Psychiatry, 1*(1), 54-65

Ratnasuriya, R. H., Eisler, I., Szmukler, G. I., & Russell, G. F. (1991). Anorexia nervosa: Outcome and prognostic factors after 20 years. *The British Journal of Psychiatry*, *158*(4), 495-502

Ravitz, P., Maunder, R., Hunter, J., Sthankiya, B., & Lancee, W. (2010). Adult attachment measures: A 25-year review. *Journal of Psychosomatic Research*, *69*(4), 419-432

Ray, E. C., Nickels, M. W., Sayeed, S., & Sax, H. C. (2003). Predicting success after gastric bypass: The role of psychosocial and behavioral factors. *Surgery*, *134*(4), 555-563

Reed, G. W., & Hill, J. O. (1993). Weight cycling: A review of the animal literature. *Obesity Research, 1*(5), 392-402

Renalds, A., Smith, T. H., & Hale, P. J. (2010). A systematic review of built environment and health. *Family and Community Health, 33*(1), 68-78

Richter, A., Gilbert, P., & McEwan, K. (2009). Development of an early memories of warmth and safeness scale and its relationship to psychopathology. *Psychology and Psychotherapy: Theory, Research and Practice*, *82*(2), 171-184

Rieger, E., Van Buren, D. J., Bishop, M., Tanofsky-Kraff, M., Welch, R., & Wilfley, D. E. (2010). An eating disorder-specific model of interpersonal psychotherapy (IPT-ED): Causal pathways and treatment implications. *Clinical Psychology Review*, *30*(4), 400-410

Rippe, J. M., & Hess, S. (1998). The role of physical activity in the prevention and management of obesity. *Journal of American Dietetic Association, 98*(2), S31-S38

Roberts, A., & Ashley, G. (1999). What are the characteristics of overweight and obese patients who achieve weight loss and what factors are most helpful? A quantitative and qualitative study of patients and interventions in a rural general practice. *Journal of Human Nutrition and Dietetics*, *12*(1), 20-27

Roberts, C. J., Campbell, I. C., & Troop, N. (2013). Increases in weight during chronic stress are partially associated with a switch in food choice towards increased carbohydrate and saturated fat intake. *European Eating Disorders Review*

Roberts, C. J., Troop, N. T., Connan, F., Treasure, J., & Campbell, I. C. (2007). The effects of stress on body weight: Biological and psychological predictors of change in BMI. *Obesity, 15*(12), 3045-3055

Roberts, J. E., Gotlib, I. H., & Kassel, J. D. (1996). Adult attachment security and symptoms of depression: The mediating roles of dysfunctional attitudes and low self-esteem. *Journal of Personality and Social Psychology, 70,* 310-320

Robertson, J., & Bowlby, J. (1952). Responses of young children to separation from their mothers. *Courrier of the International Childrn's Centre*, *2*, 131-42

Robinson, P., & Serfaty, M. (2008). Getting better byte by byte: A pilot randomised controlled trial of email therapy for bulimia nervosa and binge eating disorder. *European Eating Disorders Review*, *16*(2), 84-93

Robroek, S. J., van den Berg, T. I., Plat, J. F., & Burdorf, A. (2011). The role of obesity and lifestyle behaviours in a productive workforce. *Occupational and Environmental Medicine*, *68*(2), 134-139

Rockliff, H., Gilbert, P., McEwan, K., Lightman, S., & Glover, D. (2008). A pilot exploration of heart rate variability and salivary cortisol responses to compassion-focused imagery. *Journal of Clinical Neuropsychiatry*, *5*, 132-139

Roerig, J. L., Steffen, K. J., Mitchell, J. E., & Zunker, C. (2010). Laxative abuse: Epidemiology, diagnosis and management. *Drugs*, *70*(12), 1487-1503

Rojo, L., Conesa, L., Bermudez, O., & Livianos, L. (2006). Influence of stress in the onset of eating disorders: Data from a two-stage epidemiologic controlled study. *Psychosomatic Medicine*, *68*(4), 628-635

Rolls, B. J., Fedoroff, I. C., & Guthrie, J. F. (1991). Gender differences in eating behavior and body weight regulation. *Health Psychology, 10*(2), 133-142

Rookus, M. A., Burema, J., & Frijters, J. E. (1988). Changes in body mass index in young adults in relation to number of life events experienced. *International Journal of Obesity*, *12*(1), 29-39

Rössner, S., & Flaten, H. (1997). VLCD versus LCD in long-term treatment of obesity. *International Journal of Obesity*, *21*(1), 22-26

Rowland, M. L. (1990). Self-reported weight and height. *American Journal of Clinical Nutrition, 52,* 1125-1133

Royal College of Psychiatrists. (2012). *Eating Disorders in the UK: Service distribution, service development and training*. Council Report CR170. London: Royal College of Psychiatrists

Ruderman, A. J. (1986). Dietary restraint: A theoretical and empirical review. *Psychological Bulletin*, *99*(2), 247-262

Rudiger, J. A., Cash, T. F., Roehrig, M., & Thompson, J. K. (2007). Day-to-day body-image states: Prospective predictors of intra-individual level and variability. *Body Image*, *4*(1), 1-9

Russell, E., Koren, G., Rieder, M., & Van Uum, S. (2012). Hair cortisol as a biological marker of chronic stress: Current status, future directions and unanswered questions. *Psychoneuroendocrinology*, *37*(5), 589-601

Russell, G. F., Szmukler, G. I., Dare, C., & Eisler, I. (1987). An evaluation of family therapy in anorexia nervosa and bulimia nervosa. *Archives of General Psychiatry*, *44*(12), 1047-1056

Santor, D., & Zuroff, D. (1997). Interpersonal responses to threats to status and interpersonal relatedness: Effects of dependency and self-criticism. *British Journal of Clinical Psychology, 36*, 521-541

Sapolsky, R. M. (2005). The influence of social hierarchy on primate health. *Science*, *308*(5722), 648-652

Sarason, I. G., Johnson, J. H., & Siegel, J. M. (1978). Assessing the impact of life changes: Development of the Life Experiences Survey. *Journal of Consulting and Clinical Psychology, 46,* 932-946

Saris, W. H. M., Blair, S. N., Van Baak, M. A., Eaton, S. B., Davies, P. S. W., Di Pietro, L., Fogelholm, M., Rissanen, A., Schoeller, D., Swinburn, B., Tremblay, A., Westerterp, K. R., & Wyatt, H. (2003). How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. *Obesity Reviews*, *4*(2), 101-114

Sarlio-Lähteenkorva, S. (2001). Weight loss and quality of life among obese people. *Social Indicators Research*, *54*(3), 329-354

Sarlio-Lähteenkorva, S., & Rissanen, A. (1998). Weight loss maintenance: Determinants of long-term success. *Eating and Weight Disorders, 3*(3), 131-135

Sarlio-Lähteenkorva, S., Rissanen, A., & Kaprio, J. (2000). A descriptive study of weight loss maintenance: 6 and 15 year follow-up of initially overweight adults. *International Journal of Obesity, 24,* 116-125

Sarwer, D. B., Dilks, R. J., & West-Smith, L. (2011). Dietary intake and eating behavior after bariatric surgery: Threats to weight loss maintenance and strategies for success. *Surgery for Obesity and Related Diseases, 7,* 644-651

Sarwer, D. B., von Sydow Green, A., Vetter, M. L., & Wadden, T. A. (2009). Behavior therapy for obesity: Where are we now? *Current Opinion in Endocrinology, Diabetes and Obesity, 16,* 347-352

Sarzynski, M. A., Jacobson, P., Rankinen, T., Carlsson, B., Sjöström, L., Bouchard, C., & Carlsson, L. M. S. (2011). Associations of markers in 11 obesity candidate genes with maximal weight loss and weight regain in the SOS bariatric surgery cases. *International Journal of Obesity, 35,* 676-683

