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Improving Affect Regulation in Eating Disorders: The Case for Positive Emotions.

Augustina Rogowski
Declaration:

I confirm that this thesis has been composed by me, the work is my own and it has not been submitted for any other degree or professional qualification.

Signed:

Augustina Rogowski

Acknowledgements:

I would like to thank B-EAT (Registered Charity No. 801343) and SAGE (The University of Edinburgh) for their help with recruiting participants. Special thanks to Prof. Mick Power, Dr. Ethel Quayle, Prof. Randy Larsen, Prof. Dennis Hocevar, Dr. Paul Silvia, Prof. Sonja Lyubomirsky, Prof. Julia Buckroyd and Dr. John Fox for their help with my research project. I would also like to thank all the people who took part in my studies.
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List of Abbreviations

BM = Before Mood Induction
AM = After Mood Induction

InDys = Internal Dysfunctional emotion regulation
ExDys = External Dysfunctional emotion regulation
ExFun = External Functional emotion regulation
InFun = Internal Functional emotion regulation
ER = Emotion Regulation

Positive AI = Positive Affect Intensity
Negative AI = Negative Affect Intensity
Global AI = Global Affect Intensity
AI = Affect Intensity

SD = Standard Deviation
N = Number (e.g. of participants)

PPI = Positive Psychology Intervention
SED = Subclinically Eating-Disordered (participants)

REQ = Regulation of Emotion Questionnaire (Phillips and Power, 2007)
AIM = Affect Intensity Measure Questionnaire (Larsen, 1984)
SWLS = Satisfaction with Life Scale (Diener, 1985)
EDE-Q = Eating Disorder Examination Questionnaire (Fairburn and Beiglin, 1994)
EAT-26 = Eating Attitudes Test (Garner et al., 1982)
HADS = Hospital Anxiety and Depression Scale (Zigmond and Snaith, 1983)
EAS = Emotional Assessment Scale (Carlson et al., 1989)
ABSTRACT

Evidence from multiple studies suggests that regulation of emotions and intensity of affect may be relevant to understanding disordered eating. Emotion regulation concerns the ways in which emotions are managed in daily life, whereas Affect Intensity (Larsen et al., 1986) refers to individuals’ typical emotional reactivity. The thesis examines emotion regulation and affect in females with eating pathology (subclinical as well as clinical), and looks at ways dysfunctional regulatory strategies may be improved. The main objective of the present research was to look at the influence of experimentally-induced positive affect on the choice of emotion management strategies. Study 1 looked at typical Affect Intensity and emotion regulation in a sample of subclinically eating-disordered University of Edinburgh students. This study examined functionality of regulatory strategies, typical intensity of affect, and the effects of experimentally induced happiness and sadness on the two. Study 2 introduced the construct of creativity into the discourse on emotions and psychopathology, and looked at creative tendencies in relation to Affect Intensity, emotion regulation and psychopathology (anxiety, depression and sub-clinical eating pathology). Study 3 looked at the effects of Positive Psychology Interventions (PPIs) on emotion regulation, life satisfaction, anxiety and depression in a subclinically eating-disordered group and controls. Studies 4 and 5 were carried out in order to test and extend the results of Studies 1 and 3 with a clinical sample. In Study 4, emotion regulation, Affect Intensity and the immediate post-test effect of happiness on emotion regulation and life satisfaction were examined in females clinically diagnosed with eating disorders (i.e. anorexia nervosa, bulimia nervosa, and EDNOS). Study 5 looked at how longitudinal happiness induction influenced emotion regulation, eating behaviours and life satisfaction in eating-disordered individuals. One of the main findings across the studies was that females with subclinical and clinical forms of eating pathology tended to experience negative emotions of high intensity, and used predominantly dysfunctional regulatory strategies to manage them. Another important finding was that experimentally-induced positive emotions improved emotion regulation, and encouraged participants to choose healthier affect management strategies. The studies, their implications and contribution to theory and treatment of eating disorders are discussed.
INTRODUCTION

The thesis consists of five studies that examined emotion regulation in females with eating pathology of differing severity (subclinical and clinical forms). The main aim of research was to look at the influence of experimentally-induced positive emotions on the choice of emotion management strategies.

Chapters 1-4 provide a theoretical background to the studies. Chapter 1 discusses symptomatology, outcomes, aetiology and treatment of eating disorders (anorexia nervosa, bulimia nervosa and EDNOS) and subclinical disordered eating. In particular, Chapter 1 looks at the role of biology (e.g. addiction to endogenous opioids), sociocultural factors (e.g. mass media), trauma and individual differences (e.g. neurotic perfectionism) in development and maintenance of disordered eating. It also discusses pharmacological and psychological treatment options, available to patients with eating disorders. Chapter 2 focuses on the role of negative affect and its regulation in development and maintenance of eating pathology. Chapter 3 looks at the research on positive emotions, and their potential application to treatment of eating disorders. It also contains a section on creativity, and discusses it in the context of eating pathology and affect.

Study 1 is outlined and discussed in Chapter 4. This study looked at Affect Intensity and emotion regulation in subclinically eating disordered females. The sample consisted of 100 female University of Edinburgh students. Using the Regulation of Emotion Questionnaire (Phillips and Power, 2007) and the Affect Intensity Measure Questionnaire (Larsen, 1986), this study examined functionality of regulatory strategies, intensity of affect, and the effects of experimentally induced happiness and sadness on the two in the subclinically eating-disordered group and controls. The emotions were induced using a combination of the standard Velten mood induction procedure (Velten, 1968) and music (Mozart’s Toy Symphony in the Happiness condition; Albinoni’s Adagio in the Sadness condition; Holst’s Planets in the Neutral condition). The relationship between emotion regulation and Affect Intensity across the whole sample was also examined. The Eating Attitudes Test (EAT-26; Garner et al., 1982) was used a screening tool for eating pathology.
Chapter 5 describes and discusses Study 2. This study put creativity in the context of emotions and psychopathology, and looked at creative tendencies in relation to Affect Intensity, emotion regulation and psychopathology (sub-clinical anxiety, depression and eating pathology) in a sample of 97 female University of Edinburgh students. Creativity was measured using Creative Behaviours Checklist (Hocevar, 1980) to assess history of creative accomplishments and the Alternative Uses Task (Guildford, 1950) to measure on-line creativity. Other measures used include Affect Intensity Measure (Larsen, 1986), Eating Attitudes Test (EAT-26; Garner et al., 1982), Hospital Anxiety and Depression Scale (Zigmond and Snaith, 1983) and Regulation of Emotions Questionnaire (Phillips and Powers, 2007).

Study 3 is outlined and discussed in Chapter 6. This study was a replication of Study 1, with some important differences: (1) it looked at positive versus neutral mood in relation to emotion regulation (as opposed to positive versus negative mood, like in Study 1), (2) a different measure of eating pathology was used – Eating Disorder Examination Questionnaire (EDE-Q; Fairburn and Beglin, 1994), and (3) Positive Psychology Interventions (PPIs) were used in experimental mood manipulation. Two PPIs were administered: a Gratitude exercise (Emmons and McCullough, 2003) and a Positive Reminiscence task (Bryant et al., 2005). In Study 3 PPIs were chosen as a mood induction method in preparation and as a pilot for clinical studies with participants who would not be able to travel to Edinburgh to take part in the research project. This study looked at the effects of PPI-induced happiness vs. neutral mood on emotion regulation (Regulation of Emotion Questionnaire; Phillips and Power, 2007), life satisfaction (Satisfaction with Life Scale; Diener, 1985), anxiety and depression (Hospital Anxiety and Depression Scale; Zigmond and Snaith, 1983) in a subclinically eating-disordered group and controls.

Studies 4 and 5 are described in Chapters 7 and 8. Studies 4 and 5 were conducted in an attempt to replicate and extend the findings of Studies 1 and 3 with a clinical sample. The questionnaires used were largely the same as the ones in previous studies, and in both studies, positive emotions were induced using the Gratitude exercise (Emmons and
McCullough, 2003). In Study 4, emotion regulation (Regulation of Emotion Questionnaire; Phillips and Power, 2007), Affect Intensity (Affect Intensity Measure Questionnaire; Larsen, 1986) and the immediate post-test effect of happiness on emotion regulation and life satisfaction (Satisfaction with Life Scale; Diener, 1985) were examined in 27 individuals with eating disorders and 27 healthy controls. Study 5 looked at how happiness induction over a period of time influenced emotion regulation (Regulation of Emotion Questionnaire; Phillips and Power, 2007), eating behaviours (EDE-Q, Fairburn and Beglin, 1994) and life satisfaction (Satisfaction with Life Scale; Diener, 1985) in 27 eating-disordered females.

Chapter 8 provides a general discussion of the findings of the studies with a particular focus on the findings of Studies 4 and 5. Effectiveness of emotion induction methods that were used in the thesis is discussed. In addition, Chapter 8 critically examines the studies’ findings on emotion regulation, Affect Intensity and the role of positive affect in improving regulatory choices, and their implications for theory and treatment of eating disorders. The Chapter also provides suggestions and ideas for future research and practical application of the findings.
CHAPTER 1. EATING DISORDERS

Eating disorders have been receiving a lot of attention for the past 30 years. There is a concern among clinicians and researchers alike that anorexia nervosa, bulimia nervosa and Eating Disorder Not Otherwise Specified (EDNOS) are becoming more and more common (Hoek and van Hoeken, 2003; Palmer, 2000; van Hoeken et al., 2003). Eating disorders have very complex aetiology, and there are multiple sociocultural, biological and individual vulnerability factors that may predispose an individual to eating pathology. Because of this, anorexia, bulimia and EDNOS are extremely difficult to prevent and very hard to treat (Treasure and Schmidt, 2003). Pharmacological and psychological treatments tend to have limited effectiveness, and there is a high relapse rate (Buckroyd, 2009). In some cases eating disorders can be life-threatening. Both anorexia and bulimia are associated with low self-esteem, affective and obsessive compulsive psychopathology and carry the risk of suicide (Palmer, 2000). Because eating pathology in clinical and subclinical forms is quickly becoming a ‘social epidemic’ (Gordon, 1990), it is important to understand what causes it and why treatments are relatively ineffective. This chapter looks at our present understanding of what eating disorders are, what underpins them and what treatment options are available.

Diagnosis, Features and Prevalence

ANOREXIA NERVOSA

Anorexia nervosa is an eating disorder where preoccupation with weight and shape is characterized by significant voluntary reduction of food intake and dramatic weight loss (Palmer, 2000). This illness has a long history, dating back to biblical times (Silverman, 1995). Anorexia nervosa is cross-culturally universal (Keel and Klump, 2003) in that its incidence has been recorded in all known societies at some point in time.

An enduring illness, anorexia can affect individuals of both genders and all ages, although it is 10 times more common in young females (Walters and Kendeler, 1995). The mental health charity Mind (Registered Charity No. 219830; 2011) estimated that around one per cent of women between the ages of 15 and 30 have anorexia; it is much
rarer in men (van Hoeken et al., 2003; Szmukler et al., 1985; Turnbull et al., 1996). Gender may be one of the key vulnerability factors for developing this disorder (Treasure et al., 2010). Another one is age. Anorexia nervosa typically starts in adolescence with the average age of onset being around 17 years of age (Crisp, 1980). When it comes to anorexia, age seems to be an aetiologically important factor, as latest research shows that it may be impossible to develop anorexia later in life (Scholtz et al., 2010).

As outlined in the DSM-IV (Table 1), in order to be diagnosed with anorexia, the individual has to meet the following diagnostic criteria: (1) refusal to maintain body weight at or above what is normal for age and height (e.g. body weight less than 85% of the norm), (2) intense and morbid fear of weight gain, (3) misperception of own weight/shape (i.e. distortion of body image), excessive importance ascribed to weight (weight as a measure of self-worth), and (4) in post-menarchal females, absence of at least 3 consecutive menstrual cycles (i.e. amenorrhea; Garfinkel, 1995).

Amenorrhea has been nominated for elimination as a diagnostic criterion for anorexia (Attia and Roberto, 2009; Mitchell et al., 2005) from the upcoming fifth edition of the DSM (DSM-V). In their review of literature on amenorrhea, Attia and Roberto (2009) question its primacy and validity as a core criterion for anorexia. They posit that amenorrhea is a consequence of malnutrition and weight loss, and reflects severity of the condition, rather than being an independent accompanying or predisposing factor. In addition, they note that this criterion is irrelevant to some anorexia sufferers, as it excludes males, females taking the contraceptive pill, pre-menarchal and post-menopausal females. Therefore, their suggestion is to omit it from the core diagnostic criteria.

For diagnostic and treatment purposes, anorexia is further subdivided into two forms: binge-eating/purging and restrictive (Garfinkel, 1995). The former type refers to regular binge-eating (i.e. eating more than objectively normal in a short period of time) or purging behaviour (e.g. use of laxatives) during the current episode of the illness (Table 1). This type of anorexia symptomatically mimics periodic bulimia, with the exception of weight; it is sometimes termed bulimexia. The term restrictive anorexia stands for absence of such symptomatology during the current episode of anorexia nervosa, and
refers to consistent dietary restriction (Table 1). According to Aronson (2001), there is an equal split between the two types in the anorexic population.

**Table 1: DSM-IV Diagnostic Criteria for Anorexia Nervosa.**

<table>
<thead>
<tr>
<th></th>
<th>ANOREXIA NERVOSA</th>
</tr>
</thead>
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<tr>
<td><strong>General Diagnostic Criteria</strong></td>
<td><strong>Specific Diagnostic Criteria</strong></td>
</tr>
<tr>
<td>Refusal to maintain body weight at or above a minimally normal weight for age and height or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected.</td>
<td><strong>Restricting Type</strong></td>
</tr>
<tr>
<td>Intense fear of gaining weight or becoming fat, even though underweight.</td>
<td><strong>During the current episode of Anorexia Nervosa, the person has not regularly engaged in binge-eating or purging behaviour (self-induced vomiting or misuse of laxatives, diuretics, or enemas).</strong></td>
</tr>
<tr>
<td>Disturbance in the way one's body weight or shape is experienced, undue influence of body weight or shape on self evaluation, or denial of the seriousness of the current low body weight.</td>
<td><strong>During the current episode of Anorexia Nervosa, the person has regularly engaged in binge-eating or purging behaviour.</strong></td>
</tr>
<tr>
<td>In post-menarchal females, amenorrhea, i.e., the absence of at least 3 consecutive menstrual cycles. A woman having periods only while on hormone medication (e.g. oestrogen) still qualifies as having amenorrhea.</td>
<td></td>
</tr>
</tbody>
</table>

Source: adapted from the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV), American Psychiatric Association (2000).

Interestingly, there appear to be some differences between purging and restricting individuals. Those of former subtype tend to have a family history of obesity, higher weight prior to the onset of the illness and an impulsive personality; they also tend to be more prone to substance abuse than restricting individuals (Garfinkel, 1995; Aronson, 2001; Peat et al., 2009). These characteristics appear to make purging anorexics similar to bulimic patients. However, Eddy et al., (2002) failed to find stable differences between the two anorexia sub-types and suggested that the restricting type of anorexia ‘represents a phase in the course of anorexia, rather than a distinct subtype’ (Eddy et al.,
Their results showed that the restrictive and purging types did not differ in terms of illness course, impulsivity, suicidality, substance abuse, and other indicators of psychopathology (Eddy et al., 2002). Therefore, the evidence for the current subtyping system of anorexia remains mixed.

As part of the diagnostic procedure, clinicians may use the the BMI (Body Mass Index) to estimate whether an individual has low weight (Table 2). The individual’s BMI is calculated by dividing their weight in kilograms by their height in meters squared (BMI = kg/m²). Generally, the normal weight range is between 20 and 25; someone with a BMI below 20 would be classed as underweight, and someone with BMI over 25 - as overweight. The World Health Organization (WHO, 2010) recommends the BMI of 18.50 and 25 as cut-off points. As can be seen from Table 2, the underweight category has three levels: mild thinness (17 < BMI < 18.50), moderate thinness (16 < BMI < 17) and severe thinness (BMI < 16). The overweight category comprises pre-obesity (25 < BMI < 30) and three levels of obesity (BMI ≥ 30).

Table 2: Classification of weight, according to the Body Mass Index norms (BMI)

<table>
<thead>
<tr>
<th>Classification</th>
<th>BMI Score: Cut-Off Points</th>
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<tr>
<td>UNDERWEIGHT</td>
<td>&lt;18.50</td>
</tr>
<tr>
<td>Severe thinness</td>
<td>&lt;16</td>
</tr>
<tr>
<td>Moderate thinness</td>
<td>16 - 16.99</td>
</tr>
<tr>
<td>Mild thinness</td>
<td>17 - 18.49</td>
</tr>
<tr>
<td>NORMAL RANGE</td>
<td>18.50 - 24.99</td>
</tr>
<tr>
<td>OVERWEIGHT</td>
<td>≥25</td>
</tr>
<tr>
<td>Pre-obesity</td>
<td>25 - 29.99</td>
</tr>
<tr>
<td>OBESITY</td>
<td>≥30</td>
</tr>
<tr>
<td>Obesity class I</td>
<td>30 - 34.99</td>
</tr>
<tr>
<td>Obesity class II</td>
<td>35 - 39.99</td>
</tr>
<tr>
<td>Obesity class III</td>
<td>≥40</td>
</tr>
</tbody>
</table>

Source: adapted from the Global Database on Body Mass Index (WHO, 2010)
Anorexia nervosa is often accompanied by a major disturbance in functioning of reproductive and endocrine systems, such as loss of libido, cessation of periods (i.e. amenorrhea) and infertility in females and loss of sexual potency in males (Garfinkel, 1995). Other features of anorexia may include: brittle hair and dry skin, increase in lanugo (i.e. body hair), low blood pressure, heart problems (e.g. cardiac arrhythmias), anemia, osteoporosis, digestive system malfunction and hypothermia (DSM-IV, APA, 2000; Treasure et al., 2010). The individual may feel extremely tired, or, on the contrary, extremely awake and alert (DSM-IV, APA, 2000).

Individuals suffering from this type of eating disorder employ a wide array of pathological behaviours that are designed to decrease their weight and keep it below norm. Their weight-loss repertoire typically includes self-starvation, use of appetite suppressants, self-induced vomiting, misuse of enemas, laxatives or diuretics (Garfinkel, 1995). Anorexic individuals try to conceal their eating habits. Despite the considerable physical and psychological discomfort and effort associated with the disorder (e.g. eating restraint and hunger), individuals are typically resistant to help, and often insist that they are fine (Crisp, 1980; Garfinkel, 1995).

BULIMIA NERVOSA

This is another major type of disordered eating. An individual with bulimia nervosa struggles to control their overeating and uses pathological means to compensate for their excessive food intake (e.g. self-induced vomiting). Research and clinical practice show that it can sometimes develop out of anorexia, and there is often a considerable overlap between the symptoms of anorexia and bulimia. Since a person can shift from one to the other and back along the continuum of eating pathology, this led some researchers to suggest that anorexia and bulimia are not two discreet conditions, but stages in the course of eating pathology (Aronson, 2001; Palmer, 2000).

Bulimia is a relatively new illness, and it was only in the 1970s that it was identified as a disorder in its own right (Garfinkel, 1995). It typically develops in late teenage years and early twenties (Palmer, 2000). Just like in anorexia, gender appears to be a vulnerability factor for developing bulimia. Prevalence rates for bulimia are higher in females, as around 90% of sufferers are female (Gotestam and Agras, 1995). This
means that females may be 10 times more likely than males to be affected by this condition. According to the mental health charity Mind (2011; Registered Charity No. 219830), currently one or two per cent of women in the UK have bulimia; this makes it a more widespread eating disorder than anorexia (Hoek and van Hoeken, 2003).

DSM-IV (APA, 2000) outlines the following clinical features of bulimia nervosa: (1) recurrent episodes of excessive food consumption in a short period of time, characterized by loss of control over eating (i.e. a binge) plus (2) unhealthy and extreme compensatory weight-control behaviours, such as misuse of medicines, enemas, fasting, self-induced vomiting and excessive exercise (Table 3). On the whole, bulimia has fewer physical symptoms than anorexia, but can be just as damaging and distressing.

Table 3: DSM-IV Diagnostic Criteria for Bulimia Nervosa.

<table>
<thead>
<tr>
<th>General Diagnostic Criteria</th>
<th>Specific Diagnostic Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recurrent episodes of binge eating characterized by both:</td>
<td>Purging Type</td>
</tr>
<tr>
<td>• Eating in a discrete period of time an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.</td>
<td></td>
</tr>
<tr>
<td>• A sense of lack of control over eating during the episode</td>
<td></td>
</tr>
<tr>
<td>Recurrent inappropriate compensatory behaviour to prevent weight gain, such as self-induced vomiting, misuse of laxatives, diuretics, enemas, or other medications, fasting, or excessive exercise.</td>
<td>During the current episode of bulimia nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas</td>
</tr>
<tr>
<td>The binge eating and inappropriate compensatory behaviour both occur, on average, at least twice a week for 3 months</td>
<td></td>
</tr>
<tr>
<td>Self-evaluation is unduly influenced by body shape and weight</td>
<td></td>
</tr>
</tbody>
</table>

Source: adapted from the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV), American Psychiatric Association (2000).

Depending on the nature of the compensatory behaviour, bulimia may be classed as either purging or non-purging (Garfinkel, 1995). The former refers to abuse of medications (e.g. laxatives) and self-induced vomiting as primary means of weight
control (Table 3), whereas the latter stands for other extreme compensatory behaviours (Table 3). The usefulness of subtyping bulimia has been questioned (van Hoeken et al., 2003), because it appears that different types simply represent a different degree of severity of the disorder, as opposed to an altogether distinct form.

On cognitive and psychological levels, bulimia is characterized by the distortion of body image, weight-based self-valuation, dread of fatness and intense shame about eating behaviours. For the diagnosis to be made, the symptoms should occur at least twice a week for 3 months. Bulimia may be accompanied by such physical features as swollen glands, a puffy face and dental enamel erosion that may result from vomiting (Garfinkel, 1995). Due to erratic eating patterns and distress associated with the condition, menstrual irregularity may also be present.

EDNOS (Eating Disorder Not Otherwise Specified)
This is a third diagnostic category in DSM-IV (Table 4); approximately 50% of all referred for eating disorder assessment receive this diagnosis. It includes all the variations of eating pathology that do not meet the diagnostic criteria for anorexia and bulimia, such as binge eating disorder, as well as atypical forms of anorexia and bulimia. For instance, someone who eats normal amounts (as opposed to bingeing) and then makes themselves throw up, would be placed into this category. Despite its nosological usefulness, this diagnostic category is not narrow enough to reliably inform treatment; therefore the proposals have been made to change it in the upcoming DSM-V (Treasure et al., 2010).

Binge-eating disorder is outlined in DSM-IV (APA, 2000), and it is worth mentioning it here briefly. This thesis focuses on anorexia nervosa, bulimia nervosa, EDNOS and subclinical disordered eating, because these are the types of eating pathology that the participants in Studies 1-5 had. As none of the participants had the clinical diagnosis of a binge eating disorder, this type of eating pathology is not given detailed attention in the thesis chapters. Symptomatically the binge eating disorder is similar to bulimia nervosa in that a binge-eating patient also regularly overeats, consuming large amounts of food in short periods of time. However it differs from bulimia nervosa in that bingeing is not followed by purging and other compensatory behaviours; another
difference may be that the person tends to be concerned primarily about their loss of control over eating and the bingeing behaviour itself, rather than about the resultant weight gain. The binge-eating disorder is currently not recognised as a separate condition and falls under the EDNOS category (DSM-IV, APA, 2000).

Table 4: DSM-IV Diagnostic Criteria for Eating Disorder Not Otherwise Specified (EDNOS)

<table>
<thead>
<tr>
<th>General Diagnostic Criteria</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disorders of eating that do not meet the DSM-IV diagnostic criteria for anorexia nervosa and bulimia nervosa</td>
<td>For female patients, all of the criteria for anorexia nervosa are met except that the patient has regular menses.</td>
</tr>
<tr>
<td></td>
<td>All of the criteria for anorexia nervosa are met except that, despite significant weight loss, the patient's current weight is in the normal range.</td>
</tr>
<tr>
<td></td>
<td>All of the criteria for bulimia nervosa are met except that the binge eating and inappropriate compensatory mechanisms occur less than twice a week or for less than 3 months.</td>
</tr>
<tr>
<td></td>
<td>Binge-eating disorder: recurrent episodes of binge eating in the absence if regular inappropriate compensatory behaviour characteristic of bulimia nervosa.</td>
</tr>
</tbody>
</table>

Source: adapted from the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV), American Psychiatric Association (2000).

The three clinical diagnoses often overlap. Barretta et al. (1998) suggest that there is a continuum from normal eating to severely disordered eating, and individuals may transition from EDNOS to anorexia and to bulimia. In practice, anorexia, bulimia and EDNOS are not clear-cut and stable conditions, and patients tend to cross over from one diagnostic category into another. According to Barretta et al. (1998) the individual may make a linear transition from normal eating to bulimia, or then move back and forward on the continuum between different disordered eating conditions (e.g. from anorexia to bulimia to binge eating disorder). This suggests not only that there is a common underlying factor in all eating pathology, but also that the use of diagnosis as a guide for assigning specific treatment (e.g. family therapy for anorexia, cognitive behavioural therapy for bulimia) is not always justified.

SUBCLINICAL DISORDERED EATING

With regard to subclinical forms of eating disorders, the continuum theory of severity has been proposed (Barretta et al., 1998). As can be seen on Figure 1, under certain
circumstances normal eating may turn into subclinically disordered eating, which over time may result in the development of a full-blown eating disorder (Barretta et al., 1998). The continuity of symptoms and different degrees of intensity of eating pathology mean that there are many subclinical forms of eating disorders (Gordon, 1990). This has been confirmed in multiple studies (Button and Whitehouse, 1981; Crisp, 1980; Schmukler, 1983). Hesse-Biber et al. (1999) argue that ‘a wide range of attitudes and behaviours related to food and image exists in the ‘grey zone’ all along the two ends of continuum [healthy attitudes to eating on one end and clinical eating disorder on the other]’. This grey zone (Hesse-Biber et al., 1999) contains pathological eating behaviours and cognitions that do not meet the formal diagnostic criteria, either because of their frequency, their severity, or their nature. There is evidence to suggest that subclinical forms of eating disorders may progress to a full-blown clinical diagnosis at a later date (Barretta et al., 1998).

Figure 1: Timeline of eating pathology development

Subclinical eating pathology, including body image disturbance and the desire to be thin, typically occurs in young women of normal weight (Gray and Ford, 1985; Hesse-Biber, 1989, Zuckerman et al., 1984). Just like its clinical manifestation, subclinical eating pathology has been found to share comorbidity with mood and anxiety disorders (Touchette et al., 2010). Subclinical eating disorders appear to share a lot of the qualitative characteristics of clinical eating disorders; the behavioural, cognitive and affective symptoms (e.g. emotion recognition and management difficulties; Ridout et al., 2010) are analogous. The difference appears to be largely quantitative: e.g. the length of disordered eating duration and severity of the symptoms.

These subclinical eating disorders, or partial syndrome eating disorders (Shisslak and Crago, 1994) appear to be very common, particularly among female university students
Touchette et al. (2010) found that in their sample of 833 adolescent girls subclinical anorexia nervosa (restricting type) had a prevalence of 16.8% and the rate for bulimia was 3.8%. This is very high. For comparison, prevalence rate for full-syndrome anorexia nervosa on average equals 0.3% and 1% for bulimia in the adolescent female population (van Hoek et al., 2003). This means that the subclinical eating disorders may be more widespread than the clinical syndromes (Dancyger and Garfinkel, 1995). Despite this, there has been surprisingly little research on subclinical eating disorders.

**Outcome of Eating Disorders**

Eating disorders tend to have a broad negative influence on physical health, and social functioning of those affected. They are very debilitating and can be life-threatening. Anorexia is associated with a relatively high risk of relapse and mortality (between 5 and 8%; Herzog et al., 2000; Gordon, 1990), as well as a low recovery rate and physical complications (Kaye, 1999; Kaye et al., 1998; 2000; Mizes and Palermo, 1997; Vitello and Lederhendler, 2000). Mortality rates in anorexia are high because of the often irreversible health damage (Treasure et al., 2010; van Hoeken et al., 2003) and suicide risk (Harris and Barraclough, 1996). In comparison, bulimia, where there are fewer physical symptoms, has a much lower mortality rate of 2% (Fichter and Quadflieg, 2004).

Dramatic reduction in calorie intake and resultant weight loss, associated with anorexia, can lead to organ failure. Without adequate nutrition the anorexic’s body may begin to shut down (Palmer, 2000); for instance, it may start using the heart muscle as protein to nourish itself when the individual has used up all the fat resources (Gordon, 1990). Weakening of the heart muscles may result in a permanent heart damage, which would affect functioning of the cardiovascular system and all other bodily processes (Gordon, 1990). In addition, there is a hypothesis that anorexia may result in a brain deficit, specifically, hypothalamic abnormality. Prolonged starvation may lead to malfunctioning of the hypothalamus (Bhanji and Mattingly, 1988). The lateral hypothalamus is involved in regulation of appetite, and its dysfunction may lead to misperception of hunger signals and loss of the drive to eat. However, at present, this is
just a hypothesis, and the data come from animal studies (Hoebel and Teitelbaum, 1966; Stoving et al., 1999) and need further testing on humans.

The health outcomes of bulimia tend to be just as negative as those of anorexia. In bulimia, there is a danger of a lasting, irreversible electrolyte imbalance and hypocalemia in those misusing laxatives (these are conditions where the body is so depleted of minerals that its functioning is severely impaired), and damage of the digestive tract and teeth as a result of purging (Aronson, 2001). In addition, both anorexia and bulimia carry the risk of hormonal problems, such as menstrual irregularity, amenorrhea and infertility in women.

Eating disorders also often have a detrimental effect on the patients’ relationships (Schmidt and Treasure, 2006). This may be because they try to conceal their eating habits and focus on food at the expense of social relationships. This may ultimately lead to social withdrawal (Karatzias et al., 2009). For instance, an anorexic may avoid family dinners and friends’ birthday parties because of the fear of gaining weight, and this would limit opportunities for social interaction and support. This means that after a certain period of time individuals with eating disorders may to some extent lose their support network which would subsequently make recovery and positive change even harder for them (Karatzias et al., 2009). Eating disorders often have a devastating effect on the family of the patient, creating a stressful atmosphere and putting a strain on the relationships within the family (Ball and Ball, 1995; Hildege et al., 2005; Schmidt and Treasure, 2006). Therefore eating disorders endanger not just the physical health of those affected, but also their emotional health by disrupting their social relationships. To sum up, what makes eating disorders dangerous is physiological damage caused by erratic eating and compensation practices, and their negative impact on relationships.

**Aetiology and development of eating disorders**

There are multiple theories to account for the development of eating disorders. Factors such as eating restraint, biology, sociocultural context, family, cognition, disposition and trauma may make an individual vulnerable to disordered eating. It is interesting that anorexia and bulimia have similar aetiology and risk factors; however, at present it is
not known for sure why some people develop one as opposed to the other, and what differentiates those anorexics who go on to develop bulimia from those who do not. It is likely that there is no uniform pathway to developing eating disorders, and that a combination of several different predisposing and risk factors in the individual’s life result in the eating pathology outcome. This section gives an overview of the theories of eating disorders: from eating restraint to individual differences.

EATING RESTRAINT AND DIETING

An eating disorder may begin as an intention to lose weight by dieting (Palmer, 2000; Russell, 1995). Dieting refers to intentional restriction in the variety and quantity of food eaten, in line with a certain set of rules over a pre-determined period of time with the purpose of losing weight.

There is evidence to suggest that over time dieting may develop into a full-blown eating disorder (Hesse-Biber et al., 1999) in susceptible individuals, and this makes dieting physiologically and psychologically dangerous, especially in those with a normal BMI. In individuals with a healthy weight there is no medical need for (or benefit from) weight loss and dieting (Brownell and Rodin, 1994), and food restraint may indeed be harmful. Physiologically, a drastic restriction of calorie intake and decrease in food variety may lead to depletion of vitamin and mineral stores in the body. This in turn may lead to health problems, such as low immunity. Psychologically, dieting is a double-edge sword because on the one hand weight loss may provide the dieter with a sense of achievement and a boost to self-esteem (Garner and Bemis, 1982; Overton et al., 2005; Vitousek and Ewald, 1993) and on the other hand, it is a constant highly unpleasant battle with hunger, cravings, preoccupation with food (Keys et al., 1950) and low mood (Wilson, 1995). This has been termed a dieter's dilemma (Palmer, 2000). However, in severely overweight dieters where the excess weight creates health problems, eating restraint is typically associated with more positive outcomes (Wilson, 1995).

A possible route of development of anorexia in the process of dieting may be as follows. An individual embarking on a diet may rightly or wrongly perceive themselves
to be fat, or fatter than they really are, and to blame food and their weight for all their problems. They may pick up mass media messages about weight control methods and to take them on board (e.g. avoiding fast food). The individual may first cut down on or completely exclude certain food groups believed to be bad (e.g. fats and sugars), begin to skip meals and become fussy about what they eat. After a certain period of time, their restrictive eating is rewarded with the much-desired weight loss and a sense of achievement, which may propel the person to stick to dieting. Further down the line, the weight loss inevitably hits a plateau which is typically met with frustration; the individual further reduces their calorie intake and increases energy expenditure to promote further weight loss and to feel good about themselves again. Gradually reducing the amount and variety of the food they consume in order to stimulate further weight loss, the individual may come to a point when their daily calorie intake is dangerously low. This is how someone may gradually go from dieting to starvation.

Dieting creates an illusion of control over hunger and body weight. But this sense of control is only temporary, because it is impossible to maintain a low calorie diet over a prolonged period of time, and many dieters find themselves relapsing into their old eating habits rather soon. In some dieters, hunger may become so intense that they give in to their urge to eat during their diet and inevitably overeat. So called *yo-yo dieting*, or weight cycling (Wilson, 1995), is a common consequence of motivated food restriction. Having embarked on a diet, the individual oscillates between eating too much and too little, and rapidly loses and regains weight in cycles (Wilson, 1995). In such individuals the process of weight loss and gain is typically accompanied by fluctuations in mood, dissatisfaction with their body and disappointment with themselves (Wilson, 1995). A body that has been starved of nutrients for a significant period of time becomes impossible to control around food; the more the individual eats, the more they may want to eat (this is termed *counterregulation*; Polivy and Herman, 1995).

The causes of *counterregulation* may be psychological and physiological. Psychologically, the individual may realize that they have broken the dieting rules by bingeing, and hence lose all motivation for dieting. Physiologically, there may be a breakdown in the satiety detection process (Halmi, 1995); for instance, it is known that
in women undereating may alter the production of serotonin (5HT), so that the sense of satiety is subdued (Goodwin et al., 1987). The individual may feel shame, guilt and self-disgust after their binge, and may make themselves vomit or otherwise dysfuctionally compensate in order to get rid of the food they have eaten. This makes them feel that they have regained control over their body and have found a new effective way of weight management. This is a plausible route for developing bulimia nervosa and EDNOS (DSM-IV, APA, 2000).

However, the majority of individuals who go on a diet do not develop clinical eating disorders, which leads researchers and clinicians to believe that other risk and vulnerability factors, in addition to eating restraint, are necessary to trigger eating disorders (Wilson, 1995). It is likely that in certain individuals anorexia, bulimia and EDNOS can arise from eating restraint, and that dieting may be a risk factor if there are other vulnerability factors present.

**BIOLOGY AND GENETICS**

There is some evidence to suggest that eating disorders have biological aetiology. In particular, anorexia has been found to be partly genetic and to run in families (Klump et al., 2000; Rutherford et al., 1993). With biological factors such as brain structure abnormalities and neuroendocrine disorders, it is hard to discern which ones of them are the cause and which ones are the effects of eating pathology. This section provides an overview of biological explanations of eating disorders.

**Puberty**

Because eating disorders typically start during puberty and early adulthood, some researchers think that physiological changes associated with maturation may be directly or indirectly responsible for eating disorders (Crisp, 1980). For example, a young girl entering puberty may notice that her body is changing: her weight is increasing and her shape is becoming more womanly. This process may be perceived as scary and uncontrollable; she may not be ready to grow up. This maturational crisis (Crisp, 1980) may result in her dieting in order to lose weight and stay pre-pubescent. By dieting the girl is also unwittingly ensuring that she does not mature hormonally (a certain
percentage of body fat is needed for menarche to occur). Palmer calls this an attempt at ‘psychobiological regression’ (Palmer, 2000). Restriction of calorie intake and the desire to prevent maturational changes may develop into an obsession with weight and eating (Crisp, 1980). This means that anorexia may start because of the teenager’s motivation to reverse physiological changes at puberty and slow it down.

The connection between eating pathology and puberty is less immediately obvious for bulimia. The age of illness onset is later for bulimia (Palmer, 2000) and this eating disorder can start at any age (unlike anorexia). In addition, bulimia typically does not bring any significant or reliable reduction in weight. Therefore at a first glance it appears unlikely that bulimia may develop as a result of psychological dealing with pubescent changes. However, in some individuals it may be possible. Barretta et al. (1998) suggest that bulimia may be a phase after anorexia, and this may explain why the age statistics are higher for bulimia than they are for anorexia. Looked at this way, bulimia in adolescents may develop out of anorexia, and the anorexia may have been the result of overzealous dieting and a desire to reverse the maturational process. This is a plausible hypothesis of puberty crises resulting in eating disorders.

**Endogenous opioids and Addiction**

Another biological explanation of why anorexia is maintained even though it involves consistent considerable effort and discomfort (e.g. hunger) is that starvation alone or in combination with excessive exercise causes the brain to secrete endogenous opioids (Bergh and Sodersten, 1996; Casper, 1998). Body’s endogenous opioids (e.g. the β-endorphins) may function as negative reinforcers of eating disordered behaviours and are associated with feelings of euphoria (Davis and Claridge, 1998, Hardy and Waller, 1988). This is known as the auto-addiction opioid model of anorexia (Marazzi and Luby, 1986). The model posits that eating pathology is an addiction to eating disordered behaviours, mediated by endogenous opioids. In other words, Marazzi and Luby (1986) suggest that the mechanisms, responsible for the development and maintenance of eating disorders are the same as those in substance abuse (Marazzi and Luby, 1986). This model is supported by findings that eating-disordered patients tend to score highly on addiction measures (Davis and Claridge, 1998). Individuals with eating pathology
may become ‘addicted’ to dysfunctional weight management practices, and their obsession with food avoidance and weight-watching in its intensity mirrors that of someone with a substance dependence problem.

Elation, induced by endogenous opioids, is a pleasurable and addictive altered awareness state; it occurs because self-starvation results in a reduction in serotonin (5-HT) activity in the brain (Kaye, 1999; Kaye et al., 1998, 2000). The individual begins to feel happy and comforted when they restrain their eating, and this may make the eating disorder experience worth the discomfort and effort for them (Marazzi and Luby, 1986). They sacrifice the health of their body for a sense of achievement and control that they get when they are severely limiting the amount they eat. This sense of control spans beyond the individual’s eating into other areas of their life, and they may feel temporarily relieved and liberated from negative affect and anxiety (Davis, 2001; Gerhardt, 2009). However this positive feeling is cyclic and fleeting, and cannot be maintained because of the constant battle with hunger. The regulation of negative affect and seeking of positive emotions are the motivational routes (Davis, 2001) along which eating pathology may develop.

The auto-addiction opioid model (Marazzi and Luby, 1986) may also be applied to bulimia nervosa and EDNOS, in light of the promising recent evidence that frequent excessive consumption of sugar is associated with endogenous opioid dependence in rats (Colantuoni et al., 2002). The researchers found that food deprivation, alternating with administration of large quantities of sugar, ultimately led the rats to develop sugar withdrawal symptoms, which is indicative of the second stage of dependency (Colantuoni et al., 2002). If we apply these results to humans, it may mean that the combination of a diet high in sugar and starvation would result in opiate addiction, and this may be one of the maintaining factors in eating pathology. In other words, a bulimic patient who alternates high-sugar binges with compensatory starvation, may become biochemically addicted to this behaviour cycle. This would make it so much harder to recover from the eating disorder. More research is needed to establish whether this is in fact what occurs.
**Genetics, brain structure and neuroendocrine disorder**

Genes may be a substantial factor in the development of anorexia (Holland et al., 1984; Treasure and Holland, 1995; Strober, 1995; Strober et al., 2000). It has been shown that inheritance rates for anorexia are more than 50%, and it is frequently diagnosed in members of the same family (Klump et al., 2001; Kortegaard et al., 2001; Rutherford et al., 1993). This suggests that there is an underlying genetic make-up that makes individuals more susceptible to anorexia, but the mechanism of this is at present unclear (Polivy and Herman, 2002). In addition, anorexics have been found to have abnormalities in their brain structure, which may or may not be genetic. Chui et al. (2008) reported that patients with acute anorexia had larger lateral ventricles than those with a more mild form of the eating disorder, as well as healthy controls. In addition, those with severe anorexia were found to have smaller brain volumes (Katzman et al., 1996; Lambe et al., 1997). More research is needed to determine the temporal course of the structural abnormalities and their possible genetic origin. Furthermore, individuals with anorexia typically have an over-reactive stress response and consistently high levels of stress hormone cortisol in the brain (Hoek et al., 1998). Cortisol may be responsible for the negative affect (e.g. dysphoria) which inevitably accompanies eating disorders.

Interestingly, bulimia, unlike anorexia, has not definitively been shown to have a genetic component (Kassett et al., 1989; Keel and Klump, 2003; Winchester and Collier, 2003). In fact, it does not seem to be driven by biology at all; bulimia appears to be more of a sociocultural product. With regard to this disorder, only one major biological hypothesis has been proposed: that of low serotonin levels (Brewerton, 1995). Low serotonin could be the result of a malfunctioning neuroendocrine system in those with eating disorders (Pirke, 1995) as a consequence of stress. In particular, it has been found that individuals with bulimia show a decreased production of pituitary hormones, such as luteinising hormone (LH) and follicular stimulating hormone (FSH) (Palmer, 2000; Pirke, 1995). As with most other theories, at present it is hard to discern whether low levels of these hormones are the cause or consequence of eating disorder symptomatology.
Conclusions

Validity of the biological theories outlined above is perhaps best demonstrated by their practical applications, i.e. effectiveness of pharmacological treatment (please see Pharmacological Treatment). Bulimia has been successfully treated with selective serotonin re-uptake inhibitors (treatments that boost serotonin levels), whereas no drugs have been effective enough in treating anorexia. It is plausible that biological pathogenesis of anorexia may consist in the fact that it is partly inherited and maintained by endogenous opioids, whereas bulimia does not have a clear aetiological genetic component and may be partly caused by low serotonin levels.

SOCIOCULTURAL FACTORS

According to some researchers, it is the media obsession with weight, obesity and dieting in the last few decades that has led to the increase in incidence of eating disorders in the West (Palmer, 2000; Striegel-Moore, 1993, 1997). Peer pressure to conform to the new societal beauty ideal and family influences also may have contributed to the rising numbers of cases with eating pathology in industrialized countries. This section gives an overview of the sociocultural factors in development of eating disorders.

Mass media

In contemporary Western society there is a considerable pressure on men and women to be thin (Palmer, 2000). Female slimness, in particular, is celebrated in the media and over the past 50 years has become a social value and a measure of beauty and success (Striegel-Moore, 1993, 1997). It is not surprising that body dissatisfaction has reached epic proportions in the West. There is an increase in eating disorders as well as in obesity. Some researchers argue that the rise in eating pathology incidence (e.g. undereating and overeating) is a sociocultural product (Lindberg and Hjern, 2003). This is because eating pathology appears to thrive in societies where there is an abundance of easily available, cheap high calorie food (Polivy and Herman, 2002). Eating disorders used to be considered exclusive to the white, middle class strata of the Western culture. However the rise of obesity and eating pathology in the recent years can be observed all
over the world which suggests that it is less culture- and class-specific than previously thought (Nasser and Katzman, 2003). According to some researchers, globalization and transmission of beauty standards has contributed to the spreading of eating pathology from the industrialized countries to the rest of the world (Nasser, 1997).

Eating disorders are what Gordon (1990) calls ‘a culture-bound syndrome’, in that they are not present in all cultures at all times. Eating disorders may represent ‘a common pathway for expression of idiosyncratic personal problems and psychological distress’ of the young generation, and eating pathology may provide a ‘template for deviance’ (Gordon, 1990). In other words, in a certain sociocultural climate, eating disorders may emerge as a popular means of channelling distress. This suggests that eating pathology may be functional because it is used to signal to others that the individual is distressed and needs help. Their body ‘says’ that they are not coping with life, and their eating disorder is an expression of general distress (Gordon, 1990). Mass media may facilitate and encourage transmission of this ‘template of deviance’ (Gordon, 1990).

In the West the upsurge in eating disorders after the 1960s coincided with the return of the fashion for thinness (Russell, 1995), with the societal female body ideal gradually shrinking to a ‘size 0’ by 2010 (Orbach, 2009). This, coupled with media encouragement for dieting and prejudice against obesity, has contributed to what has been labelled as a ‘social epidemic’ (Gordon, 1990). According to the eating disorders charity B-EAT (2011), now around 1.6 million people in the UK have an eating disorder, most of whom are young people between 14 and 25 years of age; it is worth noting that this figure refers to detected cases, the actual prevalence rate could be higher (Hoek and van Hoeken, 2003).