Savage, J. S., & Birch, L. L. (2010). Patterns of weight control strategies predict differences in women's 4-year weight gain. *Obesity*, *18*(3), 513-520

Savage, J. S., Hoffman, L., & Birch, L. L. (2009). Dieting, restraint, and disinhibition predict women's weight change over 6 y. *The American Journal of Clinical Nutrition*, *90*(1), 33-40

Schmidt, U. H. (2003). Aetiology of eating disorders in the 21st century. *European Child and Adolescent Psychiatry*, *12*, 30-37

Schmidt, U. H., Bone, G., Hems, S., Lessem, J., & Treasure, J. (2002). Structured therapeutic writing tasks as an adjunct to treatment in eating disorders. *European Eating Disorders Review*, *10*(5), 299-315

Schmidt, U. H., Oldershaw, A., Jichi, F., Sternheim, L., Startup, H., McIntosh, V., Jordan, J., Tchanturia, K., Wolff, G., Rooney, M., Landau, S., & Treasure, J. (2012). Out-patient psychological therapies for adults with anorexia nervosa: Randomised controlled trial. *The British Journal of Psychiatry, 201*, 392-399

Schmidt, U., Tiller, J., Blanchard, M., Andrews, B., & Treasure, J. (1997). Is there a specific trauma precipitating anorexia nervosa? *Psychological Medicine*, *27*(03), 523-530

Schmidt, U. H., & Treasure, J. (2006). Anorexia nervosa: Valued and visible. A cognitive-interpersonal maintenance model and its implications for research and practice. *British Journal of Clinical Psychology*, *45*(3), 343-366

Schmier, J. K., Jones, M. L., & Halpern, M. T. (2006). Cost of obesity in the workplace. *Scandinavian Journal of Work, Environment and Health, 32*(1), 5-11

Selassie, M., & Sinha, A. C. (2011). The epidemiology and aetiology of obesity: A global challenge. *Best Practice and Research Clinical Anaesthesiology, 25,* 1-9

Selcuk, E., Zayas, V., Günaydin, G., Hazan, C., & Kross, E. (2012). Mental representations of attachment figures facilitate recovery following upsetting autobiographical memory recall. *Journal of Personality and Social Psychology, 103*(2), 362-378

Selye, H. (1936). A syndrome produced by diverse nocuous agents*. Nature, 38*, 32

Selye, H. (1976). *The stress of life (2nd ed.)*. New York: McGraw-Hill

Serlachius, A., Hamer, M., & Wardle, J. (2007). Stress and weight change in university students in the United Kingdom. *Physiology and Behavior*, *92*(4), 548-553

Serpell, L., Livingstone, A., Neiderman, M., & Lask, B. (2002). Anorexia nervosa: Obsessive-compulsive disorder, obsessive-compulsive personality disorder, or neither? *Clinical Psychology Review*, *22*(5), 647-669

Seymour, M., Hoerr, S. L., & Huang, Y. L. (1997). Inappropriate dieting behaviors and related lifestyle factors in young adults: Are college students different? *Journal of Nutrition Education*, *29*(1), 21-26

Shafran, R., Cooper, Z., & Fairburn, C. G. (2002). Clinical perfectionism: A cognitive-behavioural analysis. *Behaviour Research and Therapy, 40*(7), 773-791

Shah, M., Simha, V., & Garg, A. (2006). Long-term impact of bariatric surgery on body weight, comorbidities, and nutritional status. *The Journal of Clinical Endocrinology and Metabolism, 91*(11), 4223-4231

Sharpe, T. M., Ryst, E., Hinshaw, S. P., & Steiner, H. (1997). Reports of stress: A comparison between eating disordered and non-eating disordered adolescents. *Child Psychiatry and Human Development*, *28*(2), 117-132

Shea, M. E., & Pritchard, M. E. (2007). Is self-esteem the primary predictor of disordered eating? *Personality and Individual Differences*, *42*(8), 1527-1537

Shisslak, C. M., Renger, R., Sharpe, T., Crago, M., McKnight, K. M., Gray, N., Bryson, S., Estes, L. S., Parnaby, O. G., Killen, J., & Taylor, C. B. (1999). Development and evaluation of the McKnight Risk Factor Survey for assessing potential risk and protective factors for disordered eating in preadolescent and adolescent girls. *International Journal of Eating Disorders, 25*, 195-214

Shorey, H. A., & Snyder, C. R. (2006). The role of adult attachment styles in psychopathology and psychotherapy outcomes. *Review of General Psychology, 10,* 1-20

Siu, J., Giskes, K., Shaw, J., & Turrell, G. (2011). Perceived weight status may contribute to education inequalities in five-year weight change among mid-aged women. *Australian and New Zealand Journal of Public Health*, *35*(3), 284-291

Sjöström, L., Lindroos, A-K., Peltonen, M., Torgerson, J., Bouchard, C., Carlsson, B., Dahlgren, S., Larsson, B., Narbro, K., Sjöström, C. D., Sullivan, M., & Wedel, H. (2004). Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. *The New England Journal of Medicine, 351(26),* 2683-2693

Sjöström, L., Rissanen, A., Andersen, T., Boldrin, M., Golay, A., Koppeschaar, H. P., & Krempf, M. (1998). Randomised placebo-controlled trial of orlistat for weight loss and prevention of weight gain in obese patients. *Lancet, 352,* 167-173

Skender, M. L., Goodrick, K., Del Junco, D. J., Reeves, R. S., Darnell, L., Gotto, A. M., & Foreyt, J. P. (1996). Comparison of 2-year weight loss trends in behavioral treatments of obesity: Diet, exercise, and combination interventions. *Journal of American Dietetic Association, 96,* 342-346

Slatcher, R. B., & Pennebaker, J. W. (2006). How do I love thee? Let me count the words the social effects of expressive writing. *Psychological Science*, *17*(8), 660-664

Sloan, D. M., & Marx, B. P. (2004a). A closer examination of the structured written disclosure procedure. *Journal of Consulting and Clinical Psychology, 72*, 165-175

Sloan, D. M., & Marx, B. P. (2004b). Taking pen to hand: Evaluating theories underlying the written disclosure paradigm. *Clinical Psychology: Science and Practice, 11,* 121-137

Sloman, L. (2008). A new comprehensive evolutionary model of depression and anxiety. *Journal of Affective Disorders*, *106*(3), 219-228

Sloman, L., Atkinson, L., Milligan K., & Liotti, G. (2002). Attachment, social rank, and affect regulation: Speculations on an ethological approach to family interaction. *Family Process, 41*(3), 313-327

Sloman, L., Gilbert, P., & Hasey, G. (2003). Evolved mechanisms in depression: The role and interaction of attachment and social rank in depression. *Journal of Affective Disorders, 74,* 107-121

Smoller, J. W., Wadden, T. A., & Stunkard, A. J. (1987). Dieting and depression: A critical review. *Journal of Psychosomatic Research, 31,* 429-440

Smyth, J. M. (1998). Written emotional expressive: Effect sizes, outcome types, and moderating variables. *Journal of Consulting and Clinical Psychology, 66*(1), 174-184

Smyth, J. M., Heron, K. E., Wonderich, S. A., Crosby, R. D., & Thompson, K. M. (2008). The influence of reported trauma and adverse events on eating disturbance in young adults. *International Journal of Eating Disorders, 41,* 195-202

Smyth, J. M., Hockemeyer, J. R., Heron, K. E., Wonderlich, S. A., & Pennebaker, J. W. (2008). Prevalence, type, disclosure, and severity of adverse life events in college students. *Journal of American College Health*, *57*(1), 69-76

Smyth, J. M., & Pennebaker, J. W. (2008). Exploring the boundary conditions of expressive writing: In search of the right recipe. *British Journal of Health Psychology*, *13*(1), 1-7

Snyder, B., Nguyen, A., Scarbourough, T., Yu, S., & Wilson, E. (2009). Comparison of those who succeed in losing significant excessive weight after bariatric surgery and those who fail. *Surgical Endoscopy, 23,* 2302-2306

Sobal, J., & Stunkard, A. J. (1989). Socioeconomic status and obesity: A review of the literature. *Psychological Bulletin, 105*(2), 260-275

Sohlberg, S. (1990). Personality, life stress and the course of eating disorders. *Acta Psychiatrica Scandinavica*, *82*(s361), 29-33

Sørensen, T. I. A. (1995). The genetics of obesity. *Metabolism, 44*(9), 4-6

Sørensen, T. I., Rissanen, A., Korkeila, M., & Kaprio, J. (2005). Intention to lose weight, weight changes, and 18-y mortality in overweight individuals without co-morbidities. *PLoS Medicine*, *2*(6), e171

Sours, H. W., Frattali, V. P., Brand, D., Feldman, R. A., Forbes, A. L., Swanson, R. C., & Paris, A. L. (1981). Sudden death associated with very low calorie weight reduction regimes. *American Journal of Clinical Nutrition, 34,* 453-461

Spangler, G., & Grossmann, K. E. (1993). Biobehavioral organization in securely and insecurely attached infants. *Child Development*, *64*(5), 1439-1450

Speakman, J. R. (2004). Obesity: The integrated roles of environment and genetics. *The Journal of Nutrition, 134*(8), 2090S-2105S

Spiegel, D. (1999). Healing words. *The Journal of the American Medical Association*, *281*(14), 1328-1329

Spitzer, R. L., Devline, M., Walsh, B. T., Hasin, D., Wing, R., Marcus, M., Stunkard, A., Wadden, T., Yanovski, S., Agras, S., Mitchell, J., & Nonas, C. (1992). Binge eating disorder: A multi-site field trial of the diagnostic criteria. *International Journal of Eating Disorders, 11*, 191-203

Sroufe, L. A., & Waters, E. (1977). Attachment as an organizational construct. *Child Development*, 1184-1199

Stanton, A. L., Danoff-Burg, S., Sworowski, L. A., Collins, C. A., Branstetter, A. D., Rodriguez-Hanley, A., Kirk, S. B., & Austenfeld, J. L. (2002). Randomized, controlled trial of written emotional expression and benefit finding in breast cancer patients. *Journal of Clinical Oncology*, *20*(20), 4160-4168

Statistics Canada: Housing, Family and Social Statistics Division. (1991). *General Social Survey: Time Use Questionnaire GSS 7-2*. Canada: Statistics Canada

Steinhausen, H. C. (2002). The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry*, *159*(8), 1284-1293

Steinhausen, H. C., Gavez, S., & Winkler Metzke, C. (2005). Psychosocial correlates, outcome, and stability of abnormal adolescent eating behavior in community samples of young people. *International Journal of Eating Disorders*, *37*(2), 119-126

Stern, J. S., Hirsch, J., Blair, S. N., Foreyt, J. P., Frank, A., Kumanyika, S. K., Madans, J. H., Marlatt, G. A., St Jeor, S. T., & Stunkard, A. J. (1995). Weighing the options: Criteria for evaluating weight-management programs. The committee to develop criteria for evaluating the outcomes of approaches to prevent and treat obesity. *Obesity Research*, *3*(6), 591-604

Stevens, J., Chambless, L. E., Tyroler, H. A., Harp, J., Jones, D., & Arnett, D. (2001). Weight change among self-reported dieters and non-dieters in white and African American men and women. *European Journal of Epidemiology*, *17*(10), 917-923

Stevens, J., Knapp, R. G., Keil, J. E., & Verdugo, R. R. (1991). Changes in body weight and girths in black and white adults studied over a 25 year interval. *International Journal of Obesity*, *15*(12), 803-808

Stevens, J., Truesdale, K. P., McClain, J. E., & Cai, J. (2005). The definition of weight maintenance. *International Journal of Obesity*, *30*(3), 391-399

Stice, E. (1998). Prospective relation of dieting behaviors to weight change in a community sample of adolescents. *Behavior Therapy*, *29*(2), 277-297

Stice, E. (2002). Risk and maintenance factors for eating pathology: a meta-analytic review. *Psychological Bulletin*, *128*(5), 825-848

Stice, E., & Agras, W. S. (1998). Predicting onset and cessation of bulimic behaviors during adolescence: A longitudinal grouping analysis. *Behavior Therapy*, *29*(2), 257-276

Stice, E., & Shaw, H. E. (1994). Adverse effects of the media portrayed thin-ideal of women and linkages to bulimic symptomatology. *Journal of Social and Clinical Psychology, 13*, 288-308

St Jeor, S. T., Brunner, R. L., Harrington, M. E., Scott, B. J., Cutter, G. R., Brownell, K. D., Dyer, A. R., & Foreyt, J. P. (1995). Who are the weight maintainers? *Obesity Research, 3*(2), 249s-259s

Stone, A. A., & Brownell, K. D. (1994). The stress-eating paradox: Multiple daily measurements in adult males and females. *Psychology and Health*, *9*(6), 425-436

Stormer, S. M., & Thompson, J. K. (1996). Explanations of body image disturbance: A test of maturational status, negative verbal commentary, social comparison, and sociocultural hypotheses. *International Journal of Eating Disorders, 19*, 193-202

Strahan, E. J., Wilson, A. E., Cressman, K. E., & Buote, V. M. (2006). Comparing to perfection: How cultural norms for appearance affect social comparisons and self-image. *Body Image*, *3*(3), 211-227

Streiner, D. L. (1994). Figuring out factors: The use and misuse of factor analysis. *The Canadian Journal of Psychiatry, 39*, 135-140

Striegel-Moore, R. H., Dohm, F. A., Kraemer, H. C., Schreiber, G. B., Taylor, C. B., & Daniels, S. R. (2007). Risk factors for binge-eating disorders: An exploratory study. *International Journal of Eating Disorders*, *40*(6), 481-487

Striegel-Moore, R. H., Wilfley, D. E., Pike, K. M., Dohm, F. A., & Fairburn, C. G., (2000). Recurrent binge eating in black American women. *Archives of Family Medicine, 9*(1), 83-87

Strober, M., Freeman, R., DeAntonio, M., Lampert, C., & Diamond, J. (1997a). Does adjunctive fluoxetine influence the post-hospital course of restrictor-type anorexia nervosa? A 24-month prospective, longitudinal followup and comparison with historical controls. *Psychopharmacology Bulletin, 33*(3), 425-431

Strober, M., Freeman, R., & Morrell, W. (1997b). The long-term course of severe anorexia nervosa in adolescents: Survival analysis of recovery, relapse, and outcome predictors over 10–15 years in a prospective study. *International Journal of Eating Disorders*, *22*(4), 339-360

Stroud, L. R., Salovey, P., & Epel, E. S. (2002). Sex differences in stress responses: Social rejection versus achievement stress. *Biological Psychiatry*, *52*(4), 318-327

Strumia, R. (2005). Dermatologic signs in patients with eating disorders. *American Journal of Clinical Dermatology*, *6*(3), 165-173

Stubbs, C., & Lee, A. J. (2004). The obesity epidemic: Both energy intake and physical activity contribute. *Medical Journal of Australia, 181,* 489-491

Stunkard, A. J., Faith, M. S., & Allison, K. C. (2003). Depression and obesity. *Biological Psychiatry, 54,* 330-337

Stunkard, A. J., Harris, J. R., Pedersen, N. L., & McCleam, G. E. (1990). The body mass index of twins who have been reared apart. *The New England Journal of Medicine, 322*(21), 1483-1487