Passive exposure to mass media as well as active seeking of beauty and dieting advice in the media have been found to relate to idealisation of thinness, body dissatisfaction and eating problems in females (Harrison, 2001; Levine and Murnen, 2009). The following process has been proposed as a plausible mechanism of pathogenesis of bulimia and anorexia in females (Orbach, 2009, Palmer, 2000). Media images of slim celebrities make women feel unhappy with their looks and encourage dieting behaviours.
which in turn may trigger an eating disorder (Harrison, 2001; Polivy and Herman, 1985; Tiggemann and Pickering, 1996). The disparity between media-promoted ideal body and a real female body may drive women to resort to extreme measures of weight loss, such as crash dieting, ‘fad diets’, vomiting after meals or severe food restriction (Harrison, 2001; Orbach, 2009). This theory of how mass media may trigger an eating disorder is known as the *self-discrepancy theory* (Higgins et al., 1986; Harrison, 2001). According to this theory, the individual assesses the difference between their *actual self* (the way they look) and their *ought self* (the way they would like to look). If the difference between the two is perceived to be large, this creates negative affect and determination to reach the ideal by whatever means necessary. This process may be internally-driven, or stem from the outside influences (e.g. mass media). *Accessibility of discrepancy*, or how often the individual is reminded of the difference between the actual and ought self, is an important factor (Higgins et al., 1986). In case of weight, this may mean that if the individual perceives themselves to fall short of the societal attractiveness ideal and is constantly reminded about it by the mass media, they may resort to unhealthy means of reaching their desired body weight (e.g. self-starvation).

The fact that eating disorders develop only in the minority of those in the Western culture (Polivy and Herman, 2004) suggests that media pressure may merely be a triggering factor, as opposed to causal one in development of eating disorders. This is known as the *relative-rarity argument* (Levine and Murnen, 2009). The strength of self-concept was put forward as the factor that mediates the relationship between media exposure and development of eating disorders (Vartanian, 2009). According to Vartanian (2009), the extent to which the media messages about body ideal are internalized depends on stability of the individual’s self-concept which refers to the sense of self. Those with a weak self-concept may be more inclined to conform to the societal standards of beauty and to define themselves by their body weight (Vartanian, 2009). A strong sense of self, on the other hand, may be a protective factor against eating disorders and may buffer the individuals from the negative influence of mass media (Vartanian, 2009).
In conclusion, media idealization of a slim body and internalization of the ‘thin body ideal’ may provoke an eating disorder in susceptible individuals, who are already vulnerable to eating pathology by virtue of their biological and psychological make-up (Harrison, 2001; Levine and Murnen, 2009; Nasser and Katzman, 2003; Polivy and Herman, 2004). For them the ‘pursuit of thinness’ (Bruch, 1973) becomes the pursuit of success in life.

**Peer Influence**

Because the notions of weight and shape have entered our value system, they are now transmitted not just by the mass media, but also from one individual to another in a social context (Polivy and Herman, 2002). The strength of peer influence on health-related behaviours (e.g. substance use) among adolescents and young adults is well-documented (Wills et al., 2000). The same may be true with regard to weight management. By copying and learning from celebrity role models, young people may establish peer norms of weight and its control. For instance, if thinness is valued in a certain social environment (e.g. school), larger girls may be bullied into losing weight or otherwise encouraged to ‘fit in’ by their peers (Levine et al., 1994b; Ward and Gowers, 2003). Learning about slimming methods and diets also takes place; this may be how tales of self-induced vomiting being an effective weight control method get transmitted from one person to the next. However, although most young people are exposed to such messages, not all of them develop a concern for their shape or indeed, an eating disorder. This suggests that other factors, in addition to peer pressure, have a part to play. Just like media influence, peer influence may be just a contributory, but not a causal factor in eating disorders (Levine et al., 1994a, Polivy and Herman, 2002).

**Family Influence**

There are a number of theories explaining how family circumstances may contribute to the individual developing an eating disorder. First, those suffering from anorexia or bulimia often have an insecure attachment to their primary caregiver which more often than not happens to be the mother (Ward et al., 2000a, 2000b, 2001; Ward and Gowers, 2003). The dysfunctional relationship with the mother seems to be important in the development of an eating disorder (Bruch, 1973; Ward and Gowers, 2003).
Insecure attachment is often formed when a child’s physical or emotional needs are not met with responsiveness and care by the primary care giver (Gerhardt, 2009). This significantly affects their ability to self-sooth, to recognise emotions and regulate them functionally as an adult (Buckroyd, 2009). In the absence of a reliable caregiver and healthy emotion regulation skills, the child may develop a tendency to manage their emotions dysfunctionally (e.g. with food) because she/he will be unaware of how to look after their emotions appropriately (Buckroyd, 2009).

There are two main negative consequences of insecure attachment that tend to persist into adulthood. One is a tendency to regulate emotions dysfunctionally (Gerhardt, 2009). Emotion awareness and regulation in individuals with eating disorders will be discussed at length in the next chapter (Chapter 2). The other consequence is disruption in identity formation (Gerhardt, 2009). According to the psychodynamic school of thought, insecure attachment results in a poorly formed sense of self. If a primary caregiver did not reflect back to their child in the process of interaction, the child did not get a chance to internalize his/her parent. As a result of this the child’s ego identity (Erikson, 1968) could not be sufficiently formed (Allen, 2001; Fonagy and Target, 2002; Fonagy et al., 2002; Heatherton and Baumeister, 1991; Schore, 2003). In other words, the child may be left with a weak self-concept, unstable sense of self-worth and poor understanding of what he/she is as a person. The role of self-concept in disordered eating will be discussed in the Problems with Ego Development section further in this chapter. Thus it is not surprising that insecure attachment is also associated with psychiatric vulnerability, low self-esteem and difficulties in forming relationships as an adult (Gerhardt, 2009). All of these are risk factors for developing eating disorders (Treasure, 2010).

In addition, parents of women with eating disorders are often overly critical of their daughters (including their weight) and the mothers themselves tend to have dysfunctional eating problems and psychiatric disorders (MacLeod, 1981; Rodin et al., 1990; Selvini Palazzoli, 1974). This means that a girl may learn pathological eating behaviours as weight and/or emotion control methods from her mother; alternatively, a
genetic predisposition for an eating disorder may be triggered by weight-directed criticism from the mother (Agras, 1999; Ward and Gowers, 2003).

It is also likely that the eating-disordered individual’s family as a system may be dysfunctional. *Family systems theory* (FST; Minuchin et al., 1975, 1978; Selvini Palazzoli, 1974) suggests that eating disorders arise in the context of an unhealthy family structure and function to avoid dealing with deep-seated problems within the family. Selvini Palazzoli (1974) and Minuchin’s (1978) transactional approach, based on the *object-relations theory*, views the family as a cybernetic system that regulates relationships between its members. Eating disorder symptomatology reflects psychopathology of the whole family, because it develops as a result of dysfunctional family dynamics and unresolved family conflicts (Minuchin et al., 1975, 1978; Selvini Palazzoli, 1974). According to FST, the dysfunctional family is characterized by a high degree of enmeshment, overprotection, rigidity and lack of conflict resolution (Minuchin et al., 1975, 1978; Selvini Palazzoli, 1974). In other words, it is a family where the child is not given enough independence, s/he is overprotected, the rules imposed on him/her are inflexible and the family does not properly address their difficulties. This means that the negative atmosphere within the family and early attachment problems may be conducive to eating pathology to a great extent.

**Conclusions**

Media, peers and family may encourage unhealthy preoccupation with weight and shape, and pressure an individual into losing weight and thus provoke eating pathology in susceptible individuals. In addition, family environment and relationships with parents may be dysfunctional, which may activate a genetic predisposition to eating disorders. Alternatively, the failure to make a secure attachment to the primary caregiver may result in difficulties with emotion management, insufficient ego formation and tendency to regulate emotions dysfunctionally (e.g. with food).

**TRAUMA AND STRESS**

It has been found that most patients with eating disorders have experienced frequent and at times severe life stresses prior to the onset of their illness – typically, in childhood.
and young adulthood; these often include sexual and emotional abuse (Connors and Morse, 1993; Fischer et al., 2010; Lacey, 1993) and other types of traumas (Faravelli et al., 2004; Treuer et al., 2005; Schmidt et al., 1993b). Some researchers suggest that disordered eating patterns may be a way of coping with life adversities, because they allow the sufferer to regain some control over their bodies and their lives which may provide a sense of emotional relief, comfort and reward (Dallman et al, 2005; Epel et al., 2001; Schoemaker et al., 2002; Smolak and Murnen, 2002). It is also possible that eating disorders may be constructed to distract from more global psychopathology, and as a coping response to a deep-seated trauma (Faravelli et al., 2004; Treuer et al., 2005; Schmidt et al., 1993b). Instead of dealing with the underlying psychological trauma, the individual may use disordered eating and worries about weight to divert their attention away from the difficulties that they find impossible to face (e.g. traumatic memories). The trauma may be constantly causing negative affect, and disordered eating somehow manages to alleviate it (Gerhardt, 2009; Macleod, 1981; Serpel and Troop, 2003). By focusing on what can be controlled (e.g. eating), the individual may begin to feel more capable of coping with life (Macleod, 1981; Serpel and Troop, 2003). Eating disorders may develop to mask the real psychological issue that causes distress, in other words, as a maladaptive means of coping with negative affect (Serpel and Troop, 2003). Self-medicating with the use/misuse of food appears to bring emotional relief and, except in the food-related context, reduces anxiety (Gerhardt, 2009). Even though disordered eating may be useful as a coping mechanism, it is harmful for health in the long run, and it is important that the eating-disordered individual learns alternative adaptive ways of coping with traumas and life stresses.

INDIVIDUAL DIFFERENCES
Eating disorders often co-occur with neuroticism (Gordon, 1990), perfectionism (Bastiani et al., 1995; Bruch, 1973; Casper 1983; Garner et al., 1983, 1984), low self-esteem and obsession with weight and control (Gleaves et al., 2000; Fairburn et al., 1998; Sunday et al., 1995). In addition there is evidence to believe that individuals with eating disorders have an unstable sense of self-worth that is heavily dependent on their eating behaviours (Serpell, 2003; Vitousek and Hollon, 1990). According to Polivy and Herman (2002), individuals with eating disorders have a poorly formed identity which
makes them susceptible to developing eating pathology as a means of building a new one. This section briefly looks at these characteristics and their potential role in the aetiology and maintenance of eating disorders.

**Neurotic Perfectionism and Self-esteem**

Neurotic perfectionism (Davis, 1997), combining the features of both neuroticism and perfectionism, is manifested in the ‘all or nothing’ attitude, typically exhibited by eating-disordered patients (Bastiani et al., 1995; Bruch, 1973; Casper 1983; Garner et al., 1983, 1984; Sassaroli et al., 2008; Shafran et al., 2002). High neuroticism - which can be loosely defined as a predisposition to experience negative affect - is one of the characteristic risk factors for developing eating disorders (Cervera et al., 2003; Gordon, 1990). An individual with dysfunctional, or clinical, perfectionism (Frost et al., 1993; Shafran et al., 2002) shows preoccupation with high standards, achievement and goals, and has a persistent feeling of their efforts not being good enough; furthermore, they may perceive the outcome of their perfectionist efforts as a measure of self-worth. In those with eating disorders perfectionism appears to be a stable trait, rather than a general tendency, because research shows that perfectionist qualities can be observed prior to the onset of eating disorders (Fairburn et al., 1999) and following the remission of symptoms (Pia and Toro, 1999).

Perfectionism was found to be partly genetically based via its association with the Obsessive-Compulsive Personality Disorder (OCPD; Lilenfeld et al., 2000). There is evidence for co-morbidity of bulimia and the Obsessive-Compulsive Personality Disorder. There also appears to be a genetic link between OCPD and anorexia (Altman and Shankman, 2009; Lilenfeld et al., 1998; Piran et al., 1988; Wonderlich et al., 1990).

DSM-IV diagnostic criteria for OCPD include: excessive need for perfectionism and control, rigidity and preoccupation with details (DSM-IV; APA, 2000) that are severe enough to affect individual’s relationships and daily activities. It is known that people with eating disorders are excessively preoccupied with their weight, shape and eating, and are compulsive in following their weight-loss routine (Gleaves et al., 2000; Palmer, 2000; Sunday et al., 1995). It is normal, especially in women, to take pride in one’s appearance and to watch one’s figure; however in anorexia and bulimia such healthy
manifestations of femininity are taken to the extreme. This tendency to obsess and strive for perfection may be partly biologically-determined both in bulimia and anorexia.

It is well-documented that individuals with eating disorders, anorexics in particular, have a strong need to be in control (Fairburn et al., 1998). It is believed that they attempt to exert total control over their eating in a bid to achieve more command over their lives (Slade, 1982). Anorexics’ perception of themselves is typically characterized by helplessness and they may be overwhelmed by how little they are able to control their life. ‘Anorexia is chosen as an act of rebellion’ (MacLeod, 1981), and it may be a way of claiming control over one’s body and life (Szasz, 1974). In the course of anorexia, individuals may lose some of their helplessness, begin to feel more empowered, superior to others and even euphoric; regaining control over their bodies may strengthen their identity and may function to maintain eating pathology (MacLeod, 1981). They may begin to see their body as a project that they have total control over, and therefore can easily manipulate (Szasz, 1974). The sense of power anorexics gain when they manage to restrict their eating and lose weight, may give them comfort and reassurance that at least some things are within their reach (Szasz, 1974). In bulimics the cycle of loss and gain of control may be self-perpetuating. For them power over their bodies is lost when they binge, but regained when they make themselves compensate for excessive calorie intake, and they may become trapped in this cycle (Orbach, 2009). It appears that individuals with eating disorders have a need to control their eating, and take their desire to be thin to the extreme. It is a struggle between the mind that strives for weight loss and the body that tries to hold on to calories and survive.

Clinical perfectionism often goes hand in hand with low self-esteem which itself is a significant predictor of eating disorders (Cervera et al., 2003; Ghaderi and Scott, 2001; Gual et al., 2002). Self-esteem can be defined as a set of beliefs about one’s worthiness and competencies. Research suggests that there is a strong association between low self-esteem and eating disorders (Button et al., 1996; Fairburn et al., 1997, 1999; Gual et al., 2002). Individuals with anorexia, bulimia and EDNOS are typically prone to base their self-esteem on their shape and weight (Serpell, 2003; Shafran et al., 2002; Vitousek and
Hollon, 1990), and this may explain the strength of their preoccupation with their appearance and weight loss. A perfectionist strives to achieve, often at the expense of their physical health, because it is a potent means of lifting their self-esteem. In order to maintain their fragile self-esteem, an eating disordered individual may need regular affirmations of their value as a person (a boost to self-esteem) which they obtain by achieving something they ascribe importance to (Goldner et al., 2002; Garner et al., 1983). For example, an anorexic who experiences a sense of satisfaction seeing the result of their dieting efforts on the scales will have to eat less and less in order to achieve weight loss and get the self-esteem boost that feeds their perfectionism (Shafran et al., 2002). Similarly, individuals with bulimia, who also tend to be affected by perfectionism (Goldner et al., 2002; Shafran et al., 2002), may be delighted to see that their purging behaviours have compensated for their bingeing, and that weight gain was prevented. This would promote repetition of compensation behaviours as self-starvation and purging. Therefore, it appears that eating pathology may be constantly internally reinforced by the need to increase self-esteem (Garner and Bemis, 1982; Vitousek and Ewald, 1993) and this may be one of the reasons why eating disorders are typically chronic (Gordon, 1990) and hard to treat (Treasure, 2010).

**Problems with Ego Development**

It is well-known that anorexics and bulimics tend to judge their self-worth as human beings in terms of how many pounds they have gained or lost, and how successfully they are able to resist and control their hunger (Serpell, 2003; Vitousek and Hollon, 1990). The obsession with weight and absence of a robust instilled sense of self-worth are characteristic of eating pathology (Allen, 2008; Fonagy, 2002; Gerhardt, 2009; Heatherton and Baumeister, 1991; Schore, 2003). As was discussed above, insufficient ego development in those with eating disorders may be the result of an insecure attachment in childhood (Allen, 2008; Fonagy, 2002; Heatherton and Baumeister, 1991; Schore, 2003).

*Transactional analysis* (Berne, 2009) can be used to explain eating disorders in terms of an imbalance between three structural parts of the self: the Parent, the Child and the Adult (i.e. the Ego). There is a hypothesis that the source and container of the self-
worth (i.e. the Ego) is underdeveloped in sufferers of anorexia and bulimia (Buckroyd, 2009). Within the same person there may be an emotional Child and a strict, demanding Parent (an internalized image of the patient’s parents), and no Adult (the Ego) to mediate between them. The individual may lack a coherent sense of identity, because the two forces are, figuratively speaking, pulling them in the opposite directions. Although anorexics and bulimics often appear grown-up and together on the surface, this may be merely ‘pseudo-maturity’, a defensive disguise of low self-esteem (Gordon, 1990). Eating disorders may be a coping strategy, designed to develop autonomous identity and to resolve intrapsychic and existential conflicts of adulthood and womanhood in females (Bruch, 1974; MacLeod, 1981). However, as any maladaptive coping strategy, disordered eating only masks those deep-seated issues and creates a temporary solution to them, instead of resolving them. According to Erikson (1968), every individual in their personal development moves through a succession of ego identity crises that have to be resolved in order for them to develop a complete identity. It is plausible that individuals with eating disorders have been unable to resolve their adolescent crises. Adolescent girls with anorexia may reject their femininity in favour of a safe and familiar girlhood, and use their eating to reverse maturation (Macleod, 1981). As a result, their ego identities remain weak and incomplete (Macleod, 1981).

Sheila MacLeod (1981) views eating disorders as a psychoneurotic syndrome, where the symptoms stem from underlying emotional difficulties, identity crises and traumas. In her conception, physical and behavioural symptoms (e.g. eating patterns) are secondary to psychological ones. According to MacLeod (1981), the eating-disordered individual communicates via ‘the language of the symptoms’; in other words, disordered eating and changing body weight are used to convey emotions, much like words would. Like in hysterical conversion (Fairbairn, 1954), in eating disorders emotional problems are being substituted by physical and behavioural symptoms. This substitution serves a purpose of helping eating-disordered individuals to cope with their psychological difficulties (MacLeod, 1981).

Applying Winnicottian thinking to eating disorders, Susie Orbach (2009) proposes that eating disordered individuals create a false self which is maintained by ‘creating and
surviving emergencies which [provide temporary reassurance of their] physical existence’ (Orbach, 2009). According to Orbach, anorexic and bulimic girls are looking for a ‘reliable’ body, and inflict pain on themselves in order to make their bodies feel more real (Orbach, 2009). Thus, an eating disorder may give individuals a focus, a core around which they try to build their identity and their awareness of their body. This makes it functional and important to the sufferer which may explain their reluctance to look for help.

**Conclusions**

Evidence outlined above shows that individuals with eating disorders tend to have problems with their identity, which may stem from the relationship with their primary caregiver. The quest for a coherent sense of self may cause the need to constantly prove one’s worth by succeeding in an area of importance, such as weight loss. When the sense of self-worth becomes dependent on weight, this may stimulate the obsessive need for control over eating and hypervigilance of weight. Low self-esteem and attempts to raise it by excelling at weight loss may be a driving force behind eating disorders. This may be one of the mechanisms of how dieting in females with low self-esteem and perfectionistic nature may lead to a serious eating disorder. Thus disordered eating may represent search for a more stable sense of identity and a means of maintaining or obtaining self-esteem in individuals with eating disorders.

**Treatment of Eating Disorders: Challenges**

Only around 50% of individuals with anorexia recover (Steinhausen, 2002); the figure for bulimia is similar (Fairburn, 2002). Eating disorders are very hard to treat (Treasure and Schmidt, 2003), and there may be several reasons why. Firstly, eating-disordered individuals often do not consider themselves ill (i.e. they are in denial), do not seek treatment or actively resist others’ attempts to impose treatment on them (NHS Quality Improvement Scotland, 2007). The majority of those with eating disorders may be not receiving any help with their condition because they are not in treatment. This could be because anorexics may deny that they are underweight and bulimics may consider
purging a useful weight control method. Therefore they are likely to actively avoid treatment (Aronson, 1990).

Furthermore, individuals with eating disorders often are simply unable to successfully participate in psychological treatment that requires self-reflection (i.e. psychotherapy), because of the emotion awareness deficits associated with eating disorders. This will be discussed at length in Chapter 2. Because individuals with eating disorders tend to have difficulties with emotion recognition (Russell et al., 2009), they may struggle with functional regulation of affect, and eating pathology may serve this function.

Secondly, the task of clinicians is further complicated by the fact that weight loss tends to be seen in positive terms by anorexics and bulimics. They often perceive restricted eating as an achievement of will (a triumphant ‘transcending of <…> human needs’, Aronson, 2001; a ‘source of perverse satisfaction’, Palmer, 2000) and as a measure of self-worth, as opposed to a disease. Because their eating disorder may provide a sense of achievement and reward, this makes the individual reluctant to give it up and to seek treatment (Aronson, 2001; Palmer, 2000).

Thirdly, eating disorders have a high comorbidity rate with other mental illnesses such as depression and Obsessive Compulsive Disorder (Brewerton et al., 1995; Geist et al., 1998; Halmi, 1995; Karatzias et al., 2010; Rastam, 1992). Anorexia shares comorbidity with major depression and OCD (O’Brien and Vincent, 2003), whereas bulimia is co-morbid with major depression, substance abuse and the borderline personality disorder (O’Brien and Vincent, 2003). Moreover, the more severe the eating disorder, the more likely there is to be a co-morbid condition (Spindler and Milos, 2007). This complicates treatment further because it means that several mental health problems have to be tackled at once. Despite the complicated nature of eating disorders, various pharmacological and psychological treatments have been developed and used to treat eating disorders with some success. This section provides a brief overview of pharmacological treatment and a discussion of psychotherapies available to those with eating disorders.
PHARMACOLOGICAL TREATMENT

Drug treatments for eating disorders are generally of low effectiveness. This could be because such treatments are based on the premise that eating pathology is an imbalance of biochemicals in the brain (Kruger and Kennedy, 2000; Walsh, 2002) that may be overly simplistic. Alternative explanation for relative drug ineffectiveness may be that the right class of drugs for treating eating pathology has not yet been established. Pharmacological treatment appears to be more effective for treating bulimia than anorexia. At the moment, there is no effective drug for anorexia to bring about a substantial remission in symptoms (Bulik et al., 2007). Antipsychotics, anxiolytics, tricyclic antidepressants, selective serotonin reuptake inhibitors and mood stabilizers (e.g. lithium) despite their well-documented success in increasing body weight and stabilizing mood in sufferers with comorbid conditions (e.g. major depression) fail to bring about a marked improvement in anorexia (Bulik et al., 2007; Claudino et al., 2009; Kruger and Kennedy, 2000).

Psychotropic medicines (e.g. Fluoxetine/Prozac) have been used to treat bulimia with some effect. In bulimia, tricyclic antidepressants were shown to be effective in reducing binge eating; Fluoxetine in particular, is effective in interrupting the binge/purge cycle on behavioural and cognitive levels (Fairburn, 2002; Goldbloom and Olmsted, 1993; Shapiro et al., 2007; Walsh, 2002). Walsh (2002) has concluded that medication that lifts low mood (i.e. antidepressants) can be helpful in prevention of binge episodes in bulimia and EDNOS.

PSYCHOLOGICAL TREATMENT

In treatment of eating disorders, medicines may be used alongside psychotherapeutic interventions. Psychological treatment of choice for anorexia, bulimia and EDNOS is typically Cognitive Behavioural Therapy (CBT) because of its cost-effectiveness (Fairburn, 2002). However, as anorexia occurs predominantly in young people, it may also be treated with Family Therapy. Family Therapy is based on the premise that it is important to look at the family dynamics and the relationships within the family as a system in order to understand what conflicts may be provoking eating pathology (Minuchin et al., 1975, 1978). This treatment has been found to be moderately effective.
for anorexia (Dare and Eisler, 1995; Russell et al., 1987), however some researchers suggest that more studies are needed (Cottrell, 2003; Fisher et al., 2010). Interpersonal Psychotherapy (IPT) is frequently used with individuals with bulimia and binge eating disorder (Agras et al., 2000; Wilfley et al., 2002). This therapy addresses dysfunctional relationships and their possible contribution to eating pathology. It was found to be as effective as CBT in the long term – around 50% recovery rates at one year follow-up (Agras et al., 2000).

Recent treatment reviews by the Cochrane Collaboration (Hay et al., 2009 and Fisher et al., 2010) provide a good summary of research on individual psychotherapy and family therapy for anorexia nervosa. Hay et al. (2009) assessed effectiveness of a large number of psychotherapies, including Interpersonal Therapy (IPT) and Cognitive Behavioural Therapy (CBT). They found that all the therapies were approximately equal to one another in their efficacy and more research was needed before they could recommend one over the other. With regard to family therapy, Fisher et al. (2010) examined four main family-based treatment approaches: Structural Family Therapy (Minuchin, 1978), Systems Family Therapy (Selvini Palazzoli, 1978), Strategic Family Therapy (Madanes, 1981), Family-Based Therapy (Lock et al., 2005, 2006) and other related approaches in relation to other psychological and pharmacological interventions. It was concluded that there were no substantial differences in the efficacy of different treatment approaches in treating anorexia nervosa. This suggests that although different psychotherapies seem to lead to some improvement in disordered eating symptomatology, they all may be missing something important in their conceptualization of eating disorders. This would explain low to moderate efficacy of such treatments.

Cognitive Behavioural Therapy is the most widely used psychotherapeutic treatment for bulimia nervosa (Fairburn and Brownell, 2002). In CBT used for eating disorders emotions are seen as end-products of thoughts, and the focus is on reappraising cognitions associated with body shape, weight and food. A change in cognitions about food and weight is believed to lead to a change in eating behaviours, and a change in actions would in turn lead to a modification of cognitions and emotions. CBT success rate in treating eating disorders is around 40-50% (Buckroyd, 2009; Agras et al., 2000),
and there are no data to suggest that any positive effects are maintained in the long-
term.

Benefits of CBT and other talking therapies are largely dependent on the ability of the
patient to self-reflect and identify connections between cognitions, emotions and
behaviours. But if eating-disordered individuals are alexithymic (i.e. are unable to
describe their affective experience; this will be discussed at length in Chapter 2), it is
difficult to see how they would benefit from such a therapy. There is evidence that
alexithymia may prevent patients from fully responding to and benefiting from
psychotherapy (Krystal, 1982; Ogrodniczuk et al., 2005, 2010). And indeed, the
majority do not. Research suggests that cessation rate for bulimic patients following
CBT is less than 50% (Fairburn and Brownell, 2002). The figures are similar for
anorexic patients (Vitousek, 2002).

Limited effectiveness of the traditional CBT approach may stem from its focus on
treating cognitions at the expense of emotions. In CBT cognitions are seen as primary in
maintenance of eating pathology, and emotions (and behaviours) as secondary. It is
useful to point out - without delving too deeply into the Lazarus-Zajonc debate - that it
could be the other way around (Aldwin, 1984; Lazarus, 1984; Zajonc, 1984). In CBT, a
thought is a starting point (e.g. ‘I am fat’) that gives rise to an emotion (e.g. self-disgust)
and behaviours (e.g. binging). But the opposite may also be true in some cases.
Consistent negative affect (e.g. due to a succession of stressful events or a
temperamental tendency) may give rise to a chain of thoughts to match the feelings and
context (e.g. ‘I feel sad and worthless - no one likes me – it’s because I’m fat and ugly -
I must lose weight - I will stop eating’) and the result is manifested in dysfunctional
behaviours (e.g. starvation). This may be particularly true if this chronic negative affect
is highly intense. Even if the processing systems are - as Aldwin (1984) suggests -
highly complex (e.g. parallel, independent and multilevel), if there is chronically intense
negative affect, it may be useful to therapeutically address the emotions first. This is
discussed in more detail in Chapter 2.
As was mentioned above, in CBT the focus is on modifying beliefs about the body, food and self-image. In the original version of CBT, the patient was not explicitly taught how to connect with their emotions and regulate them. She/he was encouraged to manage her/his affect by challenging negative beliefs and thoughts, and changing dysfunctional eating behaviours. But if disordered eating represents an attempt to manage negative affect, it seems logical that therapy should address affect, first and foremost. The more advanced, Transdiagnostic version of CBT aims to take emotions and a number of other maintaining factors into account (Fairburn et al., 2003). Transdiagnostic CBT incorporates four new components into the traditional CBT formulation (Fairburn et al., 2003, 2009): clinical perfectionism, low self-esteem, mood intolerance (which is defined as an ‘inability to cope appropriately with certain emotional states’, Fairburn et al., 2003) and interpersonal problems. These are added to the standard CBT procedure, if a case is complex (Murphy et al., 2010).

As was discussed earlier, individuals with eating disorders often have the tendency to measure their self-worth in terms of weight loss achievement; in the absence of a stable sense of identity, they may attempt to acquire it via their body. The clinical perfectionism component of the transactional CBT addresses this problem (Fairburn et al., 2003, 2009). As part of the model, strategies to increase low self-esteem are also implemented to address core low self-esteem, which may be maintaining eating pathology (Fairburn et al., 2003, 2009). The mood intolerance component acknowledges that individuals with eating disorders have difficulty regulating their emotions functionally and struggle with affect (both positive and negative). Therefore, the treatment aims to help them to tolerate their negative emotions better (Fairburn et al., 2003, 2009). The Transdiagnostic model also addresses interpersonal difficulties, and encourages patients to work through old relationship issues and to form new healthy relationships (Fairburn et al., 2003, 2009). However, despite being more advanced, the Transdiagnostic version of the CBT therapy seems to be just as effective as traditional CBT in treating eating disorders (Fairburn et al., 2009), and not more so. This points to the fact that CBT therapies may be missing something important in their understanding of eating pathology; in other words, that the theories behind them may need to be revised.
A number of new therapies that focus primarily on and address negative emotions and their management have been developed and applied to eating disorders; some of these are known as mindfulness-based approaches (Kristeller et al., 2006). Dialectical Behaviour Therapy (DBT; Safer et al., 2001) and Acceptance and Commitment Therapy (ACT; Hayes et al., 1999) are promising new therapies that focus on healthy emotion management education and actively teach emotion regulation skills using a number of mindfulness techniques (e.g. meditation). In DBT (Safer et al., 2001) disordered eating and weight management practices in bulimia nervosa are seen as attempts at regulation of negative affect. Therefore this treatment focuses on teaching individuals more adaptive regulatory strategies, increasing the awareness of their emotional states and developing a repertoire of healthy emotion modulation behaviours. It was found that emotion awareness and affect regulation training reduce the need for eating disorder as a coping strategy and lead to the remission of binge and purge symptoms (Safer et al., 2001a, 2001b). A recent study found that eating disorder symptoms remitted in 54% of bulimics and 33% of anorexics at the two year follow-up, as a result of DBT (Kroger et al., 2010). This is an impressive result, considering that the patients had been unresponsive to other treatments (Kroger et al., 2010). However, the fact that the eating pathology was still maintained in 46% of bulimics and 67% of anorexics suggests that the DBT has to be developed further (Kroger et al., 2010).

Acceptance and Commitment Therapy (Hayes et al., 1999) in its application to disordered eating addresses emotional aspects of eating disorders (anorexia nervosa, in particular) from a slightly different angle. It emphasizes the need for control and intentional avoidance of negative thoughts and feelings about the body image as maintaining factors in anorexia. According to ACT, individuals with anorexia feel and think negatively about their bodies, and use dysfunctional weight loss strategies to address those; in other words, disordered eating is a consequence of this unwillingness to accept a negative body image (Heffner et al., 2002). ACT teaches individuals techniques that help them to recognize and accept the thoughts and feelings that cause them distress, as opposed to blocking them out. This results in more affect awareness and mastery, which has a positive influence on eating pathology (Heffner et al., 2002).
Research on the effectiveness of these therapies in treating eating disorders is still in early stages, however studies increasingly find that these therapies are successful in bringing remission in eating pathology (Heffner et al., 2002; Kristeller et al., 2006; Safer et al., 2001a, 2001b).

Interestingly, none of the established psychotherapies (i.e. IPT, CBT and family therapy) for eating disorders directly concentrate on increasing well-being (as opposed to decreasing distress) and teaching skills for eliciting positive emotions. There are positive psychotherapies that have been developed by the positive psychology movement, and these are being applied to treating eating disorders (Chapter 3). There is a need for a comprehensive approach to emotions in eating disorders, in particular, for interventions that target positive affect in addition to negative emotions. Such interventions, if used alongside other psychotherapeutic approaches, may be effective in treating eating disorders and may boost the effectiveness of CBT and other therapies (Sin and Lyubomirsky, 2009). Positive psychotherapies and interventions will be discussed in more detail in Chapter 3.

It appears that a large number of therapies (e.g. CBT, IPT and Family therapies) failed to understand primacy of affect and its regulation in eating disorders. If dysfunctional regulation of negative affect is at the core of eating pathology, then it does not make sense to focus therapy on treating cognitions. It appears likely that psychotherapies would benefit from incorporating and combining interventions that directly target negative and positive affect. Such interventions should aim to equip the patient with skills for emotional awareness, emotional language and healthy regulation of negative affect. In addition, in therapy the eating disordered patient may need to be explicitly taught how to recognize their emotions, how to self-nurture, how to cope with bad mood and what to do in order to feel happy (i.e. how to elicit and maintain positive affect). Such skills would increase their sense of mastery and self-esteem, which in turn may help ego maturation. Effective emotion management education may deem disordered eating useless to the patient, and as a result, the symptomatology may subside. More research is needed in order to establish whether that is indeed the case.
**Conclusions**

The evidence shows that pharmacological treatments are more effective in treating bulimia than anorexia, but they are still far from 100% successful. This is also largely true of psychotherapies (Treasure and Schmidt, 2001, 2002). There is an emergent interest in the role of emotions in eating disorders, and psychological therapies that address affect regulation and negative emotions (e.g. Dialectical Behaviour Therapy) are being developed. The new Transdiagnostic CBT (Fairburn et al., 2003, 2009) incorporates emotions into its theory and practice as a component of eating pathology. However, emotions and their regulation may be core factors in eating disorders, and if so, they should be more comprehensively addressed in treatment. If an eating disorder is a disorder of affect regulation, and dysfunctional eating patterns are symptomatic of that, the full spectre of emotions (i.e. negative and positive) has to be directly and comprehensively addressed in therapy. CBT and other well-established therapies are at present incomplete because they do not include development of positive emotions skills and positive affect assessment among their aims. There is a need for development of a unified approach for treating emotion regulation difficulties in eating disorders.

**CHAPTER 1 SUMMARY**

This Chapter looked at profiles, aetiology and treatment options for eating disorders. The review of literature shows that eating pathology may stem from a complex combination of extreme dieting, genetic predisposition and sociocultural influences; other risk factors include stresses, history of trauma and certain personality traits (e.g. neurotic perfectionism). The literature seems to converge on the fact that, despite their detrimental effects on the body, eating disorders may serve important functions for the sufferer. Eating disorders may provide: 1. a means of coping with a trauma and negative emotions (e.g. cognitively: an escape; emotionally: relief and comfort; socially: a cry for help; and behaviourally: control), 2. a focus for building an identity, and increasing self-esteem and 3. a source of positive emotions (a sense of achievement and pride). Pharmacotherapies (if used on their own) try to take this defence system away without building anything to replace it; therefore it is not surprising that there is a considerable relapse rate and resistance to treatment on the part of the patients. There are promising
developments in the psychotherapy field (e.g. Transdiagnostic CBT and DBT) and a wider acceptance of the role emotions play in eating disorders. The chapter that follows (Chapter 2) focuses on emotions and affect regulation in eating disorders in more detail.
CHAPTER 2. NEGATIVE AFFECT AND ITS REGULATION IN EATING DISORDERS

Chapter 2 looks in detail at emotion regulation in eating disorders and the role negative affect and its management may play in their onset and maintenance. The chapter starts by introducing the topic of emotion regulation, before moving on to discussing negative affect and its regulation in those with eating disorders, and concludes with several sections on Affect Intensity and its potential importance in emotion management and eating disorders. In this and the following chapter (Chapter 3) the terms ‘emotion’, ‘affect’, and ‘mood’ will be used interchangeably to avoid repetitiveness. In the English language, the words ‘emotion’ and ‘affect’ are synonyms (Oxford Dictionary, 2010), whereas ‘mood’ is defined as ‘a temporary state of mind or feeling’ (Oxford Dictionary, 2010). Although there are clear differences between mood and affect/emotion, in the present thesis the term ‘negative mood’ is used as an equivalent to ‘negative emotions’ and ‘negative affect’, whereas the term ‘positive mood’ should be understood as being equivalent to ‘positive emotions’ and ‘positive affect’; this is done solely for the purpose of avoiding repetitiveness.

EMOTION REGULATION

Emotion regulation concerns the ways in which emotions are experienced, expressed, and managed in daily life. From the functionalist perspective, emotions serve to inform us about situations and to provide guidance for the necessary actions (Tooby and Cosmides, 1990). An individual’s capacity to understand and become aware of their emotions may enhance their ability to respond to situations appropriately (Kostiuk and Fouts, 2002; Southam-Gerrow and Kendall, 2002; Tooby and Cosmides, 1990). The difficulty with regulating dysphoric emotions in a healthy way is a very common feature in psychopathology. This makes emotion regulation (and dysregulation) pertinent to mental health (Dalgleish and Power, 1999; Gross and Munos, 1995).

Emotions are functional, in that they alert individuals as to where they are in relation to their goals; negative emotions (e.g. sadness) may indicate that the individual has encountered an obstacle on the way to their goal; positive emotions (e.g. joy) may indicate that the individual is getting closer to their goal. As Gross (1999) points out,
even though emotions are useful, they often must be monitored and regulated. Healthy emotion regulation implies processing of emotions in ways that enhance well-being (Phillips and Power, 2007). Functional processing of an emotion involves allowing oneself to become aware of it, to experience it, to understand its cause and to respond with behaviours that maximize the chances of a positive outcome. Blocking, inhibition and repression of affect do precisely the opposite because they distance the individual from the emotion and do not allow its expression and processing; such strategies therefore tend to be unhelpful and unhealthy in the long run. However, adaptiveness of affect management tendencies may also depend on the context of their implementation (Gross, 1998; Gross, 1999; Kostiuk and Fouts, 2002; Southam-Gerow and Kendall, 2002). For instance, in some circumstances, denial (e.g. the refusal to acknowledge certain emotions) can be a helpful strategy. According to Gross and Munos (1995), emotion regulation may occur at two different points in time: before a certain emotion arises (antecedent-focused emotion regulation) and after the emotion has arisen (response-focused emotion regulation). In the former case, regulatory strategies are aimed at reducing the chances of the emotion starting (e.g. avoiding spiders if one is afraid of them). In the latter case, the regulation involves managing the emotion that has already been activated (e.g. downregulating anxiety by deep breathing if one sees a spider).

The field of emotion regulation emerged from the study of defence mechanisms (Freud, 1959) and coping (Lazarus, 1966). Unlike its predecessors the construct refers to management of not only negative, but also positive affective states. In the field of emotion regulation it is acknowledged that various aspects of positive affect (e.g. intensity) also often need to be regulated, for instance, in order to comply with cultural norms of affect expression. Emotion regulation is a narrower term than coping, and it does not address management of situations that do not have an emotional component but nevertheless require individuals to adjust to them. Gross (1999) has a very good example of a coping strategy: purchasing a city map in order to find one’s way. This action represents coping with the situation (i.e. being in an unfamiliar place), but not emotion regulation. Lazarus (1966) categorizes coping strategies in terms of their functions. A coping response can be problem-focused, where the individual aims to
directly address the situation and change it for the better; or emotion-focused, where the individual focuses on altering their emotional experience to make it more positive (Lazarus, 1966). Individuals may use one either or both types of coping responses, depending on the situation. A process that may determine selection of a particular coping response is cognitive appraisal, or interpretation of the event (Folkman et al., 1986). In primary appraisal stages, the individual makes a judgement about whether the situation is dangerous or beneficial to them physically and psychologically; in secondary appraisal stages the individual makes a decision about what strategies should be used to improve the situation (Folkman et al., 1986). Coping strategies, identified by Lazarus (1993) include: confrontive coping, distancing, self-controlling, seeking social support, planful problem solving, escape-avoidance and positive reappraisal. The emotion regulation field grew from the study of emotion-focused coping strategies, such as positive reappraisal and escape-avoidance.

Thompson (1994) suggests that flexibility and situational appropriateness of regulatory strategies determine their usefulness. He defines emotion regulation as ‘extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially in their intensive and temporal features, to accomplish one’s goals’ (Thompson, 1994). Emotion regulation can refer to management of positive and negative affective experiences when there is a need to reduce, increase or maintain their intensity and frequency. According to Gross and Thompson (2007), emotions may be managed by self-regulation (intrinsic regulation, e.g. replaying happy memories when one feels sad), or other’s regulation of one’s emotions (extrinsic regulation, e.g. using humour to cheer someone up). The processes that are being managed in the course of emotion regulation may include: physiological response, attention, cognitive appraisal and selection of appropriate behavioural responses (Thompson, 1994). Gross and Thompson (2007) suggest a Modal Model of Emotion where there are five regulatory processes: situation selection, situation modification, attentional deployment, cognitive change and response modulation. The first four refer to antecedent-focused regulation that takes place before an emotion occurs, and the fifth process (response modulation) refers to response-focused regulation because it is employed after emotions have already been elicited (Gross and Thompson, 2007). Under situation selection the authors understand avoiding or seeking emotion-eliciting situations (e.g. avoiding
spiders); *situation modification* refers to altering an emotion-eliciting situation so that it becomes less emotion-laden (e.g. physically moving away from the spider); *attentional deployment* (e.g. *distraction* and *concentration*) stands for a shift in attention that may be used to manage affect, such as diversion of attention to pleasant stimuli; *cognitive change* refers to cognitive interpretation of negative events in more helpful terms, such as reappraisal of the stressful situation (e.g. as a challenge as opposed to a difficulty) and downward social comparison (e.g. when the individual compares themselves with someone who is worse off). *Response modulation* occurs after emotions have been elicited (Gross and Thompson, 2007), and its aim is to manage thoughts, emotions and behaviours by means of various strategies (e.g. exercise, food, medicines and verbal expression of emotions).

The emotion regulation strategies that have been studied most in their relation to psychological outcomes are: reappraisal, problem-solving, emotion acceptance, suppression, avoidance and rumination (Aldao et al., 2010; Gross, 1999). The *reappraisal* strategy refers to cognitive evaluation of a negative emotion-generative event as good (positive reappraisal) or bad (negative appraisal). For example, the individual may reason with themselves to try to see how the bad situation may be good for them (Gross, 1999). *Problem-solving* refers to directly addressing the negative situation to maximize the chances of a positive outcome (Aldao et al., 2010). *Emotion acceptance* consists in recognising negative emotions and thoughts, and accepting them without judgement (Aldao et al., 2010). *Suppression* and *avoidance* refer to denying that a negative emotion exists and preventing its outward expression (Gross, 1999). *Rumination* refers to persistent focusing on the negative aspects of the situation and negative emotions, and dwelling on them (Aldao et al., 2010). Reappraisal, problem-solving and emotion acceptance were found to be associated with better psychopathological outcomes than suppression, avoidance and rumination (Aldao et al., 2010). However, it is likely that all of the strategies can be potentially functional or dysfunctional depending on the situation. For instance, rumination on a negative experience may enable personal growth and insight; whereas positive reappraisal may lead to unrealistic optimism and recklessness leading to negative outcomes.
Phillips and Power (2007) suggest that functionality of regulatory efforts rests on the effective utilization of two types of resources: *internal* (person-focused) and *external* (environment-focused). Their 21-item measure, Regulation of Emotions Questionnaire (REQ; Phillips and Power, 2007), defines responses such as *positive re-appraisal* (internal) and *advice seeking* (external) as generally healthy, adaptive responses to negative emotions, and responses such as *assault* (external) and *self-harm* (internal) as unhealthy and maladaptive (Figure 2). According to the authors, self-harm, rumination, negative social comparison, repression and de-realization are in the Internal Dysfunctional category (Phillips and Power, 2007). Positive re-appraisal, modification of goals, planning, putting the situation in perspective and concentration on a pleasant activity are Internal Functional alternatives (Figure 2). External Dysfunctional strategies include bullying, verbal/physical assault, lashing out at objects and making others feel bad (Phillips and Power, 2007). The alternative External Functional repertoire of regulatory strategies would involve non-violent expression of feelings, seeking of advice/physical contact, venting of feelings through exercise and doing something nice (Phillips and Power, 2007). The authors found that dysfunctional regulatory tendencies were associated with impaired interpersonal functioning, psychosomatic problems and lower quality of life (Phillips and Power, 2007). This suggests that effective emotion regulation skills have important implications for health - both mental and physical (Phillips and Power, 2007; Siegeman, 1993).
Regulation of Negative Affect in Eating Pathology

There is evidence to suggest that self-harm may be a powerful emotion regulation strategy (Gratz, 2003; Klonsky, 2007; Mikolajczak et al., 2009); deliberate self-harm behaviour was found to effectively relieve acute negative emotions, such as anxiety and guilt (Klonsky, 2007). The definition of self-harm is as follows: self-harm is ‘an expression of personal distress, usually made in private, by an individual who hurts him or herself” (NICE, 2004). Risk factors for self-harm include childhood abuse, insecure
attachment and emotional reactivity (Gratz, 2003). Self-harm behaviours, such as skin cutting, hair pulling, self-starvation and overeating, are typically preceded by intense negative emotions (Gratz, 2003; Mikolajczak et al., 2009). According to NICE (2004), self-harm may fulfil multiple and varied functions for those who engage in it. As well as reduction of painful affect, individuals may use self-harm in order to punish themselves, to signal to others about their distress, and to externalize their negative affect (Gratz, 2003). Self-harm may be helpful as a short-term emotion regulation strategy, but in the long run it is maladaptive (Gratz, 2003). Regulation of negative emotions by self-harm has particular relevance for individuals with eating disorders (Paul et al., 2002). Anorexic, bulimic and EDNOS individuals tend to score highly on self-injury measures (Paul et al., 2002). Disordered eating (e.g. bingeing) may represent a type of self-harm, which suggests that eating pathology may be used as a way of emotion regulation. This means that disordered eating may fulfil the same functions as other types of self-harm; in other words, by purging, bingeing and self-starvation, the eating-disordered individual may attempt to reduce their acute negative affect.