Stunkard, A., & McLaren-Hume, M. (1959). The results of treatment for obesity: A review of the literature and report of a series. *American Medical Association Archives of Internal Medicine, 103,* 79-85

Stunkard, A. J., & Rush, J. (1974). Dieting and depression re-examined: A critical review of reports of untoward responses during weight reduction for obesity. *Annals of Internal Medicine, 81,* 526-533

Sturman, E. D. (2011). Involuntary subordination and its relation to personality, mood, and submissive behavior. *Psychological Assessment*, *23*(1), 262-276

Sturman, E. D., & Mongrain, M. (2005). Self-criticism and major depression: An evolutionary perspective. *British Journal of Clinical Psychology*, *44*(4), 505-519

Sturman, E. D., & Mongrain, M. (2008). Entrapment and perceived status in graduate students experiencing a recurrence of major depression. *Canadian Journal of Behavioural Science*, *40*(3), 185-188

Sullivan, P. F. (1995). Mortality in anorexia nervosa. *American Journal of Psychiatry*, *152*(7), 1073-1074

Svetkey, L. P., Ard, J. D., Stevens, V. J., Loria, C. M., Young, D. Y., Hollis, J. F., Appel, L. J., Brantley, P. J., Kennedy, B. M., Kumanyika, S. K., Batch, B. C., Corsino, L., Lien, L.

F., & Vollmer, W. M. (2012). Predictors of long-term weight loss in adults with modest initial weight loss, by sex and race. *Obesity*, *20*(9), 1820-1828

Swallow, S. R., & Kuiper, N. A. (1988). Social comparison and negative self-evaluations: An application to depression. *Clinical Psychology Review*, *8*(1), 55-76

Sypeck, M. F., Gray, J. J., Etu, S. F., Ahrens, A. H., Mosimann, J. E., & Wiseman, C. V. (2006). Cultural representations of thinness in women, redux: *Playboy* magazine's depiction of beauty from 1979 to 1999. *Body Image*, *3*(3), 229-235

Talbot, T. S. (2010). The effects of viewing pro-eating disorder websites: a systematic review. *West Indian Medical Journal*, *59*(6), 686-697

Tangney, J. P., Wagner, P., & Gramzow, R. (1992). Proneness to shame, proneness to guilt, and psychopathology. *Journal of Abnormal Psychology, 101*, 469-478

Tate, D. F., Jeffery, R. W., Sherwood, N. E., & Wing, R. R. (2007). Long-term weight losses associated with prescription of higher physical activity goals. Are higher levels of physical activity protective against weight regain? *American Journal of Clinical Nutrition, 85*, 954-959

Taylor, C. B., Bryson, S. W., Altman, T. M., Abascal, L., Celio, A., Cunning, D., Killen, J. D., Shisslak, C. M., Crago, M., Ranger-Moore, J., Cook, P., Ruble, A., Olmsted, M. E., Kraemer, H. C., & Smolak, L. (2003). Risk factors for the onset of eating disorders in adolescent girls: Results of the McKnight longitudinal risk factor study. *American Journal of Psychiatry*, *160*(2), 248-254

Taylor, S. E., & Lobel, M. (1989). Social comparison activity under threat: Downward evaluation and upward contacts. *Psychological Review*, *96*(4), 569-575

The EHSRE Capri Workshop Group. (2006). Nutrition and reproduction in women. *Human Reproduction Update*, *12*(3), 193-207

Thomas, J. G., Butryn, M. L., Stice, E., & Lowe, M. R. (2011). A prospective test of the relation between weight change and risk for bulimia nervosa. *International Journal of Eating Disorders*, *44*(4), 295-303

Thompson, J. K., Coovert, M. D., & Stormer, S. M. (1999). Body image, social comparison, and eating disturbance: A covariance structure modeling investigation. *International Journal of Eating Disorders, 26*, 43-51

Thwaites, R., & Dagnan, D. (2004). Moderating variables in the relationship between social comparison and depression: An evolutionary perspective. *Psychology and Psychotherapy: Theory, Research and Practice*, *77*(3), 309-323

Tiggemann, M. (2004). Dietary restraint and self-esteem as predictors of weight gain over an 8-year time period. *Eating Behaviors*, *5*(3), 251-259

Tinker, J. E., & Tucker, J. A. (1997). Environmental events surrounding natural recovery from obesity. *Addictive Behaviors, 22*(4), 571-575

Tong, J., & D'Alessio, D. (2011). Eating disorders and gastrointestinal peptides. *Current Opinion in Endocrinology, Diabetes and Obesity*, *18*(1), 42-49

Treasure, J. L. (2005). Treatment of anorexia nervosa in adults. *Psychiatry*, *4*(4), 10-13

Treasure, J. L. (2007). Getting beneath the phenotype of anorexia nervosa: the search for viable endophenotypes and genotypes. *Canadian Journal of Psychiatry. 52*(4), 212-219

Treasure, J. L., & Owen, J. B. (1997). Intriguing links between animal behavior and anorexia nervosa. *International Journal of Eating Disorders*, *21*(4), 307-311

Treasure, J. L., Sepulveda, A. R., MacDonald, P., Whitaker, W., Lopez, C., Zabala, M., Kyriacou, O., & Todd, G. (2008). The assessment of the family of people with eating disorders. *European Eating Disorders Review, 16*(4), 247-255

Treasure, J., & Serpell, L. (2001). Osteoporosis in young people: Research and treatment in eating disorders. *Psychiatric Clinics of North America*, *24*(2), 359-370

Troop, N. A., Allan, S., Treasure, J. L., & Katzman, M. (2003). Social comparison and submissive behaviour in eating disorder patients. *Psychology and Psychotherapy-Theory Research and Practice, 76*, 237-249

Troop, N. A., Andrews, L., Hiskey, S., & Treasure, J. L. (2013). Social rank and symptom change in eating disorders: A 6-month longitudinal study. *Clinical Psychology and Psychotherapy*.

Troop, N. A., & Baker, A. H. (2008). The specificity of social rank in eating disorder versus depressive symptoms. *Eating Disorders*, *16*(4), 331-341

Troop, N. A., & Bifulco, A. (2002). Childhood social arena and cognitive sets in eating disorders. *British Journal of Clinical Psychology*, *41*(2), 205-211

Troop, N. A., Chilcot, J., Hutchings, L., & Varnaite, G. (2013). Expressive writing, self-criticism, and self-reassurance. *Psychology and Psychotherapy: Theory, Research and Practice, 86*(4), 374-386s

Troop, N. A., & Hiskey, S. (2013). Social defeat and PTSD symptoms following trauma. *British Journal of Clinical Psychology*.

Troop, N. A., Holbrey, A., & Treasure, J. L. (1998). Stress, coping, and crisis support in eating disorders. *International Journal of Eating Disorders*, *24*(2), 157-166

Troop, N. A., Serpell, L., & Treasure, J. L. (2001). Specificity in the relationship between depressive and eating disorder symptoms in remitted and nonremitted women. *International Journal of Eating Disorders*, *30*(3), 306-311

Troop, N. A., & Treasure, J. L. (1997). Psychosocial factors in the onset of eating disorders: Responses to life-events and difficulties. *British Journal of Medical Psychology, 70,* 373-385

Trower, P., & Gilbert, P. (1989). New theoretical conceptions of social anxiety and social phobia. *Clinical Psychology Review*, *9*(1), 19-35

Tsai, A. G., & Wadden, T. A. (2006). The evolution of Very-Low-Calorie Diets: An update and meta-analysis. *Obesity, 14*(8), 1283-1293

Tseng, M., & Fang, C. Y. (2011). Stress is associated with unfavorable patterns of dietary intake among female Chinese immigrants. *Annals of Behavioral Medicine*, *41*(3), 324-332

Tucker, J. A., Vuchinich, R. E., & Pukish, M. M. (1995). Molar environmental contexts surrounding recovery from alcohol problems by treated and untreated problem drinkers. *Experimental and Clinical Psychopharmacology, 3,* 195-204

Turk, M. W., Sereika, S. M., Yang, K., Ewing, L. J., Hravnak, M., & Burke, L. E. (2012). Psychosocial correlates of weight maintenance among black & white adults. *American Journal of Health Behavior*, *36*(3), 395-407

Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin*, *119*(3), 488-531

Van Cauter, E., & Knutson, K. L. (2008). Sleep and the epidemic of obesity in children and adults. *European Journal of Endocrinology, 159,* S59-S66

Van Der Zee, K. I., Buunk, B. P., Sanderman, R., Botke, G., & Van Den Bergh, F. (1999). The Big Five and identification-contrast processes in social comparison in adjustment to cancer treatment. *European Journal of Personality*, *13*(4), 307-326

Van Dorsten, B., & Lindley, E. M. (2011). Cognitive and behavioral approaches to the treatment of obesity. *Medical Clinics of North America, 95,* 971-988

van Duijvenbode, D. C., Hoozemans, M. J. M., Van Poppel, M. N. M., & Proper, K. I. (2009). The relationship between overweight and obesity, and sick leave: A systematic review. *International Journal of Obesity*, *33*(8), 807-816

van Hout, G. C. M., Verschure, S. K. M., & van Heck, G. L. (2005). Psychosocial predictors of success following bariatric surgery. *Obesity Surgery, 15*, 552-560

Van Lenthe, F. J., Droomers, M., Schrijvers, C. T., & Mackenbach, J. P. (2000). Socio-demographic variables and 6 year change in body mass index: Longitudinal results from the GLOBE study. *International Journal of Obesity, 24*(8), 1077-1084

van Middendorp, H., Sorbi, M. J., van Doornen, L. J., Bijlsma, J. W., & Geenen, R. (2007). Feasibility and induced cognitive-emotional change of an emotional disclosure intervention adapted for home application. *Patient Education and Counseling*, *66*(2), 177-187

Van Strien, T., Rookus, M. A., Bergers, G. P., Frijters, J. E., & Defares, P. B. (1986). Life events, emotional eating and change in body mass index. *International Journal of Obesity*, *10*(1), 29-35

Vardi, P., & Pinhas-Hamiel, O. (2000). The young hunter hypothesis: Age-related weight gain – a tribute to the thrifty theories. *Medical Hypotheses, 55*(6), 521-523

Wadden, T. A. (1993). Treatment of obesity by moderate and severe caloric restriction. *Annals of Internal Medicine, 119*(7), 688-693

Wadden, T. A., Berkowitz, R. I., Womble, L. G., Sarwer, D. B., Phelan, S., Cato, R. K., Hesson, L. A., Osei, S. Y., Kaplan, R., & Stunkard, A. J. (2005). Randomized trial of lifestyle modification and pharmacotherapy for obesity. *The New England Journal of Medicine, 353*(20), 2111-2120

Wadden, T. A., Brownell, K. D., & Foster, G. D. (2002). Obesity: Responding to the global epidemic. *Journal of Consulting and Clinical Psychology, 70*(3), 510-525

Wadden. T. A., Butryn, M. L., & Byrne, K. J. (2004). Efficacy of lifestyle modification for long-term weight control. *Obesity Research, 12,* 151S-162S

Wadden, T. A., Butryn, M. L., & Wilson, C. (2007). Lifestyle modification for the management of obesity. *Gastroenterology, 132,* 2226-2238

Wadden, T. A., Foster, G. A., Letizia, K. A., & Stunkard, A. J. (1992). A Multicenter evaluation of a proprietary weight reduction program for the treatment of marked obesity. *Archives of Internal Medicine, 152,* 961-966

Wadden, T. A., Sternberg, J. A., Letizia, K. A., Stunkard, A. J., & Foster, G. D. (1989). Treatment of obesity by very low calorie diet, behavior therapy, and their combination: A five-year perspective. *International Journal of Obesity, 13*(2), 39-46

Wadden, T. A., Stunkard, A. J., & Liebschutz, J. (1988). Three-year follow-up of the treatment of obesity by very low calorie diet, behavior therapy, and their combination. *Journal of Consulting and Clinical Psychology*, *56*(6), 925-928

Wadden, T. A., Vogt, R. A., Foster, G. D., & Anderson, D. A. (1998). Exercise and the maintenance of weight loss: 1-year follow-up of a controlled clinical trial. *Journal of Consulting and Clinical Psychology, 66*(2), 429-433

Wade, T. D., Treasure, J., & Schmidt, U. (2011). A case series evaluation of the Maudsley Model for treatment of adults with anorexia nervosa. *European Eating Disorders Review*, *19*(5), 382-389

Walsh, B. T., Kaplan, A. S., Attia, E., Olmsted, M., Parides, M., Carter, J. C., Pike, K. M., Devline, M. J., Woodside, D. B., Roberto, C. A., & Rockert, W. (2006). Fluoxetine after weight restoration in anorexia nervosa. *The Journal of the American Medical Association*, *295*(22), 2605-2612

Wane, S., van Uffelen, J. G. Z., & Brown, W. (2010). Determinants of weight gain in young women: A review of the literature. *Journals of Women's Health, 19*(7), 1327-1340

Wang, J., & Wang, X. (2012). *Structural equation modeling: Applications using Mplus.* Wiley

Ward, A., Ramsay, R., & Treasure, J. (2000). Attachment research in eating disorders. *British Journal of Medical Psychology, 73,* 35-51

Wardle, J., & Beinart, H. (1981). Binge eating: A theoretical review. *British Journal of Clinical Psychology*, *20*(2), 97-109

Wardle, J., Chida, Y., Gibson, E. L., Whitaker, K. L., & Steptoe, A. (2011). Stress and adiposity: A meta-analysis of longitudinal studies. *Obesity, 19*(4), 771-778

Wardle, J., Haase, A. M., Steptoe, A., Nillapun, M., Jonwutiwes, K., & Bellisie, F. (2004). Gender differences in food choice: the contribution of health beliefs and dieting. *Annals of Behavioral Medicine*, *27*(2), 107-116

Wardle, J., Steptoe, A., Oliver, G., & Lipsey, Z. (2000). Stress, dietary restraint and food intake. *Journal of Psychosomatic Research*, *48*(2), 195-202

Watt, M. C., McWilliams, L. A., & Campbell, A. G. (2005). Relations between anxiety sensitivity and attachment style dimensions. *Journal of Psychopathology and Behavioral Assessment, 27,* 191-200

Weems, C. F., Berman, S. L., Silverman, W. K., & Rodriguez, E. T. (2002). The relation between anxiety sensitivity and attachment style in adolescence and early adulthood. *Journal of Psychopathology and Behavioral Assessment, 24,* 159-168

Wei, M., Vogel, D. L., Ku, T., & Zakalik, R. A. (2005). Adult attachment, affect regulation, negative mood, and interpersonal problems: The mediating roles of emotional reactivity and emotional cutoff. *Journal of Counselling Psychology, 52,* 14-24

Weiselberg, E. C., Gonzalez, M., & Fisher, M. (2011). Eating disorders in the twenty-first century. *Minerva Ginecologica*, *63*(6), 531-545

Weiss, E. C., Galuska, D. A., Khan, L. K., Gillespie, C., & Serdula, M. K. (2007). Weight regain in U.S. adults who experienced substantial weight loss, 1999-2002. *American Journal of Preventive Medicine, 33*(1), 34-40

Welch, S. L., Doll, H. A., & Fairburn, C. G. (1997). Life events and the onset of bulimia nervosa: A controlled study. *Psychological Medicine*, *27*(03), 515-522