It is widely agreed that there are four basic negative emotions: anger, disgust, sadness and fear (Ekman, 1999), and a multitude of complex composite emotions (e.g. shame). In recent years, there has been quite a lot written about disordered eating being a way of managing those negative emotions and distress (Bydlowski et al., 2005; Fox and Power, 2007; Heatherton and Baumeister, 1991; Root et al., 1986; Taylor et al., 1997). Some of this research has been used to guide therapeutic interventions, and new promising therapies are being developed to address emotion regulation disorder in eating pathology (e.g. Dialectical Behaviour Therapy).

Negative affect - fear, anger, disgust, sadness and their composites - may play an important role in the maintenance of eating pathology. Fear, anger and disgust are the emotions studied most in relation to eating disorders. The fear of weight gain is one of the DSM-IV core criteria for the diagnosis of anorexia (APA, 2000), and eating dysfunction is linked to anxiety disorders (Mangweth et al., 2003). This suggests that fear is one of the central negative emotions that an eating disordered individual may have to cope with during their illness. It was also found that in those with eating
pathology the levels of anger and disgust are greater than in controls (Fox and Harrison, 2008; Miller, 1997; Troop et al., 2002; Waller et al., 2003), and that these two emotions may mutually reinforce one another (Fox and Harrison, 2008). In eating-disordered individuals, disgust is typically a response to food and their body (Davey et al., 1998; Troop et al., 2002), and high disgust sensitivity appears to be a prominent feature in eating pathology. Individuals with eating pathology also tend to score higher on anger measures than healthy controls (Tiller et al., 1995). There is research showing that bulimics had a tendency to suppress their anger, and this method of anger-processing may be a trigger for binge episodes (Milligan and Waller, 2000). This suggests that bingeing may perform the function of regulating anger in those with eating pathology. It is well-established that eating disorders have a high comorbidity rate with other mental illnesses such as depression (Brewerton et al., 1995; Geist et al., 1998; Halmi, 1995; Karatzias et al., 2009; Rastam, 1992). Bulimia shares comorbidity with major depression, substance abuse and the Borderline Personality Disorder (O’Brian and Vincent, 2003), whereas anorexia is comorbid with major depression and obsessive compulsive disorder (OCD; O’Brian and Vincent, 2003). The link with depression suggests that there is a substantial dysphoric component (e.g. sadness, low mood) in eating disorders (Brewerton et al., 1995; Halmi, 1995; Rastam, 1992).

Shame is another emotion that has been researched in relation to eating pathology. It was found that individuals with eating disorders report shame about their body (Burney and Irwin, 2000) and their disordered eating practices such as bingeing (Murray et al., 2000). The exact mechanisms of the implication of shame in eating pathology are at present unclear (Barney and Irving, 2000); however, it is conceivable that shame about disordered eating contributes to secretiveness and unwillingness to ask for help, whereas shame about the body may drive the individual to restrict their eating.

A large body of research suggests that behaviours such as food restriction and purging may help eating-disordered individuals cope with negative affect by preventing or reducing it (Cooper et al., 2004; Waller et al., 2004; Wildes et al., 2009). In other words, disordered eating may represent both antecedent- and response-focused emotion regulation (Gross and Munos, 1995). The presence of dysphoric emotions creates the
need to downregulate them; and because eating-disordered individuals tend to use maladaptive regulatory strategies (e.g. purging), negative affect experienced chronically may serve to perpetuate the dysfunctional eating patterns. Studies have found high comorbidity between major depression and eating disorders (Brewerton et al., 1995; Halmi, 1995; Rastam, 1992); this suggests that dysphoric emotions (characteristic of depression) may be an important component in eating pathology. Cooper’s *Cognitive Model* of bulimia (Cooper et al., 2004) postulates that the eating disorder is maintained by a delicate cognitive balance between positive and negative beliefs about eating/bingeing. On the one hand, overeating may provide a temporary escape from emotional distress and dissociation from the self that is perceived as unacceptable (or the identity that is incompletely formed); on the other hand, overeating may be seen negatively because it can lead to weight gain. However, it is not clear whether disordered eating does relieve emotional distress, or merely channels it away from the realm of the self and trauma, and displaces it into the realm of food and body. Nevertheless, emotion regulation appears to be central to the development and maintenance of eating pathology (Harrison et al., 2010).

To sum up, eating dysfunction is typically accompanied by enduring negative affect (Johnson and Larson, 1982; Leon et al., 1993) which appears to mediate the association between emotional processing and eating disorders (Gilboa-Schechtman et al., 2006). Consistent dysphoric mood and anxiety are generally associated with poor emotion processing (Aldao et al., 2010; Bydlowski et al., 2005; Eizaguirre et al., 2004; Fox, 2009; Gilboa-Harrison et al., 2010; Holliday et al., 2006; Jimerson et al., 1994; Schechtman et al., 2006; Taylor et al., 1996). According to Vaillant (2000) “mature mental health always involves affect recognition”; individuals with eating disorders may struggle with emotional awareness (Cochrane et al., 1993; De Zwaan et al., 1995; Guttman and Laporte, 2002; Pinaquy et al., 2003; Schmidt et al., 1993), perhaps unless the emotions are very intense. This makes emotion regulation difficult for them. There is evidence that anorexic females struggle with identifying and managing their emotions (Harrison et al., 2009). In addition, recovery from eating disorders was found to be related to improvement in emotion regulation strategies (Rawal et al., 2010). Recent research suggests that emotion management training can enhance effectiveness of CBT
(Berking et al., 2008). Moreover, disordered eating may be a dysfunctional strategy to manage negative affect in individuals with eating disorders. It was found that bulimic females experienced negative affect after bingeing, but purging helped to make them feel better (Alpers and Tuschen-Caffier, 2001). This suggests that eating disorder symptomatology may be an important emotion regulation mechanism for those with eating disorders. Therefore it seems important to address negative emotions and their regulation when treating eating disorders.

Emotional Theory of Mind and alexithymia

Recent findings (Oldershaw et al., 2009; Russell et al., 2009) show that individuals with anorexia have an underdeveloped *emotional theory of mind* (eToM); in other words, to rephrase Russell et al. (2009), they find it difficult to ‘represent … [emotional] states of others in terms of their intentions, desires and beliefs, and to use that representation to understand and predict behaviour’ (Russell et al., 2009). EToM deficits tend to be persistent and greatly impair social cognition because the latter is reliant upon emotion recognition. Although treatment seems to improve emotion recognition in anorexia sufferers, Oldershaw et al. (2009) found that recovered anorexics still struggled to identify positive emotions in themselves and others. This means that emotion-processing difficulties may affect the full spectrum of emotions: those of positive and negative valence.

One plausible contributor to the difficulty with eToM may be alexithymia (Moriguchi et al., 2006). It refers to ‘a personality construct characterized by a difficulty in identifying and describing feelings, a diminution of fantasy and a concrete and externally-oriented thinking style’ (Speranza et al., 2010). In other words, this means that individuals with eating disorders may struggle to understand what other people are feeling, and may have problems in recognition, expression and effective communication of their own feelings. Alexithymia also has a cognitive style component: *pensee operatoire* (de M’Uzan, 1974), which refers to a concrete and inflexible thinking which may also negatively affect social functioning (Gardos et al., 1984). Studies using the Toronto Alexithymia Scale (TAS-20) have shown that anorexic and bulimic patients find it difficult to identify, describe and differentiate between/among their emotional states and physical
sensations such as hunger (Cochrane et al., 1993; De Zwaan et al., 1995; Guttman and Laporte, 2002; Pinaquy et al., 2003; Schmidt et al., 1993). A high level of alexithymia is associated with a higher risk of negative outcomes for those with eating disorders (Speranza et al., 2007); interestingly, reduction in disordered-eating symptoms is associated with improvement in alexithymia (de Groot et al., 1995). Alexithymia may be the reason why eating-disordered individuals have deficits in emotional regulation (Bydowski et al., 2005; Carano et al., 2006; Gillboa-Schechtman et al., 2006; Fox and Fromm, 2009; Fox, 2009; Troop et al., 1995; Zonneville-Bender et al., 2002). Low emotional awareness coupled with an inflexible style of thinking may impair their capacity to regulate emotions appropriately and functionally (e.g. to express their feelings and ask for advice). This could explain why eating-disordered individuals may turn to dysfunctional strategies (e.g. self-starvation) as a primary means of emotional expression and management (Overton et al., 2005). When it comes to eating pathology there appears to be a complex interaction between affect, alexithymia and emotion regulation.

When speaking of alexithymia, it has to be noted that the origins of emotion recognition deficits in eating disorders remain unclear (Schmidt et al., 1993). They may result from a number of sources; alexithymic difficulties in eating disorders may be a by-product of malnutrition, a dispositional trait, a consequence of suboptimal parenting (Schore, 2003), insufficient development of socio-emotional skills (e.g. theory of mind), or indeed, a combination of certain aspects of all those. There is evidence to suggest that alexithymia is rooted in physiology, in particular, in the malfunctioning of certain cerebral structures (e.g. gray matter, Borsci et al., 2009). An alternative explanation is offered by Gilboa-Schechtman et al. (2006) who found that emotion recognition impairment in eating disorders may be due to depression and anxiety, associated with them. In other words, this means that dysphoric emotions may contribute to difficulties with emotion awareness.

Ogrodniczuk et al. (2010) found that psychotherapy may be complicated by alexithymia, because such treatments rely on the patient’s ability to reflect on their feelings. Furthermore, it was found that alexithymic tendencies were associated with
negative counter-transference that the researchers attributed to the patients’ limited expression of positive emotions in therapy. In other words, clinicians’ negative reaction, coupled with the patients’ alexithymia, lowered the effectiveness of therapy. If one applies these findings to eating disorders, one can argue that individuals with eating disorders may not possess emotional awareness to the degree, necessary for active participation in psychotherapy. This may explain why CBT and other psychotherapies have had limited success in treating eating disorders.

INTENSITY OF AFFECT

Affect Intensity (AI) is another aspect of emotional experience that is relevant to individual differences in emotional response (Larsen et al., 1986). It refers to emotional reactivity, arousal and affective variability (Larsen and Diener, 1987); in other words, to how intensely emotions are experienced by an individual and the affective range of his/her typical emotional states in everyday life (Larsen, 2009). Affect Intensity is manifested in subjective feelings (e.g. joy), physiological reactions (e.g. heart racing), and behaviour (e.g. clapping one’s hands). Individuals who are dispositionally high in Affect Intensity are more likely to experience emotions profoundly, to be more reactive to emotional stimuli and to have more variable mood in everyday life (Larsen and Diener, 1987).

When it comes to valence, Affect Intensity tendencies in pleasant and unpleasant emotions are strongly associated (Larsen and Diener, 1987). A person who experiences intense positive affect (high positive Affect Intensity) is also the one who tends to feel strong negative emotions (high negative Affect Intensity; Diener et al., 1985a). This suggests that emotional reactivity may be general, rather than emotion-specific (this is captured by the global Affect Intensity score on the Affect Intensity Measure Questionnaire, Larsen, 1986). Another interesting feature of Affect Intensity is that it is independent from emotion frequency (Diener et al., 1985a). A person who feels sadness strongly may not be the one who gets to experience it more often. This means that intensity and frequency represent two distinct unrelated aspects of emotional experience.
It has been suggested that Affect Intensity is a dimension of temperament (Larsen et al., 1986; Larsen and Diener, 1987). Rothbart and Derryberry (1981) conceptualized temperament as ‘individual differences in reactivity and self-regulation assumed to have constitutional basis’. In other words, temperament refers to affective responsiveness that is biologically based. Several facets of temperament have been proposed (Larsen, 1984): (1) emotionality (i.e. reactive intensity to stimuli), (2) sociability (i.e. the tendency to seek emotional stimulation), (3) sensory arousability (i.e. the ease with which emotions can be elicited in an individual) and (4) activity levels (i.e. the amount of energy the individual typically has). Affect Intensity was found to relate to all four dimensions of temperament (Larsen et al., 1986). In other words, individuals who typically experience intense affect are more likely to be highly reactive to emotional stimuli, to seek out (or create) circumstances that arouse their emotions and to be easily physiologically stimulated. In a nutshell, high AI individuals appear to be particularly sensitive to emotional stimuli.

Dispositional emotion intensity is assumed to be fairly stable over time and to have trait-like properties (Larsen, 1987). However, more recent evidence suggests that there may be a mood bias, as there appears to be a strong association between Affect Intensity and emotional state. Heide and Gronhaug (1996) found that self-ratings on the 40-item Affect Intensity Measure (Larsen, 1986) - the most widely used scale for AI - are vulnerable to mood-affecting situational factors (e.g. exposure to a funny film). In their study, the self-reported AI was found to be higher in the affect groups as compared to the neutral controls; participants in the negative mood group reported their felt arousal to be more intense, than those in the happy and neutral conditions. Thus Heide and Gronhaug’s (1996) findings suggest that Affect Intensity may be susceptible to mood manipulation; and more research is needed on the stability and robustness of the Affect Intensity construct.

**Affect Intensity and Emotion Regulation**

There is a gap in the literature with regard to the relationship between affect regulation and its intensity. However, studies that exist on the subject seem to suggest that extremes in intensity (i.e. very high and very low Affect Intensity) would be equally unconducive to healthy emotion regulation (Larsen, 2009). There is some research on
arousal regulation and Affect Intensity (Larsen et al., 1986). The term *arousal regulation* primarily refers to the physiological component of emotions; whereas *emotion regulation* is a broader concept that includes not only its physiological aspects, but also cognitive and behavioural ones. The *Arousal Regulation Theory of Affect Intensity* (Larsen, 2009) posits that individuals regulate their arousal so as to achieve the level that is optimal for them (e.g. increasing their arousal by engaging in risk taking). Larsen (2000) suggests that individuals have a preferred set point of arousal that they experience as comfortable, continuously compare their current emotions to that set point and use regulatory strategies to return to it. These strategies may be aimed at the person themselves (i.e. internal) or at the environment (i.e. external). This is an interesting model because it puts Affect Intensity in the context of arousal regulation. However, arousal is a general (rather than specific) hedonically neutral affective state, whereas emotions have specific hedonic properties. For this reason arousal regulation is not the same as emotion regulation.

It has been found that high Affect Intensity is associated with high baseline physiological reactivity (Larsen, 2009), extraversion, poor self-perceived control of negative emotions (Flett et al., 1989) and more perceived distress in challenging situations (Eisenberg and Okun, 1996). Larsen (2009) argued that “high AI subjects may not become aware of their emotional reactions until those reactions become quite strong”. They show insensitivity to the subtleties of their emotional experience coupled with a lack of confidence in their ability to manage emotions. Emotional arousal of high intensity creates the need to down-regulate it, and high AI individuals tend to use emotion-focused coping strategies (Flett et al., 1996) as well as avoidance (e.g. social diversion and distraction) in order to reach emotional homeostasis. In other words, they try to manage affect internally as well as by resorting to the help of others. However, low AI does not necessarily mean that an individual will be more adept at managing their emotions (Gross, 1998; Gross, 1999; Kostiuk and Fouts, 2002; Southam-Gerow and Kendall, 2002), because low AI is associated with alexithymia (Jacob and Hautekeete, 1999). As was discussed in the section on *Emotional Theory of Mind and Alexithymia*, difficulties in identifying emotions and describing them tend to be related to emotion dysregulation.
It is possible that bingeing and compensatory behaviours may represent extreme attempts at emotion regulation. If the affect is so intense as to be unbearable in those with eating disorders, it may explain why direct, extreme mood regulation strategies (e.g. bingeing) are implemented. They allow fast venting of distress and may not be as demanding in terms of time and effort as functional regulatory strategies are (e.g. positive reappraisal). Some healthy regulatory strategies require considerable cognitive, physical or emotional effort. For instance, in order to conduct a positive reappraisal of a negative event (this is an Internal Functional regulatory strategy, Phillips and Power, 2007) that has given rise to intensely dysphoric affect, it is necessary to spend time as well as cognitive and emotional resources trying to come up with arguments of how this event could be beneficial. In this example, positive reappraisal process is further complicated by the mood incongruence of the task; it may be extremely difficult in a state of intensely negative affect to reason positively. If one thinks about turning to social resources (e.g. advice-seeking) in order to cope with negative affect (i.e. External Functional emotion regulation, Philips and Power, 2007), the situation is similar.

Families of individuals with eating disorders often have dysfunctional family dynamics, which means that it may be more difficult for them to ask for support with their intense negative emotions than it is to address them dysfunctionally on their own. This suggests that individuals with eating disorders may lack the social and emotional resources, required for functional emotion regulation. It appears that it may be easier and quicker to reduce/address intense negative affect by resorting to extreme mood modification methods, even though those are dysfunctional and damaging in the long run. The intense negative affect hypothesis may explain why individuals turn to dysfunctional regulatory strategies in the first place. If it is confirmed, there are important implications for therapeutic practice. With regards to therapeutic treatment it would mean that it may be important to primarily address the affect component in therapy.

**Affect Intensity (AI) and Eating Pathology**

Because of the emotional instability component, AI has a part to play in psychopathology. Gratz (2006) showed that high AI scores characterize women with a history of self-harm. Borderline Personality Disorder, somatization, addiction,
maladjustment and substance abuse are known correlates of AI (Flett and Hewitt, 1995; Larson, 2009; Thornberg and Lyvers, 2006), and Vujanovic et al. (2006) showed that anxiety and panic disorder are some of the likely long-term outcomes for high AI individuals. Therefore the evidence shows that high global and negative Affect Intensity tend to be associated with negative psychological outcomes.

At present it is not known what intensity the affect experienced by eating-disordered individuals is, i.e. whether it is flat, high, low, or oscillating between the three most of the time. Attempts to deduce from the alexithymia findings are inconclusive, pointing to the possibility that typical affect in those with eating disorders may be either flat or high in intensity. On the one hand, it is plausible that emotions need to be of certain intensity in order to be detected, and alexithymic difficulties may negatively impact on emotion detection if the affect is low in intensity. On the other hand, it is clear that emotions can exist outside conscious awareness (Winkielman and Berridge, 2004). The fact that eating-disordered individuals do not have sufficient emotional language to identify and describe their emotions does not necessarily mean flattened affect. Clinical evidence shows that patients who report experiencing emotions cannot always identify the thoughts behind those emotions and put them into words (Fox and Power, 2007).

Alternatively, it may be possible that there are fluctuations between flat and intense negative effect in individuals with eating disorders. The flattened affect may be the result of a prolonged experience of intense emotions, and represents successful achievement of the emotion downregulation goal. If an individual had been feeling certain emotions (e.g. sadness) intensely and/or for a long time, and the (conscious or unconscious) goal was to decrease the intensity of these emotions (e.g. to return to their comfortable set level of arousal, Larsen, 2000), certain powerful coping strategies may have been employed to regulate it (e.g. bingeing) which resulted in radical reduction of the emotion (i.e. flatness). This way emotional state was successfully (if dysfunctionally) managed. In other words, an overwhelming negative emotion may deplete the individual’s emotional and cognitive resources, leaving him/her emotionally unresponsive until the next intensely negative experience. This suggests that the imbalance of affect may be a feature of eating disorders.
To date Affect Intensity had not been studied in the context of eating pathology, but the evidence outlined above points to the possibility for some interesting findings. It seems equally possible that individuals with eating pathology would have either higher or lower global and negative AI than healthy controls. Because of the alexithymia link, one could expect that eating dysfunction would be associated with a diminished level of AI; however, the relationship between anxiety, psychological maladjustment and emotional arousal suggested that eating problems would be associated with high Affect Intensity (global and negative, in particular). It would also be interesting to see whether there are any differences in the intensity of positive and negative emotions as experienced by those with eating pathology as compared to controls.

**Research Aims (Chapter 2)**

This chapter looked at the evidence for the nature and role of negative emotions and their regulation in eating pathology. Researchers seem to agree that limited emotion awareness, lack of emotional language and affect regulation deficits are associated with development and maintenance of eating pathology. High frequency of negative emotions also features in the literature as one of the characteristic factors in eating disorders. The chapter shows that it is worth investigating emotion regulation and intensity of negative and positive affect in those with eating pathology, because it may shed light on the origins and maintenance of eating disorders. The following research aims arose from the discussion above:

1. To examine emotion regulation and Affect Intensity in individuals with eating pathology and controls
2. To look at the effects of experimentally-induced happiness, sadness and neutral mood on emotion regulation and Affect Intensity in individuals with eating pathology and controls in order to establish robustness of the Affect Intensity and emotion regulation (as measured by the Regulation of Emotion Questionnaire, Phillips and Power, 2007) constructs.

For studies addressing the above research aims, please refer to Chapters 4, 6, 7 and 8.
CHAPTER 3. POSITIVE AFFECT AND CREATIVITY IN EATING DISORDERS

Positive affect in eating disorders has been largely overlooked, and there is very little (if anything) known about how positive emotions are experienced by individuals with eating pathology (Fox and Power, 2009) and how positive psychology can be used to aid their recovery. This section looks at the effects of happiness on mental and physical health, as well as at positive interventions. Since this section deals with the domain of positive psychology, it also includes a section on creativity. There is tentative evidence to suggest that eating pathology may be associated with creativity, and if so, this may have implications for treatment. This chapter covers all of the above topics in detail.

Positive emotions and their value

Positive affect is one of the central subject matters of positive psychology (Seligman, 2007). It is a branch of psychology that explores conditions and attributes, consistent with personal growth and well-being (Seligman, 2007). Positive emotions are ‘brief experiences that feel good in the present and increase the chances that one will feel good in the future’ (Froh, 2009). Positive emotions and states include joy, happiness, contentment, satisfaction, gladness, hope and gratitude. Seligman (2007) categorizes positive emotions into three distinct groups: those relating to the past (e.g. pride), the future (e.g. optimism) and present (e.g. relaxation and bliss).

In positive psychology happiness has been divided into two main types: eudaimonic and hedonic (Ryff et al., 2004). Eudaimonic happiness results from personal development, serving others and finding meaning (Ryan and Deci, 2001); and hedonic (or subjective) well-being stems from pleasures and transitory positive affect (Diener, 1984). Eudaimonic happiness tends to be associated with better psychological and physical outcomes than hedonic happiness because of its association with meaning and personal growth (Ryff et al., 2004; Seligman, 2007). However, some researchers argued that both types of well-being are equally valuable; according to Biswas-Diener et al. (2009), the two types of happiness are interconnected and build on each other. The researchers suggested that the classification of happiness into eudaimonic and hedonic is unnecessary because the two types represent two different approaches to the study of positive emotions, rather than two qualitatively distinct kinds of happiness.
In this Chapter and further in the thesis, the terms ‘positive affect’, ‘happiness’, ‘positive emotions’, ‘positive mood’, ‘subjective well-being’ and ‘joy’ will be used interchangeably to prevent repetitiveness.

Seligman (2007) proposes the following formula for measuring happiness: Enduring Level of Happiness = Set Range + Circumstances + Factors Under Voluntary Control (Seligman, 2007). The Set Range, or genetically pre-determined baseline happiness level, accounts for 50% of the total happiness score (Figure 3). This level cannot be sustainably changed, and individuals tend to return to their baseline level of happiness following happy and sad events. People habituate to happiness and adversity to an equal degree and after a while come back to their set point of happiness; this is known as hedonic adaptation (Seligman, 2007; Lyubomirsky, 2011). However Circumstances (10%) and Factors Under Voluntary Control (40%) can be influenced to some degree to affect the Enduring Level of Happiness. Some circumstances such as marital status can be changed to improve well-being, others such as age cannot. Factors Under Voluntary Control have the most potential to push enduring happiness level to the upper part of the default happiness level; such factors include satisfaction about the past, pleasure and positive emotions in the present and optimism about the future. These can be achieved by practising gratitude, actively pursuing happiness in everyday life and choosing a more positive outlook (Seligman, 2007).

Figure 3: Level of Happiness Determinants (after Lyubomirsky, 2008).
It is important to apply findings on positive affect to clinical psychology because - in the words of Sheldon and Lyubomirsky (2004) - ‘happiness is a central criterion of mental health’. If one was going to give a definition of happiness mathematically, subjective well-being would equal the sum of life satisfaction and a high ratio of positive to negative affect (Diener et al., 1991; Larsen et al., 1985). In other words, happiness can be characterized to some extent by the frequency of joyful experiences in one’s life. Although emotion intensity is important for mental health (e.g. it helps to draw the line between healthy joy and pathological mania), therapeutic effects of positive emotions have to do with their frequency (Fredrickson and Losada, 2005). Fredrikson and Losada (2005) have found that a ratio of positive to negative affect of 2.9 (or 3 to 1) indicates robust mental health. This ratio is known as the Losada line, after the psychologist who discovered it.

The Losada ratio is a pre-condition for the upward spiral of development (Fredrickson, 1998). The Broaden-and-Build Theory (Fredrickson, 1998) proposes that positive emotions have several important roles: (1) they broaden thought-action repertoires (Fredrickson, 1998) which means that attention, problem-solving skills, creativity and cognition are all enhanced if the individual is experiencing positive affect; and (2) they can trigger upward development spirals in which positive affect and broad-minded coping (or healthy, flexible coping with adversity) mutually influence each other and grow together over time (Fredrickson and Joiner, 2002). This suggests that by encouraging functional emotion regulation, positive affect makes a contribution to long-term emotional well-being, and improved emotion regulation in turn aids personal growth and increases well-being. Several other studies have also found that happiness is related to functional coping strategies (Fredrickson and Losada, 2005; Lyubomirsky et al., 2005).

There is evidence to suggest that positive mood is a protective factor in mental and physical health, and can even have a therapeutic effect (Lyubomirsky and Dickerhoof, 2010). It has been found that frequent experience of positive emotions is associated with growth, resilience and flourishing, even in adverse circumstances (Fredrickson, 1998; Fredrickson et al., 2001; Fredrickson and Losada, 2005). Happiness is associated with a
reduction in psychopathic symptomatology, prevention of relapse and lower incidence of unhealthy behaviours (Lyubomirsky et al., 2005).

Moreover, happiness can reverse the impact of negative emotions on psychological, cognitive and physiological levels (Fredrickson, 1998; Fredrickson et al., 2000; Fredrickson and Losada, 2005; Fredrickson and Levenson, 1998; Howell et al., 2007; Lyubomirsky et al., 2006). Izard (1991) suggested that one of the key functions of joy is ‘recuperation from stress and strain’. Melnechuk (1988) found that positive emotions help immune system and encourage recovery. In addition, positive affect increases longevity (Danner et al., 2001) and protects general health (Ryff and Singer, 1998). Fredrickson et al. (1998; 2000) found that positive emotions managed ‘to undo the [health-damaging] cardiovascular aftereffects of negative emotions’. Their study in 2000 provided evidence that positive emotions do not just replace negative emotions, but actively undo them on the physiological level. These findings mean that positive affect helps the body (e.g. the cardiovascular system) to return to homeostatic levels of arousal and to recover from physiological changes, associated with negative emotions (Fredrickson, 2000). This may be the mechanism whereby positive emotions influence health.

Another function of joy is facilitation of social interactions (Izard, 1991). Fredrikson’s (1998) Broaden-and-Build Theory suggests that positive affect helps to build lasting resources, such as social connections (Fredrickson, 2001; Fredrickson and Losada, 2005). Positive emotions activate broad thought-action repertoires; in other words, they widen one’s spectre of responses and increase one’s cognitive flexibility and creativity, which helps to build social and other resources (Fredrickson, 1998). Positive emotions encourage exploration, play and social interactions, which are useful for increasing social resources and strengthening relationships. The benefits of joy do not end there - the feelings of happiness are also accompanied by an increase in self-confidence and self-esteem (Izard, 1991). Positive emotions also appear to be at the foundation of strengths and virtues (Seligman, 2007), which may be another way in which they help to build resources. All of these factors make positive affect important for long-term health and well-being.
positive Therapies

Several therapies that aim to increase positive affect and encourage personal growth are currently being developed. Positive psychotherapies (PPT) is based on the theory that increasing happiness and building meaning is an effective treatment strategy for emotional disorders (Seligman et al., 2006). Depression and eating disorders have a low mood component and are characterized by a lack of positive emotions, and Seligman et al. (2006) argue that positive psychotherapies can be a useful complement to other forms of therapy and may help to prevent relapse in such disorders. The Well-being therapy (WBT) is a promising new therapy, and it provides evidence that distress can be prevented and counteracted by positive emotions (Ruini and Fava, 2003, 2009). WBT aims to develop a habit of focusing on the positives in one’s life and to help individuals become more aware of their emotions. This therapy is based on Ryff’s Model of Well-Being (1989) which suggests that in order to experience psychological wellness a person has to develop such attributes as: environmental mastery, purpose in life, autonomy, self-acceptance and positive relationships with others, and to strive for personal development (Ryff, 1989). Developing these attributes is the focus of Well-Being Therapy (Ruini and Fava, 2003, 2009). It has been successfully implemented in adolescent samples (Ruini and Fava, 2003, 2009), and is being tested in other populations.

Positive emotions in eating pathology

To date there had been no systematic study of positive emotions in individuals with eating disorders (Fox and Power, 2009). The scarce literature that exists does not offer a comprehensive, coherent positive affect model for eating disorders; indeed, the findings on the topic are sketchy. It seems that eating-disordered individuals’ alexithymic difficulties extend to positive emotions. Oldershaw et al. (2009) found that even after emotion recognition training in anorexia sufferers, recovered anorexics still struggled to identify positive emotions in themselves and others.

There is evidence that females with eating disorders do experience positive affect, and that it may be related to the eating pathology symptomatology (Overton et al., 2005).
This is not surprising. As was discussed in Chapter 1, biochemically, disordered eating is often associated with feelings of euphoria and positive affect (Davis and Crardge, 1998; Hardy and Waller, 1988). Weight loss and control may provide a sense of achievement and pride (Palmer, 2000; Vitousek and Ewald, 1993).

It appears that positive emotions may have a complex influence on eating and exercising behaviours. It was found that individuals with eating disorders were more likely to engage in physical activity when in a positive mood (Vansteelandt et al., 2007); however, this study did not subdivide participants into groups according to their diagnosis, so it is not known whether these findings apply to all three eating disorders (anorexia, bulimia and EDNOS), or are more diagnosis-specific. The implications of these findings differ in accordance with diagnoses. Physical activity may be dangerous for those with anorexia whose bodies may not be strong enough for exercise; however, it is a healthy distraction strategy and in moderation may be beneficial for those with bulimia and EDNOS. With regard to bulimic and EDNOS symptoms, another study showed that dysphoric binge eaters who believed that food would lift their mood, tended to choose foods that were higher in calories (Dingemans et al., 2009). In other words, the food choice represented the individual’s attempt to change their mood from sadness to happiness. These studies suggest that it is important to investigate positive emotions in eating disorders.

**Positive interventions and eating disorders**

Positive psychology has been applied to prevention and treatment of eating disorders in a number of ways. In their article on the role of positive psychology in prevention of disordered eating, Steck et al. (2004) outline their proposal for re-conceptualization of eating disorders in line with the positive psychology tenets. They believe that eating pathology can be prevented if it is placed in the context of ‘subjective well-being, constructive individual traits and positive institutions’ (Steck et al., 2004). The concepts of *subjective well-being* and *constructive individual traits* refer to positive emotions and positive personal qualities; under *positive institutions* Steck et al., (2004) understand social institutions (e.g. schools) that have the potential to foster the development of individual character strengths and to enhance well-being. Steck et al.’s (2004)
reformulation of eating disorders in the positive psychology context appears to be empirically supported. Increasing positive emotions, encouraging development of individual character strengths and providing information about the fashion industry (e.g. how unrealistic media images are) as well as about healthy eating have all been found to be protective factors for eating pathology and body dissatisfaction in adolescents (Phelps et al., 1999a; Phelps et al., 1999b; Posavac et al., 2001). In multiple studies, positive psychology interventions (PPIs) successfully and significantly reduced depressive symptoms, and the results were maintained at the 1-year follow-up (Seligman et al., 2006). This suggests that positive psychology has a lot to offer to prevention of psychopathology in general, and eating disorders in particular.

The positive psychology treatment techniques that have been used with some success to address eating pathology are mindful eating (Proulx, 2008) and savouring (Bryant, 2003; Bryant and Veroff, 2007). These approaches are based on developing associations between food and positive emotions (e.g. gratitude, relaxation – in savouring) as well as increasing awareness and acceptance of eating and hunger (in mindful eating) in order to make eating a more pleasant experience. Proulx (2008) found that when bulimic females were taught how to appreciate and enjoy food, they were less likely to engage in unhealthy and extreme eating behaviours at the 2-month follow-up. This suggests that eating pathology may be alleviated by interventions that aim to increase positive affect. The concepts of savouring and mindfulness have been incorporated into therapies: Dialectical Behaviour Therapy, Acceptance and Commitment Therapy and mindfulness therapies have all been applied to treating eating disorders with promising results (Heffner et al., 2002; Kroger et al., 2010; Safer et al., 2001a, 2001b). This was discussed in more detail in the Psychological Treatment section of the thesis (Chapter 1).

There is another way of applying positive psychology to eating disorders. As was discussed in Chapter 2, eating pathology is often accompanied by low mood (Johnson and Larson, 1982; Leon et al., 1993). Eating disorder symptomatology may represent a dysfunctional attempt at regulating negative emotions (Bydlowski et al., 2005; Fox and Power, 2007; Heatherton and Baumeister, 1991). Therefore it is plausible that reduction
of dysphoric affect would be associated with improvement in disordered eating. If negative affect encourages maladaptive coping, then decreasing the frequency of dysphoric emotions would lead to healthier emotion regulation and perhaps, over time, to remission of eating pathology. One way to decrease negative affect may be by focusing a one’s attention on positive emotions and increasing their frequency. Positive psychologists have developed a number of specific techniques (*positive psychology interventions*, or PPIs) that induce positive affect. Doing acts of kindness, imagining one’s best possible self, replaying positive memories, and regularly expressing gratitude have been found to effectively reduce negative affect and increase well-being both in the short and long term (Emmons and McCullough, 2004; Lyubomirsky et al., 2003; Forduce, 1983; Sin and Lyubomirsky, 2009; Seligman et al., 2002; Wiseman, 2009).

**Gratitude and ‘Count Your Blessings’ Intervention**

Gratitude is ‘being aware of and thankful for the good things that happen’ (Seligman et al., 2005). It has been conceptualized as an emotion (i.e. the feeling of thankfulness), a general worldview, a virtue and a trait (Emmons, 2009). According to Seligman (2002), in order to be happy one needs to be ‘satisfied about the past, optimistic about the future and happy in the present’. Expressing gratitude – either in writing, in thoughts, or in person – achieves all three objectives. It makes the individual realize that good things happened to them in the past, which makes them feel happy in the present and optimistic about the future (Seligman, 2002). Gratitude increases both eudaimonic and hedonic types of happiness because it lifts mood in the short term (*hedonic happiness*) and helps find meaning and contentment in the long term (*eudaimonic happiness*). According to Emmons (2009) grateful thinking may prevent hedonic adaption, by encouraging one to pay attention to the good things in life and to appreciate them consciously. This means that regular practice of gratitude may raise and maintain one’s default happiness level.

Gratitude expression is associated with good psychological and physical health (Emmons and McCulloogh, 2004), as well as optimal social functioning, optimism, life satisfaction and well-being (Emmons and McCullough, 2003; Kashdan et al., 2006; Seligman et al., 2005; Watson et al., 1988). In addition, gratitude enhances resilience in
the face of adversity and buffers against emotional disorders such as depression, anxiety and substance abuse (Bono et al., 2004; Emmons, 2007; Emmons and McCullough, 2003; Fredrickson et al., 2003; Kendler, 2003; McCullough et al., 2002). It may even protect against cardiovascular dysfunction and strengthen the immune system (Emmons, 2009).

Gratitude is outward directed, and this makes it an interpersonal emotion (Emmons and Shelton, 2002). In line with the Broaden-and-Build Theory (Fredrickson, 1998) expression of gratitude would promote strengthening of social ties and an increase in social capital. This indeed seems to be the case. Emmons (2007) reports that participants in his gratitude study reported ‘feeling closer and more connected to others and were more likely to help others’ (Emmons, 2007). This means that gratitude encourages building of relationships and social networks which can be valuable resources in the future (Bartlett and DeSteno, 2006; McCullough et al., 2008; Tsang, 2006). Nowak and Roch (2006) suggested that gratitude is evolutionarily adaptive because it inspires reciprocal altruism (Trivers, 1972) which is altruism in relation to the benefactor, as well as upstream reciprocity (Nowak and Roch, 2006) which is altruistic behaviour in relation to a third party. Emmons (2009) writes that ‘there is a general consensus that gratitude can be regarded as a moral emotion in that it leads to behavior intended to benefit others’. McCullough et al. (2001, 2008) discovered three functions of gratitude. It is (1) a moral barometer because it signals to the grateful person that someone has acted in their interest, (2) a moral motive because it encourages pro-social behaviours in the giver of gratitude, (3) a moral reinforcer because it encourages pro-social behaviours in the recipient of gratitude. In other words, expressing appreciation leads to positive social outcomes for the person who is grateful, for the benefactor and for the rest of society.

One of the most successful happiness-enhancing methods is counting one’s blessings. The gratitude exercises may involve writing down several things one is thankful for (i.e. journaling) or writing a gratitude letter to the benefactor (Emmons and McCullough, 2003; Lyubomirsky et al., 2005). The exercises seem to be remarkably effective in inducing sustained positive mood. Emmons and McCullough (2003) found that there
was a 25% increase in happiness in the group that did gratitude exercises once a week for 10 weeks, compared to controls who did a neutral task. In addition, they were also more likely to look after their health better (e.g. exercise more), and reported more optimism about the future. Similarly, Seligman et al. (2005) found that in their randomized longitudinal study those in the gratitude condition scored lower on depression and higher on well-being at the 6 months follow-up than those in the neutral condition.

The intervention can be done at any time interval (e.g. weekly). However there is evidence for habituation effects; Lyubomirsky et al. (2005) found that doing the gratitude task once a week, as opposed to several times a week, was the optimal frequency. This gratitude approach is somewhat similar to positive reappraisal (Internal Functional emotion regulation strategy, Phillips and Power, 2007) because it gets the individual to re-interpret past and present stressful life experiences in a positive light and to focus on what’s going right for them (Sheldon and Lyubomirsky, 2006). It is therefore not surprising that expressing gratitude is associated with increased well-being, and this is true both for nonclinical and clinical samples (Carson et al., 2010; Sin and Lyubomirsky, 2009). A recent pilot study by Carson et al. (2010) looked at the impact of gratitude exercises on service users with long-term mental illnesses. They found that longitudinal gratitude intervention led to a significant increase in subjective well-being, life satisfaction, social feelings and environmental mastery. In addition, their participants reported that they had enjoyed keeping a gratitude journal. Carson et al.’s (2010) study with a clinical sample is very promising, and more research into PPIs in clinical populations is needed.

Individuals who are typically low in positive emotions (e.g. those with depressive symptoms) may particularly benefit from doing gratitude exercises (Froh et al., 2009). This is because they have a longer way to go before they reach their emotional ceiling (Froh et al., 2009), and therefore may be more responsive to the gratitude intervention than individuals who do not have a negative/positive affect imbalance. Expressing gratitude may help to start a cycle of positive affect in those with depressive symptomatology: gratitude expression elicits positive emotions, which in turn elicits
more gratitude which further increases feelings of well-being. Moreover, the positive effects of regular expression of gratitude tend to be maintained in the long term (Seligman et al., 2005). This makes gratitude intervention a valuable tool for mood modification.

Emmons and Shelton (2002) acknowledge that consistent expression of gratitude may be difficult and taxing. As a happiness-enhancing skill, gratitude may need to be consciously cultivated because most people tend to have a tendency to take things for granted (i.e. to habituate to positives) and to dwell excessively on the negatives. According to Emmons and Shelton (2002) grateful thinking is a useful skill to learn and it is worth investing time and energy into it.

**Positive Reminiscence Intervention**

Positive reminiscence, or replaying happy memories in one’s head, in writing or talking, is another effective method for inducing positive mood (Bryant et al., 2005; Wiseman, 2009; Westerhof et al., 2010). Happy memories can refer to the events of years ago or very recent past (e.g. yesterday). Positive reminiscence is related to savouring, which is ‘the capacity to attend to, appreciate and enhance the positive experiences of one’s life’ (Bryant et al., 2009). In other words, when an individual is replaying happy memories in their head, they are focusing on the past events, savouring them, and thereby eliciting positive affect.

Positive reminiscence has been tested considerably less than the gratitude approach. There is a lot of evidence for positive influence of reminiscence on social functioning and well-being in older adults (Cook, 1998; Westerhof et al., 2010). According to Webster (1993, 1997), reliving and sharing happy memories appears to serve several positive functions; it may help (1) to build an identity (Erikson, 1963), (2) to strengthen identity continuity, (3) to come to terms with one’s mortality, (4) to remind one of helpful coping strategies that were used before, (5) to combat boredom, and (6) to foster intimacy and connections with others (Webster, 1993, 1997). In addition, positive reminiscence has been consistently found to improve self-esteem in older adults.
(Westerhof et al., 2010). All these are adaptively important factors. However, recent studies show that the positive effects of reminiscence are not age-specific, and younger people seem to derive just as many benefits from it (Bryant et al., 2005; Habermas and Paha, 2000). Bryant et al., (2005) found that the positive reminiscence exercise had significant benefits in 98% of cases; the participants reported an increase in their subjective well-being, thankfulness for an opportunity to return to happy times and to gain new insights. Bryant et al. (2005) discovered that vividness of happy memories considerably enhanced participants’ feelings of happiness.

The purpose of the positive reminiscence task is typically to get the individual to focus on good memories, and to thereby elicit positive emotions. When doing this exercise, participants may be asked to think back and recall (or alternatively, to write down or discuss) happy events or images from their past in as much detail as they can (Bryant et al., 2005), while avoiding trying to analyse them (Lyubomirsky, 2006). Analysing happy memories, as opposed to just replaying them, may result in negative, as opposed to positive mood (Lyubomirsky et al., 2006).

Positive reminiscence may be seen as relating to some aspects of the Well-Being Therapy (Ruini et al., 2003). In this therapy individuals learn to identify their emotions and to pay attention to the positive events in the present, via working through positive events and feelings associated with them from the recent past (self-observation stage, Ruini et al., 2003). Increased self-awareness and feelings of well-being, as well as lower levels of dysphoric symptoms and psychosomatic complaints are some of the benefits of this approach (Ruini et al., 2003, 2009). Therefore, positive reminiscence intervention has several well-being, awareness and self-esteem benefits, and is an effective and easy to implement method of positive mood induction.

**Conclusions**

Positive affect had not been properly studied in the context of eating disorders. Evidence from positive psychology studies suggests that it has palliative benefits and may be used for therapeutic purposes in treating mental and physical illness (Sin and
Lyubomirsky, 2009). Gratitude and Happy Memories exercises are some of the most effective methods of positive mood induction, and they appear easy to administer to an eating-disordered sample. It seems worthwhile to study positive emotions in individuals with eating disorders using the PPIs and to examine whether positive affect has an impact on emotion regulation and eating pathology. The expectation is that the gratitude induction will have an overall positive effect on emotion regulation. Since gratitude is a social emotion, it is likely that the Gratitude task will primarily affect emotion regulation that involves other people (External Functional ER and External Dysfunctional ER; Phillips and Power, 2007). However, in addition to being an interpersonal emotion, gratitude is also a positive emotion, therefore it is also plausible that it will influence emotion regulation from the inside (Internal Functional ER and Internal Dysfunctional ER; Phillips and Power, 2007). With regard to happy memories, it is more difficult to predict which aspects of emotion regulation it would affect. Because it contributes to identity formation and strengthening, as well as to social bonding, it is expected that positive reminiscence will have a general positive effect on emotion regulation in females with eating pathology. Chapters 4, 6, 7 and 8 describe and discuss the studies that looked at positive emotions in the context of emotion regulation and eating pathology.