Wendel-Vos, W., Droomers, M., Kremers, S., Brug, J., & van Lenthe, F. (2007). Potential environmental determinants of physical activity in adults: A systematic review. *Obesity Reviews, 8,* 425-440

Westerterp-Plantenga, M. S., Kempen, K. P. G., & Saris, W. H. M. (1998). Determinants of weight maintenance in women after diet-induced weight reduction. *International Journal of Obesity*, *22*(1), 1-6

Westover, S. A., & Lanyon, R. I. (1990). The maintenance of weight loss after behavioral treatment: A review. *Behavior Modification, 14*(2), 123-137

Wilfley, D. E., Schwartz, M. B., Spurrell, E. B., & Fairburn, C. G. (2000). Using the eating disorder examination to identify the specific psychopathology of binge eating disorder. *International Journal of Eating Disorders*, *27*(3), 259-269

Will, J. C., Williamson, D. F., Ford, E. S., Calle, E. E., & Thun, M. J. (2002). Intentional weight loss and 13-year diabetes incidence in overweight adults. *American Journal of Public Health*, *92*(8), 1245-1248

Williams, G. J., Power, K. G., Millar, H. R., Freeman, C. P., Yellowlees, A., Dowds, T., Walker, M., Campsie, L., MacPherson, F., & Jackson, M. A. (1993). Comparison of eating disorders and other dietary/weight groups on measures of perceived control, assertiveness, self-esteem and self-directed hostility. *International Journal of Eating Disorders, 14*, 27-32

Williamson, D. F. (1993). Descriptive epidemiology of body weight and weight change in US adults. *Annals of Internal Medicine*, *119*(7), 646-649

Williamson, D. F., Kahn, H. S., Remington, P. L., & Anda, R. F. (1990). The 10-year incidence of overweight and major weight gain in US adults. *Archives of Internal Medicine, 150,* 665-672

Wills, T. A. (1981). Downward comparison principles in social psychology. *Psychological Bulletin*, *90*(2), 245-271

Wilson, D. R. (2010). Health consequences of childhood sexual abuse. *Perspectives in Psychiatric Care, 46*(1), 56-64

Wilson, G. T., & Fairburn, C. G. (1993). Cognitive treatments for eating disorders. *Journal of Consulting and Clinical Psychology*, *61*(2), 261-269

Wilson, G. T., & Shafran, R. (2005). Eating disorders guidelines from NICE. *Lancet*, *365*(9453), 79-81

Wing, J. F., Schutte, N. S., & Byrne, B. (2006). The effect of positive writing on emotional intelligence and life satisfaction. *Journal of Clinical Psychology*, *62*(10), 1291-1302

Wing, R. R. (1992). Weight cycling in humans: A review of the literature. *Annals of Behavioral Medicine, 14*(2), 113-119

Wing, R. R. (1993). Weight cycling: The public concern and the scientific data. *Obesity Research, 1*(5), 390-391

Wing, R. R., & Hill, J. O. (2001). Successful weight loss maintenance. *Annual Review of Nutrition, 21,* 323-341

Wing, R. R., & Phelan, S. (2005). Long-term weight loss maintenance. *The American Journal of Clinical Nutrition*, *82*(1), 222S-225S

Wonderlich, S. A., Brewerton, T. D., Jocic, Z., Dansky, B. S., & Abbott, D. W. (1997). Relationship of childhood sexual abuse and eating disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*(8), 1107-1115

Woodside, D. B. (1995). A review of anorexia nervosa and bulimia nervosa. *Current Problems in Pediatrics*, *25*(2), 67-89

World Health Organization, Expert Consultation (2004). Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *The Lancet, 363,* 157-163

Wright, J., & Chung, M. C. (2001). Mastery or mystery? Therapeutic writing: A review of the literature. *British Journal of Guidance and Counselling, 29*(3), 277-291

Wyatt, R., & Gilbert, P. (1998). Dimensions of perfectionism: A study exploring their relationship with perceived social rank and status. *Personality and Individual Differences*, *24*(1), 71-79

Ybema, J. F., & Buunk, B. P. (1995). Affective responses to social comparison: A study among disabled individuals. *British Journal of Social Psychology*, *34*(3), 279-292

Zachrisson, H. D., & Skårderud, F. (2010). Feelings of insecurity: Review of attachment and eating disorders. *European Eating Disorders Review, 18,* 97-106

Zandian, M., Ioakimidis, I., Bergh, C., & Södersten, P. (2007). Cause and treatment of anorexia nervosa. *Physiology and Behavior*, *92*(1), 283-290

Zhou, Y-H., Ma, X-Q., Wu, C., Lu, J., Zhang, S-S., Guo, J., Wu, S-Q., Ye, X-F., Xu, F-F., & He, J. (2012) Effect of anti-obesity drug on cardiovascular Risk factors: A systematic review and meta-analysis of randomized controlled trials. *PLoS ONE, 7*(6), e39062

Zuroff, D. C., & Fitzpatrick, D. K. (1995). Depressive personality styles: Implications for adult attachment. *Personality and Individual Differences, 18,* 253-265

Zuroff, D. C., Fournier, M. A., Patall, E. A., & Leybman, M. J. (2010). Steps toward an evolutionary personality psychology: Individual differences in the social rank domain. *Canadian Psychology, 51*, 58-66

Appendices

Self-report measures

A. Food Frequency Questionnaire (Cade & Margetts, 1988)

FOOD FREQUENCY QUESTIONNAIRE

Please read the following instructions carefully before completing the questionnaire

1. Please answer **ALL** of the questions.
2. Select the answer that best applies to your situation and **TICK** the appropriate column.
3. Only tick **ONE** box on **EACH** line (i.e. one tick per food).
4. If you make a mistake, put a cross through the incorrect tick and then tick the correct answer.

Example:

How often do you eat the following foods?

	2 or more times a day	**Every day**	**3-5 times a week**	**1-2 times a week**	**1-3 times a month**	**Rarely/ never**
Whole	√					
Semi-skimmed				√		
Skimmed						√

HOW OFTEN DO YOU EAT THE FOLLOWING FOODS?

Dairy Products	**2 or more times a day**	**Every day**	**3-5 times a week**	**1-2 times a week**	**1-3 times a month**	**Rarely/ never**
Whole milk (including in tea of coffee)						
Semi-skimmed milk (including in tea						

of coffee)						
Skimmed milk (including in tea of coffee)						
Butter						
Margarine (e.g. Stork, Clover)						
Polyunsaturated margarine (e.g. flora, sunflower)						
Low fat spreads (e.g. Outline, Gold)						
Ice cream						
Yoghurt, Fromage Frais						
Full Fat Cheese (e.g. cheddar, cream cheese)						
Low fat cheese (e.g. cottage, light cream cheese)						
Eggs - fried						
Eggs - not fried (boiled, poached, in baking)						
Cheese and / or egg dishes						

Meats and Fish	**2 or more times a day**	**Every day**	**3-5 times a week**	**1-2 times a week**	**1-3 times a month**	**Rarely/ never**
Beef - roast/steak						
Lamb - roast/chops						
Pork - roast / chops						
Chicken, turkey or other poultry						
Bacon or gammon						
Meat dishes (e.g. stew, curry, chilli)						
Canned meats (e.g. corned beef, ham)						
Meat pies, sausage rolls or pasties						
Sausages or beef burgers						

Liver, kidney, pate (and other offal products)						
Fish and sea food - not fried						
Fish and sea food - fried						
Fish and sea food - Canned (e.g. tuna)						

Breads and Cereals	**2 or more times a day**	**Every day**	**3-5 times a week**	**1-2 times a week**	**1-3 times a month**	**Rarely/ never**
White bread						
Brown/granary bread						
Wholemeal bread (including chapattis)						
Sweet biscuits (plain and chocolate)						
Crackers/crisp bread						
Cakes/buns/pastries						
Puddings (e.g. fruit pies, cheesecake)						
Breakfast cereal - High fibre (e.g. bran flakes)						
Breakfast cereal - Ordinary (e.g. cornflakes)						
Breakfast cereal - Muesli						
Rice or pasta						

Fruit and Vegetables	**2 or more times a day**	**Every day**	**3-5 times a week**	**1-2 times a week**	**1-3 times a month**	**Rarely/ never**
Apples, pears						
Oranges, grapefruit and/or other citrus fruit						
Bananas						
Green vegetables (e.g. peas,						

cabbage)						
Carrots, tomatoes (fresh or canned)						
Other vegetables (including salad vegetables)						
Baked beans						
Other beans/lentils (e.g. dahl)						
Vegetable dishes (e.g. stew, curry - NO meat)						
Potatoes - chips, roasted, fried						
Potatoes - not fried (boiled, baked)						

Beverages	**2 or more times a day**	**Every day**	**3-5 times a week**	**1-2 times a week**	**1-3 times a month**	**Rarely/ never**
Beer or lager						
Wine, sherry or spirits						
Tea or coffee						
Squash or fizzy drinks						
Low calorie drinks						
Pure fruit juices						

Miscellaneous	**2 or more times a day**	**Every day**	**3-5 times a week**	**1-2 times a week**	**1-3 times a month**	**Rarely/ never**
Chocolate						
Sweets						
Sweet spreads (e.g. jam, marmalade)						
Sugar (e.g. in coffee, tea, on cereal)						
Crisps and savoury snacks						
Nuts (including peanut butter)						
Sauces and pickles (e.g. ketchup, Branston)						
Salad oils, dressings, mayonnaise						

B. Eating Disorder Examination Questionnaire (Fairburn & Beglin, 1994)

EATING CONCERNS

The following questions are concerned with the past four weeks only (28 days). Please read each question carefully and tick the appropriate box.

Please answer all the questions.

	On how many days out of the past 28 days:	**No days**	**1-5 days**	**6-12 days**	**13-15 days**	**16-22 days**	**23-27 days**	**Every day**
1.	Have you been deliberately trying to limit the amount of food you eat to influence your shape or weight?							
2.	Have you gone for long periods of time (8 hours or more) without eating anything in order to influence your shape or weight?							
3.	Have you tried to avoid eating foods, which you like in order to influence your shape or weight?							
4.	Have you tried to follow definite rules regarding your eating in order to influence your shape or weight; for example, a calorie limit, a set amount of food, or rules about what or when you should eat?							
5.	Have you wanted your stomach to be empty?							
6.	Has thinking about food or its calorie content made it much more difficult to concentrate on things you're interested in; for example, read, watch TV or follow a conversation?							
7.	Have you been afraid of losing control over eating?							
8.	Have you had episodes of binge eating?							
9.	Have you eaten in secret? (Do not count binges)							
10.	Have you definitely wanted your stomach to be flat?							
11.	Has thinking about shape or weight made it more difficult to concentrate on things you are interested in; e.g., read, watch TV or follow a conversation?							
12.	Have you had a definite fear that you might gain weight or become fat?							
13.	Have you felt fat?							
14.	Have you had a strong desire to lose weight?							

	Over the past 4 weeks (28 days):	None of the time	A few of the times	Less than ½ the time	Half the time	More than ½ the time	Most of the time	Every time
15.	On what proportion of times that you have eaten have you **felt guilty** because of the effect on your shape or weight? *(Do not count binges)*							
16.	Have there been any times when you have felt that you have eaten what other people would regard as an unusually large amount of food given the circumstances? **If yes:** a. How many such episodes have you had over the past 4 weeks? b. During how many of these episodes of overeating did you have a sense of having lost control over your eating?	**Please circle:** **NO** **YES**						
17.	Have you had other episodes of eating in which you have had a sense of having lost control and eaten too much, but have **not** eaten an unusually large amount of food given the circumstances? **If yes:** How many such episodes have you had over the past 4 weeks?	**Please circle:** **NO** **YES**						
18.	Have you made yourself sick (vomit) as a means of controlling your shape or weight? **If yes:** How many times have you done this over the past 4 weeks?	**Please circle:** **NO** **YES**						
19.	Have you taken laxatives as a means of controlling your shape or weight? **If yes:** How many times have you done this over the past 4 weeks?	**Please circle:** **NO** **YES**						
20.	Have you taken diuretics (water tablets) as a means of controlling your shape or weight? **If yes:** How many times have you done this over the past 4 weeks?	**Please circle:** **NO** **YES**						
21.	Have you exercised **hard** as a means of controlling your shape or weight? **If yes:**	**Please circle:** **NO** **YES**						

	How many times have you done this over the past 4 weeks?	

	Over the past 4 weeks (28 days):	Not at all		Slightly		Moderately		Markedly
		0	1	2	3	4	5	6
22.	Has your weight influenced how you think about (judge) yourself as a person?							
23.	Has your shape influenced how you think about (judge) yourself as a person?							
24.	How much would it upset you if you had to weigh yourself once a week for the next four weeks?							
25.	How dissatisfied have you felt about your weight?							
26.	How dissatisfied have you felt about your shape?							
27.	How concerned have you been about other people seeing you eat?							
28.	How uncomfortable have you felt seeing your body; for example, in shop window reflections, while undressing or taking a bath or shower?							
29.	How uncomfortable have you felt about others seeing your body; for example, in communal changing rooms, when swimming or wearing tight clothes?							

C. Vulnerable Attachment Style Questionnaire (Bifulco et al., 2003)

RELATIONSHIP STYLES

Below are a number of statements concerning the way people feel about themselves in relation to others. Please indicate whether you agree or disagree with the description as it applies to you by circling a number from 1 to 5. Please complete items as you generally feel rather than how you feel right now.

There are no "right" or "wrong" answers.

		Strongly disagree	Disagree	Unsure	Agree	Strongly agree
1	I take my time getting to know people	1	2	3	4	5
2	I rely on others to help me make decisions	1	2	3	4	5
3	People let me down a lot	1	2	3	4	5
4	I miss the company of others when I am alone	1	2	3	4	5
5	It's best not to get too emotionally close to other people	1	2	3	4	5
6	I worry a lot if people I live with arrive back later than expected	1	2	3	4	5
7	I usually rely on advice from others when I've got a problem	1	2	3	4	5
8	I feel uncomfortable when people get too close to me	1	2	3	4	5
9	People close to me often get on my nerves	1	2	3	4	5
10	I feel people are against me	1	2	3	4	5
11	I worry about things happening to close family and friends	1	2	3	4	5
12	I often get into arguments	1	2	3	4	5
13	I am clingy with others	1	2	3	4	5
14	I look forward to spending time on my own	1	2	3	4	5
15	I like making decisions on my own	1	2	3	4	5
16	I get anxious when people close to me are away	1	2	3	4	5
17	I feel uneasy when others confide in me	1	2	3	4	5

18	I find it hard to trust others	1	2	3	4	5
19	Having people around me can be a nuisance	1	2	3	4	5
20	I feel people haven't done enough for me	1	2	3	4	5
21	It's important to have people around me	1	2	3	4	5
22	I find it difficult to confide in people	1	2	3	4	5

D. Short Depression-Happiness Scale (Joseph et al., 2004)

MOOD SCALE

This questionnaire is interested in your feelings over the **last 7 days**. Please indicate the degree to which you agree with each of the following statements by ticking the response that most closely matches how you have felt over the **last 7 days**.