Creativity
This section provides the background to the concept of creativity, and its relationship with psychopathology, emotion regulation and Affect Intensity (Larsen, 1984). There are two main lines of creativity research that this section will pursue: one concerns the relationship between creativity, Affect Intensity, emotion regulation and general psychopathology and the other one looks at creativity in the context of eating pathology. Creativity is one of the most complex psychological phenomena (Runco and Sakamoto, 1999). It is also one of the most commonplace, and can be seen in most forms of human activity - from culture, sport and science to more mundane tasks (e.g. cooking). Despite creativity being a subject of considerable scientific interest in the past 50 years, it remains unclear whether the concept has its roots in intelligence, personality, mental health, or emotion processing.
Creativity can be broadly defined as ‘the ability to produce work that is both novel [i.e. original, unexpected] and appropriate [for its purpose, i.e. useful]’ (Sternberg and Lubart, 1999). The concept seems to elude a clear uniform definition and is typically defined by the specific tests used to measure it (Guilford, 1950). For research purposes creativity has been operationalized in several ways: autobiographically (i.e. self-reported creative interests and accomplishments; Hocevar, 1980), as part of the personality structure (e.g. certain facets of the Openness to Experience factor in the Big-Five), as convergent thinking (i.e. finding a correct solution to a problem by original means) and as divergent thinking (e.g. adopting a clever and unconventional approach to a problem; Guilford, 1950; Silvia et al., 2008). Each of the tests taps into different aspects of creativity and most researchers tend to favour one assessment method over the others. The two most widely used measures to measure creativity are the Alternative Uses task (Guilford, 1950) and the Creative Behaviour Checklist (Hocevar, 1980). Guilford (1950) favoured the divergent thinking approach to creativity and on-line creativity tests; his famous task consists in asking participants to name original uses for an every-day object, and the novelty and number of their responses are assessed. Unlike Guilford, Hocevar (1981, 1989) suggested that real life manifestations of creativity (e.g. creative accomplishments) are its most useful and accurate indicators. His approach has been in looking at individuals’ self-reports of their achievements across six domains: fine arts, literature, crafts, music, performing arts and maths and science. Both approaches have their merits, and it appears that it would be useful to combine the two assessment methods in order to get a more complete picture of creativity.

CREATIVITY: AFFECT INTENSITY AND EMOTION REGULATION
There is some evidence which points to the fact that creative people may typically experience emotions of higher intensity than average; this may be particularly true of negative emotions (Mitchell, 1972). According to Martindale (1989), creative people have a tendency to physiologically overreact to stimuli at both sensory and emotional level; in addition, they tend to exhibit a ‘higher basal level of arousal’ (Martindale, 1999), both of which could be a result of their biogenetic makeup. This hypersensitivity
to internal and external stimuli appears to be reflected in their anxiety scores which tend to be high (Martindale, 1999).

Multiple researchers report higher incidence of affect disorders and mental illness in the creative population (Prentky, 1989; Jamison, 1993). This is not surprising if we assume that creative people are higher in Affect Intensity (AI). Somatization, poor adjustment, hypomania, substance abuse are known correlates of AI (Flett and Hewitt, 1995; Thornberg and Lyvers, 2006) and Vujanovic et al. (2006) showed that anxiety and panic disorder are potential long-term outcomes for high AI individuals. Some of these mental health difficulties are often observed in creative populations as well (Prentky, 1989). As an alternative to the biogenetic hypothesis, it is also conceivable that strong emotions may stimulate creative expression and that creative activities provide a safe outlet for heightened affect. The ability to engage one’s emotions and to be responsive to affective stimuli appears to be important for creative process. Hoppe and Kyle (1990) found that paucity of affect (e.g. lowered awareness of own and others’ emotional states) has a negative influence on expressiveness, originality and imagination that are pertinent to creative performance. This means that individuals high in creativity may score highly in affect intensity as well.

There are reasons to believe that creativity would be associated with dysfunctional emotion management. Evidence shows that creative individuals tend to be impulsive, risk-taking and often have a substance abuse problem (Martindale 1989; Sternberg, 1999); these qualities would presumably be non-conducive to healthy emotion regulation choices. In addition, creativity has been found to be associated with a wide spectrum of psychopathology (e.g. anxiety, depression and bipolar disorder; Jamison, 1993; Martindale, 1999; Prentky, 1989), and this may also impact on how affect is regulated. Therefore, it is likely that creative individuals would tend to use unhealthy ways to regulate their emotions (e.g. self-harm). It is also plausible that creative activity per se may represent an emotion management strategy (e.g. distraction), that is used to channel and express affect.
CREATIVITY AND EATING PATHOLOGY

If eating disorders are a means of emotion expression, this may suggest the individuals with anorexia and bulimia may be creative. It is likely that eating disordered individuals creatively use a symbolic language where emotions are conveyed and processed via the medium of the body (Gerhardt, 2009) as opposed to words. There is some clinical literature on creativity-based treatments such as arts therapies (Crisp, 1980; Waller and Gilroy, 2000; Dokter, 1995). However, there has been virtually no research looking at the relationship of creativity to eating pathology.

Although creativity per se is a healthy activity, creative abilities and eating pathology (anorexia nervosa in particular) may be associated in a number of ways. There are some dispositional and clinical similarities between highly creative individuals and those suffering from eating disorders. Characteristics like perseverance, a high need for order, fragmented personal identity (Bruch, 1978), physiological hypersensitivity (Martindale, 1989; Bruch, 1978), high Neuroticism and anxiety (Dowd, 1989) are common in both. In addition, in both creative process and eating disorders there is an observed tendency to manage negative emotions with the use of substances such as food (e.g. restriction of food intake), alcohol or drugs (Kerr, 2009; Martindale, 1989; Bruch, 1978). This can be seen as an attempt to regulate affect at the more profound level - by altering the biochemistry of the brain. It could be that the apparent similarities are due to a common factor underlying both eating pathology and creativity (e.g. high Affect Intensity); in other words, the tendency to be emotionally reactive may be implicated in stimulation of creative expression, and may also be a risk factor for developing an eating disorder.

Individuals suffering from eating disorders appear to be creative in their approach to food and weight management. The methods they resort to in order to hide fluctuations in weight, the amount of food consumed, or the weight loss strategies they develop are often very inventive and original (Bruch, 1978). Eating-disordered individuals meet a lot of criteria for Sternberg and Lubart’s (Sternberg, 1999) Theory of Creativity; according to their theory, an individual has to have the right combination of intellectual abilities, relevant background knowledge, styles of thinking, personality, motivation and
environmental conditions in order to become creative in a certain domain. A stereotypical eating-disordered patient tends to have extensive knowledge of food and calories, a rigid external-oriented thinking style, a neurotic and perfectionistic personality, a motivation to lose weight and a dysfunctional family environment, all of which are conducive to developing an unconventional and original approach to eating. Some eating-disordered patients appear to be highly original in the art domain - clinical evidence (e.g. patients’ drawings in art therapy) demonstrates originality and unconventionality of their perception of themselves and the world. They often approach a simple task (e.g. to draw themselves) from an unexpected angle and go on to produce work of high creative merit: complex, creative and original (Crowl, 1994; Crisp, 1980; Dokter, 1995; Luzzatto, 1980; Waller and Gilroy, 2000). The subjects for drawing are typically assigned by the art therapist, and the drawing process taps into divergent thinking abilities. This suggests that individuals with eating pathology may be creative when it comes to such tasks.

On the other hand, it is plausible that eating pathology would be negatively related to creativity. There is evidence that patients with eating pathology tend to exhibit difficulties with identifying and describing their emotional states (i.e. alexithymia) and as Hoppe and Kyle (1990) showed, insufficient awareness of own and others emotions is not conducive to expressiveness, originality and imagination, required for creativity. So it may be that this numbing of affect and lack of emotional language - dispositional or resulting from malnutrition - would be an obstacle to creative self-expression. The arguments outlined above suggest that there may be a relationship between eating pathology and creative tendencies. If the evidence that the clinically or subclinically eating-disordered population is more creative than normal controls can be provided, creativity may be incorporated as an element of psychological interventions. The creativity intervention may be used to teach emotional regulation by means of more adaptive tools (e.g. art supplies) than the body and food. Study 2 (Chapter 5) examined the relationship between creativity and eating pathology.
Research Aims (Chapter 3)

This chapter gave an overview of positive emotions, positive psychology interventions and creativity and made suggestions for their application to the study of eating disorders. The following research aims arose from the literature review above:

1. To examine the effect of positive psychology interventions (in particular, gratitude and positive reminiscence) on emotion regulation in individuals with eating pathology (please refer to Chapters 6, 7 and 8)
2. To test whether there is a relationship between Affect Intensity, emotion regulation, eating pathology and creativity (please refer to Chapter 5).

Timeline of research development

Original research aim of studying emotion regulation in eating disorders developed over the course of the PhD program to include emotion induction, creativity and Affect Intensity components. Study 1 (Chapter 4) examined emotion regulation and Affect Intensity in female students with subclinical eating pathology, as well as the effects of happiness, sadness and neutral mood (induced via a combination of the Velten Mood Induction procedure and Music) on emotion regulation and Affect Intensity. The study was conducted in 2008. Study 2 (Chapter 5) was an exploratory study that looked at the relationship between creativity, emotional disorder (anxiety and depression), Affect Intensity, emotion regulation and eating pathology. In light of the evidence, gathered in Study 2, the creativity hypothesis was abandoned, and the subsequent research focused on the effects of emotions on affect regulation in females with eating pathology. The study was conducted in 2008.

Study 3 (Chapter 6) looked at the effects of positive psychology interventions (gratitude and positive reminiscence) on emotion regulation, general well-being, anxiety and depression levels in subclinically eating-disordered females. The study was carried out in 2009. The choice of PPIs as the mood induction method in Study 3 was explained by the intention to test their usefulness and ease of implementation in a potential longitudinal study with a clinical sample, where regular mood induction would be required. Due to the gratitude intervention proving a more
effective mood induction tool, the positive reminiscence exercise was not used in subsequent studies. The gratitude intervention proved to be a simple and effective method of inducing positive mood (in comparison with the more complex Music + Velten Mood Induction procedure), and was therefore used in subsequent studies.

Study 4 (Chapter 7) examined emotion regulation and Affect Intensity, as well as the influence of a one-off gratitude intervention on regulation of emotion, in females with clinical eating pathology (anorexia nervosa, bulimia nervosa and EDNOS). Study 5 (Chapter 8) was a longitudinal analogue to Study 4, in that it looked at the effects of gratitude-induced happiness on emotion regulation and eating pathology in females with eating disorders over the course of 4 weeks. Both studies were carried out in 2010.
CHAPTER 4. Study 1: Emotion Regulation and Affect Intensity in a subclinically eating-disordered sample and controls: the effects of happiness, sadness and neutral mood.

INTRODUCTION

Study 1 addressed two main questions: (1) is it possible that emotional dysregulation in eating pathology is related to intensity of affect? and (2) can positive emotions improve emotion regulation in eating pathology? Regulation of emotions and Affect Intensity are two aspects of emotional experience that may be relevant to our understanding of disordered eating. Individuals with eating pathology tend to experience frequent and varied dysphoric emotions (e.g. sadness, anger, fear and disgust), and dysfunctional regulation of such emotions appears to play an important part in maintenance of eating pathology (Bydlowski et al., 2005; Fox and Power, 2007; Heatherton and Baumeister, 1991; Root et al., 1986; Taylor et al., 1997). Disordered eating may represent an attempt at managing negative feelings (Cooper et al., 2004; Waller et al., 2004; Wildes et al., 2009). It is not clear why emotion dysregulation occurs in individuals with eating pathology; one possibility is that it may be the result of an insufficiently developed emotional theory of mind (Oldershaw et al., 2009; Russell et al., 2009) and difficulties with recognizing and expressing feelings (i.e. alexithymia), associated with it. Alexithymia may be one of the plausible reasons for why eating-disordered individuals have deficits in emotion regulation (Bydlowski et al., 2005; Carano et al., 2006; Gillboa-Schechtman et al., 2006; Fox and Fromm, 2009; Fox, 2009; Troop et al., 1995; Zonneville-Bender et al., 2002). The origins of alexithymia are contested, and at present there is no consensus on where it stems from (Schmidt et al., 1993): some researchers attribute it to malfunctioning of certain cerebral structures (Borsci et al., 2009), while others – to the effects of psychological factors, such as depression and anxiety, on emotional processing mechanisms (Gilboa-Schechtmann et al., 2006).

Another possibility is that the trouble with identifying, communicating and regulating emotions may be linked to one of the inherent properties of negative affect, namely its intensity. The tendency to experience affect of low intensity was found to be associated with alexithymia in a study by Jacob and Hautekeete (1999). In other words, individuals who typically experience flattened affect, tend to have more difficulties in recognizing, describing and expressing their feelings. However, Jacob and Hautekeete’s (1999) study
did not look at alexithymia in eating disorders, and it would not be viable to make generalizations to an eating-disordered population from this one study. Because of the well-documented link with alexithymia, it is possible that eating-disordered individuals would report having emotions of low intensity; however, the opposite may also be true. Literature suggests that emotions characterized by high intensity are important and frequent accompaniers of psychopathology: from self-harm (Gratz, 2006) to addictions, somatization, emotional suppression and the borderline personality disorder (Flett and Hewitt, 1995; Larson, 2009; Thornberg and Lyvers, 2006). The Affect Intensity construct (Larsen, 1987) is also an indicator of variability of affect (i.e. how changeable it is). It is plausible that individuals with eating disorders may experience affect that fluctuates (e.g. from highly intense to completely flat). This emotional imbalance may contribute to alexithymia, which in turn may impair emotion regulation. In practice, this may mean that individuals with eating pathology may experience unstable fluctuating affect, and in episodes of emotional flatness or extreme negative affect, they may exhibit alexithymic difficulties and mismanage their emotions, using food and eating.

The present study focused on exploring the functionality of regulatory strategies and intensity of affect in a subclinically eating-disordered group (SED) and controls. It also looked at the effects of experimentally induced happiness, sadness and neutral mood on emotion regulation and Affect Intensity. One of the most significant psychology findings of the past two decades was that positive affect could benefit mental and physical health. It was discovered that happiness could reverse the impact of negative emotions - on psychological, cognitive and physiological levels (Fredrickson, 1998; Fredrickson et al., 2000; Fredrickson and Losada, 2005; Fredrickson and Levenson, 1998; Howell et al., 2007; Izard, 1991; Lyubomirsky et al., 2006). In addition, it was found that frequent experience of positive emotions was associated with growth, resilience and flourishing even in adverse circumstances (Fredrickson, 1998; Fredrickson et al., 2001; Fredrickson and Losada, 2005).

According to the Broader-and-Build Theory (Fredrickson, 1998), positive emotions encourage broad-minded coping (or healthy, flexible coping with adversity). It is worth looking at happiness induction as a possible way of improving emotion regulation in
individuals with eating pathology because if it works, such an improvement may contribute to effective treatment of disordered eating. It is possible that positive affect would encourage individuals with eating pathology to select healthier emotion regulation strategies, and this in turn would deem disordered eating as an emotion regulation mechanism unnecessary.

One of the most obvious hedonic antitheses to happiness (i.e. positive affect) is sadness (i.e. negative affect). The effects of sadness on emotion regulation would presumably be diametrically opposite to those of happiness. In other words, if happiness would improve adaptiveness of emotion regulation, then sadness would encourage dysfunctional coping. Literature on dysphoric emotions suggests that negative affect prompts eating-disordered individuals to engage in disordered eating as a means of down-regulating it (Cooper et al., 2004; Waller et al., 2004; Wildes et al., 2009). Therefore, sadness may be unconducive to healthy emotion regulation. This is an area worth researching because of its potential implications for treatment of eating disorders. There is little consensus regarding the influence of emotions on Affect Intensity. Some studies have found it to have trait-like properties and be a stable characteristic (Larsen, 1987), presumably rooted in temperament and biology; whereas others found that it could be easily manipulated and was highly variable (Heide and Gronhaug, 1996). Because of the possible implication of Affect Intensity in emotion regulation in individuals with eating pathology, it seemed worthwhile to test these findings, and to examine the effects of happiness, sadness and neutral mood on Affect Intensity.

To sum up, Study 1 looked at functionality of emotion regulation and Affect Intensity (Global, Positive and Negative AI), as well as the relationship between the two, in females with and without eating pathology, using the Regulation of Emotion Questionnaire (Phillips and Power, 2007) and the Affect Intensity Measure Questionnaire (Larsen, 1986). It was predicted that disordered eating would be related to dysfunctional regulatory tendencies, and that Affect Intensity (Negative, Positive and Global) would be associated with eating pathology (the direction of the interaction was not specified). The relationship between Affect Intensity and emotion regulation was
also explored, and it was predicted that the tendency to experience strong and variable affect would not be conducive to functional emotion regulation.

The study also examined the effects of experimentally-induced happiness, sadness and neutral mood on functionality of emotion regulation and Affect Intensity. It was predicted that unlike sadness and neutral mood, happiness would have a positive effect on emotion regulation, making it more functional. With regard to Affect Intensity, it was predicted that mood induction would not have any effect on the tendency to experience emotions intensely.

METHOD

Participants and Recruitment
One hundred University of Edinburgh Psychology students (mean age = 20.6 years) replied to an on-line advert on SAGE, the University of Edinburgh job database, expressing their wish to take part in the study. Participants were screened for eating pathology using EAT-26 (Garner et al., 1982). The mean eating pathology score in the sample was 10.71 (SD = 11.05). The score of 11 was used as a cut-off point for subclinical eating pathology. All the participants were female and Caucasian. Being a native English speaker was the main inclusion criterion for the study, and all the participants were from English-speaking countries (United Kingdom: N= 97, United States: N = 3). The participants’ age ranged from 18 to 33 years (mean = 20.5, SD = 2.67).

The average scores on the emotion regulation measure (REQ, Phillips and Power, 2007) were calculated; Internal Dysfunctional emotion regulation mean was 7.29 (SD = 3.56); External Dysfunctional emotion regulation mean was 2.90 (SD = 2.23); Internal Functional emotion regulation mean was 7.23 (SD = 2.05) and External Functional emotion regulation mean was 14.51 (SD = 3.59). The averages and dispersion of scores on the affect intensity scale (AIM, Larsen, 1984) were also computed; Global Affect Intensity mean was 3.73 (SD = .46); Positive Affect Intensity mean was 3.71 (SD =
.54), and Negative Affect Intensity mean was 3.79 (SD = .59). The mean anxiety and depression scores (HADS, Zigmond and Snaith, 1983) were also calculated; anxiety mean was 8.84 (SD = 4.05), and depression mean was 3.66 (SD = 2.79).

**Design**

Study 1 was of a mixed design: participants were assigned to two separate experimental conditions: Happiness (N = 34) and (Sadness N = 33) and one control condition (N = 33), and each participant filled in a set of questionnaires on two occasions (before and after mood induction). Figure 4 shows the flow and attrition of participants in the study in detail.

Figure 4: A consort diagram showing the flow and attrition of participants in Study 1
Ethics

Study 1 received ethical approval from the School of Psychology, Philosophy and Language Sciences Ethics Committee (University of Edinburgh). Participants were informed that the main research aims concerned eating habits and emotions, and that the experimental procedure would involve listening to a piece of music and completing questionnaires. The participants signed the informed consent forms, stating their wish to take part in the study. They were reminded of their right to withdraw at any time and assured that all of their responses would be kept anonymous and confidential in order to protect their privacy. At the end of the session, the participants were fully debriefed on the purposes of the study, and those in the Sadness condition were given a small unexpected gift to lift their mood to the pre-manipulation level. The gift was a discount voucher.

Procedure

Potential participants were told that the experiment would last for around 1 ½ hours and they would be paid £10 for their time. The approximate procedure time was calculated as follows: 40mins for the first set of questionnaires + 12 minutes of mood induction + 40mins for the second set of questionnaires. Following completion of the informed consent form, they were asked to indicate their availability over the subsequent four weeks, and were allocated a time slot. The sessions were scheduled in 2-hour time slots with 3 participants per slot. The experiment required use of computers and took place in computer cubicles in the University of Edinburgh’s Psychology Department. Each participant had their own individual computer cubicle. The scheduling was flexible to suit the participants, and sessions ran from 9am till 6pm on weekdays (10 slots per day) over the course of 4 weeks. For participants’ convenience and to guide the experimental process, procedure instructions and music were computerized using Microsoft Power Point. In the first part of the study, participants were randomly allocated to three mood groups (happy, sad and neutral) and presented with a set of paper-and-pen questionnaires: Emotion Assessment Scale (EAS; Carlson et al., 1989), Regulation of Emotion Questionnaire (Phillips and Power, 2007) and Affect Intensity Measure (Larsen, 1984). The second part of the study involved mood manipulation. Happy, sad and neutral moods were induced using a combination of the Velten Mood Induction
Procedure (Velten, 1968) and happy/sad/neutral 12-minute music excerpts (Albinoni’s Adagio in the Sad condition; Mozart’s Toy Symphony in the Happy condition; Holst’s Planets in the Neutral condition), presented to each participant on the computer screen and through the headphones. Following that, effectiveness of emotion manipulation was assessed by the EAS (Carlson et al., 1989) and the battery of the questionnaires was re-administered. Manipulation checks were performed on the data, and those who failed to respond to mood induction (N = 9) were excluded from the statistical analyses that looked at mood effects. Manipulation checks involved assessing the change in scores on the Happiness and Sadness scales of the EAS (Carlson et al., 1989) as a result of mood induction.

**Measures**

1. The Eating Attitudes Test (EAT-26; Garner et al., 1982) assesses the risk of eating pathology and is commonly used as a screening tool. It consists of 26 items that refer to the past 6 months. EAT-26 (Garner et al., 1982) has three subscales: Dieting, Bulimia and Food Preoccupation, and Oral Control. The composite score of 20 or over on the test may indicate an eating disorder. [Appendix 5]

2. Affect Intensity Measure Questionnaire (Larsen, 1984) assesses typical depth of emotion processing and measures Global, Positive and Negative emotions intensity. Items are phrased so as to highlight the intensity aspect of emotions. The scale consists of 40 statements (e.g. ‘When I accomplish something difficult, I feel delighted or elated’), and the respondent has to rate the frequency of each statement from Never to Always. [Appendix 2]

3. Regulation of Emotion Questionnaire (REQ, Phillips and Power, 2007) measures functionality of affect management along four subscales (Internal Dysfunctional, Internal Functional, External Dysfunctional and External Functional emotion regulation). It is a 21-item questionnaire with statements
about different emotion regulation strategies like ‘I ask others for advice’ with a frequency scale from Never to Always. The respondent is asked how often they use certain regulatory strategies: ‘In general, how do you respond to your emotions?’ In Study 1 participants completed this questionnaire in its original form prior to mood induction. Because this questionnaire measures a general tendency to regulate emotions in particular ways at present, it had to be adapted to meaningfully capture any change after mood induction. Therefore, after mood manipulation, participants completed the adapted version of the Regulation of Emotion Questionnaire. The instructions focused on the future intentions and likelihood of using different regulatory strategies, and the question was changed to: ‘How will you respond to your emotions?’. All the statements were adapted to refer to the future (e.g. ‘I shall talk to someone about how I feel’ instead of ‘I talk to someone about how I feel’), and the response scale (from Never to Always) remained the same. [Appendix 1]

Phillips and Power (2007) had reported that the original Regulation of Emotion Questionnaire (REQ) had acceptable internal consistency with an average Cronbach’s alpha coefficient of 0.72 (the coefficients ranged from 0.66 to 0.76). As part of the present study, reliability testing was carried out to ensure equivalence of the two versions of REQ (original and revised). Reliability analysis showed that the average Cronbach’s alpha coefficient for the Revised REQ was 0.68 which indicates acceptable internal consistency. In the neutral condition the Cronbach’s alpha was 0.69 for Internal Dysfunctional emotion regulation, 0.63 for External Dysfunctional emotion regulation, 0.68 for Internal Functional emotion regulation and 0.71 for External Functional emotion regulation in the Revised REQ. For comparison, in the present study, the average Cronbach’s alpha coefficient for the original REQ questionnaire was 0.65. In the neutral condition the Cronbach’s alpha was 0.79 for Internal Dysfunctional emotion regulation, 0.61 for External Dysfunctional emotion regulation, 0.62 for Internal Functional emotion regulation and 0.61 for External Functional emotion regulation in the original version of the REQ. This suggests that the revised version of REQ is as reliable as the original REQ questionnaire.
The original REQ reliably measures current emotion regulation tendencies, whereas the revised REQ reliably assesses future emotion regulation intentions.

4. The Emotion Assessment Scale (EAS; Carlson et al., 1989) measures current mood and assesses effectiveness of mood induction. It is a list of 24 mood adjectives such as Happy or Disgusted, and the participants are asked to indicate the extent to which they experience those emotions by putting a slash on the line that goes from Least Likely to Most Likely. The answers are then added up to make up a score for each of the eight emotions (Happiness, Sadness, Disgust, Fear, Guilt, Surprise, Anxiety and Anger). [Appendix 9]

5. Mood Manipulation: a combination of Music and the Velten Mood Induction Procedure (Velten, 1968) was used to induce happy, sad and neutral mood. There is evidence that effectiveness of these two methods of mood manipulation is enhanced by combining them (Albersnagel, 1988; Clark, 1983). Happiness and sadness were chosen for induction because these are clearly emotions of opposite valence (positive – negative). The Velten Mood Induction Procedure (Velten, 1968) is a well-established mood induction method. It can be used to induce 3 different moods: happy, sad and neutral. Participants are asked to read 60 self-referent statements that are designed to elicit either happiness (e.g. ‘I feel good today!’), sadness (e.g. ‘I feel a little low today’) or 60 general sentences to elicit neutral mood (e.g. ‘Nothing can be burned that has already been burned once’). In addition to the Velten statements [Appendix 8], three 12 minute excerpts of classical music were chosen. Albinoni’s Adagio G-Minor was used to elicit sadness because it had been shown to be effective in inducing mood of this valence (Eich and Metcalfe, 1989; Gerrards-Hesse et al., 1994). Mozart’s Toy Symphony was the music excerpt of choice for the Happiness condition; it had been previously successfully used to induce positive mood and elation (Gerrards-Hesse et al., 1994; Mayer et al., 1990). Holst’s Neptune - the Mystic (from the Planets suite) was used to induce neutral mood in the control condition; it had been shown to be an effective neutral mood induction method (Gerrards-Hesse et al., 1994; McFarland, 1984). In the present study, the Velten
statements were put on Microsoft Power Point presentation slides, and the music files were attached to the presentation. Participants were asked to slowly go through the slides and read the statements, while listening to the music through their headphones.

RESULTS
Data screening procedures were performed. The data were checked for accuracy by computing frequencies and minimum/maximum statistics for the variables using Statistical Package for the Social Sciences (SPSS). Visual checking of the resultant analyses against the questionnaire data ensured that there were no errors in the data file, and all the scores fell within the range of possible scores for the scales. Skewness, kurtosis and z-scores were also computed for each dependent variable (please see Appendix 13). All z-scores fell below 3.3, which indicated reasonably good normal distribution. Visual checking of histograms and scatterplots confirmed that the scores on the dependent variables were reasonably normally distributed, the relationships between them were linear and the data tended to have similar variability. As part of the t-tests, Levene’s test was performed in order to ensure homogeneity of variance; in case of all the variables the Levene test was non-significant (p > 0.05). The cases that have missing values were excluded from analyses that included that variable.

The findings show that the combination of Music and Velten procedure was effective in inducing the required mood in more than 85% of cases. Out of one hundred participants, 34 took part in the Happiness condition, 33 in the Sadness condition and 33 were controls. Using the combination of music and the Velten procedure, Happiness was successfully induced in 29 cases (85%) and Sadness was effectively induced in 29 cases (88%). As can be seen from Table 5, in the happiness condition, participants scored between 0 and 25.70 (Mean = 7.92, SD = 7.12) on Sadness before mood induction; after happiness induction, their Sadness scores dropped to between 0 and 14.30 (Mean = 2.62, SD = 3.22). In addition, before mood induction, they scored between 0.70 and 28.40 (Mean = 15.28, SD = 7.82) on happiness; after happiness induction their scores rose to between 0.70 and 35 (Mean = 21.61, SD = 8.30). As Table 5 shows, in the sadness condition, participants scored between 0 and 28.80 (Mean = 7.11, SD = 7.17)
on Sadness before mood induction; their Sadness scores rose to between .40 and 31.70 (Mean = 14.56, SD = 8.03) after Sadness induction. Participants in the Sadness condition also scored between 2.70 and 30.10 (Mean = 15.63, SD = 7.08) on Happiness prior to Sadness induction; after Sadness induction their scores dropped to between .20 and 22.40 (Mean = 5.58, SD = 5.34). The group comparison (subclinical group versus controls) was not included in the mood induction analyses because it was not part of the original design. As can be seen from Table 5, in the neutral condition participants scored on average 6.35 on Sadness before mood induction (SD = 6.57); these scores dropped to 5.82 (SD = 5.62) following neutral mood induction. Their happiness scores also dropped slightly from 15.57 (SD = 5.62) to 11.48 (SD = 7.86) following neutral mood induction. The decrease in happiness as well as sadness scores represents successful neutralization of mood in this condition.

Table 5: Descriptive Statistics for Happiness, Sadness and Neutral Conditions

<table>
<thead>
<tr>
<th>Condition</th>
<th>N</th>
<th>Minimum</th>
<th>Maximum</th>
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<td><strong>Happiness</strong></td>
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<tr>
<td>Pre-Induction Sadness</td>
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<td>25.70</td>
<td>7.92</td>
<td>7.12</td>
</tr>
<tr>
<td>Pre-Induction Happiness</td>
<td>34</td>
<td>.70</td>
<td>28.40</td>
<td>15.28</td>
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N = Number of participants, SD = Standard Deviation
A series of t-tests, mixed between-within subjects ANOVAs and post-hocs was conducted to assess the impact of three different emotional states (Happy, Sad and Neutral) on participants’ self-reported Emotion Regulation strategies (Internal Dysfunctional ER (InDys), Internal Functional ER (InFun), External Dysfunctional ER (ExDys) and External Functional ER (ExFun)) and Affect Intensity (Global, Positive and Negative AI) scores in two conditions (subclinically eating-disordered and controls) across two time periods (pre mood induction - Time 1 and post mood induction - Time 2). The data were checked to ensure no violation of the assumptions of normality, linearity and homoscedasticity. Histograms and scatterplots showed that the scores on the variables were reasonably normally distributed, the relationships between them were linear and the data tended to have similar variability.

Functionality of emotion regulation and Affect Intensity in the subclinically eating disordered sample and controls.

Bonferroni adjustment was performed, and the alpha value (0.05) was divided by 7 (the number of tests). The new alpha level was 0.007 after rounding, and it was used as a cut-off. A series of Independent Samples t-tests was conducted to compare Emotion Regulation and Affect Intensity scores for subclinically eating disordered participants (SED) and controls (Figure 5). There was a significant difference in InDys scores for SED (Mean = 9.87, SD = 4.32) and controls (Mean = 6.87, SD = 3.23); t (89) = 3.09, p < 0.007 (two-tailed). The magnitude of difference in the means (mean difference = 3, 95% CI: -4.92 to -1.07) was large (eta squared = 0.10). There was no significant difference in InFun scores for SED (Mean = 7, SD = 1.77) and controls (Mean = 7.29, SD = 2.11); t (89) = .5, p = .62 (two-tailed). There was no significant difference in ExDys scores for SED (Mean = 3.93, SD = 2.49) and controls (Mean = 2.67, SD = 2.02); t (89) = 2.12, p = .036 (two-tailed). There was no significant difference in ExFun scores for SED (Mean = 13.73, SD = 4.70) and controls (Mean = 14.49, SD = 3.34); t (89) = .74, p = .46 (two-tailed). In summary, compared to controls, the SED group reported significantly more Internally Dysfunctional regulatory strategies (Figure 5).
ER = Emotion Regulation

A series of Independent Samples t-tests was conducted to compare Affect Intensity Scores (Global AI, Positive AI and Negative AI) for subclinically eating disordered participants (SED) and controls (Figure 6). There was a significant difference in Global Affect Intensity scores for SED (Mean = 3.96, SD = .45) and controls (Mean = 3.69, SD = .46); t (89) = 2.10, p < 0.007 (two-tailed). The magnitude of difference in the means (mean difference = .27, 95% CI: -.52 to -.01 was small (eta squared = 0.05). There was no significant difference in Positive AI scores for SED (Mean = 3.77, SD = .67) and controls (Mean = 3.68, SD = .51); t (89) = .61, p = .54 (two-tailed). There was a significant difference in Negative AI scores, with SED scoring higher (Mean = 4.25, SD = .50) than controls (Mean = 3.75, SD = .54); t (89) = 3.29, p < 0.007 (two-tailed). The magnitude of difference in the means (mean difference = 0.5, 95% CI: -.80 to -.20) was medium (eta squared = .11).
In summary, the SED group scored higher on Global Affect Intensity and Negative Affect Intensity than controls.

Effect of happiness and sadness on functionality of Emotion Regulation in the subclinically eating disordered sample and controls.

Internal Dysfunctional Emotion Regulation (InDys)
A mixed between-within subjects ANOVA was conducted to assess the impact of three different emotional states (Happy, Sad and Neutral) on participants’ self-reported Internal Dysfunctional emotion regulation scores (InDys) across two time periods (pre mood induction: Time 1 and post mood induction: Time 2). Table 6 and Figure 7 show the results. There was no significant main effect for time, Wilks’ Lambda =1, F
(1, 88) = .30, p = .59, partial eta squared = .59. There was a significant interaction between mood condition and time, Wilks’ Lambda = .75, F (2, 88) = 14.51, p < 0.05, partial eta squared = .25. Post hoc tests were conducted to check where the significance lay. There was a significant decline in InDys scores, following the happiness induction (Time 1: Mean = 7.79, SD = 3.42; Time 2: Mean = 6.31, SD = 3.42), t (28) = 4.20, p < 0.05 (two-tailed). The mean decrease in InDys scores in the happiness condition was 1.48 with a 95% confidence interval rating from .76 to 2.21. The eta squared statistic (0.39) indicated a large effect size. In addition, there was a significant increase in InDys scores in the sadness condition (Time 1: Mean = 7.28, SD = 3.62; Time 2: Mean = 8.24, SD = 4.15); t (28) = 2.57, p < 0.05 (two-tailed). The mean increase in InDys scores in the sadness condition was 0.93 with a 95% confidence interval rating from -1.74 to -0.19. The eta squared statistic (0.19) indicated a large effect size. The changes in the InDys scores in the Neutral condition were not significant (Time 1: Mean = 7.06, SD = 3.77; Time 2: Mean = 7.27, SD = 3.33), t (33) = .88, p = .39. There was no significant main effect of the three mood conditions on InDys scores F (2, 88) = .34, p = .71, partial eta squared = .008.

Table 6: Internal Dysfunctional ER Scores in 3 Conditions Before and After Mood Induction

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mean</th>
<th>SD</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Induction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Happy</td>
<td>7.79</td>
<td>3.42</td>
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<tr>
<td>Sad</td>
<td>7.28</td>
<td>3.62</td>
<td>29</td>
</tr>
<tr>
<td>Neutral</td>
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<td>3.77</td>
<td>33</td>
</tr>
<tr>
<td>Total</td>
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<td>3.59</td>
<td>91</td>
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<tr>
<td>Post-Induction</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Happy</td>
<td>6.31</td>
<td>3.42</td>
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<tr>
<td>Sad</td>
<td>8.24</td>
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<tr>
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<td>33</td>
</tr>
<tr>
<td>Total</td>
<td>7.27</td>
<td>3.68</td>
<td>91</td>
</tr>
</tbody>
</table>

N = Number (of participants), SD = Standard Deviation
Internal Functional Emotion Regulation (InFun)
A mixed between-within subjects ANOVA was conducted to assess the impact of three different emotional states (Happy, Sad and Neutral) on participants’ self-reported Internal Functional emotion regulation scores (InFun) across two time periods (pre mood induction - Time 1 and post mood induction - Time 2). Table 7 shows InFun scores before and after mood induction. There was a significant main effect for time, Wilks’ Lambda = .19, F (1, 88) = 384.60, p < 0.05, partial eta squared = .81 with all three groups showing an increase in InFun scores across two time periods. There was a significant interaction between mood groups and time, Wilks’ Lambda = .88, F (2, 88) = 5.76, p < 0.05, partial eta squared = .12. Post hoc tests were conducted to check where the significance lay. There was a significant increase in InFun scores, following the
happiness induction (Time 1: Mean = 7.03, SD = 2.21; Time 2: Mean = 12.24, SD = 3.15), \( t (28) = 16.31, p < 0.05 \) (two-tailed). The mean increase in InFun scores in the happiness condition was 5.21 with a 95% confidence interval rating from -5.86 to -4.55. The eta squared statistic (0.9) indicated a large effect size. The InFun scores also increased following the sadness induction (Time 1: Mean = 7.21, SD = 2.32; Time 2: Mean = 10.55, SD = 3.61), \( t (28) = 6.57, p < 0.05 \) (two-tailed). The mean increase in InFun scores in the sadness condition was 3.34 with a 95% confidence interval rating from -4.39 to -2.30. The eta squared statistic (0.61) indicated a large effect size.

The InFun scores also significantly rose the neutral mood condition (Time 1: Mean = 7.45, SD = 1.68; Time 2: Mean = 11.97, SD = 2.44); \( t (32) = 14.65, p < 0.05 \) (two-tailed). The mean increase in the InFun scores was 4.57 with a 95% confidence interval rating from -5.14 to -3.88. The eta squared statistic (0.87) indicated a large effect size.

The main effect, comparing the three mood conditions was not significant, \( F (2, 88) = 1.10, p = .34 \), partial eta squared = .024.

Table 7: Internal Functional ER Scores Before and After Mood Induction

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mean</th>
<th>SD</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Induction</td>
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<td></td>
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</tr>
<tr>
<td>Happy</td>
<td>7.03</td>
<td>2.21</td>
<td>29</td>
</tr>
<tr>
<td>Sad</td>
<td>7.21</td>
<td>2.32</td>
<td>29</td>
</tr>
<tr>
<td>Neutral</td>
<td>7.45</td>
<td>1.68</td>
<td>33</td>
</tr>
<tr>
<td>Total</td>
<td>7.24</td>
<td>2.06</td>
<td>91</td>
</tr>
<tr>
<td>Post-Induction</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Happy</td>
<td>12.24</td>
<td>3.15</td>
<td>29</td>
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<tr>
<td>Sad</td>
<td>10.55</td>
<td>3.61</td>
<td>29</td>
</tr>
<tr>
<td>Neutral</td>
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<td>2.44</td>
<td>33</td>
</tr>
<tr>
<td>Total</td>
<td>11.60</td>
<td>3.13</td>
<td>91</td>
</tr>
</tbody>
</table>

N = Number (of participants), SD = Standard Deviation

External Dysfunctional ER (ExDys)

A mixed between-within subjects ANOVA was conducted to assess the impact of three different emotional states (Happy, Sad and Neutral) on participants’ self-reported External Dysfunctional emotion regulation scores (ExDys) across two time periods (pre mood induction - Time 1 and post mood induction - Time 2). Table 8 and Figure 8 show ExDys scores before and after mood induction. There was a significant main effect for time, Wilks’ Lambda = .84, \( F (1, 88) = 17.10, p < 0.05 \), partial eta squared =
with all three groups showing a decrease in ExDys scores over time. There was a significant interaction between mood group and time, Wilks’ Lambda = .90, F (2, 88) = 4.77, p < 0.05, partial eta squared = .10. There was a significant decrease in ExDys scores in the happiness condition (Time 1: Mean = 3.07, SD = 2.27; Time 2: Mean = 1.55, SD = 1.53), t (28) = 4.25, p < 0.05 (two-tailed). The mean decrease in ExDys scores was 1.52 with a 95% confidence interval rating from .79 to 2.25. The eta squared statistic (0.39) was large. There was a significant decrease in ExDys scores in the neutral condition (Time 1: Mean = 2.30, SD = 1.67; Time 2: Mean = 1.64, SD = 1.69), t (32) = 4.30, p < 0.05 (two-tailed). The mean decrease in ExDys scores was 0.66 with a 95% confidence interval rating from .35 to .98. The eta squared statistic (0.37) was large. The changes in the ExDys scores in the sadness condition were not significant (Time 1: Mean = 3.34, SD = 2.41; Time 2: Mean = 3.24, SD = 3.18), t (28) = .25, p = .81 (two-tailed).

The main effect comparing the three mood groups was significant, F (2, 88) = 3.60, p < 0.05, partial eta squared = .07, suggesting a difference in ExDys scores between mood conditions. Tukey HSD test showed that the ExDys scores in the Sad (Time 1: Mean = 3.34, SD = 2.41; Time 2: Mean = 3.24, SD = 3.18) and Neutral (Time 1: Mean = 2.30, SD = 1.67; Time 2: Mean = 1.64, SD = 1.69) conditions were significantly different (mean difference = 1.32, 95% CI: .11 to 2.53, p < 0.05).
Figure 8: Changes in External Dysfunctional emotion regulation scores across 3 mood conditions

Time 1: before mood induction, Time 2: after mood induction

Table 8: External Dysfunctional emotion regulation before and after mood induction

<table>
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<th>Condition</th>
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<td>2.41</td>
<td>29</td>
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<tr>
<td>Neutral</td>
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N = Number (of participants), SD = Standard Deviation
External Functional ER (ExFun)

A mixed between-within subjects ANOVA was conducted to assess the impact of three different emotional states (Happy, Sad and Neutral) on participants’ self-reported

**External Functional emotion regulation scores** (ExFun) across two time periods (pre mood induction - Time 1 and post mood induction - Time 2). Figure 9 and Table 9 show ExFun scores before and after mood manipulation. There was no significant main effect for time, Wilks’ Lambda = .10, F (1, 88) = .217, p = .64, partial eta squared = .00. There was a significant interaction between mood group and time, Wilks’ Lambda = .84, F (2, 88) = 8.35, p < 0.05, partial eta squared = .16. There was a significant increase in ExFun scores in the happiness condition (Time 1: Mean = 13.69, SD = 4.06; Time 2: Mean = 14.93, SD = 3.76), t (28) = 4.25, p < 0.05 (two-tailed). The mean increase in ExFun scores was 1.24 with a 95% confidence interval rating from -1.84 to -.64. The eta squared statistic (0.39) was large. The changes in ExFun scores in the sadness condition were not significant (Time 1: Mean = 14.10, SD = 3.02; Time 2: Mean = 13.41, SD = 3.85), t (28) = 1.51, p = .14 (two-tailed). The changes in ExFun scores in the neutral condition were also non-significant (Time 1: Mean = 15.18, SD = 3.51; Time 2: Mean = 14.91, SD = 4.09), t (32) = 1, p = 0.32 (two-tailed). The main effect comparing the three mood groups was not significant, F (2, 88) = .99, p = .38, partial eta squared = .02.

In summary, happiness induction led to a significant decline in InDys and ExDys scores as well as an increase in InFun and ExFun scores. InDys and InFun levels significantly increased in the sadness condition; ExDys scores decreased and InFun levels increased in the neutral condition.
Figure 9: Changes in External Functional emotion regulation in 3 mood conditions

Table 9: External Functional emotion regulation before and after mood induction

<table>
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<td>Pre-Induction</td>
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<td></td>
</tr>
<tr>
<td>Happy</td>
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<tr>
<td>Sad</td>
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<td>3.93</td>
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</table>

N = Number (of participants), SD = Standard Deviation

Time 1: before mood induction, Time 2: after mood induction
Effect of happiness and sadness on Affect Intensity in the subclinically eating disordered sample and controls.

A mixed between-within subjects analysis of variance (ANOVA) was conducted to assess the impact of three different mood conditions (happy, sad and neutral) on Affect Intensity Scores. Table 10 and Figure 10 show Global Affect Intensity scores before and after mood induction.

Global Affect Intensity

There was a significant main effect for time in Global Affect Intensity scores, Wilks’ Lambda = .92, F (1, 88) = 8.03, p = .01, partial eta squared = .08. Figure 10 shows that Global AI scores decreased in Sadness (Time 1: Mean = 3.84, SD = 0.43; Time 2: Mean = 3.69, SD = 0.43) and Neutral (Time 1: Mean = 3.58, SD = 0.42; Time 2: Mean = 3.49, SD = 0.53) conditions and increased in the Happiness condition (Time 1: Mean = 3.80, SD = 0.51; Time 2: Mean = 3.88, SD = 0.55). There was a significant interaction between mood condition and time, Wilks’ Lambda = .81, F (2, 88) = 10.11, p < 0.01, partial eta squared = .19. There was a significant increase in scores in the happiness condition (Time 1: Mean = 3.80, SD = 0.51; Time 2: Mean = 3.88, SD = 0.55), t (28) = 2.09, p < 0.05 (two-tailed). The mean increase in scores was 0.08 with a 95% confidence interval rating from -0.14 to -0.001. The eta squared statistic (0.13) was large. The changes in scores in the sadness condition were also significant (Time 1: Mean = 3.84, SD = 0.43; Time 2: Mean = 3.69, SD = 0.43), t (28) = 2.09, p < 0.01. The mean decrease in scores was 0.15 with a 95% confidence interval rating from 0.09 to 0.21. The eta squared statistic (0.50) was large. The changes in scores in the neutral condition also reached significance (Time 1: Mean = 3.58, SD = 0.42; Time 2: Mean = 3.49, SD = 0.53), t (32) = 2.35, p < 0.05 (two-tailed). The mean decrease in scores was 0.09 with a 95% confidence interval rating from 0.01 to 0.18. The eta squared statistic (0.15) was large. The main effect comparing the three mood groups was also significant, F (2, 88) = 3.58, p = .032, partial eta squared = .07. Tukey HSD test showed that the difference between Global AI scores in Happy and Neutral conditions was significant (mean difference = .30, p < 0.05, 95% CI: 0.02 to .59).
Table 10: Global Affect Intensity Before and After Mood Induction

<table>
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<th>Condition</th>
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<th>SD</th>
<th>N</th>
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<tr>
<td>Sad</td>
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<td>.43</td>
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</tr>
<tr>
<td>Neutral</td>
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<td>.42</td>
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</tr>
<tr>
<td>Total</td>
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<td>.46</td>
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</tr>
<tr>
<td>Happy</td>
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</tr>
<tr>
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<tr>
<td>Total</td>
<td>3.68</td>
<td>.53</td>
<td>91</td>
</tr>
</tbody>
</table>

N = Number (of participants), SD = Standard Deviation

Figure 10: Global Affect Intensity Across Three Experimental Conditions (Happiness, Sadness and Neutral Mood).

Time 1: before mood induction, Time 2: after mood induction
Positive Affect Intensity

There was a significant main effect for time in Positive AI scores, Wilks’ Lambda = .92, F (1, 87) = 7.99, p < 0.05, partial eta squared = .08. Figure 11 shows that Positive AI scores decreased in Sadness (Time 1: Mean = 3.85, SD = .48; Time 2: Mean = 3.57, SD = .52) and Neutral (Time 1: Mean = 3.50, SD = .54; Time 2: Mean = 3.34, SD = .62) conditions and increased in the Happiness condition (Time 1: Mean = 3.77, SD = .53; Time 2: Mean = 3.95, SD = .62). Table 11 shows Positive AI scores before and after mood induction. There was a significant interaction between mood conditions and time, Wilks’ Lambda = .70, F (2, 87) = 18.28, p < 0.01, partial eta squared = .30. There was a significant increase in scores in the happiness condition (Time 1: Mean = 3.77, SD = .53; Time 2: Mean = 3.95, SD = .62), t (28) = 3.03, p < 0.01. The mean increase in scores was 0.18 with a 95% confidence interval rating from -.28 to .05. The eta squared statistic (0.25) was large. The changes in scores in the sadness condition were also significant (Time 1: Mean = 3.85, SD = .48; Time 2: Mean = 3.57, SD = .52), t (27) = 5.89, p < 0.001. The mean increase in scores was 0.28 with a 95% confidence interval rating from .18 to .37. The eta squared statistic (0.56) was large. The changes in scores in the neutral condition also reached significance (Time 1: Mean = 3.50, SD = .54; Time 2: Mean = 3.34, SD = .62), t (32) = 2.85, p < 0.01. The mean increase in scores was 0.16 with a 95% confidence interval rating from 0.04 to .26. The eta squared statistic (0.20) was large.

The main effect comparing the three mood groups was also significant, F (2, 87) = 5.42, p < 0.05, partial eta squared = .11. Tukey HSD showed that there were significant differences in Positive AI scores between Happy and Neutral conditions (mean difference = .44, p < 0.05, 95% CI: .11 to .76).
Table 11: Positive Affect Intensity Before and After Mood Induction

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mean</th>
<th>SD</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Induction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Happy</td>
<td>3.77</td>
<td>.53</td>
<td>29</td>
</tr>
<tr>
<td>Sad</td>
<td>3.85</td>
<td>.48</td>
<td>28</td>
</tr>
<tr>
<td>Neutral</td>
<td>3.50</td>
<td>.54</td>
<td>33</td>
</tr>
<tr>
<td>Total</td>
<td>3.70</td>
<td>.54</td>
<td>90</td>
</tr>
<tr>
<td>Post-Induction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Happy</td>
<td>3.94</td>
<td>.62</td>
<td>29</td>
</tr>
<tr>
<td>Sad</td>
<td>3.57</td>
<td>.52</td>
<td>28</td>
</tr>
<tr>
<td>Neutral</td>
<td>3.34</td>
<td>.62</td>
<td>33</td>
</tr>
<tr>
<td>Total</td>
<td>3.61</td>
<td>.63</td>
<td>90</td>
</tr>
</tbody>
</table>

N = Number (of participants), SD = Standard Deviation

Figure 11: Positive Affect Intensity Across Three Experimental Conditions (Happiness, Sadness and Neutral Mood).

Time 1: before mood induction, Time 2: after mood induction
Negative Affect Intensity

Table 12 and Figure 12 display the results for Negative Affect Intensity. The main effect for time was non-significant, Wilks’ Lambda = 1, F (1, 86) = .001, p = .98, partial eta squared = 0. The interaction between time and mood conditions also did not reach significance, Wilks’ Lambda = .94, F (2, 86) = 2.81, p = .06, partial eta squared = .06. The main effect comparing the three mood groups was also non-significant, F (2, 86) = .43, p = .65, partial eta squared = .01.

Table 12: Negative Affect Intensity Before and After Mood Induction

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mean</th>
<th>SD</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pre-Induction</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Happy</td>
<td>3.90</td>
<td>.65</td>
<td>28</td>
</tr>
<tr>
<td>Sad</td>
<td>3.86</td>
<td>.51</td>
<td>28</td>
</tr>
<tr>
<td>Neutral</td>
<td>3.76</td>
<td>.51</td>
<td>33</td>
</tr>
<tr>
<td>Total</td>
<td>3.84</td>
<td>.56</td>
<td>89</td>
</tr>
<tr>
<td><strong>Post-Induction</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Happy</td>
<td>3.81</td>
<td>.70</td>
<td>28</td>
</tr>
<tr>
<td>Sad</td>
<td>3.94</td>
<td>.63</td>
<td>28</td>
</tr>
<tr>
<td>Neutral</td>
<td>3.78</td>
<td>.60</td>
<td>33</td>
</tr>
<tr>
<td>Total</td>
<td>3.84</td>
<td>.64</td>
<td>89</td>
</tr>
</tbody>
</table>

N = Number (of participants), SD = Standard Deviation
In summary, happiness and sadness had a significant effect on Positive and Global Affect Intensity in the SED group and controls, but not on Negative Affect Intensity.

The relationship between Affect Intensity and emotion regulation in the full sample.

Pearson correlation was used to explore the relationship between Affect Intensity and Emotion Regulation in the whole sample (N = 100); the correlations are shown in Table 13. The data were checked to ensure no violation of the assumptions of normality, linearity and homoscedasticity. There was a moderate positive correlation between Global Affect Intensity and Internal Dysfunctional ER, $r = .30$, N = 100, $p < .01$ and Global Affect Intensity and External Dysfunctional ER, $r = .31$, N = 100, $p < .01$;
There was a moderate positive correlation between Positive Affect Intensity and External Functional ER, \( r = .21, N = 100, p < .05 \), and between Positive Affect Intensity and External Dysfunctional ER, \( r = .23, N = 100, p < .05 \). There was a strong positive correlation between Negative Affect Intensity and Internal Dysfunctional ER, \( r = .53, N = 100, p < .01 \); a medium positive correlation between negative Affect Intensity and External Dysfunctional ER, \( r = .25, N = 100, p < .05 \); and a medium negative correlation between negative Affect Intensity and External Functional ER, \( r = -.24, N = 100, p < .05 \).

Table 13: Correlations between Affect Intensity (AI) and Emotion Regulation (ER)

<table>
<thead>
<tr>
<th></th>
<th>Global AI</th>
<th>Positive AI</th>
<th>Negative AI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Internal Dysfunctional ER</strong></td>
<td>Pearson Correlation</td>
<td>( .30^{**} )</td>
<td>.01</td>
</tr>
<tr>
<td></td>
<td>Significance</td>
<td>.00</td>
<td>.94</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td><strong>Internal Functional ER</strong></td>
<td>Pearson Correlation</td>
<td>.01</td>
<td>.07</td>
</tr>
<tr>
<td></td>
<td>Significance</td>
<td>.93</td>
<td>.49</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td><strong>External Dysfunctional ER</strong></td>
<td>Pearson Correlation</td>
<td>( .31^{**} )</td>
<td>.23</td>
</tr>
<tr>
<td></td>
<td>Significance</td>
<td>.00</td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td><strong>External Functional ER</strong></td>
<td>Pearson Correlation</td>
<td>.03</td>
<td>.21^{*}</td>
</tr>
<tr>
<td></td>
<td>Significance</td>
<td>.75</td>
<td>.04</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

* significance at \( p < 0.05 \)
** significance at \( p < 0.01 \)

\( N = \) Number of participants

In summary, across the whole sample, high Global AI was associated with dysfunctional regulatory styles. In addition, high Negative AI was associated with dysfunctional emotion regulation and high Positive AI was associated with external functional emotion regulation.
Main findings

1. Eating pathology was associated with Internal Dysfunctional emotion regulation as well as high Global and Negative Affect Intensity.
2. The combination of Music + the Velten Mood Induction procedure was very effective in inducing happiness and sadness (success rate $\geq 85\%$).
3. Out of the three experimentally induced emotions, happiness had the best emotion regulation outcomes. Positive emotions led to a global improvement in emotion management.
4. Sadness led to an increase in the tendency to regulate emotions internally, without resorting to the help of others. This is consistent with the key properties of sadness such as withdrawal and introspection (Power and Dalgleish, 2008).
5. Mood induction did not significantly affect Negative Intensity scores, but it did influence Global Affect Intensity and Positive Affect Intensity.
6. High negative Affect Intensity and Global Affect Intensity were associated with dysfunctional emotion regulation.

Discussion

*Emotion Regulation and Affect Intensity in Subclinical Eating Disorders*

The hypothesis that eating pathology would be associated with dysfunctional emotion regulation, as measured by the Regulation of Emotions Questionnaire (Phillips and Power, 2007), was largely supported. The study found that individuals with eating pathology used Internal Dysfunctional emotion regulation strategies significantly more than healthy controls. These strategies include: self-harm, rumination, negative social comparison, repression and de-realization (Philips and Power, 2007). This means that individuals with eating pathology may tend to harm themselves, dwell on negative feelings, compare themselves unfavourably with others, keep feelings inside and daydream as an attempt to manage their emotions. All of these are unhealthy regulatory strategies that are generally associated with psychopathology and ill health (Aldao et al., 2010; Gratz, 2003; Gross, 1999; Phillips and Power, 2007). These strategies discourage healthy expression of disturbing emotions (e.g. verbal expression) and attempt to block them from the individual’s awareness. In this way, the individual’s distress remains
private, and there is no opportunity for social support. In addition, the individual is prevented from consciously working through their emotions and effectively soothing themselves. The nature of the relationship between these strategies and eating pathology is interesting. It could be that disordered eating represents a variant of self-harm (Gratz, 2003). In other words, individuals may use food and eating to punish themselves, and thereby deal with the negative feelings that they have about themselves (e.g. self-disgust), or those elicited by stressors (e.g. fear). In this way, disordered eating would be just another one of the internal dysfunctional regulatory strategies (Gratz, 2003).

It is possible that individuals with eating pathology may use the Internal Dysfunctional strategies because the alternative healthy regulation strategies have simply not been learned. This is supported by empirical research on attachment in individuals with eating disorders; it shows that individuals with eating disorders often had a primary caregiver who was not attuned enough to their needs as a child and failed to teach him/her to recognize and soothe his/her emotions (Schore, 2003). Therefore, the individual may not have learned how to become emotionally aware (i.e. to recognize what they are feeling) and healthy regulatory strategies. Functional emotion regulation strategies include: positive reappraisal, modification of goals, expression of feelings, advice and physical contact seeking and distraction (Phillips and Power, 2007). In other words, it is healthy to express emotions, and work through them, alone and with other people. These strategies are invariably associated with positive psychological outcomes (Phillips and Power, 2007).

However, the finding that there were no significant differences in External Functional and Internal Functional strategies between those with eating pathology and controls, suggests that both groups may use them equally frequently. In other words, individuals with eating pathology may know of the adaptive alternatives to dysfunctional regulatory strategies. Therefore, it is interesting that they use unhealthy strategies more. One explanation may be that functional emotion regulation strategies are not effective enough in managing negative emotions in those with eating pathology. It could be that they experience emotions of such intensity that more extreme regulatory strategies (e.g. self-harm) are required in order to bring affect under control. This fits in with the findings that students with eating pathology had significantly higher Global Affect...
Intensity as well as Negative Affect Intensity. This means that these females are highly emotionally reactive and typically experience negative emotions very profoundly. Larsen (2009) argued that ‘high AI subjects may not become aware of their emotional reactions until those reactions become quite strong’; to paraphrase Larsen (2009) and put his words in the context of Study 1 findings: females with eating pathology ‘may not become aware of their emotional reactions until those reactions become quite strong’. This means that alexithymia (i.e. difficulty in identifying and expressing feelings) in such individuals may reflect an episode of flattened affect, and moreover, that they may experience fluctuations between intensely negative emotions and flattened affect. Literature shows that individuals high in AI tend to experience highly variable moods in their daily life (Larsen and Diener, 1987); this means that their emotions may fluctuate widely across the affective range in a short space of time. Default emotional functioning of this kind (i.e. mood disturbance) would presumably be unconducive to healthy emotion regulation. This topic deserves a qualitative exploration in a clinical sample.

The tendency to experience affect profoundly may originate from the interplay between genetics, life stresses and traumas (Rothbart et al., 2000) because Affect Intensity is a component of temperament (Larsen et al., 1986; Larsen and Diener, 1987). It is plausible that individuals with eating pathology are particularly sensitive to negative emotions and may get easily overwhelmed by them. In these circumstances, such individuals may only be able to manage their emotions in ways that do not require a lot of emotional or cognitive resources. Functional regulatory strategies often require a lot of effort: positive reappraisal, for instance, requires assessment of a negative situation with a goal of seeing the silver lining. It engages such cognitive processes as concentration, reasoning and memory. This and other Internal Functional strategies may be difficult and time-consuming in the circumstances of overwhelming affect. External Functional strategies may also be rather demanding. For instance, advice-seeking requires the individual to identify a person they can turn to, to make the effort of finding them, to formulate their feelings into words, to explain the situation to them and to then receive and process their advice. When the individual is in *acute* distress and has trouble expressing their emotions, it may be much easier for them to attempt to deal with their feelings dysfunctionally and on their own, rather than to consciously engage
in functional emotion regulation and seek help. Self-harm, rumination, negative social comparison, repression and de-realization require little more than succumbing to the negative affect. They are easy to implement and may be somewhat helpful in suppressing acute negative affect (even though dysfunctionally). This may explain why individuals with eating pathology choose dysfunctional regulatory strategies.

It was interesting that individuals with eating pathology did not significantly differ from healthy controls on External Dysfunctional emotion regulation. These include such strategies as: bullying, verbal assault, physical assault and making others feel bad (Phillips and Power, 2007). It does not mean that these strategies of emotion management are not used, but rather that they do not distinguish those with eating pathology from those without. It is conceivable that these strategies also require considerable effort. For instance, bullying requires the individual to select a target, formulate the insult and deliver it. As eating-disordered individuals are not typically verbally expressive of their feelings (Bydlowski et al., 2005), it is unlikely that they would use words to cope with their negative emotions. Therefore, it is not surprising that such individuals do not use External Dysfunctional strategies more than do controls. However, it has to be noted that although the Bonferroni adjustment of the alpha level in External Dysfunctional scores was insignificant, conventional p = .036 is significant; therefore we need to be cautious when interpreting the results as meaning that there are no differences between the groups in External Dysfunctional emotion regulation.

In summary, individuals with eating pathology experience negative affect very intensely and tend to use Internal Dysfunctional strategies to regulate it (e.g. self-harm). It is suggested that such strategies are chosen because they are less taxing than functional regulatory strategies and Externally Dysfunctional emotion management.

Effects of happiness, sadness and neutral mood on emotion regulation and Affect Intensity

Happiness (i.e. a positive emotion) was compared with sadness (i.e. the emotion of opposite valence - negative) and neutral mood (i.e. state of neutral valence) in their effects on emotion regulation and Affect Intensity in those with subclinical eating pathology and controls. In this study sadness was chosen as an emotion for comparison
with positive mood because it is the most straightforward antithesis to happiness. However, it is not the only one, and any negative emotion (e.g. anger, fear, disgust) would also be in a different valence category from happiness (i.e. negative). In the future studies, it would be interesting to compare the effects of happiness and other negative emotions (e.g. disgust, fear and anger) on emotion regulation in individuals with eating pathology. The neutral mood subsumes such affective states as relaxation, comfort and lack of extreme emotions. The hypotheses that a happy mood would have a positive effect on emotion regulation, and the opposite would be true of sadness, were largely confirmed. The study showed that positive affect led to a wide improvement in emotion regulation strategies; it reduced the inclination to use Internal Dysfunctional and External Dysfunctional regulatory strategies, and increased the use of Internal Functional and External Functional methods. However, none of the emotions led to a significant change in Negative Affect Intensity.

Happiness is an emotion, interesting in its potential application to the field of clinical psychology. Multiple studies have shown and continue to show that positive affect has palliative and healing properties when it comes to physical and mental illnesses (Fredrickson, 1998; Fredrickson et al., 2000; Fredrickson and Losada, 2005; Fredrickson and Levenson, 1998; Howell et al., 2007; Lyubomirsky et al., 2005, 2006). Sadness as a dysphoric emotion has been studied in relation to eating pathology (Brewerton et al., 1995; Halmi, 1995; Rastam, 1992); however, to the author’s knowledge, there had not been studies where it was experimentally induced in eating-disordered participants with a view of examining emotion regulation. Positive affect led to a reduction in Internal Dysfunctional regulatory strategies, whereas negative affect led to an increase in those strategies in both subclinically eating-disordered sample and controls. Internal Dysfunctional scores did not change significantly in the neutral condition. This means that when an individual is in a good mood, they are less likely to choose strategies such as self-harm, rumination, negative social comparison, repression and de-realization (Philips and Power, 2007). Conversely, when the individual is sad, they are more likely to engage in those processes. These findings provide support for the body of literature on the benefits of positive emotions for cognition and social functioning (Fredrickson, 1998; Fredrickson et al., 2000;
Fredrickson and Losada, 2005; Fredrickson and Levenson, 1998; Howell et al., 2007; Lyubomirsky et al., 2005, 2006). Positive affect may be beneficial for emotion regulation because it encourages flexible thinking and approaching others for help (Fredrickson, 1998; Izen, 1991), whereas negative emotions promote restricted thinking and isolation from others.

Another finding was that participants reported themselves to be equally likely to engage in Internal Functional emotion regulation, following happiness, sadness and neutral mood induction. This suggests that an individual in any of those moods may use strategies, such as positive reappraisal, modification of goals and putting things in perspective (Phillips and Power, 2007), when attempting to regulate their emotions. These findings are interesting, because they suggest that mood is not a factor when it comes to Internal Functional emotion regulation. Perhaps, this affect management style is influenced by other negative emotions (e.g. anger, disgust etc.) that were not the object of the study. Alternatively, it is also possible that Internal Functional emotion regulation does not have emotional origins, and instead represents learned cognitive skills. External Dysfunctional scores decreased in the happiness condition, as well as in the neutral condition. Sadness had no significant effect on these scores. This means that an individual in a happy or relaxed mood is less likely to use bullying, verbal assault and physical assault as emotion regulation methods. It is interesting that sadness did not affect External Dysfunctional scores. This may be because these strategies are not used to manage this particular emotion; perhaps, other affective states, such as anger would have had a stronger effect on external dysfunctional strategies when experimentally induced.

Happiness led to an increase in External Functional scores, whereas sadness and neutral mood had no effect. This suggests that an individual in a happy mood would choose healthy emotion regulation strategies, such as expression of feelings and advice seeking when managing their affect. The finding that sadness and neutral mood did not have an effect on external functional scores shows that these affective states may be of little relevance to this type of emotion regulation.
If positive affect had such a beneficial influence on emotion regulation, it is plausible that it may be useful in bringing remission in eating pathology. If happiness encourages healthy emotion regulation, it may be possible that it will discourage disordered eating. Eating pathology may be a manifestation of self-harm (i.e. Internal Dysfunctional emotion regulation strategy), and therefore, improving emotion regulation by means of positive emotion induction may lead to remission in eating pathology. It is likely that regular longitudinal positive emotion induction would be needed in order to achieve such results. Positive emotion induction over time would result in increased frequency in positive affect, which may have a palliative influence. Because of their un-doing properties (Fredrickson and Levenson, 1998) positive emotions may reverse the effects of dysphoric emotions that individuals with eating pathology typically experience, thereby improving their well-being on physical and psychological levels (Fredrickson, 1998; Fredrickson et al., 2000; Fredrickson and Losada, 2005; Fredrickson and Levenson, 1998).

The results show that mood induction did not influence Negative Affect Intensity scores, which suggest that it may be a stable personal characteristic. Mood had a significant impact on Global and Positive Affect Intensity, which may mean that these constructs are less robust. The results partly support Larsen’s (1987) findings on stability of Affect Intensity, as well as those of Heide and Gronhaug (1996) who found that emotion intensity was susceptible to mood manipulation. This means that a tendency to experience negative emotions profoundly may be a consistent dispositional factor, whereas general affective responsiveness (Global AI) and the tendency to experience positive emotions (Positive AI) intensity may be amenable to change.

In summary, happy mood led to a reduction in dysfunctional emotion regulation strategies (both internal and external) and an increase in functional affect management (both internal and external) in both subclinically eating-disordered group and healthy controls. Sadness led to a significant increase in internal regulatory strategies (both functional and dysfunctional). Neutral mood was associated with higher internal functional emotion regulation and lower external dysfunctional affect management. Mood had no effect on negative Affect Intensity. Perhaps, the most interesting results
are those on the effects of happiness on emotion regulation, and subsequent research in this thesis focused on positive affect. Studies 3, 4 and 5 further look at the role of positive emotions in affect regulation, and examine the effects of increasing the frequency of positive emotions on affect regulation in subclinical samples (Studies 3 and 4), and in a longitudinal clinical study (Study 5).

**Relationship between Emotion Regulation and Affect Intensity**

The study is the first of its kind in exploring the relationship between emotion regulation and intensity of affect in a sample with eating pathology and healthy controls. It was found that high Global Affect Intensity, Positive Affect Intensity and Negative Affect Intensity were associated with dysfunctional emotion regulation strategies (either internal, or external, or both). This suggests that those who have a dispositional tendency to experience profound affect (in particular, negative) are likely to manage their emotions in unhealthy ways (e.g. rumination, bullying etc.). Following this line of thought, it may mean that the tendency to experience intense emotions may be incompatible with healthy affect regulation. Previous studies suggest that the three types of Affect Intensity are strongly related (Diener et al., 1985a); the person who tends to experience strong negative emotions (e.g. rage), would also feel positive emotions intensely (e.g. euphoria). In other words, they may have an imbalance of affect, where their mood may fluctuate between extreme positivity and extreme negativity. Extreme mood fluctuations may be unconducive to healthy emotion regulation because strong affect presumably overwhelms the person, and makes it more difficult for them to consider their options. When it comes to affect management, healthy emotion regulation strategies (e.g. positive reappraisal) may not be automatic responses to affect, they may require conscious cognitive processing of all possible emotion management options and selection of the ones that are best suited to a given situation. Functional regulatory strategies may draw heavily on the individual’s resources (e.g. cognitive). On the contrary, the dysfunctional regulatory strategies (e.g. negative social comparison) may be automatic and easy to carry out, not requiring such considerable effort. In this case, the tendency to experience intense affect may interfere with the cognitive processes that may be involved in regulatory strategies selection,
such as reasoning and memory, resulting in the choice of the least resource-demanding strategies, i.e. the dysfunctional ones.

Out of the three, intensity of negative affect was associated with the worst affect regulation outcomes: high Negative Affect Intensity was linked with dysfunctional emotion regulation (internal and external), as well as low external functional regulation. This means that a person dispositionally high in Negative Affect Intensity is more likely to use such strategies as self-harm, repression and assault as a means of down-regulating their emotions, and less likely to ask others for help and express their feelings. Being high in Global Affect Intensity is slightly better because it does not seem to affect functional emotion regulation. Those with high global Affect Intensity would tend to use dysfunctional emotion regulation strategies, such as self-harm and assault, but it appears that this does not make them less likely to choose healthy strategies. The final finding was that positive Affect Intensity was associated with external regulatory styles (functional and dysfunctional). In one way, this is not surprising, considering the properties of positive emotions – they encourage approach behaviours (Izard, 1991); therefore it is logical that the individual who is high in positive Affect Intensity would turn outwards when managing their emotions. However, it is interesting that high positive Affect Intensity was associated with dysfunctional emotion regulation (e.g. bullying and assault). These are approach behaviours, but they are negative. It is likely that this finding refers to the general imbalance in emotions, of which positive Affect Intensity is a feature. This suggests that extremely intense emotions, be they negative or positive, may contribute to poor emotion regulation. This is true for both subclinically eating-disordered individuals and healthy controls.

**Eating Pathology, Affect Intensity and Emotion Regulation**

Taken together, Study 1 findings suggest that individuals with eating pathology have a tendency to experience negative affect intensely (and frequently, as the literature suggests, Johnson and Larson, 1982; Leon et al., 1993), and this may interfere with their emotion regulation (Figure 13). Generally, the tendency to experience extreme positive and negative emotions was associated with dysfunctional emotion regulation, and may point to an imbalance in mood which may be unconducive to healthy affect.
management choices. It was found that individuals with eating pathology tend to use internally dysfunctional affect regulation strategies (e.g. self-harm, rumination) significantly more than controls. It is suggested that these strategies are chosen because they may be less demanding of cognitive resources than functional regulatory methods. The final finding was that happiness induction led to a significant improvement in emotion regulation in subclinically eating-disordered sample and controls. This suggests that positive emotions - via their effect on emotion regulation - may potentially contribute to remission of eating pathology; therefore, it may be important to study positive affect in individuals with eating disorders. Study 1 findings may have important implications for theory and treatment of eating disorders, if they are replicated with a clinical sample. The implications for theory would consist in the fact that negative affect – in particular, its intensity and frequency – may need to be included in aetiological models of eating disorders. The implications for treatment would be in higher priority given to addressing negative and positive affect when treating eating disorders therapeutically.

Figure 13: The relationship between intensity and frequency of negative affect, dysfunctional emotion regulation and eating pathology.

It is plausible that negative affect of high intensity and frequency promotes dysfunctional emotion regulation strategies, which in turn encourage disordered eating. Eating pathology, in its turn, may create negative emotions, and the individual with eating pathology may be caught in this cycle. Substituting negative affect with positive
affect – which was found to improve emotion regulation – may lead to the disruption of this cycle (Figure 14). Increasing the frequency of positive affect may reverse the effects of negative emotions, and benefit emotion regulation, making it more functional. This in turn may lead to a remission in disordered eating.

Figure 14: The relationship between positive affect, functional emotion regulation and eating pathology.

These hypotheses are further investigated in Studies 3, 4, and 5, and the implications are discussed in detail in the relevant chapters.

Methodological improvements in subsequent studies (Studies 3, 4 and 5)
In Study 1 a combination of music and the Velten Mood Induction Procedure (Velten, 1968) was used to manipulate mood. This method is rather complex, because it required supervision and extensive instructions on how to use the Microsoft Power Point presentations, specially designed for mood induction. It may be unfeasible to use this mood manipulation method longitudinally, especially if the presence of the researcher cannot be guaranteed (e.g. in long distance studies) and if the participant has no access to a computer. For such studies emotion induction methods have to be simplified, so that they can be easily adapted for longitudinal and/or postal surveys. Because the subsequent studies will focus on positive emotions, alternative positive affect induction methods were researched; in particular, Positive Psychology Interventions (PPIs) such as a gratitude task and happy memories exercise. These do not require supervision or
any technical equipment, are simple to carry out, and can be done at participants’ convenience. In subsequent studies (Studies 3, 4 and 5) neutral mood was used as a control condition, as it is the author’s belief that inducing sadness in subclinically and clinically eating-disordered participants may be unethical.

Another methodological improvement concerns the eating pathology measure. EAT-26 (Garner et al., 1982) was chosen in Study 1 because it was a straightforward test that gave a single score on eating pathology. This is a relatively simple, general measure of eating pathology that was well-suited for the explorative nature of the study; however, it would not be sophisticated enough for clinical purposes (Rivas et al., 2010). Because the study’s hypotheses were largely confirmed and had to be tested with a clinical sample, there was a need for a more sensitive eating disorder measure that had better clinical validity. In subsequent studies EAT-26 (Garner et al., 1982) was changed for the Eating Disorder Examination Questionnaire (EDE-Q; Fairburn and Beglin, 1994). A study by Engelsen and Laberg (2001) found that EDE-Q (Fairburn and Beglin, 1994) was more sensitive to identifying eating disorders than the short version of the Eating Attitudes Test (EAT-12, Garner et al., 1982). The EDE-Q consists of four subscales (Restraint, Eating Concern, Shape Concern and Weight Concern) that can be summed to obtain a composite eating pathology score; this scale was shown to have good reliability and validity in clinical and normal populations (Fairburn and Beglin, 1994; Mond et al., 2004; Peterson et al., 2007). The next study on emotion regulation in eating disorders (Study 3) tested alternative positive mood induction methods (e.g. the gratitude task) and their effect on emotion regulation in subclinically eating-disordered females. Participants were screened for eating pathology using EDE-Q (Fairburn and Beglin, 1994). Then the procedure was replicated with a clinical sample – in a one-off emotion induction study (Study 4), and a longitudinal study (Study 5). Emotion regulation, Affect Intensity and positive emotions in females with eating pathology are the subject of this thesis.
CHAPTER 5. Study 2: Emotion Regulation, Affect Intensity, Sub-Clinical Eating Pathology and Creativity in a student sample.

INTRODUCTION

Study 2 took place simultaneously with Study 1, and the research aims were to look at creativity in general psychopathology (i.e. disordered eating, anxiety and depression), as well as to examine the relationship between creativity and Affect Intensity (global, positive and negative). The Creativity section in Chapter 3 discussed the background to the study in more detail. Creativity can be broadly defined as ‘the ability to produce work that is both novel and appropriate’ (Sternberg and Lubart, 1999). It is a construct that is difficult to operationalize, and there is a lot of disagreement on its definitions and measurement strategies. Creativity has been defined and measured as a thinking style; other researchers conceptualized creativity as an unconventional and original approach to a problem in an experimental setting i.e. divergent thinking (e.g. the Alternative Uses Task; Guilford, 1950; Silvia et al., 2008). Alternatively, it is often assessed in terms of a history of achievements in creative spheres, when a participant is asked to fill in an inventory listing their creative accomplishments (e.g. Creative Behaviour Inventory, Hocevar, 1980). The divergent thinking and the autobiographical approaches appear to complement each another, because they cover different aspects of creativity; the Alternative Uses Task (Guillford, 1950) is an on-line creativity test that allows to estimate creative abilities at present and the Creative Behaviour Inventory (Hocevar, 1980) is a comprehensive list of achievements in different creative domains, relating to the past. Therefore, in order to get as complete assessment of creativity as possible, it would make sense to combine the two methods in one study.

Creativity and psychopathology (anxiety, depression and eating disorders).

There appears to be a relationship between creative tendencies and poor mental health. Creative individuals tend to suffer from affect disorders, anxiety, substance abuse and psychoses (Jamison, 1993; Martindale, 1999; Post, 1994; Prentky, 1989). The direction of this relationship is at present unclear. Creative activities may be conducive to mental illness, or alternatively, that those who are suffering from poor mental health, tend to achieve more in creative domains. This raises interesting questions about the possibility of psychopathological origins and outcomes of creativity. It is unclear what exactly it is
about creativity that affects mental health, and whether this is specific to certain creative pursuits (e.g. music as opposed to science). It may be worthwhile to look at clinical measures of emotional disorder (e.g. anxiety and depression) in relation to a wide range of creative behaviours (e.g. in different spheres, as well as relating to the past and present), in order to explore this relationship further. The studies that have been done before tended to use their preferred method to measure creativity; however, a combination of methods may give a more complete picture when it comes to such a contested construct as creativity.

To the author’s knowledge, there has been no research explicitly looking at creativity in relation to sub-clinical eating pathology. There appear to be some interesting similarities in the emotional and cognitive profiles of those who score highly on creativity and those with eating disorders. Firstly, both creative and eating-disordered individuals tend to score higher on psychopathology measures (e.g. anxiety; Dowd, 1989; Martindale, 1999; Touchette et al., 2010) as well as on dispositional characteristics, such as neuroticism (Dowd, 1989; Gordon, 1990) and unstable identity (Polivy and Herman, 2002). Secondly, both creative and eating-disordered individuals tend to self-medicate with the use (or misuse) of substances, e.g. food, alcohol or drugs (Kerr, 2009; Martindale, 1989, Bruch, 1978). Thirdly, those with eating pathology show creativity in their approach to food and weight management. For instance, they often develop highly complex and inventive methods of weight loss (Bruch, 1978). Fourthly, evidence from art therapy studies shows that eating-disordered clients often produce highly complex and original work in drawing and painting (Crowl, 1994; Crisp, 1980; Dokter, 1995; Luzzatto, 1980; Waller and Gilroy, 2000) when they are given a task to express their feelings on paper.

This therapeutic approach does not require skills of verbal reflection on emotions and events, and allows free flow and symbolic expression of feelings, which allows them to rise to the surface and reach awareness (Dokter, 1995). Individuals with eating pathology often have difficulties recognizing, distinguishing and expressing their emotions in words (i.e. alexithymia, Cochrane et al., 1993; De Zwaan et al., 1995, Schmidt et al., 1993), and instead may use their body and eating to signal to others that
they are in distress (Gerhardt, 2009). In art therapy they are given the chance to learn to externalize and communicate their emotions in the way that is familiar to them - i.e. by manipulating objects - but the means of self-expression are no longer their body and food, but paper and painting supplies. If eating pathology is conducive to creativity in this way, it may mean that more effort should be made to incorporate art therapy elements that foster creativity into treatment of eating disorders. However, it is also possible that eating pathology would be an obstacle to creativity. Alexithymia was found to correlate with low expressiveness, as well as lack of originality and imagination (Hoppe and Kyle, 1990), all of which are incompatible with creative accomplishments (Kerr, 2009). Therefore, it is plausible that lack of emotional language in eating disorders - dispositional or resulting from malnutrition - would present a hindrance to creative self-expression; this means that individuals with eating pathology would be less creative than healthy controls.

**Creativity, Affect Intensity and Emotion Regulation.**

Prior to the present research there had been no studies, looking at how creative individuals regulate their emotions and how intense and variable their affect is. To the author’s knowledge there have not been any studies examining the relationship between emotion regulation and creativity. However, there is some indirect evidence suggesting that there may be potential for interesting findings. In particular, creativity may be associated with dysfunctional emotion management. Disinhibition traits such as impulsivity and sensation-seeking (Martindale, 1989; Sternberg, 1999) tend to be associated with creativity, which may explain why the rates of substance abuse are higher in creative population (Martindale, 1989). The lack of control at emotional and behavioural level that often accompanies creative expression suggests that creativity may be negatively related to functionality of emotion regulation. In other words, people who are more creative may resort to less adaptive strategies when trying to manage their emotions. In the context of Phillips and Power’s (2007) model, this would mean that creativity in some or all of the domains would be associated with self-harm, rumination, negative social comparison, repression and de-realization (Internal Dysfunctional strategies) as well as bullying, verbal/physical assault, lashing out at objects and making others feel bad (External Dysfunctional strategies).
Creativity and emotion regulation is a chicken-and-egg issue. It is unclear whether innate creativity may drive people to choose certain emotion regulation strategies (e.g. self-harm), or whether creativity itself is an emotion regulation strategy (e.g. a type of distraction). It is plausible that individuals are compelled to engage in creative activity in order to decrease dysphoric mood and to obtain relief from negative emotions by giving them a physical expression (e.g. on canvas, as in painting). This makes creativity relevant to the affect intensity concept (Larsen, 1984): if one finds that creativity is associated with intense emotions, it may explain why creative individuals may turn to psychopharmacological means (e.g. drugs) of affect management. This could represent an attempt to regulate emotions on a biochemical level. If the positive or negative affect is strong enough to be overwhelming, then constructive, healthy emotion regulation strategies (e.g. positive reappraisal or advice seeking) may not be fast-acting or potent enough to restore emotional balance. Therefore an individual may opt for maladaptive strategies (e.g. verbal assault or self-harm) which may require less cognitive and emotional effort. This suggests that people higher in creativity may show dysfunctionality in their emotion regulation. Study 2 tested this assumption among several others, and looked at the relationship between creativity, psychopathology (anxiety, depression and eating disorders), intensity of affect and emotion regulation.

The present exploratory study put creativity in the context of emotions and psychopathology, and looked at creativity on a divergent thinking task (Alternative Uses Task; Guilford, 1950) and six different domains (Literature, Fine Arts, Crafts, Music, Performing Arts, Maths and Science; Creative Behaviour Inventory, Hocevar, 1980) in relation to Affect Intensity (Larsen, 1984), emotion regulation, and psychopathology (anxiety, depression and eating pathology). The following hypotheses were put forward:

1. Creativity (on-line and in the 6 domains) would be associated with higher levels of anxiety and depression;
2. There would be a relationship between creativity (on-line and in the 6 domains) and sub-clinical eating pathology;
3. Creativity (on-line and in the 6 domains) would be associated with dysfunctional emotion regulation (i.e. high Internal/External Dysfunctional and low
Internal/External Functional ER) and high Affect Intensity (Global, Positive and Negative AI).

METHOD

Participants and Recruitment
A hundred University of Edinburgh students who participated in Study 1 were invited to take part in Study 2. The sample (mean age = 20.5 years) consisted of ninety seven of them who agreed to participate. Participants were screened for eating pathology, using EAT-26 (Garner et al., 1982), and the mean eating pathology score in the sample was 10.89 (SD = 11.17). The participants’ age ranged from 18 to 33 (mean = 20.5, SD = 2.53). All were female and Caucasian. The participants were full-time undergraduate students at the University of Edinburgh. Being a native English speaker was the main inclusion criterion for the study and all the participants were from English-speaking countries (United Kingdom: N= 94, United States: N = 3).

The means for creativity scores in the sample were calculated. On average participants scored 4.70 on on-line creativity (SD = 1.95), 10.12 on creative accomplishments in Literature (SD = 6.21), 10.08 on creativity in Music (SD = 7.50), 21.21 on creative achievements in Crafts (SD = 9.10), 10.65 on creativity in Performing Arts (SD = 6.67), 2.51 on creative accomplishments in Maths and Science (SD = 2.58), and 9.13 on creativity in Fine Arts (SD = 5.15). Participants’ mean scores on the emotion regulation measure (REQ, Phillips and Power, 2007) and affect intensity scale (AIM, Larsen, 1984) were computed. Participants scored 7.31 (SD = 3.56) on Internal Dysfunctional emotion regulation, 7.19 (SD = 2.02) on Internal Functional emotion regulation, 2.90 on External Dysfunctional emotion regulation (SD = 2.23), and 14. 52 on External Functional emotion regulation (SD = 3.63). Participants scored on average 3.73 on Global Affect Intensity (SD = .46), 3.72 on Positive Affect Intensity (SD = .54) and 3.80 on Negative Affect Intensity (SD = .59). Participants’ mean scores on anxiety and depression measure (HADS, Zigmond and Snaith, 1983) were: 8.84 on anxiety (SD = 4.06) and 3.57 (SD = 2.68) on depression.
Ethics
Study 2 received ethical approval from the School of Psychology, Philosophy and Language Sciences Ethics Committee (University of Edinburgh). Participants were told that the main research aims concerned creativity and emotions. They were informed of their right to withdraw at any time without stating a reason, and assured that all of their responses would be kept anonymous and confidential in order to protect their privacy. At the end of the session, the participants were fully debriefed on the purposes of the study.

Design
Study 2 was a correlational study that aimed to explore associations between creativity, depression, anxiety, affect intensity, emotion regulation and eating pathology. The data were collected from one sample at one point in time, and there were no experimental and control groups.

Procedure
The study used the data on eating pathology, emotion regulation and Affect Intensity, collected in Study 1. In addition, participants were asked to complete two different creativity tasks (Creative Behaviour Inventory, Hocevar, 1980; Alternative Uses Task; Guilford, 1950) and a depression and anxiety measure (Hospital Anxiety and Depression Scale; Zigmond and Snaith, 1983). The participants who took part in Study 1 were invited to take part in Study 2. They were told that the creativity experiment would take approximately 1 hour, and that they would be paid £6 for their contribution. Participants were asked to indicate their availability over the subsequent two weeks, and were allocated into 1 ½ hour time slots with 10 participants per slot. A large classroom in the School of Psychology building was booked for this purpose. The sessions ran from 9am till 6pm on weekdays over the course of 2 weeks. The creativity part of the study was timed and the participants were given 15 minutes to complete the Alternative Uses Task (Guilford, 1950). The remainder of the time (approximately 45 minutes) they spent completing the questionnaires.
**Measures**

The following questionnaires and scales were used:

1. The Eating Attitudes Test (EAT-26; Garner et al., 1982) assesses the risk of eating pathology and is commonly used as a screening tool. It consists of 26 items that refer to the past 6 months. EAT-26 (Garner et al., 1982) has three subscales: Dieting, Bulimia and Food Preoccupation and Oral Control. The composite score of 20 or over on the test may indicate an eating disorder. [Appendix 5]

2. Affect Intensity Measure Questionnaire (Larsen, 1984) assesses typical depth of emotion processing and measures global, positive and negative emotions intensity. Items are phrased so as to highlight the intensity aspect of emotions. The scale consists of 40 statements (e.g. ‘When I accomplish something difficult, I feel delighted or elated’), and the respondent has to rate the frequency of each statement from *Never* to *Always*. [Appendix 2]

3. Regulation of Emotion Questionnaire (REQ, Phillips and Power, 2007) measures functionality of affect management along four subscales (Internal Dysfunctional/Functional and External Dysfunctional/Functional emotion regulation). It is a 21-item questionnaire with statements about different emotion regulation strategies like ‘I ask others for advice’ with a frequency scale from *Never* to *Always*. [Appendix 1]

4. The Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983) measures depression and anxiety. It consists of 14 statements (e.g. ‘I feel tense or wound up’) with 4 answer options (e.g. from *Most of the time* to *Not at all*). [Appendix 7]

5. Creative Behaviour Inventory (Hocevar, 1980) is a 90-item creativity measure, and it is used to assess personal history of creative behaviours and achievements in Fine Arts, Maths/Science, Performing Arts, Music, Literature and Crafts. The
respondent is asked to report on how many occasions they have carried out different creative activities, e.g. ‘Painted an original picture’. [Appendix 10]

6. The Alternative Uses Task (Guilford, 1950) is an on-line measure of creativity. It is a task measuring divergent thinking. The respondent is asked to name as many possible uses of a common object (e.g. a shoe) as possible. Responses can be scored in a number of ways, e.g. in terms of originality, novelty or uniqueness. In the current study, the snapshot scoring method (Silvia, 2009) was used to assess creativity of responses. Each set of responses was scored on creativity by two independent raters on a 1 (not creative) to 5 (very creative) point scale. The average of the set scores, given by the two raters, was recorded as an index of creativity.

RESULTS

Data screening procedures were performed. The data were checked for accuracy by computing frequencies and minimum/maximum statistics for the variables using Statistical Package for the Social Sciences (SPSS). Visual checking of the resultant analyses against the questionnaire data ensured that there were no errors in the data file, and all the scores fell within the range of possible scores for the scales. Skewness, kurtosis and z-scores were also computed for each dependent variable (please see Appendix 14). All z-scores fell below 3.3, which indicated reasonably good normal distribution. Visual checking of histograms and scatterplots confirmed that the scores on the dependent variables were reasonably normally distributed, the relationships between them were linear and the data tended to have similar variability.

Creativity, Emotional Disorder (anxiety and depression) and Affect Intensity.

Pearson correlation was used to explore the relationship between Anxiety, Depression, Affect Intensity, on-line creativity and 6 Creativity domains (Table 14).
Statistically Significant Correlations ($p < 0.05$)

Literature scores were positively correlated with depression ($r = .25, N = 97, p < .05$) as well as anxiety ($r = .26, N = 97, p < .05$). Fine Arts scores were positively correlated with anxiety ($r = .21, N = 100, p < .05$) and depression ($r = .23, N = 97, p < .05$).

Table 14. Correlations among Depression, Anxiety, Affect Intensity and Creativity

<table>
<thead>
<tr>
<th></th>
<th>On-line Creativity</th>
<th>Literature</th>
<th>Music</th>
<th>Crafts</th>
<th>Performing Arts</th>
<th>Maths/Science</th>
<th>Fine Arts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>.16</td>
<td>.25*</td>
<td>.50</td>
<td>.14</td>
<td>.3</td>
<td>-.08</td>
<td>.23*</td>
</tr>
<tr>
<td>Anxiety</td>
<td>.03</td>
<td>.26*</td>
<td>-.12</td>
<td>.08</td>
<td>.06</td>
<td>0</td>
<td>.21*</td>
</tr>
<tr>
<td>Global AI</td>
<td>.04</td>
<td>-.81</td>
<td>.12</td>
<td>-.09</td>
<td>.01</td>
<td>.15</td>
<td>.12</td>
</tr>
<tr>
<td>Positive AI</td>
<td>-.01</td>
<td>-.01</td>
<td>.07</td>
<td>.15</td>
<td>.17</td>
<td>.04</td>
<td>.08</td>
</tr>
<tr>
<td>Negative AI</td>
<td>.08</td>
<td>.12</td>
<td>.09</td>
<td>.01</td>
<td>-.05</td>
<td>-.12</td>
<td>.09</td>
</tr>
</tbody>
</table>

* significance at .05 level
AI = Affect Intensity

Non-Significant Correlations ($p > 0.05$).

None of the following correlations reached statistical significance. On-line Creativity was positively correlated with Depression ($r = 0.16, N = 97, p > 0.05$), Anxiety ($r = .03, N = 97, p > 0.05$), Global Affect Intensity ($r = 0.04, N = 97, p > 0.05$) as well as Negative Affect Intensity ($r = .08, N = 97, p > 0.05$), and negatively with Positive Affect Intensity ($r = -0.1, N = 97, p > 0.05$). Creativity in Literature was negatively associated with Global Affect Intensity ($r = -.81, N = 97, p > 0.05$) as well as Positive Affect Intensity ($r = -.01, N = 97, p > 0.05$), and positively with Negative Affect Intensity ($r = .12, N = 97, p > 0.05$). Creativity in Music was positively correlated with Depression ($r = 0.5, N = 97, p > 0.05$), negatively with Anxiety ($r = -.12, N = 97, p > 0.05$), positively with Global Affect Intensity ($r = 0.12, N = 97, p > 0.05$) as well as Negative Affect Intensity ($r = .09, N = 97, p > 0.05$), and Positive Affect Intensity ($r = 0.07, N = 97, p > 0.05$). Creativity in Crafts was positively correlated with Depression ($r = 0.14, N = 97, p > 0.05$), Anxiety ($r = .08, N = 97, p > 0.05$), negatively with Global Affect Intensity ($r = -.0.09, N = 97, p > 0.05$) and positively with Negative Affect Intensity ($r = .15, N = 97, p > 0.05$), and Positive Affect Intensity ($r = 0.01, N = 97, p >
Creativity in Performing Arts was positively correlated with Depression ($r = .3$, $N = 97$, $p > 0.05$), Anxiety ($r = .06$, $N = 97$, $p > 0.05$), Global AI ($r = .01$, $N = 97$, $p > 0.05$), as well as Positive Affect Intensity ($r = .17$, $N = 97$, $p > 0.05$), and negatively with Negative Affect Intensity ($r = -0.05$, $N = 97$, $p > 0.05$). Creativity in Maths and Science was negatively associated with Depression ($r = -.08$, $N = 97$, $p > 0.05$) as well as Negative Affect Intensity ($r = -.12$, $N = 97$, $p > 0.05$), and positively with Anxiety ($r = 0$, $N = 97$, $p > 0.05$), Global Affect Intensity ($r = .15$, $N = 97$, $p > 0.05$), and Positive Affect Intensity ($r = .04$, $N = 97$, $p > 0.05$). Creativity in Fine Arts was positively correlated with Global Affect Intensity ($r = .12$, $N = 97$, $p > 0.05$), Positive Affect Intensity ($r = .08$, $N = 97$, $p > 0.05$) and Negative Affect Intensity ($r = .09$, $N = 97$, $p > 0.05$). In summary, Creativity in Crafts, Performing Arts, Music and Maths/Science was not significantly associated with Affect Intensity, depression and anxiety. The same was true for on-line creativity. However, creativity in Literature and Fine Arts was significantly associated with depression and anxiety.

Creativity and Emotion Regulation

Pearson correlation was used to explore the relationship between Emotion Regulation, on-line creativity and the 6 Creativity domains (Table 15). Preliminary analyses were performed to ensure no violation of the assumptions of normality, linearity and homoscedasticity.

**Statistically Significant Correlations ($p < 0.05$)**

**Literature** scores were positively correlated with **Internal Dysfunctional** ER ($r = .33$, $N = 97$, $p < .05$) and negatively with **External Functional** ER ($r = -.27$, $N = 97$, $p < .05$). **Fine Arts** scores were positively correlated with **Internal Dysfunctional** ER ($r = .25$, $N = 97$, $p < .05$), and **Internal Functional** ER ($r = .23$, $N = 97$, $p < .05$).
Table 15: Correlations among Emotion Regulation facets and Creativity

<table>
<thead>
<tr>
<th></th>
<th>On-line Creativity</th>
<th>Literature</th>
<th>Music</th>
<th>Crafts</th>
<th>Performing Arts</th>
<th>Maths/Science</th>
<th>Fine Arts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internal Dysfunctional ER</td>
<td>.07</td>
<td>.33*</td>
<td>-.02</td>
<td>.15</td>
<td>.06</td>
<td>-.3</td>
<td>.33*</td>
</tr>
<tr>
<td>Internal Functional ER</td>
<td>-.06</td>
<td>.17</td>
<td>0</td>
<td>.11</td>
<td>.12</td>
<td>.11</td>
<td>.23*</td>
</tr>
<tr>
<td>External Dysfunctional ER</td>
<td>-.02</td>
<td>.12</td>
<td>.03</td>
<td>-.01</td>
<td>.09</td>
<td>-.01</td>
<td>.06</td>
</tr>
<tr>
<td>External Functional ER</td>
<td>.31</td>
<td>-.27*</td>
<td>-.1</td>
<td>.09</td>
<td>.13</td>
<td>.16</td>
<td>-.04</td>
</tr>
</tbody>
</table>

* Significance at .05 level
ER = Emotion regulation

Non-Significant Correlations (p > 0.05)
None of the following correlations reached statistical significance. On-line Creativity was not associated with Internal Dysfunctional ER (r = .07, N = 97, p > 0.05), External Functional ER (r = .31, N = 97, p > 0.05), Internal Functional ER (r = -.06, N = 97, p > 0.05) and External Dysfunctional ER (r = -.02, N = 97, p > 0.05). Creativity in Literature did not correlate with Internal Functional ER (r = .17, N = 97, p > 0.05) and External Dysfunctional ER (r = .12, N = 97, p > 0.05). Creativity in Music was not associated with Internal Dysfunctional ER (r = -.02, N = 97, p > 0.05), External Functional ER (r = -.1, N = 97, p > 0.05), Internal Functional ER (r = 0, N = 97, p > 0.05) and External Dysfunctional ER (r = .03, N = 97, p > 0.05). Creativity in Crafts was not associated with Internal Dysfunctional ER (r = .15, N = 97, p > 0.05), Internal Functional ER (r = .11, N = 97, p > 0.05), External Functional ER (r = .09, N = 97, p > 0.05), and External Dysfunctional ER (r = -.01, N = 97, p > 0.05). Creativity in Performing Arts was not associated with Internal Dysfunctional ER (r = .06, N = 97, p > 0.05), Internal Functional ER (r = .12, N = 97, p > 0.05), External Functional ER (r =
.09, N = 97, p > 0.05), and External Dysfunctional ER (r = 0.13, N = 97, p > 0.05). Creativity in Maths and Science did not correlate with Internal Functional ER (r = .11, N = 97, p > 0.05), External Functional ER (r = .16, N = 97, p > 0.05), Internal Dysfunctional ER (r = -.3, N = 97, p > 0.05) and External Dysfunctional ER (r = -.01, N = 97, p > 0.05). Creativity in Fine Arts was not associated with External Dysfunctional ER (r = .06, N = 97, p > 0.05) and External Functional ER (r = -.04, N = 97, p > 0.05).

In summary, Creativity in Crafts, Performing Arts, Music and Maths/Science was not significantly associated with emotion regulation strategies. The same was true for on-line creativity. However, creativity in Literature was positively associated with Internal Dysfunctional emotion regulation and negatively with External Functional emotion regulation; whereas creativity in Fine Arts was related to Internal Functional and Internal Dysfunctional emotion regulation.

Creativity and subclinical eating pathology.

Pearson correlation was used to explore the relationship between eating pathology, on-line creativity and the 6 Creativity domains (Table 16). Preliminary analyses were performed to ensure no violation of the assumptions of normality, linearity and homoscedasticity. There were no significant correlations between creativity (on-line and the 6 dimensions) and eating pathology; none of the following correlations reached statistical significance. Eating Pathology was positively correlated with On-line Creativity (r = .05, N = 97, p > 0.05), Creativity in Music (r = .02, N = 97, p > 0.05), Creativity in Crafts (r = .07, N = 97, p > 0.05), Creativity in Performing Arts (r = .11, N = 97, p > 0.05), Creativity in Maths and Science (r = .11, N = 97, p > 0.05) and Creativity in Fine Arts (r = .11, N = 97, p > 0.05). Eating pathology was negatively correlated with Creativity in Literature (r = -.02, N = 97, p > 0.05).

Table 16: Correlations between Creativity and Sub-Clinical Eating Pathology

<table>
<thead>
<tr>
<th>Eating Pathology</th>
<th>On-line Creativity</th>
<th>Literature</th>
<th>Music</th>
<th>Crafts</th>
<th>Performing Arts</th>
<th>Maths/Science</th>
<th>Fine Arts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eating Pathology</td>
<td>r</td>
<td>.05</td>
<td>-.02</td>
<td>.02</td>
<td>.07</td>
<td>.11</td>
<td>.11</td>
</tr>
<tr>
<td>Sig.</td>
<td>.64</td>
<td>.89</td>
<td>.84</td>
<td>.49</td>
<td>.30</td>
<td>.30</td>
<td>.28</td>
</tr>
</tbody>
</table>

r = Pearson correlation coefficient
Sig. = Significance
Main findings

1. Literature scores were positively correlated with Internal Dysfunctional emotion regulation (ER), sub-clinical depression, anxiety and negatively with External Functional ER;
2. Fine Arts scores were positively correlated with Internal ER (functional and dysfunctional), as well as sub-clinical anxiety and depression;
3. Online creativity as well as creativity in Music, Crafts, Performing Arts and Maths/Science were not associated with any negative outcomes (i.e. sub-clinical depression, anxiety, emotion dysregulation, high affect intensity and eating pathology).
4. There were no significant correlations between creativity and eating pathology, and between creativity and Affect Intensity (Global, Negative and Positive AI).

DISCUSSION

Creativity and Sub-Clinical Psychopathology

The results show that out of all the creativity domains only creativity in Literature and Fine Arts were associated with emotional disorder (sub-clinical anxiety and depression). These findings support those of Post (1994), who found high prevalence of different types of psychopathology (e.g. depression) in creative populations. Creativity on the divergent thinking test (Alternative Uses Task, Guilford, 1950), as well as personal history of creative accomplishments in Music, Crafts, Performing Arts, Maths and Science were not associated with those types of psychopathology.

These findings are interesting because they suggest that there may be a qualitative difference between the types of creativity across different domains (e.g. Music versus Literature). It may be that individuals who are anxious and depressed excel in certain creative activities (e.g. Fine Arts), as opposed to others (e.g. Maths and Science). Furthermore, it is plausible that a certain degree of emotional dysfunction and/or a psychological crisis (e.g. sub-clinical depression) are necessary for the
literary and artistic creativity to emerge. For instance, the impulse to write or to paint may arise in the context of dysphoric emotions, and creative activity may be used as a means of down-regulating negative affect, expression and working through feelings. Alternatively, it is conceivable that individuals who engage in creative activities in the literature and fine arts domains are more depressed and anxious, because these activities may contribute to their symptomatology. Literature and fine arts are largely solitary activities, unlike performing arts, and therefore they may contribute to social isolation. Social withdrawal may trigger and perpetuate depression and anxiety symptoms in susceptible individuals. However, because this was a correlational study, it is not possible to say with confidence where the direction of the effect lay. In future studies it may be worthwhile exploring qualitative differences between individuals, who are creative in different domains, as a means of establishing the exact nature of the relationship between creativity and psychopathology.

There were no significant associations between creativity (on-line and in the 6 domains) and eating pathology. This suggests that eating pathology is not related to creative accomplishments, and that individuals who may be creative in weight management, are not as creative in other domains. However, because there were no negative associations between creativity and eating pathology, this means that disordered eating and its psychopathological concomitants (e.g. alexithymia, dysphoria and anxiety) are not obstacles to creative accomplishments in Music, Performing Arts, Crafts, Fine Arts, Literature, Maths and Science. The finding that psychopathology may not be a hindrance to creativity is interesting, and in future studies it may be worthwhile to look at symptoms such as alexithymia and dysphoria in the context of creativity. It may be that creativity in certain domains (e.g. Literature and Fine Arts) is enhanced by psychopathologic tendencies (e.g. depression).
Study 2 showed that there were no significant associations between creativity (online and in the 6 domains) and Affect Intensity (Positive, Negative and Global AI). In other words, compared to their less creative counterparts, creative individuals may not experience positive and negative emotions of higher intensity and their affect may not be more variable. This means that intensity and changeability of affect may not be implicated in creative behaviours. There were some interesting findings with regard to emotion regulation. Study 2 showed that creativity in Literature and Fine Arts was associated with higher Internal Dysfunctional emotion regulation tendencies. This means that individuals who engage in writing and painting are more likely to use such unhealthy regulatory strategies as self-harm, rumination, negative social comparison, repression and de-realization (Phillips and Power, 2007). In other words, they tend to hurt themselves, dwell on negative events, compare themselves unfavourably with those who are better off, hide their emotions and daydream as a means of managing their affect. It is plausible that some of those strategies may be useful in fostering creativity in Literature and Fine Arts. For instance, daydreaming is a solitary activity that may be conducive to self-expression in writing because it engages imagination. In addition, it was found that those who were creative in literature were significantly less likely to use External Functional strategies, such as sharing feelings with others, advice and contact seeking, distraction and exercise (Phillips and Power, 2007). Therefore, it is conceivable that literary-gifted individuals prefer to work through their feelings on their own, perhaps with the use of writing, as opposed to reaching out to others for help.

Another interesting finding was that creativity in Fine Arts was associated with Internal Functional emotion regulation. This means that those who are gifted in fine arts, are more likely to use such healthy strategies as positive reappraisal, modification of goals, planning, perspective and concentration on a different activity (Phillips and Power, 2007). In other words, such individuals may address their
feelings on their own, in an adaptive manner; taken together with the finding on Internal Dysfunctional ER, this is rather puzzling. The finding that creativity in Fine Arts is associated with both Internal Functional and Internal Dysfunctional emotion regulation, points to the conclusion that individuals who are talented in fine arts, may predominantly manage their emotions internally, rather than externalizing their affect and involving others in its regulation. Therefore, there may be something about this type of creative activity that makes the individual resort to primarily internal strategies when addressing their emotions. It is plausible that for individuals, creative in the fine arts domain, painting and drawing may be the internal emotion regulation strategies that help down-regulate and channel their affect. More research is needed to establish whether that indeed is the case.

Conclusions
As creative accomplishments in Crafts, Music, Performing Arts, Maths and Science, as well as online creativity, were not associated with sub-clinical psychopathology and poor emotion regulation, it may be that negative psychological outcomes in creativity are specific to certain domains i.e. Literature and Fine Arts. Study 2 shows that individuals who are gifted in literature and fine arts are more likely to be sub-clinically anxious and depressed, and to use unhealthy internalizing strategies (e.g. self-harm and rumination) to manage their emotions. Creativity in Literature was found to be associated with the worst emotion regulation and psychopathological outcomes. The findings suggest that creative individuals do not tend to experience highly intense and variable affect, or to suffer from sub-clinical eating pathology, which suggests that the origins of their creativity may lay elsewhere. It may be worthwhile researching creativity in literature and fine arts further in order to understand what makes these domains so psychologically dysfunctional. Despite the interesting findings, it was decided that the thesis should follow the research line of Study 1, and focus on emotion regulation in eating pathology. Therefore, the theme of creativity and psychopathology was set aside at this point.

INTRODUCTION
Study 3 further examined the relationship between eating pathology and emotion regulation, adding a number of other important factors such as anxiety, depression and life satisfaction to the analysis. Several studies had shown that individuals with eating pathology tended to be anxious, depressed and dissatisfied with their lives (Bydlowski et al., 2005; Fox and Harrison, 2008; Heatherton and Baumeister, 1991; Kitzantas et al., 2003; Valois et al., 2003). Study 3 looked at the effects of positive mood on sub-clinical anxiety, depression and life satisfaction, as well as emotion regulation, in a subclinically eating-disordered sample and controls. Positive affect had been shown to reverse the effects of negative emotions on physical and psychological levels (Fredrickson, 1998; Fredrickson et al., 2000; Fredrickson and Losada, 2005; Fredrickson and Levenson, 1998; Howell et al., 2007; Lyubomirsky et al., 2006), and it was interesting to test these findings with depression and anxiety in individuals with eating pathology and controls.

If positive affect influences such aspects of eating disorders as depressed mood, anxiety and emotion regulation, this means that positive mood induction can be potentially used to treat eating pathology. This was discussed in more depth in Chapters 2 and 3. It was also important to assess satisfaction with life in individuals with eating pathology because it is a good indicator of a general level of happiness and could provide a glimpse into how positive emotions are experienced by those with eating pathology. In addition, it was interesting to see whether life satisfaction was implicated in emotion regulation choices. It seemed plausible that an individual who was unhappy with their life, would tend to use dysfunctional methods to regulate their emotions, and that the use of unhealthy emotion management techniques (e.g. self-harm, bullying) would contribute to dissatisfaction with life. Phillips and Power (2007) found that emotion regulation was associated with quality of life, which is a well-being concept related to life satisfaction. They showed that there was a negative association between unhealthy emotion regulation and quality of life; the more dysfunctional the individual’s
regulatory strategies were, the lower quality of life they reported (Phillips and Power, 2007). Therefore, it seemed plausible that the same would be true of life satisfaction. In other words, it was likely that there would be a relationship between emotion regulation and life satisfaction.

**Positive Psychology Interventions (PPIs)**

PPIs, such as the Gratitude exercise and Positive Reminiscence task, are often used as positive mood induction methods in positive psychology studies; this was discussed in more detail in Chapter 3. The evidence shows that PPIs have a therapeutic effect on mental and physical health through evoking positive emotions (Emmons, 2009). In addition, PPIs are reportedly associated with a sustained long-term increase in subjective well-being (Emmons and McCullough, 2004; Lyubomirsky et al., 2006; Seligman et al., 2005; Wiseman, 2009). In particular, expression of gratitude contributes to resilience and may protect against psychiatric disorders, such as depression and anxiety as well as substance abuse (Bono et al., 2004; Emmons, 2007; Emmons and McCullough, 2003; Fredrickson et al., 2003; Kashdan and Breen, 2007; Kendler, 2003; McCullough et al., 2002). Gratitude is a social emotion, and as such is associated with pro-social behaviours and improved social functioning (Bartlett and DeSteno, 2006; McCullough et al., 2008; Tsang, 2006). The Gratitude task makes participants focus on the good in their lives, and through that increases their feelings of well-being (i.e. induces positive affect).

Another effective PPI is Positive Reminiscence, or recollecting happy memories from the past, and it was also found to be associated with a marked increase in subjective well-being (Bryant et al., 2005), self-esteem (Westerhof et al., 2010) and strengthening of social ties (Webster, 1993, 1997). For the purposes of Study 3, the most important property of these two PPIs is their capacity to reliably induce positive affect. In Study 3, the Gratitude exercise and the Positive Reminiscence task were used as a mood manipulation method to elicit positive emotions.

Study 1 (Chapter 4) had found that experimentally-induced happiness led to a reduction in dysfunctional emotion regulation in a sample of subclinically eating-disordered
females and controls. This suggests that positive emotions may improve the choice of emotion regulation strategies. The mood manipulation method used in Study 1 (a combination of music and the Velten Mood Induction procedure) is too complex and laborious for positive affect induction, and PPIs seemed like a good alternative. Positive Psychology Interventions (PPIs) are a popular method of increasing positive affect due to their ease of implementation and apparent effectiveness. Exercises such as writing a gratitude log or recalling happy memories can be easily completed when convenient and do not require neither supervision, nor special equipment (e.g. a computer). This suggested that PPIs could be used for experimental mood induction, especially if implementation over a period of time was required. One of the purposes of the study was to establish how effective in positive mood induction and easy in implementation two popular Positive Psychology Interventions (the Gratitude exercise and the Positive Reminiscence task) were. This information was required in order to methodologically prepare for the studies with clinical samples recruited long-distance (Studies 4 and 5), where the presence of the researcher at the mood induction stage could not be guaranteed.

Other research aims were as follows:

1. To study the effects of PPI-induced positive affect on emotion regulation, subclinical symptoms of anxiety, depression and life satisfaction in a subclinically eating-disordered sample and controls. This study looked at the effects of PPI-induced happiness versus neutral mood on regulation of emotion (Regulation of Emotion Questionnaire; Phillips and Power, 2007), satisfaction with life (Satisfaction with Life Scale; Diener, 1985), as well as anxiety and depression levels (The Hospital Anxiety and Depression Scale, Zigmond and Snaith, 1983), in a subclinically eating-disordered group (SED) and controls.

2. To examine the relationship between sub-clinical eating pathology (as measured by the Eating Disorder Examination Questionnaire; Fairburn and Beglin, 1994), emotion regulation (REQ, Phillips and Power, 2007), life satisfaction (SWLS; Diener, 1985), sub-clinical anxiety and depression (HADS, Zigmond and Snaith, 1983).
3. To examine the level of concordance between two measures of eating pathology: the SCOFF questionnaire (Morgan, 1999) and EDE-Q (Fairburn and Beglin, 1994), in order to establish whether the SCOFF questionnaire (Morgan, 1999) was sensitive enough in detecting eating pathology to be used for screening controls in subsequent studies, instead of the longer and more complex EDE-Q (Fairburn and Beglin, 1994).

METHOD

Participants and Recruitment
In this study the sample consisted of 101 female University of Edinburgh students (mean age = 21.6). Like in previous studies, they were recruited via the University of Edinburgh on-line job database, SAGE. Ninety-eight participants were Caucasian, and three were of Asian ethnicity. Being a native English speaker was the main inclusion criterion for the study and all the participants were from English-speaking countries (United Kingdom: N= 90, United States: N = 5, Australia: N = 3, Hong Kong: N = 3). All the participants were full-time students at the University of Edinburgh (undergraduate N = 80, postgraduate N = 21). Participants’ age ranged from 18 to 35 (mean = 21.6, SD = 3.35).

Participants’ mean scores on the emotion regulation scale (REQ, Phillips and Power, 2007) and life satisfaction measure (SWLS, Diener, 1985) were computed. On average participants scored 7.17 (SD = 2.55) on Internal Dysfunctional emotion regulation, 11.94 (SD = 2.85) on Internal Functional emotion regulation, 2.57 on External Dysfunctional regulation (SD = 1.74), 13.70 on External Functional emotion regulation (SD = 3.46), and 25.27 on Life Satisfaction (SD = 5.76). Participants’ mean scores on anxiety and depression measure (HADS, Zigmond and Snaith, 1983) were: 9.04 on anxiety (SD = 3.33) and 3.56 (SD = 2.50) on depression. The Eating Disorder Examination Questionnaire (EDE-Q; Fairburn and Beglin, 1994) and the SCOFF Questionnaire (Morgan et al., 1999) were used to screen for eating pathology; these instruments are more robust than EAT-26 (Garner et al., 1982) and the EDE-Q
(Fairburn and Beglin, 1994) is typically used as part of the diagnostic process in clinical settings. The mean eating pathology score in the sample, as measured by the EDE-Q (Fairburn and Beglin, 1994) was 1.92 (SD = 1.23); the mean SCOFF questionnaire score (Morgan et al., 1999) was 1.28 (SD = 1.23). Those who scored 1 or more on the SCOFF questionnaire (Morgan et al., 1999) constituted the subclinically eating disordered group (N = 50).

**Design**

Study 3 was of a mixed design. Participants were randomly allocated to one of the three conditions (Gratitude, Happy Memories or Neutral), and completed a set of questionnaires before (Time 1) and after (Time 2) relevant mood induction. The change in scores from Time 1 and Time 2 was assessed for each participant, and the differences between the experimental groups were examined. Because participant numbers were small, the sample was not divided into groups according to eating pathology, and eating pathology scores were used in the correlational analyses only. Figure 15 shows the flow and attrition of participants in the study in more detail.

Figure 15: A consort diagram showing the flow and attrition of participants in Study 3
**Ethics**

Study 3 received the ethical approval from the School of Health in Social Science Ethics Committee (University of Edinburgh). Participants were told that they would be taking part in a study looking at affect, well-being and eating pathology. They signed a consent form that clearly stated their right to withdraw from the study at any time, and included data anonymity and confidentiality guarantees. At the end of the study participants were fully debriefed on the aims of the research project.

**Procedure**

Potential participants were informed that the study would take around 1 1/2 hours, and that they would receive £10 for their time. They were given a choice of 2-hour time slots over the course of four weeks (9am to 6pm on weekdays), and chose the day/time that suited them best. Two participants were scheduled per time slot. The completion of the first and second sets of questionnaires was not timed, but the mood induction in between was (15 minutes). After participants filled in the first set of questionnaires, the experimenter explained the interventions procedure and gave instructions on how to do the exercises. After 15 minutes, the participants were told that the mood induction was over and presented with the second set of questionnaires. Participants were randomly allocated to one of the positive mood conditions (Gratitude: N = 33; Happy Memories: N = 34) and the neutral condition (N = 34). First they completed a questionnaire set, consisting of the Emotion Assessment Scale (EAS, Carlson et al., 1989), Regulation of Emotion Questionnaire (REQ, Phillips and Power, 2007), Satisfaction with Life Scale (Diener, 1985), and the Hospital Anxiety and Depression Scale (HADS, Zigmond and Snaith, 1983). Then either a happy or neutral mood was induced, using Positive Psychology Interventions or a neutral task. Gratitude (Group 1) and Happy Memories (Group 2) exercises were used to induce positive mood in the experimental sample. A neutral task was used in the control group. The effectiveness of mood induction was assessed using the Emotional Assessment Scale (EAS; Carlson et al., 1989), and the questionnaire set was re-administered. It was originally expected that the sample would be larger, however it was impossible to recruit sufficient numbers in the three groups. For the purposes of statistical analysis, the data from the two PPIs were combined into one condition (Happiness) and compared to the controls’ data (Neutral Mood).
Measures

1. The Eating Disorder Examination Questionnaire (EDE-Q; Fairburn and Beglin, 1994) measures eating pathology. It is a 36-item measure, assessing frequency of disordered eating behaviours, thoughts and concerns over the past 28 days. There are 4 subscales: Restraint, Weight Concern, Eating Concern and Shape Concern; they are summed to give an overall score. [Appendix 4]

2. The SCOFF Questionnaire (Morgan et al., 1999) detects eating disorders. This 5-item questionnaire asks about eating behaviours and thoughts about weight and shape (e.g. ‘Have you recently lost more than one stone in a 3 month period?’), with the answer options Yes and No. [Appendix 6]

3. Regulation of Emotion Questionnaire (REQ, Phillips and Power, 2007) measures functionality of affect management along four subscales (Internal/External Dysfunctional and Internal/External Functional emotion regulation). It is a 21-item questionnaire with statements about different emotion regulation strategies (e.g. ‘I ask others for advice’) with a frequency scale from Never to Always. The respondent is asked how often they use certain regulatory strategies: ‘In general, how do you respond to your emotions?’. In Study 3 participants completed this questionnaire in its original form prior to mood induction. Because this questionnaire measures a general tendency to regulate emotions in particular ways at present, it had to be adapted to meaningfully capture any change after the mood induction. Therefore, after mood manipulation, participants completed an adapted version of the Regulation of Emotion Questionnaire. The instructions focused on the future intentions and likelihood of using certain regulatory strategies, and the question was changed to: ‘How will you respond to your emotions?’. All the statements were adapted to refer to the future (e.g. ‘I shall talk to someone about how I feel’ instead of ‘I talk to someone about how I feel’); the response scale (from Never to Always) remained the same. [Appendix 1]

4. The Satisfaction with Life Scale (SWLS; Diener, 1985) measures subjective well-being. It is a 5-item questionnaire, and respondents are asked to rate their agreement
with statements such as ‘The conditions of my life are excellent’ from Strongly Disagree to Strongly Agree. [Appendix 3]

5. The Emotion Assessment Scale (EAS; Carlson et al., 1989) measures current mood and assesses effectiveness of mood induction. It is a list of 24 mood adjectives such as ‘Happy’ or ‘Disgusted’, and the participants are asked to indicate the extent to which they are experiencing those emotions by putting a slash on the line that goes from Least Likely to Most Likely. The answers are then added up to make up a score for each of the eight emotions (Happiness, Sadness, Disgust, Fear, Surprise, Guilt, Anxiety and Anger). [Appendix 9]

6. The Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983) measures depression and anxiety. It consists of 14 statements (e.g. ‘I feel tense or wound up’) with 4 answer options (e.g. from Most of the time to Not at all).

7. Emotion Induction

In positive psychology Gratitude exercises are commonly used to induce positive mood. In this study participants were asked to spend around 10-15 minutes thinking about and listing the things in their life that they are grateful for: “Please spend a few minutes thinking of things in your life that you are grateful for. These might include particular supportive relationships, sacrifices or contributions that others have made for you, facts about your life such as your advantages and opportunities, or even gratitude for life itself, and the world we live in. The gratitude could refer to the past, the present or the future. It can be about anything, even little things. In all of these cases you are identifying previously unappreciated aspects of your life, for which you can be thankful. You may not have thought about yourself in this way before, but research suggests that doing so can have a strong positive effect on your life satisfaction. Please use the space below to write about the things you are thankful for” (participant instructions, based on Emmons and McCullough, 2003).

There is no standard procedure for the Happy Memories exercise; participants are typically simply asked to recall some happy memories with or without a memento to help them. In this study participants were asked to write about their happy memories
without the use of mementos. The exercise was based on the *Self-Observation of Episodes of Well-Being* technique from the Well-Being Therapy (Ruini et al., 2003, 2009) which was shown to be effective in focusing attention on the positive aspects of life. The instructions were as follows: “Have you felt ecstatic, happy, joyful, pleased, glad or contented over the past 2 weeks? Can you please write down what positive emotions you felt and what it was that made you feel them? In other words, what was the trigger? Please write in the space provided below and on the next page. You can go in as much or as little detail as you wish. Please continue on the back of the page if necessary. This exercise should take around 15 minutes and it’s important that you don’t rush it”.

Neutral exercises that are used to induce mood of neutral valence in a control group typically ask participants to describe some general aspects of their life that are likely to be emotionally neutral (Sheldon and Lyubomirsky, 2006). In this study, participants were encouraged to spend 10-15 minutes writing about their typical day: “Please use the space below to write about your typical day, and the kinds of things that happen during it and outline your typical day in as much detail as you can”. These instructions were based on Sheldon and Lyubomirsky (2006).

RESULTS

Data screening procedures were performed. The data were checked for accuracy by computing frequencies and minimum/maximum statistics for the variables using Statistical Package for the Social Sciences (SPSS). Visual checking of the resultant analyses against the questionnaire data ensured that there were no errors in the data file, and all the scores fell within the range of possible scores for the scales. Skewness, kurtosis and z-scores were also computed for each dependent variable (please see Appendix 15). All z-scores fell below 3.3, which indicated reasonably good normal distribution. Visual checking of histograms and scatterplots confirmed that the scores on the dependent variables were reasonably normally distributed, the relationships between them were linear and the data tended to have similar variability. As part of the t-tests, Levene’s test was performed in order to ensure homogeneity of variance; in case of all
the variables the Levene test was non-significant (p > 0.05). The cases that have missing values were excluded from analyses that included that variable.

PPI effectiveness
Study 3 found that both PPIs were effective in inducing positive mood. The Happy Memories (Positive Reminiscence) exercise was effective in 50% of cases (in 17 out of 34 participants), whereas the Gratitude exercise was more effective and worked in 61% of cases (in 20 out of 33 participants). As can be seen from Table 17, in the Positive Reminiscence condition, Sadness scores ranged between .10 and 15.40 (Mean = 3.41, SD = 3.63) before the Positive Reminiscence task; the scores changed to between 0 and 16.30 (Mean = 2.46, SD = 3.73), following the task. Happiness scores changed from between 2.40 and 23.40 (Mean = 13.75, SD = 5.50) before Positive Reminiscence task to between 1.90 and 25.90 (Mean = 14.53, SD = 6.90) after the task. In the Gratitude condition, Sadness decreased from between 0 and 9.80 (Mean = 4.23, SD = 3.17) to between 0 and 9.10 (Mean = 2, SD = 2.13); and Happiness increased from between 5.70 and 22.80 (Mean = 14.03, SD = 4.88) to between 4.50 and 26.70 (Mean = 15.45, SD = 6.37). To sum up, both in the Positive Reminiscence and the Gratitude conditions, happiness on average increased and sadness on average decreased as a result of positive mood induction. The overall results for happiness induction for 67 participants were as follows: mean Sadness decreased from 3.81 (SD = 3.41) to 2.23 (SD = 3.03), and mean happiness increased from 13.89 (SD = 5.17) to 14.98 (SD = 6.61). In order to achieve reasonable numbers in the conditions, the two mood groups (Positive Reminiscence and Gratitude) were combined in statistical analyses; only the cases where positive mood induction was successful (Positive Reminiscence: N = 17; Gratitude: N = 20) were included in the experimental condition (N = 37). For the purposes of this study it is not important to know whether the changes from Time 1 to Time 2 were statistically significant; it is enough to know that positive mood induction was successful.
<table>
<thead>
<tr>
<th>Condition</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>SD</th>
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N = Number (of participants), SD = Standard Deviation
Effects of happiness induction versus neutral mood on emotion regulation and life satisfaction in the subclinically eating-disordered sample and controls.

A series of mixed between-within subjects analyses of variance was conducted to assess the impact of two different mood conditions (Happiness and Neutral Mood) on participants’ scores on the Regulation of Emotions Questionnaire (Phillips and Power, 2007)) and the Life Satisfaction Scale (Diener, 1985) at two time periods: pre- and post-interventions. Table 18 shows emotion regulation, life satisfaction, anxiety and depression scores before and after mood induction in the Happiness group and controls. The Happiness group consisted of participants who successfully completed the Positive Reminiscence (N = 17) exercise and the Gratitude task (N = 20). The two groups were combined in statistical analyses in order to provide a satisfactory number in the experimental condition (N = 37) to compare with the Controls (N = 34). The data were checked to ensure no violation of the assumptions of normality, linearity and homoscedasticity. Histograms and scatterplots showed that the scores on the variables were reasonably normally distributed, the relationships between them were linear and the data tended to have similar variability. Only the cases where positive mood induction was successful were included in the statistical analyses on the effects of mood induction (N = 37).
Table 18: Regulation of Emotion, Anxiety, Depression and Life Satisfaction Scores Before and After Happiness and Neutral Mood Interventions

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<td></td>
<td>2. Post-Intervention</td>
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<tr>
<td>Anxiety (HADS)</td>
<td>1. Pre-Intervention</td>
<td>37</td>
</tr>
<tr>
<td></td>
<td>2. Post-Intervention</td>
<td>37</td>
</tr>
<tr>
<td>Depression (HADS)</td>
<td>1. Pre-Intervention</td>
<td>37</td>
</tr>
<tr>
<td></td>
<td>2. Post-Intervention</td>
<td>37</td>
</tr>
</tbody>
</table>

*Significant (p < 0.05)
ER = Emotion Regulation, N = Number (of participants), SD = Standard Deviation

**Internal Dysfunctional ER (InDys)**

There was a significant main effect for time, Wilks’ Lambda = .92, F (1, 69) = 5.89, p < 0.05, partial eta squared = .08. Internal Dysfunctional emotion regulation scores decreased after the induction of both Happiness (Time 1: mean = 6.76, SD = 2.10; Time 2: mean = 6.30, SD = 2.89), and Neutral mood (Time 1: mean = 8.21, SD = 2.90; Time 2: mean = 7.79, SD = 2.83). The interaction between time and mood was non-significant, Wilks’ Lambda = 1, F (1, 67) = .02, p = .89, partial eta squared = 0. There was a significant difference between the two conditions, F (1, 69) = 5.76, p < 0.05, partial eta squared = .08. Internal Dysfunctional ER scores are shown on Figure 16.
There was a significant main effect for time in External Dysfunctional scores, Wilks’ Lambda = .8, $F(1, 69) = 20.61, p < 0.01$, partial eta squared = .23, with both positive (Time 1: mean = 2.43, SD = 1.59; Time 2: mean = 1.57, SD = 1.80), and neutral (Time 1: mean = 2.88, SD = 2.18; Time 2: mean = 2.56, SD = 2.36), conditions showing a reduction in ExDys scores. The interaction between time and mood was also significant, Wilks’ Lambda = .94, $F(1, 69) = 4.23, p < 0.05$, partial eta squared = .06, with the scores in the positive mood condition declining significantly (Time 1: mean = 2.43, SD = 1.59; Time 2: mean = 1.57, SD = 1.80). There were no significant between-subjects effects, $F(1, 69) = 2.50, p = .12$, partial eta squared = .03. External Dysfunctional ER scores are shown in Figure 17.
Figure 17: External Dysfunctional scores in the Neutral and Positive Mood conditions

The main effects for time in External Functional ER were significant, Wilks’ Lambda = .92, F (1, 69) = 5.59, p < 0.05, partial eta squared = .07, which means that scores in both conditions changed from Time 1 to Time 2. The interaction between time and mood was also significant, Wilks’ Lambda = .91, F (1, 69) = 7.12, p < 0.05, partial eta squared = .09. Positive affect was more effective in increasing the External Functional emotion regulation scores (Time 1: mean = 14.32, SD = 2.79; Time 2: mean = 15.30, SD = 3.58), compared to Neutral mood (Time 1: mean = 12.79, SD = 3.85; Time 2: mean = 12.74, SD = 4.35), F (1, 69) = 5.81, p < 0.05; partial eta squared = .08. External Functional ER scores are shown in Figure 18.
There was no significant main effect for time in Internal Functional ER, Wilks’ Lambda = 1, F (1, 69) = .061, p = .81, partial eta squared = .001. The interaction between time and mood was also non-significant, Wilks’ Lambda = 1, F (1, 69) = .16, p = .7, partial eta squared = .002. There were no between-subject effects; neither Happiness (Time 1: mean = 11.84, SD = 2.78; Time 2: mean = 11.86, SD = 3.41), nor Neutral mood induction (Time 1: mean = 11.88, SD = 3.20; Time 2: mean = 11.76, SD = 3.69) significantly affected Internal Functional Emotion Regulation scores; F (1, 69) = .001, p = .97. Internal Functional ER scores are shown in Figure 19.
Life Satisfaction

The main effect for Life Satisfaction was statistically significant, Wilks’ Lambda = .93, F (1, 69) = 4.76, p < 0.05, partial eta squared = .06. The interaction between time and condition was also significant, Wilks’ Lambda = .86, F (1, 69) = 11.33, p < 0.01, partial eta squared = .14; positive affect was effective in increasing Life Satisfaction scores (Time 1: mean = 25.08, SD = 5.14; Time 2: mean = 26.46, SD = 5.24), compared to Neutral mood (Time 1: mean = 25.38, SD = 6.39; Time 2: mean = 25.09, SD = 6.30). There were no significant between-subject effects, F (1, 69) = .15, p = .70, partial eta squared = .002. Figure 20 shows satisfaction with life scores in the two mood conditions.
Sub-clinical Anxiety

There was a significant main effect for time in Anxiety, Wilks’ Lambda = .93, F (1, 69) = 5.42, p < .05, partial eta squared = .07. Anxiety decreased in both Happiness (Time 1: mean = 8.70, SD = 3.53; Time 2: mean = 7.97, SD = 3.82), and Neutral mood induction (Time 1: mean = 9.18, SD = 3.25; Time 2: mean = 8.88, SD = 3.33). The interaction between time and mood was non-significant, Wilks’ Lambda = .99, F (1, 69) = 98, p = .32, partial eta squared = .014. There were no significant between-subjects effects, F (1, 69) = .74, p = .4, partial eta squared = .011. Figure 21 shows anxiety scores in the two mood conditions.
Sub-Clinical Depression

There was no significant main effect for time in Depression, Wilks’ Lambda = .98, F (1, 67) = 1.32, p = .25, partial eta squared = .02. The interaction between time and condition was also non-significant, Wilks’ Lambda = .98, F (1, 67) = 1.32, p = .25, partial eta squared = .02. Neither Happiness (Time 1: mean = 3.22, SD = 2.29; Time 2: mean = 2.86, SD = 2.63), nor Neutral mood induction (Time 1: mean = 4, SD = 2.73; Time 2: mean = 4, SD = 2.74) significantly affected Depression scores. There were no significant between-subject effects, F (1, 67) = 2.50, p = .12, partial eta squared = .036. Figure 22 shows depression scores in the two mood conditions.
The relationship between sub-clinical eating pathology and emotion regulation, life satisfaction, anxiety and depression

The relationship between eating pathology (EDE-Q; Fairburn and Beglin, 1994), Life Satisfaction (Diener, 1985), Depression (HADS; Zigmond and Snaith, 1983), Anxiety (HADS; Zigmond and Snaith, 1983) and emotion regulation (REQ; Phillips and Power, 2007) was investigated using Pearson product-moment correlation coefficient (Table 19).

Statistically Significant Findings ($p < 0.05$)

Consistent with Study 1, eating pathology (as measured by the overall EDE-Q score) was positively associated with Internal Dysfunctional emotion regulation ($r = .28, N = 101, p < 0.01$), negatively with life satisfaction ($r = -.26, N = 101, p < 0.01$), positively with depression ($r = .25, N = 101, p < 0.01$) and anxiety ($r = .29, N = 101, p < 0.05$).
Restraint was positively associated with Internal Dysfunctional ER ($r = .21$, $N = 101$, $p < 0.05$). Eating Concern was positively associated with Internal Dysfunctional ER ($r = .26$, $N = 101$, $p < 0.01$), Anxiety ($r = .25$, $N = 101$, $p < 0.05$), Depression ($r = .23$, $N = 101$, $p < 0.05$) and negatively with Life Satisfaction ($r = -.24$, $N = 101$, $p < 0.05$).

Shape Concern was positively associated with Internal Dysfunctional ER ($r = .28$, $N = 101$, $p < 0.01$), Anxiety ($r = .29$, $N = 101$, $p < 0.01$), Depression ($r = .27$, $N = 101$, $p < 0.01$) and negatively with Life Satisfaction ($r = -.37$, $N = 101$, $p < 0.01$).

Weight Concern was positively associated with Internal Dysfunctional ER ($r = .25$, $N = 101$, $p < 0.05$), Anxiety ($r = .30$, $N = 101$, $p < 0.05$), Depression ($r = .26$, $N = 101$, $p < 0.05$) and negatively with Life Satisfaction ($r = -.37$, $N = 101$, $p < 0.01$).

Table 19: Correlations between EDE-Q facets and Emotion Regulation, Life Satisfaction, Anxiety and Depression.

<table>
<thead>
<tr>
<th>Overall EDE-Q</th>
<th>Restraint</th>
<th>Eating Concern</th>
<th>Shape Concern</th>
<th>Weight Concern</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internal Dysfunctional ER</td>
<td>.28**</td>
<td>.21*</td>
<td>.26**</td>
<td>.25*</td>
</tr>
<tr>
<td>Internal Functional ER</td>
<td>.04</td>
<td>.05</td>
<td>.09</td>
<td>.03</td>
</tr>
<tr>
<td>External Dysfunctional ER</td>
<td>-.01</td>
<td>-.04</td>
<td>.02</td>
<td>-.02</td>
</tr>
<tr>
<td>External Functional ER</td>
<td>-.04</td>
<td>-.05</td>
<td>-.07</td>
<td>-.02</td>
</tr>
<tr>
<td>Anxiety</td>
<td>.29*</td>
<td>.19</td>
<td>.25*</td>
<td>.29**</td>
</tr>
<tr>
<td>Depression</td>
<td>.25**</td>
<td>.12</td>
<td>.23*</td>
<td>.27**</td>
</tr>
<tr>
<td>Life Satisfaction</td>
<td>-.26**</td>
<td>-.14</td>
<td>-.24*</td>
<td>-.37**</td>
</tr>
<tr>
<td>* Significant ($p &lt; 0.05$)</td>
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<td></td>
<td></td>
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<tr>
<td>** Significant ($p &lt; 0.01$)</td>
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</tbody>
</table>

Non-Significant Findings ($p > 0.05$)

None of the following correlations reached statistical significance.

The overall EDE-Q scores did not correlate with Internal Functional ER ($r = .04$, $N = 101$, $p > 0.05$), External Dysfunctional ER ($r = -.01$, $N = 101$, $p > 0.05$) and External Functional ER ($r = -.04$, $N = 101$, $p > 0.05$). Restraint score was not associated with Internal Functional ER ($r = 0.05$, $N = 101$, $p > 0.05$), Anxiety ($r = .19$, $N = 101$, $p > 0.05$), Depression ($r = .12$, $N = 101$, $p > 0.05$), External Dysfunctional ER ($r = -.04$, $N = 101$, $p > 0.05$), External Functional ER ($r = -.05$, $N = 101$, $p > 0.05$) and Life Satisfaction scores ($r = -.14$, $N = 101$, $p > 0.05$). Eating Concern was not associated
with Internal Functional ER ($r = .09, N = 101, p > 0.05$), External Dysfunctional ER ($r = .03, N = 101, p > 0.05$), and External Functional ER ($r = -.02, N = 101, p > 0.05$). Shape Concern was not associated with External Dysfunctional ER ($r = .02, N = 101, p > 0.05$), Internal Functional ER ($r = -.01, N = 101, p > 0.05$) and External Functional ER ($r = -.07, N = 101, p > 0.05$). Weight Concern did not correlate with Internal Functional ER ($r = .03, N = 101, p > 0.05$), External Dysfunctional ER ($r = -.02, N = 101, p > 0.05$) and External Functional ER ($r = -.02, N = 101, p > 0.05$).

The relationship between emotion regulation and life satisfaction, anxiety and depression

The relationships between emotion regulation, life satisfaction, anxiety and depression were investigated using Pearson product-moment correlation coefficient (Table 20).

**Statistically Significant Findings (p < 0.05)**

Internal Dysfunctional Emotion Regulation was positively associated with Anxiety ($r = .52, N = 101, p < 0.01$) and Depression ($r = .37, N = 101, p < 0.01$), and negatively with Life Satisfaction ($r = -.40, N = 101, p < 0.01$). External Dysfunctional Emotion Regulation was negatively correlated with Life Satisfaction ($r = -.21, N = 101, p < 0.05$) and positively with Anxiety ($r = .27, N = 101, p < 0.01$). External Functional Emotion Regulation was negatively associated with Depression ($r = -.29, N = 101, p < 0.01$).

**Non-Significant Findings (p > 0.05)**

None of the following correlations reached statistical significance. Internal Functional Emotion Regulation was not associated with Life Satisfaction ($r = .09, N = 101, p > 0.05$), Anxiety ($r = .02, N = 101, p > 0.05$) and Depression scores ($r = -.02, N = 101, p >
External Dysfunctional Emotion Regulation did not correlate with Depression scores ($r = .09, N = 101, p > 0.05$). External Functional ER was not associated with Life Satisfaction ($r = .17, N = 101, p > 0.05$) and Anxiety ($r = .03, N = 101, p > 0.05$).

*The SCOFF questionnaire (Morgan et al., 1999) and the Eating Disorder Examination Questionnaire (EDE-Q, Fairburn and Beglin, 1994)*

The relationship between eating pathology as measured by the EDE-Q (Fairburn and Beglin, 1994) and the SCOFF questionnaire (Morgan et al., 1999) was investigated using Pearson product-moment correlation coefficient (Table 21). The SCOFF score positively correlated with overall EDE-Q ($r = .67, N = 101, p < 0.01$), Restraint ($r = .51, N = 101, p < 0.01$), Eating Concern ($r = .69, N = 101, p < 0.01$), Shape Concern ($r = .59, N = 101, p < 0.01$) and Weight Concern ($r = .60, N = 101, p < 0.01$).

**Table 21: Correlations between the SCOFF questionnaire and facets of the EDE-Q**

<table>
<thead>
<tr>
<th>SCOFF score</th>
<th>Overall EDE-Q</th>
<th>Restraint</th>
<th>Eating Concern</th>
<th>Shape Concern</th>
<th>Weight Concern</th>
</tr>
</thead>
<tbody>
<tr>
<td>* .67*</td>
<td>.51*</td>
<td>.69*</td>
<td>.59*</td>
<td>.60*</td>
<td></td>
</tr>
</tbody>
</table>

*Significant ($p<0.01$).

The SCOFF questionnaire was highly and reliably correlated with EDE-Q and its facets, which means that it may be an alternative, accurate and time-efficient way of screening for eating pathology.

**Main findings**

1. The PPIs were easy to implement and successfully used to induce positive mood. The Gratitude exercise was more effective (success rate = 61%) than the Positive Reminiscence task (success rate = 50%).

2. Sub-clinical eating pathology was associated with dysfunctional emotion regulation, sub-clinical depression, anxiety and low life satisfaction.

3. Positive affect, induced using the PPIs, had a significant effect on emotion regulation, increasing functional ER and life satisfaction, as well as reducing dysfunctional affect management (Internal Dysfunctional ER, External Dysfunctional ER and anxiety).
4. Dysfunctional emotion regulation was associated with sub-clinical anxiety, depression and low life satisfaction.

5. The SCOFF questionnaire (Morgan et al., 1999) was highly correlated with EDE-Q (Fairburn and Beglin, 1994) and its facets, which makes it useful and quick tool for screening for eating pathology.

DISCUSSION

Positive Psychology Interventions (PPIs): Effectiveness of the Gratitude and Positive Reminiscence Tasks

The results showed that both PPIs were successful in inducing positive mood in the sample. If one looks at the means, one can see that both exercises led to a reduction of sadness and an increase in happiness. However, the Gratitude exercise was 11% more effective than the Positive Reminiscence task in eliciting positive affect. The Gratitude method is well-researched and comes with instructions (Emmons and McCullough, 2003), whereas no such consistency exists for the happy memories method. Therefore, the relative lower effectiveness of the Happy Memories exercise may be attributable to the phrasing of participant instructions that were used in Study 3. The Positive Reminiscence task would benefit from standardisation of participant instructions (which is lacking at present), and this may potentially increase its effectiveness.

Study 3 showed that both methods are equally simple to implement, and can be carried out at anytime, anywhere, even when the presence of the researcher cannot be ensured. This makes them ideal for longitudinal mood induction. The finding that the Gratitude task was more effective suggests that in the future studies, it may be worth to select it, as opposed to the Positive Reminiscence method, both for short-term and long-term mood induction. This exercise may be easily used for regular positive mood induction in long distance studies, where the researcher cannot be present at the induction or has to collect the data by post. This makes the Gratitude task a valuable tool for positive mood induction.
Influence of Positive Psychology Interventions on emotion regulation and life satisfaction.

On the whole, the results provide support for Study 1, showing that positive emotions, induced using PPIs, had a significant effect on emotion regulation and life satisfaction. Happiness, elicited by the Gratitude and Happy Memories exercise, led to a steep reduction in External Dysfunctional ER (Phillips and Power, 2007) and an increase in External Functional ER (Phillips and Power, 2007) and life satisfaction (Diener, 1985).

Following mood induction, Internal Dysfunctional ER scores declined in both control and positive affect conditions. This means that both neutral and positive emotional states have a beneficial effect on Internal Dysfunctional ER, reducing the likelihood of such behaviours as self-harm, rumination, negative social comparison, repression and de-realization (Phillips and Power, 2007). Mood had no effect on Internal Functional ER, which means that individuals in both neutral and positive emotional states may use such healthy strategies as positive reappraisal, planning, modification of goals, putting things in perspective and concentration (Phillips and Power, 2007). However, Figure 18 shows that the effects of neutral mood and positive affect are inverse: there is a sharp increase in Internal Functional ER scores from Time 1 to Time 2 in the happiness condition, and a decline in the scores in the neutral conditions. This means that the lack of statistically significant difference in mood effects may be attributable to error or insufficient participant numbers in the conditions.

External Dysfunctional ER significantly declined following positive affect induction. This suggests that positive emotions may discourage such unhealthy regulatory practices as bullying, verbal abuse and physical assault (Phillips and Power, 2007). In addition, happiness significantly boosted External Functional ER; in other words, individuals who are experiencing positive mood, are more likely to choose such healthy strategies as expression of feelings, advice and contact seeking, exercise and distraction, when managing their emotions (Phillips and Power, 2007). These findings suggest that positive affect may foster functional emotion regulation. In addition, positive affect led to a significant increase in life satisfaction. There was a sharp escalation of satisfaction with life scores in the happiness condition, whereas the opposite was true for neutral
mood – life satisfaction declined from Time 1 to Time 2. This means that positive mood may make people feel better about their lives and experience increased well-being.

The results show that anxiety declined in both neutral and happiness conditions. However, Figure 20 suggests that anxiety scores decreased more sharply in the happiness condition. This may be a result of error, or alternatively, it may be possible that both neutral emotional states (e.g. relaxation) and positive affect are not conducive to anxiety. Although there were no significant effects of mood on depression, Figure 21 shows that it decreased sharply in the happiness group and remained more or less stable in the neutral group. This discrepancy between statistical and graph data may be attributable to error.

There are several instances where the significance results do not support the data, depicted on the graphs. The graphs may show group differences, but these are not reflected in the statistical analyses. In future studies, it may be worthwhile using a larger sample size in order to minimize the chance of such errors. In this study only those who successfully underwent mood induction were included (N = 37) in the statistical analyses on the effects of positive mood, and this number may be too low for statistical significance to be detected. In future studies, it would be helpful to use a much larger experimental group, taking into account the failure rate of PPIs (50% for the Positive Reminiscence task and 39% for the Gratitude task) to account for data that will be excluded from analyses. For example, out of a 100 participants who do the Positive Reminiscence tasks, around 50 will fail to respond to mood induction, and subsequently will not be included in the analyses so as not to cloud the effects. This means a loss of around half of participants from the sample. It is important to know how effective a PPI is because recruitment aims should be informed by the average failure rates of PPIs. This study is the first of its kind in providing the data on the effectiveness of the PPIs (the Gratitude exercise and the Positive Reminiscence task).

It is possible that the effects of positive versus neutral mood did not always reach statistical significance because of the type of control mood used (i.e. neutral affective state). In Study 1, where happiness was compared to sadness, the results were clearer in showing a beneficial effect of positive affect on emotion regulation. This may be because in Study 1 two diametrically opposed emotions were induced (i.e. sadness and
happiness), therefore any discrepancies in their effects on emotion regulation were easily visible and measurable. It is safe to assume that in terms of valence, the figurative distance between positive emotions and neutral mood is much shorter than that between positive emotions and negative emotions (Figure 23). Therefore, neutral mood and positive emotions may share some affective components, which in Study 3 resulted in similarity in their effects on emotion regulation and other measures. Even though the direction of the change in scores was the same in the control and happiness conditions, the influence of positive mood was more dramatic, as can be seen from the graphs in the Results section. Therefore, the use of the neutral mood as a control condition was justified.

Figure 23: Figurative distance between emotions

**Emotion regulation and satisfaction with life in eating pathology.**

The results showed that the EDE-Q and its facets (Restraint, Eating Concern, Shape Concern and Weight Concern; Fairburn and Beglin, 1994) were positively associated with Internal Dysfunctional ER. This means that individuals with eating pathology may be more likely to use such unhealthy emotion regulation strategies as: self-harm, rumination, negative social comparison, repression and de-realization (Phillips and Power, 2007). This supports Study 1 findings in showing that Internal Dysfunctional ER strategies are associated with eating pathology, even if it is assessed by different means: EAT-26 (Garner et al., 1982) and EDE-Q (Fairburn and Beglin, 1994).

Another finding was that eating pathology was associated with low life satisfaction (Diener, 1985). In other words, individuals with eating pathology may be less likely to perceive their lives as happy, and the more severe their eating pathology, the less happy
the participants were. Therefore, Study 3 showed that disordered eating may be incompatible with subjective well-being. These findings support those of Kitzantas et al. (2003) who found that low life satisfaction was typical of individuals with disordered eating, as well as the results of Valois et al.’s study (2003) who also showed that disordered weight loss behaviours (e.g. vomiting) and negative body perceptions could significantly impact life satisfaction. Considering that eating pathology is often a source of distress and anxiety to the sufferer (Bydlowski et al., 2005; Fox and Harrison, 2008; Heatherton and Baumeister, 1991), it is not surprising that it would negatively affect their satisfaction with life.

In addition, eating pathology was associated with depression and anxiety, as measured by the HADS (Zigmond and Snaith, 1983). This means that individuals with eating pathology may tend to be depressed and anxious. A wealth of literature supports this finding (Brewerton et al., 1995; Geist et al., 1998; Halmi, 1995; Karatzias et al., 2009; O’Brian and Vincent, 2003; Rastam, 1992). To sum up, Study 3 showed that eating pathology is associated with dysfunctional emotion regulation, emotional disorder (anxiety and depression) and low subjective well-being. More research is needed in order to establish the direction of these relationships.

*Emotion Regulation in the context of sub-clinical anxiety, depression and life satisfaction.*

Study 3 showed that dysfunctional emotion management correlated with emotional disorder (sub-clinical anxiety and depression; HADS, Zigmond and Snaith, 1983) and low subjective well-being (life satisfaction; SWLS, Diener, 1985). Internal Dysfunctional ER was positively related to anxiety and depression, and negatively to life satisfaction. This means that individuals who typically use Internal Dysfunctional strategies (e.g. rumination) are likely to be anxious, depressed and unhappy. However it is not clear whether dysfunctional emotion regulation causes anxiety, depression and low life satisfaction, or whether it is the other way around. Anxiety, depression and general dissatisfaction with one’s life may lead one to use Internal Dysfunctional strategies, such as self-harm, rumination, negative social comparison, de-realization and repression (Phillips and Power, 2007). It was also found that External Dysfunctional ER was associated with anxiety and low life satisfaction, but not depression. This means
that individuals who use External Dysfunctional ER strategies, may be more likely to be anxious and dissatisfied with their lives. Such strategies include bullying, as well as verbal and physical assault (Phillips and Power, 2007). Alternatively, it is possible that those who are anxious and unhappy may opt for External Dysfunctional regulatory strategies. Regardless of the direction of the effects, it is clear that dysfunctional emotion management (internal and external) is associated with negative psychological outcomes (anxiety, depression and dissatisfaction with life). Future research is needed to determine the direction of this relationship.

With regards to healthy regulatory strategies, it was found that External Functional ER was negatively associated with depression. This may mean that using such healthy strategies as distraction, advice and contact seeking when managing emotions may be a protective factor for mental health. Alternatively, it is plausible that those who are not depressed tend to choose more functional regulatory strategies. In either case, the results show that functional emotion regulation is related to positive mental health. It is interesting that Internal Functional ER was not related to any positive (life satisfaction) or negative (anxiety and depression) outcomes. This means that there may be no connection between choosing Internal Functional regulatory strategies (such as positive reappraisal, modification of goals, planning, putting things in perspective and concentration on a pleasant activity; Phillips and Power, 2007) and emotional health (anxiety, depression) and well-being (life satisfaction).

*The relationship between the SCOFF questionnaire (Morgan et al., 1999) and EDE-Q (Fairburn and Beglin, 1994)*

The results show that the SCOFF questionnaire (Morgan et al., 1999) was consistently associated with the overall score on the EDE-Q (Fairburn and Beglin, 1994) and all four of its facets (Restraint, Eating Concern, Shape Concern and Weight Concern). The SCOFF scale (Morgan et al., 1999) is a much shorter measure of eating pathology compared to the EDE-Q (Fairburn and Beglin, 1994), and the results mean that the SCOFF scale (Morgan et al., 1999) can be used as a quick, convenient and easy way to control for presence of eating disorders when screening potential controls. Those who score 0 on the SCOFF questionnaire (Morgan et al., 1999) are unlikely to engage in disordered eating, and may be selected as a healthy control group in studies on eating.
disorders. The scale efficiently gives a rough indication of whether an individual has eating pathology. This can then be followed up with EDE-Q (Fairburn and Beglin, 1994) or other more detailed eating disorder measures in the individuals where the SCOFF questionnaire (Morgan et al., 1999) detected eating pathology.

**Summary**

The results suggest that PPIs (in particular, the Gratitude task) can be successfully used as emotion induction methods, especially if their failure rates are taken into account. The SCOFF questionnaire (Morgan et al., 1999) can be used as a quick and convenient method of screening potential controls in studies with clinical and subclinical populations. Study 3 findings supported those of Study 1 in showing that individuals with eating pathology (i.e. subclinical eating disorders) tended to use unhealthy emotion regulation strategies and that a one-off induction of positive emotions may improve affect management choices. Taking the findings of Study 1 and Study 3 further, it is plausible that increasing the frequency of positive affect in a clinical sample would lead to healthier emotion regulation over time. It is possible that longitudinal positive emotion induction may result in consistent improvement in emotion regulation skills, which may contribute to remission of eating pathology.
CHAPTER 7. Study 4: Positive affect, emotion regulation, life satisfaction and affect intensity in females with eating disorders.

INTRODUCTION
Studies 1 and 3 showed that eating pathology was associated with dysfunctional emotion regulation, sub-clinical anxiety, depression, low life satisfaction as well as high Global and Negative Affect Intensity in a subclinically eating-disordered sample and controls. Study 3 demonstrated that Positive Psychology Interventions (PPIs) may be easily implemented and successfully used to induce positive mood. Out of the two methods tested, the Gratitude exercise was the most effective (success rate = 61%) in comparison with the Positive Reminiscence task (success rate = 50%). Studies 1 and 3 also found that positive emotions led to a significant improvement in emotion regulation and emotional reactivity (Global Affect Intensity), but did not significantly affect Negative Intensity scores in subclinically eating-disordered sample and controls. Another finding of importance was that dysfunctional emotion regulation was associated with high Negative Affect Intensity, Global Affect Intensity, anxiety and depression.

Studies 4 and 5 were conducted to test the results of the two previous studies (Studies 1 and 3) with a clinical sample and to look at the effects of one-off and longitudinal positive affect induction on emotion regulation and disordered eating. Emotion regulation (Regulation of Emotion Questionnaire; Philips and Power, 2007), Affect Intensity (Affect Intensity Measure; Larsen, 1986) and the immediate post-test effect of happiness on emotion regulation and life satisfaction (Satisfaction with Life Scale; Diener, 1985) were examined in females with eating disorders. In both studies positive emotions were induced using the Gratitude exercise. Because individuals with eating disorders already had the burden of their illness to deal with, it would have been unethical to induce negative affect in them to see whether that would have worsened their emotion regulation and eating pathology. For that reason neutral mood was used a control condition, even though there was the possibility of a weaker effect than could have been obtained from using sadness or another negative emotion as a comparison condition.
In **Study 4** the following research aims were pursued:

1. To examine emotion regulation and Affect Intensity in individuals with eating disorders and healthy controls.

2. To look at the effects of a one-off positive versus neutral mood induction on the choice of regulatory strategies and satisfaction with life.

**METHOD**

**Participants and Recruitment**

In the clinical sub-sample, there were 27 females, aged 18 to 60 years old (mean age = 29 years), who were suffering from anorexia nervosa (N = 19), EDNOS (N = 1) and bulimia nervosa (N = 7). They all had been clinically diagnosed as having an eating disorder, and were receiving treatment for it at the time of the study. Potential participants were recruited with the help from the eating disorders charity B-EAT (Registered Charity No. 801343). Potential participants were told they were invited to participate in a study that looked at affect, emotion regulation and eating behaviours; they received a detailed information sheet about the study [Appendix 12]. A group of non-eating disordered controls (N = 27; mean age = 23 years) was also recruited via SAGE, the University of Edinburgh job database. Potential controls had been asked to complete the SCOFF Questionnaire (Morgan et al., 1999) to screen for eating pathology. Only those who scored zero on the SCOFF questionnaire (Morgan et al., 1999) were invited to take part in the study as the control group.

The experimental sub-sample (N = 27) and controls (N = 27) were matched according to gender (all were female) and age. The participants in the control group were full-time students at the University of Edinburgh (undergraduate N = 15, postgraduate N = 12). The controls were British, native English speakers of Caucasian (N = 25) and Asian ethnicity (N = 2). In the clinical group, 13 participants were in full-time higher education, 13 worked full or part-time and one was retired. All the participants in the clinical group were British, Caucasian and native English speakers. Eating-disordered participants in Study 4 were all pro-recovery (i.e. motivated to recover from their eating
disorders), and they received no financial incentive for participation in the study. All the participants in the clinical group were British, Caucasian and native English speakers. Mean scores on emotion regulation, affect intensity and life satisfaction were computed for the non-eating disordered controls and the clinical group. The control group scored on average 6.81 (SD = 3.02) on Internal Dysfunctional emotion regulation, 12.04 (SD = 3.02) on Internal Functional affect management, 2.55 (SD = 1.78) on External Dysfunctional emotion regulation and 14.30 (SD = 4.15) on External Functional emotion regulation. The controls’ mean score on Global Affect Intensity was 3.72 (SD = .44); 3.71 (SD = .55) on Positive Affect Intensity; and 3.78 (SD = .53) on Negative Affect Intensity. The non-eating-disordered control group scored on average 25.92 (SD = 5.30) on life satisfaction.

**Design**
Study 4 was of a mixed design; each participant was randomly assigned to one of the two conditions (Happy or Neutral mood) and filled in the questionnaires on two occasions (before and after mood induction). Study 5 also had a mixed design; there was one experimental group (Happy mood) and one control group (Neutral mood) who completed questionnaire sets on three separate occasions over one month. Figure 24 is showing the flow and attrition of participants in the study in more detail.
Ethics

Study 4 received the ethical approval from the School of Health in Social Science Ethics Committee (University of Edinburgh). It strictly observed the British Psychological Society's Code of Conduct. Prior to obtaining informed consent, all the participants were given detailed information about the aims and procedures of the studies, the opportunity to contact the researcher with any questions, and were invited to consult their clinician about their wish to participate in the studies [Information Sheet, Appendix 12]. They were informed about their right to withdraw at any time without giving a reason and with no consequence to them [Informed Consent Sheet, Appendix 11], as well as told about their right to anonymity and confidentiality. Following the completion of the studies, participants received thank you cards and an information sheet about positive psychology interventions to thank them for their contribution.
Individual feedback was not given unless specifically requested by the participants. All the data collected during the studies were kept anonymous and confidential at all times.

**Procedure**

The data in Study 4 were collected by post. Questionnaire packs containing mood induction tasks and relevant scales were put together and posted to the participants for completion. Participants sent completed questionnaire packs back to the author using pre-paid addressed envelopes. The participants were told that they would be required to spend around 1 hour of uninterrupted time filling in the questionnaires. They were instructed to go through the sections of the questionnaire pack in order. The questionnaire pack was designed to accommodate the mood induction method: the gratitude exercise was placed between the two sets of questionnaires so that the participants would come to it after completing the pre-induction questionnaire set and before the post-induction set. They were asked to fill in the questionnaire pack at their earliest convenience and to send it back to the experimenter within 2 weeks of receipt. None of the participants were paid for taking part in the study. In the first part of the study, the eating-disordered participants and healthy volunteers filled out the Emotion Assessment Scale (EAS; Carlson et al., 1989) to measure their immediate mood, as well as the Eating Disorder Examination Questionnaire (EDE-Q, Fairburn and Beglin, 1994), Affect Intensity Measure Questionnaire (AIM, Larsen, 1984), Satisfaction with Life Scale (SWLS; Diener, 1985) and Regulation of Emotion Questionnaire (REQ, Philips and Power, 2007). Then participants were randomly assigned to the Happy or Neutral condition, and the relevant mood was induced, using one of the two writing tasks: the Gratitude task (Happy condition, N = 32) and a neutral task (Neutral condition, N = 22). Effectiveness of emotion manipulation was assessed by the EAS scale (Carlson et al., 1989). Following the mood induction, the questionnaire set was re-administered to participants for completion. Participants’ mean eating pathology score on the EDE-Q (Fairburn and Beglin, 1994) was 2.91 (SD = 1.31).

**Measures**

1. The Eating Disorder Examination Questionnaire (EDE-Q; Fairburn and Beglin, 1994) assesses eating pathology. It is a 36-item measure, assessing frequency of
disordered eating behaviours, thoughts and concerns over the past 28 days. There are 4 subscales: Restraint, Weight Concern, Eating Concern and Shape Concern; they can be summed to give the overall score. [Appendix 4]

2. The SCOFF Questionnaire (Morgan et al., 1999) detects eating disorders. This 5-item questionnaire asks about eating behaviours and thoughts about weight and shape (e.g. ‘Have you recently lost more than one stone in a 3 month period?’), with the answer options Yes and No. [Appendix 6]

3. Affect Intensity Measure Questionnaire (AIM; Larsen, 1984) assesses typical depth of emotion processing and measures global, positive and negative emotional intensity. Items are phrased so as to highlight the intensity aspect of emotions. The scale consists of 40 statements (e.g. ‘When I accomplish something difficult, I feel delighted or elated’), and the respondent has to rate the frequency of each statement from Never to Always. [Appendix 2]

4. Regulation of Emotion Questionnaire (REQ; Phillips and Power, 2007) measures functionality of affect management along four subscales (Internal Dysfunctional, Internal Functional, External Dysfunctional and External Functional emotion management). It is a 21-item questionnaire with statements about different emotion regulation strategies like ‘I ask others for advice’ with a frequency scale from Never to Always. The respondent is asked how often they use certain regulatory strategies: ‘In general, how do you respond to your emotions?’. In Study 4 participants completed this questionnaire in its original form prior to mood induction. Because this questionnaire measures a general tendency to regulate emotions in particular ways at present, it had to be adapted to meaningfully capture any change post mood induction. Therefore, after mood manipulation, participants completed the adapted version of the Regulation of Emotion Questionnaire. The instructions focused on the future intentions and likelihood of using certain regulatory strategies, and the question was changed to: ‘How will you respond to your emotions?’. All the statements were adapted to refer to the future (e.g. ‘I shall talk to someone about how I feel’ instead of ‘I talk to someone about how I feel’), and the response scale
(from *Never* to *Always*) remained the same. In Study 5 only the adapted version of the questionnaire was used. [Appendix 1]

5. The Satisfaction with Life Scale (Diener, 1985) measures subjective well-being. It is a 5-item questionnaire, and respondents are asked to rate their agreement with statements such as ‘The conditions of my life are excellent’ from *Strongly Disagree* to *Strongly Agree*. [Appendix 3]

6. The Emotion Assessment Scale (EAS; Carlson et al., 1989) measures current mood and assesses effectiveness of mood induction. It is a list of 24 mood adjectives such as ‘Happy’ or ‘Disgusted’, and the participants are asked to indicate the extent to which they experience those emotions by putting a slash on the line that goes from *Least Likely* to *Most Likely*. The answers are then added up to make up a score for each of the eight emotions (Happiness, Sadness, Disgust, Fear, Guilt, Surprise, Anxiety and Anger). [Appendix 9]

7. Mood Induction
The Gratitude journal method was used to induce positive mood. Participants were asked to spend around 10-15 minutes thinking about and listing the things in their life that they are grateful for: *Please spend a few minutes thinking of things in your life that you are grateful for. These might include particular supportive relationships, sacrifices or contributions that others have made for you, facts about your life such as your advantages and opportunities, or even gratitude for life itself, and the world we live in. The gratitude could refer to the past, the present or the future. It can be about anything, even little things. In all of these cases you are identifying previously unappreciated aspects of your life, for which you can be thankful. You may not have thought about yourself in this way before, but research suggests that doing so can have a strong positive effect on your life satisfaction. Please use the space below to write about the things you are thankful for”* (participant instructions, based on Emmons and McCullough, 2003).

In the neutral condition, participants were encouraged to write about their typical day (on-line mood induction, Study 4); this exercise is typically used to induce
mood of neutral valence in a control sample (Sheldon and Lyubomirsky, 2006). The instructions were as follows: ‘Please use the space below to write about your typical day, and the kinds of things that happen during it and outline your typical day in as much detail as you can’ (participant instructions, based on Sheldon and Lyubomirsky, 2006).

RESULTS

Data screening procedures were performed. The data were checked for accuracy by computing frequencies and minimum/maximum statistics for the variables using Statistical Package for the Social Sciences (SPSS). Visual checking of the resultant analyses against the questionnaire data ensured that there were no errors in the data file, and all the scores fell within the range of possible scores for the scales. Skewness, kurtosis and z-scores were also computed for each dependent variable (please see Appendix 16). All z-scores fell below 3.3, which indicated reasonably good normal distribution. Visual checking of histograms and scatterplots confirmed that the scores on the dependent variables were reasonably normally distributed, the relationships between them were linear and the data tended to have similar variability. As part of the t-tests, Levene’s test was performed in order to ensure homogeneity of variance; in case of all the variables the Levene test was non-significant (p > 0.05). The cases that have missing values were excluded from analyses that included that variable.

Results show that the Gratitude exercise was successful in 24 out of 32 cases (75%) in inducing positive mood. As can be seen from Table 22, pre-induction happiness was between .20 and 21 (Mean = 9.45, SD = 5.93) before the Gratitude task, and rose to between 2.30 and 23.90 (Mean = 12.56, SD = 6.50) after the task.

Table 22: Descriptive Statistics for the Happiness Condition

<table>
<thead>
<tr>
<th>Condition</th>
<th>N</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gratitude Pre-Induction</td>
<td>24</td>
<td>.20</td>
<td>21.00</td>
<td>9.45</td>
<td>5.93</td>
</tr>
<tr>
<td>Happiness</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-Induction Happiness</td>
<td>24</td>
<td>2.30</td>
<td>23.90</td>
<td>12.56</td>
<td>6.50</td>
</tr>
</tbody>
</table>

N = Number (of participants), SD = Standard Deviation
Only the data from participants who successfully underwent mood induction (N = 24) were included in the statistical analyses as the experimental group. The author is aware that this number is very small, however there was no opportunity to recruit more participants.

Independent samples t-tests
Bonferroni adjustment was performed for the effects that could not be predicted from previous studies (i.e. baseline emotion profiles and life satisfaction), and the alpha value (0.05) was divided by 9 (the number of tests). The new alpha value was 0.005 after rounding, and it was used as a cut-off. A series of t-tests was conducted to compare the baseline scores on the Emotion Assessment Scale (EAS, Carlson et al., 1989), Regulation of Emotion Questionnaire (Phillips and Power, 2007), Life Satisfaction Scale (Diener, 1985) and Affect Intensity Measure (Larsen, 1984) for participants with and without eating disorders. The groups differed significantly on almost every measure, with the exception of baseline Surprise (EAS), External Dysfunctional ER and Global Affect Intensity scores.

EAS Baseline Emotion Profiles (Figure 25):
Participants with eating disorders were significantly higher in baseline Sadness (M = 13.5, SD = 7.14) than controls (M = 4.7, SD = 3.98); t (40.75) = 5.62, p < 0.005 (two-tailed). The magnitude of the difference in the means (mean difference = 8.8, 95% CI: -12.02 to -5.66) was very large (eta squared = .38).
Figure 25: Baseline emotion profiles of participants with eating disorders and controls (Sadness, Anger, Guilt, Anxiety, Happiness, Surprise, Fear and Disgust).

Participants with eating disorders also scored significantly higher on baseline Anger ($M = 12.92, SD = 8.80$) than controls ($M = 3.80, SD = 2.67$); $t (30.76) = 5.15, p < 0.005$ (two-tailed). The magnitude of the difference in the means (mean difference = 9.12, 95% CI: -12.73 to -5.50) was very large (eta squared = .34). Participants with eating disorders were significantly higher in baseline Guilt ($M = 13.6, SD = 7.85$) than controls ($M = 2.7, SD = 2.20$); $t (30.06) = 6.94, p < 0.005$ (two-tailed). The magnitude of the difference in the means (mean difference = 10.9, 95% CI: -14.09 to -7.68) was very large (eta squared = .48). Participants with eating disorders also scored significantly higher on baseline Anxiety ($M = 16.41, SD = 8.11$) than controls ($M = 10.06, SD = 5.42$); $t (45.39) = 3.38, p < 0.005$ (two-tailed). The magnitude of the difference in the means (mean difference = 6.35, 95% CI: -10.12 to -2.56) was large (eta squared = .18). Participants with eating disorders were significantly higher in baseline Fear ($M = 12.65, SD = 9.07$) than controls ($M = 3.94, SD = 3.20$); $t (32.39) = 4.71, p < 0.005$ (two-tailed). The magnitude of the difference in the means (mean difference = 8.71, 95% CI: -12.49
to -4.95) was very large (eta squared = .30). Participants with eating disorders also scored significantly higher on baseline Disgust (M = 12.10, SD = 8.73) than controls (M = 2.66, SD = 2.73); t (31.04) = 5.36, p < 0.005 (two-tailed). The magnitude of the difference in the means (mean difference = 9.44, 95% CI: -13.03 to -5.84) was large (eta squared = .17). Participants with eating disorders were significantly lower in baseline Happiness (M = 6.86, SD = 5.68) than controls (M = 13.11, SD = 5.06); t (52) = 4.27, p < 0.005 (two-tailed). The magnitude of the difference in the means (mean difference = 6.25, 95% CI: 3.31 to 9.19) was very large (eta squared = .26). Participants with eating disorders (M = 3.75, SD = 3.95) did not significantly differ from controls (M = 4.02, SD = 3.37) in baseline Surprise; t (52) = .27, p = .79.

**Emotion Regulation (Figure 26):**
Participants with eating disorders were significantly higher in Internal Dysfunctional ER (M = 11.48, SD = 3.59) than controls (M = 6.81, SD = 3.02); t (52) = 5.17, p < 0.05 (two-tailed). The magnitude of the difference in the means (mean difference = 4.67, 95% CI: -6.48 to -2.85) was very large (eta squared = .34).
They also scored significantly lower on External Functional ER ($M = 9.44$, $SD = 3.85$) than controls ($M = 14.30$, $SD = 4.15$); $t(52) = 4.45$, $p < 0.05$ (two-tailed). The magnitude of the difference in the means (mean difference = 4.86, 95% CI: 2.67 to 7.04) was very large (eta squared = .27). Participants with eating disorders were lower in Internal Functional ER ($M = 9.63$, $SD = 2.95$) than controls ($M = 12.04$, $SD = 3.02$), $t(52) = 2.95$, $p < 0.05$ (two-tailed). They also scored higher on External Dysfunctional ER ($M = 3.44$, $SD = 3.12$) than controls ($M = 2.55$, $SD = 3.11$); this difference also reached statistical significance: $t(52) = 1.29$, $p < 0.01$ (two-tailed). The magnitude of the difference in the means (mean difference = 0.89, 95% CI: -2.27 to .50) was small (eta squared = .03).

**Affect Intensity and Life Satisfaction (Figure 27):**

Participants with eating disorders were also significantly higher in Negative Affect Intensity ($M = 4.50$, $SD = .64$) than controls ($M = 3.78$, $SD = .53$); $t(52) = 4.51$, $p <$
0.05 (two-tailed). The magnitude of the difference in the means (mean difference = 0.72, 95% CI: -1.04 to -.40) was very large (eta squared = .28).

Figure 27: Differences in Baseline Life Satisfaction, Global Affect Intensity, Negative and Positive Affect Intensity in Participants with Eating Disorders and Controls

They also scored lower on Positive Affect Intensity (M = 3.31, SD = .72) than controls (M = 3.70, SD = .55); t (52) = 2.26, p < 0.05 (two-tailed). Participants with eating disorders were higher on Global Affect Intensity (M = 3.78, SD = .52) than controls (M = 3.72, SD = .44); however this difference did not reach statistical significance: t (52) = .53, p > 0.05 (two-tailed). Participants with eating disorders were significantly lower in Life Satisfaction (M = 14.44, SD = 6.59) than controls (M = 25.93, SD = 5.30); t (38) = 5.47, p < 0.005, (two-tailed). The magnitude of the difference in the means (mean difference = 11.49, 95% CI: 7.23 to 15.72) was very large (eta squared = .44).

Correlations

The relationships between eating pathology, life satisfaction, Affect Intensity and emotion regulation were investigated using Pearson product-moment correlation
The data were checked to ensure no violation of the assumptions of normality, linearity and homoscedasticity. Histograms and scatterplots showed that the scores on the variables were reasonably normally distributed, the relationships between them were linear and the data tended to have similar variability. There was a medium positive correlation between the Overall EDE-Q score and Internal Dysfunctional Emotion Regulation ($r = .42$, $N = 43$, $p < 0.05$), with high levels of eating pathology associated with higher levels of internal dysfunctional emotion regulation.

**Table 23: Correlations between Emotion Regulation, Life Satisfaction, Affect Intensity and EDE-Q facets**

<table>
<thead>
<tr>
<th></th>
<th>Internal Dys. ER</th>
<th>Internal Fun. ER</th>
<th>External Dys. ER</th>
<th>External Fun. ER</th>
<th>Global Affect Intensity</th>
<th>Negative Affect Intensity</th>
<th>Positive Affect Intensity</th>
<th>Overall EDE-Q</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall EDE-Q</td>
<td>.42*</td>
<td>-.17</td>
<td>.19</td>
<td>-.42*</td>
<td>.27</td>
<td>.25</td>
<td>.12</td>
<td>-</td>
</tr>
<tr>
<td>Restraint</td>
<td>.43*</td>
<td>-.17</td>
<td>.21</td>
<td>-.42*</td>
<td>.22</td>
<td>.32</td>
<td>.02</td>
<td>-</td>
</tr>
<tr>
<td>Eating Concern</td>
<td>.35</td>
<td>-.40*</td>
<td>.14</td>
<td>-.65*</td>
<td>.20</td>
<td>.30</td>
<td>.01</td>
<td>-</td>
</tr>
<tr>
<td>Shape Concern</td>
<td>.26</td>
<td>.27</td>
<td>.20</td>
<td>-.15</td>
<td>.06</td>
<td>-.03</td>
<td>.09</td>
<td>-</td>
</tr>
<tr>
<td>Weight Concern</td>
<td>.26</td>
<td>-.07</td>
<td>.08</td>
<td>-.35</td>
<td>.29</td>
<td>.18</td>
<td>.20</td>
<td>-</td>
</tr>
<tr>
<td>Life Satisfaction</td>
<td>-.63*</td>
<td>.36</td>
<td>-.36*</td>
<td>.42*</td>
<td>-.12</td>
<td>-.75*</td>
<td>.34</td>
<td>-.36</td>
</tr>
</tbody>
</table>

*Significant ($p < 0.05$)

ER = Emotion Regulation

The overall EDE-Q scores also negatively correlated with External Functional Emotion regulation ($r = -.42$, $N = 43$, $p < 0.05$). The Overall EDE-Q score did not correlate with External Dysfunctional ER ($r = .19$, $N = 30$, $p = .31$), Global Affect Intensity ($r = .27$, $N = 30$, $p = .15$), Negative Affect Intensity ($r = .25$, $N = 30$, $p = .08$), Positive Affect Intensity ($r = .12$, $N = 30$, $p = .17$) and Internal Functional ER ($r = -.17$, $N = 30$, $p = .38$). Restraint score positively correlated with Internal Dysfunctional ER ($r = .43$, $N = 30$, $p < 0.05$) and negatively with External Functional ER ($r = -.42$, $N = 30$, $p < 0.05$). In addition, Restraint was not associated with Internal Functional ER ($r = -.17$, $N = 30$, $p = .37$), External Dysfunctional ER ($r = .21$, $N = 30$, $p = .26$), Global Affect Intensity ($r = .22$, $N = 30$, $p = .25$), Negative Affect Intensity ($r = .32$, $N = 30$, $p = .08$) and Positive Affect Intensity ($r = .02$, $N = 30$, $p = .89$).
Eating Concern scores were negatively associated with External Functional ER ($r = -0.65, N = 30, p < 0.05$) and Internal Functional ER ($r = -0.40, N = 30, p < 0.05$). Eating Concern was not associated with Internal Dysfunctional ER ($r = 0.35, N = 30, p = 0.06$), External Dysfunctional ER ($r = 0.14, N = 30, p = 0.47$), Global Affect Intensity ($r = 0.22, N = 30, p = 0.29$), Negative Affect Intensity ($r = 0.30, N = 30, p = 0.10$) and Positive Affect Intensity ($r = 0.01, N = 30, p = 0.94$).

Two eating pathology facets (Weight Concern and Shape Concern) did not significantly correlate with emotion regulation factors, Affect Intensity and life satisfaction. Weight Concern was not associated with Internal Dysfunctional ER ($r = 0.26, N = 30, p = 0.16$), External Dysfunctional ER ($r = 0.08, N = 30, p = 0.66$), Global Affect Intensity ($r = 0.29, N = 30, p = 0.12$), Negative Affect Intensity ($r = 0.18, N = 30, p = 0.44$), Positive Affect Intensity ($r = 0.20, N = 30, p = 0.30$), Internal Functional ER ($r = -0.07, N = 30, p = 0.71$) and External Functional ER ($r = -0.35, N = 30, p = 0.60$). Shape Concern scores did not correlate with Internal Dysfunctional ER ($r = 0.26, N = 30, p = 0.17$), Internal Functional ER ($r = 0.27, N = 30, p = 0.15$), External Dysfunctional ER ($r = -0.20, N = 30, p = 0.29$), Global Affect Intensity ($r = 0.06, N = 30, p = 0.77$), Positive Affect Intensity ($r = 0.09, N = 30, p = 0.65$), External Functional ER ($r = -0.15, N = 30, p = 0.43$) and Negative Affect Intensity ($r = -0.03, N = 30, p = 0.86$).

Life Satisfaction negatively correlated with Internal Dysfunctional ER ($r = -0.63, N = 30, p < 0.05$), External Dysfunctional ER ($r = -0.36, N = 30, p < 0.05$), as well as Negative Affect Intensity ($r = -0.75, N = 30, p < 0.05$) and positively with External Functional ER ($r = 0.42, N = 30, p < 0.05$). The correlation between life satisfaction and Overall EDE-Q did not reach significance ($r = -0.36, N = 30, p = 0.05$). Life Satisfaction was not associated with Internal Functional ER ($r = 0.36, N = 30, p = 0.05$), Positive Affect Intensity ($r = 0.34, N = 30, p = 0.07$) and Global Affect Intensity ($r = -0.12, N = 30, p = 0.51$). As Table 24 shows, Global Affect Intensity was not associated with Internal Dysfunctional ER ($r = 0.15, N = 43, p = 0.39$), Internal Functional ER ($r = 0.19, N = 43, p = 0.21$), External Dysfunctional ER ($r = 0.27, N = 43, p = 0.08$) and External Functional ER ($r = 0.23, N = 43, p = 0.14$).
Table 24: Correlations between Emotion Regulation and Affect Intensity

<table>
<thead>
<tr>
<th></th>
<th>Internal Dysfunctional ER</th>
<th>Internal Functional ER</th>
<th>External Dysfunctional ER</th>
<th>External Functional ER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Global Affect Intensity</td>
<td>.15</td>
<td>.19</td>
<td>.27</td>
<td>.23</td>
</tr>
<tr>
<td>Negative Affect Intensity</td>
<td><strong>.66</strong>*</td>
<td>-.38*</td>
<td><strong>.45</strong>*</td>
<td>-.22</td>
</tr>
<tr>
<td>Positive Affect Intensity</td>
<td>-.21</td>
<td><strong>.43</strong>*</td>
<td>0.15</td>
<td><strong>.36</strong>*</td>
</tr>
</tbody>
</table>

*Significant (p < 0.05)
ER = Emotion Regulation

The relationships between Affect Intensity (Global, Negative and Positive) and Emotion Regulation was also examined (Table 24). There was no relationship between Global Affect Intensity and emotion regulation, however, Negative Affect Intensity positively correlated with Internal Dysfunctional ER (r = .66, N = 43, p < 0.05) and External Dysfunctional ER (r = .45, N = 43, p < 0.05), and negatively with Internal Functional ER (r = -.38, N = 43, p < 0.05). Positive Affect Intensity positively correlated with Internal Functional ER (r = .43, N = 43, p < 0.05) and External Functional ER (r = .36, N = 43, p < 0.05).

Negative Affect Intensity was negatively correlated with External Functional ER (r = - .22, N = 43, p = .16), but this relationship did not reach statistical significance. Positive Affect Intensity negatively correlated with Internal Dysfunctional ER (r = -.21, N = 43, p = .17) and positively with External Dysfunctional ER (r = .15, N = 43, p = .92). However, these correlations did not reach statistical significance.

**Mixed ANOVAs**
A series of mixed between-within subjects analyses of variance was conducted to assess the impact of two different interventions (Gratitude and Neutral) on participants’ scores on the facets of the Regulation of Emotions Questionnaire (ER) and Life Satisfaction Scale at two time periods: pre- and post-intervention.
### Emotion Regulation

Table 25: Regulation of Emotion and Life Satisfaction scores in the full sample before and after the Gratitude and Neutral Interventions

<table>
<thead>
<tr>
<th></th>
<th>Gratitude</th>
<th></th>
<th>Neutral</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Mean</td>
<td>SD</td>
<td>N</td>
</tr>
<tr>
<td><strong>Internal Dysfunctional ER</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Pre-Intervention</td>
<td>22</td>
<td>9.5</td>
<td>4.80</td>
<td>21</td>
</tr>
<tr>
<td><strong>Internal Functional ER</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Pre-Intervention</td>
<td>22</td>
<td>11.5</td>
<td>3.41</td>
<td>21</td>
</tr>
<tr>
<td>2. Post-Intervention</td>
<td>22</td>
<td>12</td>
<td>3.45</td>
<td>21</td>
</tr>
<tr>
<td><strong>External Dysfunctional ER</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Pre-Intervention</td>
<td>22</td>
<td>4.09*</td>
<td>3.04</td>
<td>21</td>
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<td>22</td>
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<tr>
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</tr>
<tr>
<td>2. Post-Intervention</td>
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<td>5.65</td>
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<td><strong>Life Satisfaction</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Pre-Intervention</td>
<td>17</td>
<td>19.12</td>
<td>7.86</td>
<td>13</td>
</tr>
<tr>
<td>2. Post-Intervention</td>
<td>17</td>
<td>21.00</td>
<td>8.05</td>
<td>13</td>
</tr>
</tbody>
</table>

*Significant (p < 0.01)

ER = Emotion Regulation, N = Number (of participants), SD = Standard Deviation

**External Dysfunctional ER (Table 25 and Figure 28):**

There was a significant main effect for time, Wilks’ Lambda = .71, F (1, 41) = 16.97, p < 0.01, partial eta squared = .29, with both the Gratitude group (Time 1: M = 4.09, SD = 3.04; Time 2: M = 3.36, SD = 2.87) and Neutral group (Time 1: M = 2.33, SD = 2.06; Time 2: M = 1.86, SD = 1.77) showing a reduction in External Dysfunctional scores post-intervention.

There was no significant interaction between intervention type and time, Wilks’ Lambda = .98, F (1, 41) = .74, p > 0.05, partial eta squared = .02. The between-group differences were significant, F (1, 41) = 4.74, p < 0.05, partial eta squared = .10.
Figure 28: External Dysfunctional emotion regulation scores pre- and post-interventions

Time 1: before mood induction, Time 2: after mood induction

Internal Dysfunctional ER (Table 25 and Figure 29):
There was no significant main effect for time, Wilks’ Lambda = 1, F (1, 41) = .002, p > 0.05, partial eta squared = 0. There was no significant interaction between intervention type and time, Wilks’ Lambda = .92, F (1, 41) = 3.67, p > 0.05, partial eta squared = .08, although it was approaching significance (p = .06). InDys scores in the Gratitude condition declined (Time 1: M = 9.5, SD = 4.80; Time 2: M = 9.14, SD = 5.18), whereas those in the Neutral condition increased (Time 1: M = 9, SD = 3.02; Time 2: M = 9.38, SD = 3.40). The between-group differences were non-significant, F (1, 41) = .01, p > 0.05, partial eta squared = 0.
Figure 29: Internal Dysfunctional emotion regulation scores pre- and post-Interventions

Internal Functional ER (Table 25 and Figure 30): There was no significant main effect for time, Wilks’ Lambda = .97, F (1, 41) = 1.43, p > 0.05, partial eta squared = .03. Figure 28 shows that both in the Gratitude group (Time 1: M = 11.5, SD = 3.42; Time 2: M = 12, SD = 3.45) and Neutral group (Time 1: M = 10.57, SD = 2.71; Time 2: M = 10.62, SD = 3.18) InFun scores increased post-induction. There was no significant interaction between intervention type and time, Wilks’ Lambda = .98, F (1, 41) = .97, p > 0.05, partial eta squared = .02. The between-group differences were non-significant, F (1, 41) = .23, p > 0.05, partial eta squared = .03.
Figure 30: Internal Functional emotion regulation scores pre- and post-interventions

Time 1: before mood induction, Time 2: after mood induction

External Functional ER (Table 26 and Figure 31):
There was no significant main effect for time, Wilks’ Lambda = .99, F (1, 41) = .31 p > 0.05, partial eta squared = .01, Figure 29 shows that ExFun scores in the Gratitude group (Time 1: M = 11.86, SD = 5.47; Time 2: M = 11.86, SD = 5.65) remained the same post-induction, whereas those in the Neutral group (Time 1: M = 12.05, SD = 3.48; Time 2: M = 11.81, SD = 3.61) decreased. There was no significant interaction between intervention type and time, Wilks’ Lambda = .99, F (1, 41) = .31, p > 0.05, partial eta squared = .01. The between-group differences were also non-significant, F (1, 41) = 0, p > 0.05, partial eta squared = 0.
Life Satisfaction (Table 25 and Figure 32):

There were no significant main effects for time, Wilks’ Lambda = .91, F (1, 28) = 2.91, p = .10, partial eta squared = .09. The interaction between time and mood was approaching statistical significance, Wilks’ Lambda = .87, F (1, 28) = 4.04, p = .05, partial eta squared = .13. There was an increase in Life Satisfaction scores in the happiness condition (Time 1: M = 19.12, SD = 8.11; Time 2: M = 21, SD = 8.05), whereas Life Satisfaction slightly decreased in neutral condition (Time 1: M = 15.61, SD = 8.11; Time 2: M = 15.46, SD = 8.86). The between-group differences were not significant, F (1, 28) = 2.31, p = .14, partial eta squared = .08. It has to be noted that changes in Life Satisfaction in the whole sample (N = 30), were approaching statistical significance (Table 25 and Fig. 33). Perhaps, in a study with a larger sample size the effects of mood on satisfaction with life would have reached statistical significance.
Figure 32: Life Satisfaction Scores Pre- and Post- Mood Induction.

Unlike previous studies which looked at the effects of mood induction in the full sample (i.e. both in those with eating pathology and those without), here the effects of mood induction on emotion regulation and life satisfaction are also explored separately in the clinical and control samples. Despite the small participant numbers in the groups, it was decided to be a worthwhile approach that suited the exploratory nature of the study.

**Eating-Disordered Sample and Controls**

Unlike previous studies which looked at the effects of mood induction in the full sample (i.e. both in those with eating pathology and those without), here the effects of mood induction on emotion regulation and life satisfaction are also explored separately in the clinical and control samples. Despite the small participant numbers in the groups, it was decided to be a worthwhile approach that suited the exploratory nature of the study.

**Eating-Disordered Participants**

Table 26 presents the data on the effect of the Gratitude and neutral interventions on emotion regulation (Internal Dysfunctional ER, Internal Functional ER, External
Dysfunctional ER and External Functional ER) and life satisfactions in the eating-disordered sample.

Table 26: Regulation of Emotion and Life Satisfaction Scores in the Eating-Disordered Sample Before and After the Gratitude and Neutral Interventions

<table>
<thead>
<tr>
<th></th>
<th>Gratitude</th>
<th>Neutral</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Internal Dysfunctional ER</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time Period</td>
<td>N</td>
<td>Mean</td>
</tr>
<tr>
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</tr>
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<td>2. Post-Intervention</td>
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</tr>
<tr>
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<tr>
<td>Time Period</td>
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<td>Mean</td>
</tr>
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<tr>
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<td></td>
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<tr>
<td>Time Period</td>
<td>N</td>
<td>Mean</td>
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<td>4.17*</td>
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<td><strong>External Functional ER</strong></td>
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<td></td>
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<tr>
<td>Time Period</td>
<td>N</td>
<td>Mean</td>
</tr>
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<td>2. Post-Intervention</td>
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<tr>
<td><strong>Life Satisfaction</strong></td>
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<tr>
<td>Time Period</td>
<td>N</td>
<td>Mean</td>
</tr>
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<td>1. Pre-Intervention</td>
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<td>15.83</td>
</tr>
<tr>
<td>2. Post-Intervention</td>
<td>12</td>
<td>17.58</td>
</tr>
</tbody>
</table>

*Significant (p < 0.05)
ER = Emotion Regulation, N = Number (of participants), SD = Standard Deviation

Internal Dysfunctional emotion regulation:
There were no significant main effects for time, Wilks’ Lambda = .91, F (1, 20) = 1.88, p > 0.05, partial eta squared = .09. The interaction between time and mood was also non-significant, Wilks’ Lambda = .97, F (1, 20) = .60, p > 0.05, partial eta squared = .03. There was a slight increase in InDys scores in the happiness condition (Time 1: M = 12.25, SD = 4.30; Time 2: M = 12.41, SD = 4.50), and in the neutral condition (Time 1: M = 10.30, SD = 2.90; Time 2: M = 10.90, SD = 3.54), as Table 26 shows. The between-group differences were not significant, F (1, 20) = 1.09, p > 0.05, partial eta squared = .05.

Internal Functional emotion regulation:
There were no significant main effects for time, Wilks’ Lambda = .91, F (1, 20) = 1.9, p > 0.05, partial eta squared = .09. The interaction between time and mood was also non-significant, Wilks’ Lambda = .91, F (1, 20) = 1.90, p > 0.05, partial eta squared = .09. In
the eating-disordered sample, Internal Functional ER increased in the Gratitude condition (Time 1: M= 10.50, SD= 2.84; Time 2: M= 11.25, SD= 2.93) and stayed the same in the Neutral condition (Time 1: M = 9.10, SD = 1.97; Time 2: M = 9.10, SD = 2.28), as Table 26 shows. The between-group differences were not significant, F (1, 20) = 2.76, p > 0.05, partial eta squared = .12.

External Dysfunctional emotion regulation:
There was a significant main effect for time, Wilks’ Lambda = .82, F (1, 20) = 4.52, p < 0.05, partial eta squared = .18. External Dysfunctional ER scores decreased both in the Gratitude (Time 1: M = 4.75, SD = 3.52; Time 2: M = 4.17, SD = 3.24) and the Neutral (Time 1: M = 2.50, SD = 2.71; Time 2: M = 2.10, SD = 2.28) conditions, as Figure 33 and Table 26 show. The interaction between time and mood was non-significant, Wilks’ Lambda = .99, F (1, 20) = .16 p > 0.05, partial eta squared = .01. The between-group differences were not significant, F (1, 20) = 2.87, p > 0.05, partial eta squared = .13. Because the numbers are small, differences in the effects of the two conditions failed to reach statistical significance. However, there is an indication of where those differences are. Changes in the External Dysfunctional ER scores from Time 1 to Time 2 were larger in the Gratitude condition (mean difference = 0.58), than in the Neutral condition (mean difference = .40).
External Functional emotion regulation:
There were no significant main effects for time, Wilks’ Lambda = .82, F (1, 20) = 4.42, p > 0.05, partial eta squared = .18. The interaction between time and mood was also non-significant, Wilks’ Lambda = .98, F (1, 20) = .41, p > 0.05, partial eta squared = .02. In the eating-disordered sample, External Functional ER decreased both in the Gratitude condition (Time 1: M = 9.33, SD = 4.79; Time 2: M = 8.58, SD = 4.12) and the Neutral condition (Time 1: M = 9.90, SD = 2.33; Time 2: M = 9.50, SD = 2.32), as Table 26 shows. The between-group differences were not significant, F (1, 20) = .23, p > 0.05, partial eta squared = .01.

Life Satisfaction:
There were no significant main effects for time, Wilks’ Lambda = .87, F (1, 20) = 3.09, p > 0.05, partial eta squared = .13. The interaction between time and mood was also
non-significant, Wilks’ Lambda = .93, F (1, 20) = 1.55, p > 0.05, partial eta squared = .07. In the eating-disordered sample, Life Satisfaction increased both in the Gratitude condition (Time 1: M = 15.83, SD = 6.67; Time 2: M = 17.58, SD = 7.10) and the Neutral condition (Time 1: M = 14.10, SD = 7.72; Time 2: M = 14.40, SD = 8.63), as Table 26 shows. The between-group differences were also not significant, F (1, 20) = .60, p > 0.05, partial eta squared = .03.

Non-eating disordered participants

Table 27 presents the data on the effect of the Gratitude and neutral interventions on emotion regulation (Internal Dysfunctional ER, Internal Functional ER, External Dysfunctional ER and External Functional ER) and life satisfactions in the control sample.

Table 27: Regulation of Emotion and Life Satisfaction Scores in the Control Sample Before and After the Gratitude and Neutral Interventions

<table>
<thead>
<tr>
<th></th>
<th>Gratitude</th>
<th>Neutral</th>
</tr>
</thead>
<tbody>
<tr>
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<td>N  Mean  SD</td>
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<td>12 5.2* 2.53</td>
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<td>5 29.20 1.30</td>
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*Significant (p < 0.05)

ER = Emotion Regulation, N = Number (of participants), SD = Standard Deviation

Internal Dysfunctional emotion regulation:
There were no significant main effects for time, Wilks’ Lambda = .87, F (1, 19) = 2.82, p > 0.05, partial eta squared = .13. The interaction between time and mood was
statistically significant, Wilks’ Lambda = .76, F (1, 19) = 5.88, p < 0.05, partial eta squared = .24. As Figure 34 and Table 27 show, there was a decrease in InDys scores in the happiness condition (Time 1: M = 6.2, SD = 2.97; Time 2: M = 5.2, SD = 2.53), whereas in neutral condition InDys scores slightly increased (Time 1: M = 7.81, SD = 2.71; Time 2: M = 8, SD = 2.72). The between-group differences were not significant, F (1, 19) = 3.56, p < 0.05, partial eta squared = .16.

Figure 34: Internal Dysfunctional emotion regulation scores pre- and post-intervention in the control sample

Internal Functional emotion regulation:
There were no significant main effects for time, Wilks’ Lambda = .99, F (1, 19) = 0.14, p > 0.05, partial eta squared = .01. The interaction between time and mood was also non-significant, Wilks’ Lambda = .10, F (1, 19) = 0.02, p > 0.05, partial eta squared = .09. In the control sample, Internal Functional ER increased both in the Gratitude condition (Time 1: M = 12.70, SD = 3.80; Time 2: M = 12.90, SD = 3.95) and the
Neutral condition (Time 1: M = 11.91, SD = 2.66; Time 2: M = 12, SD = 3.35), as Table 27 shows. The between-group differences were not significant, F (1, 19) = .33, p > 0.05, partial eta squared = .02.

External Dysfunctional emotion regulation:
There was a significant main effect for time, Wilks’ Lambda = .55, F (1, 19) = 15.26, p < 0.05, partial eta squared = .44. External Dysfunctional ER scores decreased both in the Gratitude (Time 1: M = 3.30, SD = 2.26; Time 2: M = 2.40, SD = 2.12) and the Neutral (Time 1: M = 2.18, SD = 1.32; Time 2: M = 1.64, SD = 1.21) conditions, as Figure 35 shows. The interaction between time and intervention was non-significant, Wilks’ Lambda = .95, F (1, 19) = .92, p > 0.05, partial eta squared = .05. The between-group differences were not significant, F (1, 19) = 1.57, p > 0.05, partial eta squared = .08. Because the numbers are small, differences in the effects of the two conditions failed to reach statistical significance. However, there is an indication of where those differences are. Changes in the External Dysfunctional ER scores from Time 1 to Time 2 were larger in the Gratitude condition (mean difference = 0.90), than in the Neutral condition (mean difference = 0.54).
External Dysfunctional emotion regulation scores pre- and post-intervention in the control sample

There were no significant main effects for time, Wilks’ Lambda = .90, F (1, 19) = 2.05, p > 0.05, partial eta squared = .01. The interaction between time and mood was also non-significant, Wilks’ Lambda = .86, F (1, 19) = 3.15, p > 0.05, partial eta squared = .14. In the control sample, External Functional ER increased in the Gratitude condition (Time 1: M = 14.90, SD = 4.79; Time 2: M = 15.80, SD = 4.71) and decreased in the Neutral condition (Time 1: M = 14, SD = 3.25; Time 2: M = 13.91, SD = 3.33), as Table 27 shows. The between-group differences were not significant, F (1, 19) = .64, p > 0.05, partial eta squared = .03.

Life Satisfaction:
There were no significant main effects for time, Wilks’ Lambda = .99, F (1, 6) = 0.06, p > 0.05, partial eta squared = .01. The interaction between time and mood was also non-
significant, Wilks’ Lambda = .66, F (1, 6) = 3.15, p > 0.05, partial eta squared = .34. In
the control sample, Life Satisfaction increased in the Gratitude condition (Time 1: M =
27, SD = 3.81; Time 2: M = 29.20, SD = 1.30) and decreased in the Neutral condition
(Time 1: M = 20.67, SD = 8.74; Time 2: M = 19, SD = 10.53), as Table 27 shows.
The between-group differences were not significant, F (1, 6) = 3.72, p > 0.05, partial eta
squared = .38.

Main findings (Study 4)

1. Participants in the clinical sample:
   a) had significantly higher levels of baseline negative affect (Sadness, Anger, Guilt,
      Anxiety, Fear and Disgust) and significantly lower levels of baseline positive affect
      (Happiness) than controls.
   b) were significantly more likely to manage their emotions in a dysfunctional way
      (Internal Dysfunctional ER) and less likely to use Functional ER (Internal and
      External) strategies than controls.
   c) reported significantly higher Negative Affect Intensity and lower Positive Affect
      Intensity, as well as lower Life Satisfaction than controls.

2. Eating pathology and the EDE-Q facets were positively related to Internal
   Dysfunctional ER and negatively to Functional ER (External and Internal). This
   suggests that individuals with eating disorders may be more likely to use dysfunctional
   emotion regulation strategies when attempting to regulate their emotions.

3. Negative Affect Intensity positively correlated with Internal Dysfunctional ER and
   negatively with Internal Functional ER; whereas Positive Affect Intensity positively
   correlated with functional ER (External and Internal). This may mean that individuals
   who are high in negative Affect Intensity and low in positive Affect Intensity may be
   more likely to use dysfunctional emotion regulation mechanisms. Those who are high in
   positive Affect Intensity may be more inclined to choose healthy emotion management
   strategies.
4. The Gratitude intervention led to a reduction in Internal Dysfunctional scores (in the control sample only) and in External Dysfunctional scores (in both clinical and control samples). It had no effect on Life Satisfaction, Internal and External Functional scores. The one-off Gratitude intervention was successful in reducing both internal and external dysfunctional emotion regulation scores in the control sample. However, in the clinical sample it only positively affected external dysfunctional scores.
CHAPTER 8: Study 5: The effects of longitudinal positive emotion induction on emotion regulation in an eating-disordered sample.

INTRODUCTION

Study 5 looked at how happiness induction *over a period of time* influenced emotion regulation, eating behaviours and life satisfaction in eating-disordered individuals. It aimed to establish whether systematic happiness induction over four weeks had a sustained positive effect on emotion regulation, life satisfaction and disordered eating, compared to neutral mood. Study 5 also contained a qualitative component: the participants were invited to give written feedback on the gratitude intervention and to write about their experience of positive and negative affect (e.g. frequency, intensity and context).

METHOD

Participants and Recruitment

Only individuals with eating disorders took part in this study (N = 27, mean age = 29 years). The sample was exclusively female and largely consisted from the same participants who participated in Study 4 (bulimia nervosa N = 20; anorexia nervosa N = 7). Out of the total 40 participants that had originally given consent to take part in Study 5, only 27 followed the study through from the beginning to the end, and only these cases were included in the study. Participants were all pro-recovery (i.e. motivated to recover from their eating disorders), and they received no financial incentive for participation in the study. Twenty six participants were Caucasian, and one was of Latin American ethnicity. All were British and native English speakers. Out of the 27 participants, 16 were full-time university students, 10 were in employment and one was retired. Mean scores on emotion regulation and life satisfaction were computed for the sample. The participants scored on average 11.48 (SD = 3.59) on Internal Dysfunctional emotion regulation; 8.89 (SD = 2.75) on Internal Functional affect management; 3.44 (SD = 3.12) on External Dysfunctional emotion regulation and 8.81 (SD = 3.42) on External Functional emotion regulation. Their mean score on Global Affect Intensity
was 3.78 (SD = .52); 3.31 (SD = .72) on Positive Affect Intensity; and 4.50 (SD = .64) on Negative Affect Intensity.

**Design**

Study 5 had a mixed design; there was one experimental group (Happy mood) and one control group (Neutral mood) who completed questionnaire sets on three separate occasions over one month. Figure 36 is showing the flow and attrition of participants in the study in more detail.

*Figure 36: A consort diagram showing the flow and attrition of participants in Study 5*
**Ethics**

Study 5 received ethical approval from the School of Health in Social Science Ethics Committee (University of Edinburgh). Both studies strictly observed the British Psychological Society's Code of Conduct. Prior to obtaining informed consent, all the participants were given detailed information about the aims and procedures of the studies, the opportunity to contact the author with any questions, and were invited to consult their clinician about their wish to participate in the studies [Information Sheet, Appendix 12]. They were informed about their right to withdraw at any time without giving a reason and with no consequence to them [Informed Consent Sheet, Appendix 11], as well as told about their right to anonymity and confidentiality. In Study 5, participants were encouraged to leave feedback on the interventions, and were given ongoing support by e-mail and phone throughout the month to ensure they were happy with the procedure. Following the completion of the studies, participants received thank you cards and an information sheet about positive psychology interventions to thank them for their contribution. Individual feedback was not given unless specifically requested by the participants. All the data collected during the studies were kept anonymous and confidential at all times.

**Procedure**

The data in Study 5 were collected by post. Questionnaire packs containing mood induction tasks and relevant scales were put together and posted to the participants for completion. Participants sent completed questionnaire packs back to the author using pre-paid addressed envelopes. In this study, 16 eating-disordered participants were asked to do the Gratitude exercise every week for 4 weeks. The other 11 were in the control group and did a neutral writing task over the same period of time. The procedure was similar to that of Study 4. The participants were asked to strictly observe the order of the sections and to return filled-out questionnaire packs within 1 week of receipt. At Week 1, the participants completed the first pack of questionnaires. At Week 2, they filled out the second pack and at Week 4 - the third. At Week 3, the participants were asked to do the gratitude exercise and Emotional Assessment Scale (Carlson et al., 1989) before and after it. Questionnaire packs were posted weekly upon the receipt of
the filled-out questionnaire packs. None of the participants were paid for their participation.

Prior to starting the interventions, all the participants filled in measures, assessing severity of their eating disorder (EDE-Q; Fairburn and Beglin, 1994), Affect Intensity (AIM; Larsen, 1984), emotion regulation (REQ; Philips and Power, 2007) and life satisfaction (SWLS; Diener, 1985). Participants’ happiness levels were monitored weekly. They were asked to spend at least 15 minutes at a time on the writing exercises (per week). The Gratitude intervention was aimed at getting the participants to focus on positive life experiences and to maximise the frequency of positive affect in their daily lives. Following the activity, participants were asked to rate their mood on the EAS scale (Carlson et al., 1989). For both groups emotion regulation, life satisfaction and eating pathology were assessed immediately post-induction, and then again in 2-weeks’ and in 4-weeks’ time. Participants’ mean eating pathology score on the EDE-Q (Fairburn and Beglin, 1994) was 3.05, (SD = 1.29). Participants were encouraged to leave feedback on the interventions, and were invited to answer the following questions: ‘Do you experience positive emotions (e.g. joy, contentment)? If yes, how frequent are they? In what context do you experience them? In other words, what makes you happy? Please feel free to write about anything else that may be relevant to your experience of emotions’.

Measures

1. The Eating Disorder Examination Questionnaire (EDE-Q; Fairburn and Beglin, 1994) assesses eating pathology. It is a 36-item measure, assessing frequency of disordered eating behaviours, thoughts and concerns over the past 28 days. There are 4 subscales: Restraint, Weight Concern, Eating Concern and Shape Concern; they can be summed to give the overall score. [Appendix 4]

2. Regulation of Emotion Questionnaire (REQ; Phillips and Power, 2007) measures functionality of affect management along four subscales (Internal Dysfunctional, Internal Functional, External Dysfunctional and External Functional emotion management). It is a 21-item questionnaire with statements
about different emotion regulation strategies like ‘I ask others for advice’ with a frequency scale from *Never* to *Always*. The respondent is asked how often they use certain regulatory strategies: ‘In general, how do you respond to your emotions?’ In Study 4 participants completed this questionnaire in its original form prior to mood induction. Because this questionnaire measures a general tendency to regulate emotions in particular ways at present, it had to be adapted to meaningfully capture any change post mood induction. Therefore, after mood manipulation, participants completed the adapted version of the Regulation of Emotion Questionnaire. The instructions focused on the future intentions and likelihood of using certain regulatory strategies, and the question was changed to: ‘How will you respond to your emotions?’ All the statements were adapted to refer to the future (e.g. ‘I shall talk to someone about how I feel’ instead of ‘I talk to someone about how I feel’), and the response scale (from *Never* to *Always*) remained the same. In Study 5 only the adapted version of the questionnaire was used. [Appendix 1]

3. The Satisfaction with Life Scale (Diener, 1985) measures subjective well-being. It is a 5-item questionnaire, and respondents are asked to rate their agreement with statements such as ‘The conditions of my life are excellent’ from *Strongly Disagree* to *Strongly Agree*. [Appendix 3]

4. The Emotion Assessment Scale (EAS; Carlson et al., 1989) measures current mood and assesses effectiveness of mood induction. It is a list of 24 mood adjectives such as ‘Happy’ or ‘Disgusted’, and the participants are asked to indicate the extent to which they experience those emotions by putting a slash on the line that goes from *Least Likely* to *Most Likely*. The answers are then added up to make up a score for each of the eight emotions (Happiness, Sadness, Disgust, Fear, Guilt, Surprise, Anxiety and Anger). [Appendix 9]
5. Mood Induction

The Gratitude journal method was used to induce positive mood. Participants were asked to spend around 10-15 minutes thinking about and listing the things in their life that they are grateful for: ‘Please spend a few minutes thinking of things in your life that you are grateful for. These might include particular supportive relationships, sacrifices or contributions that others have made for you, facts about your life such as your advantages and opportunities, or even gratitude for life itself, and the world we live in. The gratitude could refer to the past, the present or the future. It can be about anything, even little things. In all of these cases you are identifying previously unappreciated aspects of your life, for which you can be thankful. You may not have thought about yourself in this way before, but research suggests that doing so can have a strong positive effect on your life satisfaction. Please use the space below to write about the things you are thankful for”’ (participant instructions, based on Emmons and McCullough, 2003).

In the neutral condition, participants were asked to pay more attention to daily happenings in their life over the period of the study (longitudinal mood induction): ‘You have been randomly assigned to pay more attention to the daily details of your life. ‘Pay more attention to your life’ means that you take notice of the ordinary details of your life that you wouldn’t typically think about. These might include particular classes or meetings you attend, typical interactions with acquaintances, typical thoughts that you have during the day or your typical schedule as you move through the day. In all of these cases, you may be helped to better identify problem areas in your life, and to take action to change them’ (participant instructions, based on Sheldon and Lyubomirsky, 2006).

RESULTS

Data screening procedures were performed. The data were checked for accuracy by computing frequencies and minimum/maximum statistics for the variables using
Statistical Package for the Social Sciences (SPSS). Visual checking of the resultant analyses against the questionnaire data ensured that there were no errors in the data file, and all the scores fell within the range of possible scores for the scales. Skewness, kurtosis and z-scores were also computed for each dependent variable (please see Appendix 17). All z-scores fell below 3.3, which indicated reasonably good normal distribution. Visual checking of histograms and scatterplots confirmed that the scores on the dependent variables were reasonably normally distributed, the relationships between them were linear and the data tended to have similar variability. As part of the t-tests, Levene’s test was performed in order to ensure homogeneity of variance; in case of all the variables the Levene test was non-significant ($p > 0.05$). The cases that have missing values were excluded from analyses that included that variable.

The results show that the Gratitude intervention was effective in inducing positive mood in 13 out of 16 cases at Week 1 (81%), in 13 out of 16 at Week 2 (81%), in 12 out of 14 at Week 3 (approximately 86%) and in 11 out of 14 at Week 4 (78.5%). As can be seen from Table 28, at Week 1, average happiness score was 6.64 (SD = 4.83); immediately after the gratitude task, the happiness average rose to 11.92 (SD = 6.78). At Week 2, participants scored 5.81 on happiness (SD = 4.56), and their happiness average increased to 11.25 (SD = 5.83) following the gratitude task. At Week 3, participants scored on average 6.99 (SD = 4.80) on happiness before mood induction, and after mood induction, their happiness levels increased to an average of 11.52 (SD = 5.35). At Week 4, their happiness level was on average 8.54 (SD = 6.65), and it rose to 12.84 (SD = 7.07) after they did the gratitude task. Therefore, eating-disordered participants experienced an increase of baseline happiness by approximately 2 points, if we compare Week 1 (Mean = 6.64) to Week 4 (Mean = 8.54). The Gratitude exercise increased their immediate post-test positive affect almost two-fold; increase in happiness immediately post-test was as follows - Week 1: 1.7 times, Week 2: 1.9 times, Week 3: 1.6 times, Week 4: 1.5 times).
Table 28: Descriptive Statistics for Happiness Scores

<table>
<thead>
<tr>
<th>Condition</th>
<th>N</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gratitude/Happiness</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-Induction Happiness – Week 1</td>
<td>16</td>
<td>.20</td>
<td>17.40</td>
<td>6.64</td>
<td>4.83</td>
</tr>
<tr>
<td>Post-Induction Happiness – Week 1</td>
<td>16</td>
<td>3</td>
<td>24</td>
<td>11.92</td>
<td>6.78</td>
</tr>
<tr>
<td>Pre-Induction Happiness – Week 2</td>
<td>15</td>
<td>1.20</td>
<td>14.50</td>
<td>5.81</td>
<td>4.56</td>
</tr>
<tr>
<td>Post-Induction Happiness – Week 2</td>
<td>15</td>
<td>1.20</td>
<td>18.70</td>
<td>11.25</td>
<td>5.83</td>
</tr>
<tr>
<td>Pre-Induction Happiness – Week 3</td>
<td>14</td>
<td>1.20</td>
<td>17.40</td>
<td>6.99</td>
<td>4.80</td>
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<tr>
<td>Post-Induction Happiness – Week 3</td>
<td>14</td>
<td>4.90</td>
<td>18.80</td>
<td>11.52</td>
<td>5.35</td>
</tr>
<tr>
<td>Pre-Induction Happiness – Week 4</td>
<td>14</td>
<td>1.20</td>
<td>20.70</td>
<td>8.54</td>
<td>6.65</td>
</tr>
<tr>
<td>Post-Induction Happiness – Week 4</td>
<td>14</td>
<td>.60</td>
<td>27.00</td>
<td>12.84</td>
<td>7.07</td>
</tr>
</tbody>
</table>

N = Number (of participants), SD = Standard Deviation

Effects of emotion induction on emotion regulation

A series of mixed between-within subjects analyses of variance was conducted to assess the impact of two different interventions (Gratitude and Neutral) on participants’ scores on the facets of the Regulation of Emotions Questionnaire (ER) across three time periods: pre-intervention, 2-week follow-up and 4-week follow-up (Table 29). The data were checked to ensure no violation of the assumptions of normality, linearity and homoscedasticity. Histograms and scatterplots showed that the scores on the variables were reasonably normally distributed, the relationships between them were linear and the data tended to have similar variability.

The Gratitude Condition

For Internal Dysfunctional scores (InDys), there was a significant interaction between intervention type and time, Wilks Lambda = .38, F (2, 24) = 19.55, p < 0.05, partial eta squared = .62, with the gratitude group showing a steady significant reduction in InDys scores across three time periods (Time 1: M = 11.88, SD = 3.76; Time 2: M = 10.75, SD = 3.47; Time 3: M = 8, SD = 3.12; p < 0.05).
Table 29: Regulation of Emotion Questionnaire scores for the Gratitude and Neutral Interventions across three time periods

<table>
<thead>
<tr>
<th></th>
<th>Gratitude Mean</th>
<th>Neutral Mean</th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Internal Dysfunctional ER</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Pre-Intervention</td>
<td>16 11.88*</td>
<td>11 10.91</td>
<td>16</td>
<td>3.76</td>
<td>3.5</td>
<td>11</td>
<td>3.4</td>
<td>3.12</td>
</tr>
<tr>
<td>2. 2-week follow up</td>
<td>16 10.75*</td>
<td>11 11.36</td>
<td>16</td>
<td>3.47</td>
<td>2.9</td>
<td>11</td>
<td>3.06</td>
<td>3.12</td>
</tr>
<tr>
<td>3. 4-week follow up</td>
<td>16 8*</td>
<td>11 11.64</td>
<td>16</td>
<td>3.12</td>
<td>3.12</td>
<td>11</td>
<td>3.12</td>
<td>3.12</td>
</tr>
<tr>
<td><strong>Internal Functional ER</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Pre-Intervention</td>
<td>16 9.06*</td>
<td>11 8.64</td>
<td>16</td>
<td>3.02</td>
<td>2.42</td>
<td>11</td>
<td>3.64</td>
<td>3.12</td>
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<tr>
<td>2. 2-week follow up</td>
<td>16 10.31*</td>
<td>11 8.36</td>
<td>16</td>
<td>2.96</td>
<td>3.12</td>
<td>11</td>
<td>3.27</td>
<td>3.12</td>
</tr>
<tr>
<td>3. 4-week follow up</td>
<td>16 10.13</td>
<td>11 8.55</td>
<td>16</td>
<td>2.94</td>
<td>2.66</td>
<td>11</td>
<td>3.12</td>
<td>2.66</td>
</tr>
<tr>
<td><strong>External Dysfunctional ER</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Pre-Intervention</td>
<td>16 4.19*</td>
<td>11 2.36</td>
<td>16</td>
<td>3.29</td>
<td>2.67</td>
<td>11</td>
<td>3.12</td>
<td>2.77</td>
</tr>
<tr>
<td>2. 2-week follow up</td>
<td>16 2.75*</td>
<td>11 2.27</td>
<td>16</td>
<td>2.21</td>
<td>2.41</td>
<td>11</td>
<td>2.27</td>
<td>2.27</td>
</tr>
<tr>
<td>3. 4-week follow up</td>
<td>16 1.88*</td>
<td>11 2.18</td>
<td>16</td>
<td>1.4</td>
<td>2.27</td>
<td>11</td>
<td>2.18</td>
<td>2.27</td>
</tr>
<tr>
<td><strong>External Functional ER</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Pre-Intervention</td>
<td>16 7.81*</td>
<td>11 10.27</td>
<td>16</td>
<td>3.66</td>
<td>2.53</td>
<td>11</td>
<td>3.54</td>
<td>2.53</td>
</tr>
<tr>
<td>2. 2-week follow up</td>
<td>16 9.31*</td>
<td>11 10.09</td>
<td>16</td>
<td>3.74</td>
<td>2.51</td>
<td>11</td>
<td>3.74</td>
<td>2.51</td>
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<tr>
<td>3. 4-week follow up</td>
<td>16 10.88*</td>
<td>11 10.55</td>
<td>16</td>
<td>4.10</td>
<td>2.98</td>
<td>11</td>
<td>4.10</td>
<td>2.98</td>
</tr>
</tbody>
</table>

*Significant (p < 0.05)
N = Number (of participants), SD = Standard Deviation

For Internal Functional scores (InFun), there was a significant interaction between intervention type and time, Wilks Lambda = .68, F (2, 24) = 5.54, p < 0.05, partial eta squared = .32, with the gratitude group showing a significant increase in InFun scores across three time periods (Time 1: M = 9.06, SD = 3.02; Time 2: M = 10.31, SD = 2.96; Time 3: M = 10.13, SD = 2.94; p < 0.05). The change of scores from week 2 to week 4 was NOT significant (p > 0.05). For External Dysfunctional scores (ExDys), there was a significant interaction between intervention type and time, Wilks Lambda = .73, F (2, 24) = 4.38, p < 0.05, partial eta squared = .27, with the gratitude group showing a significant reduction in ExDys scores across three time periods (Time 1: M = 4.19, SD = 3.29; Time 2: M = 2.75, SD = 2.21; Time 3: M = 1.88, SD = 1.4; p < 0.05). For External Functional scores (ExFun), there was a significant interaction between intervention type and time, Wilks Lambda = .72, F (2, 24) = 4.75, p < 0.05, partial eta squared = .28, with the gratitude group showing a significant increase in ExFun scores across three time periods (Time 1: M = 7.81, SD = 3.66; Time 2: M = 9.31, SD = 3.74; Time 3: M = 10.88, SD = 4.10; p < 0.05).
The Neutral Condition

None of the results in the Neutral Condition reached statistical significance. For Internal Dysfunctional ER, there was an increase in scores from Time 1 (M = 10.91; SD = 3.5) to Time 2 (M = 11.36, SD = 2.9) to Time 3 (M = 11.64, SD = 3.12). These results were not significant (p > 0.05). For Internal Functional ER, there was an increase in scores from Time 1 (M = 10.91; SD = 3.5) to Time 2 (M = 11.36, SD = 2.9) to Time 3 (M = 11.64, SD = 3.12). These results were not significant (p > 0.05). For External Dysfunctional ER, there was a decrease from Time 1 (M = 2.36, SD = 2.67) to Time 2 (M = 2.27, SD = 2.41) to Time 3 (M = 2.18, SD = 2.27). These results did not reach significance (p > 0.05). For External Functional ER, there was a decrease in scores from Time 1 (M = 10.27, SD = 2.53) to Time 2 (M = 10.09, SD = 2.51), and then the scores increased at Time 3 (M = 10.55, SD = 2.98). These results were non-significant (p > 0.05).

Effects of mood induction on life satisfaction and eating pathology

A series of mixed between-within subjects analyses of variance was conducted to assess the impact of two different interventions (Gratitude and Neutral) on participants’ eating pathology and life satisfaction scores across two time periods: pre-intervention and at the 4th week follow-up (Table 30).

Statistically Significant Findings (p < 0.05)

There was a significant main effect for Restraint, Wilks Lambda = .52, F (1, 25) = 22.68, p < 0.05, partial eta squared = .48, with both groups showing a reduction in Restraint. There was a significant main effect for the overall EDE-Q score, Wilks Lambda = .85, F (1, 25) = 4.33, p < 0.05, partial eta squared = .15, with both groups showing a reduction in eating pathology.
Table 30: Life Satisfaction Scale and EDE-Q Scores for the Gratitude and Neutral Interventions At Two Time Periods

<table>
<thead>
<tr>
<th></th>
<th>Gratitude</th>
<th>Neutral</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Time Period</td>
<td>N</td>
</tr>
<tr>
<td>Life Satisfaction</td>
<td>1. Pre-Intervention</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>2. 4-week follow-up</td>
<td>16</td>
</tr>
<tr>
<td>EDE-Q: Restraint</td>
<td>1. Pre-Intervention</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>2. 4-week follow-up</td>
<td>16</td>
</tr>
<tr>
<td>EDE-Q: Eating Concern</td>
<td>1. Pre-Intervention</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>2. 4-week follow-up</td>
<td>16</td>
</tr>
<tr>
<td>EDE-Q: Shape Concern</td>
<td>1. Pre-Intervention</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>2. 4-week follow-up</td>
<td>16</td>
</tr>
<tr>
<td>EDE-Q: Weight Concern</td>
<td>1. Pre-Intervention</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>2. 4-week follow-up</td>
<td>16</td>
</tr>
<tr>
<td>Overall EDE-Q</td>
<td>1. Pre-Intervention</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>2. 4-week follow-up</td>
<td>16</td>
</tr>
</tbody>
</table>

*Significant (p < 0.05)
N = Number (of participants), SD = Standard Deviation

Statistically Non-Significant Findings (p>0.05)

There were no significant differences within and between the two groups on Life Satisfaction, Eating Concern, Shape Concern and Weight Concern. There was an increase in Life Satisfaction in both the gratitude condition (Time 1: M = 14.94, SD = 5.98; Time 2: M = 17.88, SD = 5.87) and neutral condition (Time 1: M = 13.45, SD = 7.63; Time 2: M = 13.64, SD = 6.87). However, these results did not reach statistical significance (p > 0.05). Although Life Satisfaction scores in the Gratitude condition on average rose from 14.94 (SD = 5.98) to 17.88 (SD = 5.87) and Figure 37 shows differences between mood conditions, these results were non-significant. This may be because the numbers in the mood conditions were too low; it would be worthwhile to test these findings with a larger clinical sample.
Figure 37: Life Satisfaction Scores Pre- and Post- Mood Induction at Week 1 and Week 4.

Time 1: before mood induction, Time 2: after mood induction

The Gratitude group showed a decrease in Eating Concern (Time 1: M = 3.63, SD = 2.25; Time 2: M = 2.45, SD = 1.18) and Weight Concern (Time 1: M = 2.29, SD = 2.62; Time 2: M = 2.25, SD = 2.23). They also reported an increase in Shape Concern (Time 1: M = 2.54, SD = 1.18; Time 2: M = 2.63, SD = 1.11). However, these results did not reach statistical significance (p > 0.05). The Neutral group showed an increase in Eating Concern (Time 1: M = 2.51, SD = 1.95; Time 2; M = 2.78, SD = 2.29), and a decrease in Shape Concern (Time 1: M = 2.65, SD = 1.28; Time 2: M = 2.24, SD = 1.86) and Weight Concern (Time 1: M = 2.35, SD = 2.33; Time 2: M = 2.02, SD = 1.98). These results were statistically non-significant (p > 0.05).
Main findings (Study 5)

1. There was a significant gradual decline in dysfunctional (Internal and External Dysfunctional ER) and an increase in functional emotion regulation (Internal and External Functional ER) in the happiness condition over the course of one month. This means that longitudinal administration of the gratitude intervention may have improved functionality of emotion regulation in the sample with eating disorders.

2. Over the course of one month, eating pathology decreased in both happiness and neutral conditions. Decrease in Restraint and overall eating pathology (Overall EDE-Q) were the ones that reached statistical significance.

Study 5: Inductive Thematic Analysis (Clinical Sample)
Participants with eating disorders were encouraged to leave comments as they were completing the questionnaire sets and to answer questions about their emotions. Topics of particular interest were participants’ subjective experience of positive and negative emotions, as well as their opinion of the Gratitude exercise. The majority of participants took this opportunity, and wrote about their positive and negative affect as well as about the Gratitude intervention; their written reports were thematically analysed. The thematic analysis procedure followed Braun and Clarke’s (2006) guidelines and was primarily data-driven, rather than theory-based. First, the data corpus was semantically coded in a systematic way, and following that, the codes were combined into themes. The generated themes were then reviewed for internal homogeneity and external heterogeneity. The themes that were consistent, recurrent, distinctive and coherent were selected and are reported below. The data corpus neatly fitted into two main categories: feedback on the gratitude intervention (Figures 35 and 36) and participants’ subjective accounts of affect (Figure 37). The key themes within each category are highlighted in Bold. Participants’ original spelling and punctuation were preserved.
The Gratitu...intervention

The majority of participants initially found the gratitude exercise challenging (‘The writing exercise was hard but made me realize how much I have. This then makes me feel guilty that I am not as grateful as I should be and probably don’t deserve these privileges’, ID 5). Most participants commented on the guilt accompanying positive emotions, and the theme of guilt was one of the most prevalent. Participants reported the Gratitude exercise resulted in positive emotions, and positive emotions led to the feelings of guilt (Figure 38). However, the majority of participants were able to overcome guilt and negative thinking, associated with it, to complete the exercise and to benefit from it. One participant commented: ‘Excellent exercise - felt really unnatural to start with as usually any reflective writing I do is full of negative emotions. I usually would feel guilty thinking about all the good things in my life and still being ill but it felt I was ‘allowed’ to acknowledge them as it was a task I was asked to complete - interesting!’ (ID 6).

Figure 38: Emotion Sequence Induced by the Gratitude Intervention

As can be seen from Figure 39, participants reported that the Gratitude intervention had a number of psychological, cognitive and physical benefits. On the psychological level, the writing exercise led to an increase in positive emotions and subjective well-being, and the majority of participants reported feeling noticeably happier, calmer and more relaxed after the exercise (‘Thinking of all the things I have to be grateful for makes me feel better. It makes me see more positives in my life to make me appreciate my life more<...> It makes me see how much is good in my life… (ID 7); ‘I feel much happier now I have thought about what I have to look forward to and I am quite excited’ (ID 9); ‘I’d say that my most dominant feeling after the writing exercise is a sense of inner peace and calm and sort of harmony. I feel like smiling, not laughing, content, not ecstatic. Also safe – reassured somehow. Pleased that I am able to experience these things, connect with my memories and really value and appreciate things’ (ID 14)).
On the cognitive level, some participants reported that the exercise helped them shift their attention from the negative in their life to the positive, which encouraged positive thinking, a renewed sense of purpose, optimism and a more balanced life perspective. Most of them commented that the exercise brought to awareness their default negative mindset (‘Thank you for this opportunity – I feel I am learning things about myself! I never realized how hard I find it to think about the positive things in my life without feeling guilty’ (ID 2); ‘I am realizing that negative emotions affect me more than positive ones, as if my focus in unbalanced. Also [I] feel ... I focus so much on negativity <...> Since I was ill with my eating disorder, I have developed a habit of focusing on negative things too much, I often forget positive things’ (ID 4)), and enhanced their awareness and appreciation of their blessings (positive awareness) (‘It [the writing exercise] made me see I have a lot to be grateful for, I am lucky. It helped...')
to remind me to appreciate what and how much I have (ID 7); ‘When I think about it, I have a lot in my life to make me feel happy’ (ID 4). On the physical level, some participants reported reduction of tiredness and an increase in physical energy (‘When I started writing today. I felt so tired and really a bit ‘flat’ – not really connected with my feelings at all. Recalling events and especially this last gratefulness exercise has helped me reconnect with times when I have felt more energetic, connected and happy. And now I feel more energetic, happy, and connected in this moment’ (ID 14)).

**Self-reports of affect**

The key themes for negative affect were: *frequency*, *dominance* and *stability*. As illustrated by the Figure 40, negative affect was reported by the majority to be frequent and enduring. Most said negative emotions dominated their lives and were easily elicited and did not require stimulation from outside (‘The continual conversation I have with myself is not a happy one. It saddens me that after so many years I don’t solve the conundrum. I won’t allow myself to take it by the horns and say ‘no more’, stop…I worry that I’m a generally miserable person. I dwell on my negative feelings and perhaps dismiss how often I have positive feelings’ (ID 10)).

Figure 40: Participants’ reports of positive and negative affect
The key themes for positive emotions were: **infrequency**, **instability** and **inaccessibility**. According to their reports, the vast majority of the eating-disordered females experienced positive emotions rarely or not at all (‘I don’t often feel like I experience positive emotions. I have feelings that are not exactly positive. It is very hard to describe - feeling OK just