		Never	Rarely	Sometimes	Often
1	I felt dissatisfied with my life	❍	❍	❍	❍
2	I felt happy	❍	❍	❍	❍
3	I felt cheerless	❍	❍	❍	❍
4	I felt pleased with the way I am	❍	❍	❍	❍
5	I felt that life was enjoyable	❍	❍	❍	❍
6	I felt that life was meaningless	❍	❍	❍	❍

E. Forms of Self-Criticising/Attacking and Self-Reassuring Scale (Gilbert et al., 2004)

SELF-CRITICISING/ATTACKING AND SELF-REASSURING SCALE

When things go wrong in our lives or don't work out as we hoped, and we feel we could have done better, we sometimes have *negative* and *self-critical thoughts* and feelings. These may take the form of feeling worthless, useless or inferior etc. However, people can also try to be supportive of themselves. Below are a series of thoughts and feelings that people sometimes have. Read each statement carefully and circle the number that best describes how much each statement is true for you.

Please use the scale below:

Not at all like me	A little bit like me	Moderately like me	Quite a bit like me	Extremely like me
0	1	2	3	4

When things go wrong for me:

1.	I am easily disappointed with myself.	0	1	2	3	4
2.	There is a part of me that puts me down.	0	1	2	3	4
3.	I am able to remind myself of positive things about myself.	0	1	2	3	4
4.	I find it difficult to control my anger and frustration at myself.	0	1	2	3	4
5.	I find it easy to forgive myself.	0	1	2	3	4
6.	There is a part of me that feels I am not good enough.	0	1	2	3	4
7.	I feel beaten down by my own self-critical thoughts.	0	1	2	3	4
8.	I still like being me.	0	1	2	3	4
9.	I have become so angry with myself that I want to hurt or injure myself.	0	1	2	3	4
10.	I have a sense of disgust with myself.	0	1	2	3	4
11.	I can still feel lovable and acceptable.	0	1	2	3	4
12.	I stop caring about myself.	0	1	2	3	4
13.	I find it easy to like myself.	0	1	2	3	4
14.	I remember and dwell on my failings.	0	1	2	3	4

15.	I call myself names.	0	1	2	3	4
16.	I am gentle and supportive with myself.	0	1	2	3	4
17.	I can't accept failures and setbacks without feeling inadequate.	0	1	2	3	4
18.	I think I deserve my self-criticism.	0	1	2	3	4
19.	I am able to care and look after myself.	0	1	2	3	4
20.	There is a part of me that wants to get rid of the bits I don't like.	0	1	2	3	4
21.	I encourage myself for the future.	0	1	2	3	4
22.	I do not like being me.	0	1	2	3	4

F. Social Comparison Rating Scale (Allan & Gilbert, 1995)

PERCEPTIONS OF SELF

Please circle a number on each line which best describes the way in which you see yourself in **comparison to others**.

Example:

Short	1	2	3	4	5	6	7	8	9	10 Tall

If you put a mark at 3 this means you see yourself as shorter than others. If you put a mark at 5 (middle) about average; and a mark at 7 somewhat taller.

If you understand the above instructions please proceed. Circle one number on each line according to how you see yourself in relation to others.

In relation to others I feel…

Inferior	1	2	3	4	5	6	7	8	9	10 Superior
Incompetent	1	2	3	4	5	6	7	8	9	10 More competent
Unlikeable	1	2	3	4	5	6	7	8	9	10 More likeable
Left out	1	2	3	4	5	6	7	8	9	10 Accepted
Different	1	2	3	4	5	6	7	8	9	10 Same
Untalented	1	2	3	4	5	6	7	8	9	10 More talented
Weaker	1	2	3	4	5	6	7	8	9	10 Stronger
Unconfident	1	2	3	4	5	6	7	8	9	10 More confident
Undesirable	1	2	3	4	5	6	7	8	9	10 More desirable
Unattractive	1	2	3	4	5	6	7	8	9	10 More attractive
An outsider	1	2	3	4	5	6	7	8	9	10 An insider

G. Perceived Stress Scale-4 (Cohen & Williamson, 1988)

STRESS PERCEPTIONS

The questions in this scale ask you about your feelings and thoughts during **the last month**. In each case, please indicate with a check how often you felt or thought a certain way.

	Never	**Almost Never**	**Sometimes**	**Fairly Often**	**Very Often**
1. In the last month, how often have you felt that you were unable to control the important things in your life?	0	1	2	3	4
2. In the last month, how often have you felt confident about your ability to handle your personal problems?	0	1	2	3	4
3. In the last month, how often have you felt that things were going your way?	0	1	2	3	4
4. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?	0	1	2	3	4

H. Self-criticism/reassurance questions developed for study three (Troop, 2013)

How did you feel about the way you responded or the way you coped?
Sometimes when things go wrong, people can be very critical of themselves – were you very critical of yourself?

- In what way?
- What sorts of things did you do or say to yourself?

(For example, sometimes when people are upset they see it as evidence that they are weak or worthless)

- How often would you say or do these things to yourself?
- What was it that you were critical of? (causing the problem, getting upset, not coping as effectively as you would like)

On the other hand, sometimes when people experience problems they can be very self-soothing or self-reassuring – is this something you did?

- In what way were you self-reassuring or soothing?
- What sorts of things did you do or say to yourself?

(For example, sometimes when people are upset they say things like "It's okay to feel this way")

- How often would you say or do these things to yourself?
- How easy did you find it to be self-soothing?

Do you think differently now?

Self-compassion

"Instead of mercilessly judging and criticizing oneself for various inadequacies or shortcomings, self-compassion means people are kind and understanding to themselves when confronted with personal failings" (Neff, 2003)

Use the same 4-point scale as other measures but bear in mind we need evidence of **level** and **amount** of self-criticism/soothing.

Paul Gilbert used factor analysis and found that self-reassurance formed one scale but self-criticism formed two, inadequate self and hated self. The items for these scales are below and can be used as guides when rating.

Inadequate self

- I am easily disappointed with myself
- There is a part of me that puts me down
- I find it difficult to control my anger and frustration at myself
- There is a part of me that feels I am not good enough
- I feel beaten down by my own self-critical thoughts
- There is a part of me that wants to get rid of the bits I don't like
- I can't accept failures and setbacks without feeling inadequate
- I think I deserve my self-criticism
- I remember and dwell on my failings

Hated self

- I have become so angry with myself that I want to hurt or injure myself
- I have a sense of disgust with myself
- I stop caring about myself
- I call myself names
- I do not like being me

Reassured self

- I am able to remind myself of positive things about myself
- I find it easy to forgive myself
- I still like being me
- I can still feel lovable and acceptable
- I find it easy to like myself
- I am gentle and supportive with myself
- I am able to care and look after myself

- I encourage myself for the future

Examples

- "I could have coped better" versus "I used to stand in front of the mirror insulting myself" for *some* versus *marked* self-criticism respectively
- "I didn't think I should have coped differently versus "I would tell myself that I am still a worthwhile person who deserved better than the way I was treated" for *some* versus *marked* self-reassurance/soothing respectively

Note that we need evidence of this kind of self-criticism/soothing at the time of the crisis, not just in retrospect.

Example: "Looking back I really beat myself up for getting upset but I realise now that it was perfectly natural – of course I was upset, my boyfriend had just dumped me" – might be self-soothing currently but was self-critical at the time. Could still record both responses, rate for the time of the crisis but note that responses have changed since then (hence the "Do you think differently now?" question)

In the same way that self-criticism splits into Hated Self and Inadequate Self, self-compassion may split into different types (e.g. self-soothing versus self-acceptance). I have no evidence for this and the factor analysis of the FSCRS only splits self-criticism. For the time being just make one rating for self-reassurance. Maybe with examples emerging from the interviews we can re-evaluate this.

Rating schedule

Inadequate self _____

- 1 – marked
- 2 – moderate
- 3 – some
- 4 – little/none

Give details:

Hated self _____

- 1 – marked
- 2 – moderate
- 3 – some
- 4 – little/none

Give details:

Reassured self _____

- 1 – marked
- 2 – moderate
- 3 – some
- 4 – little/none

Give details: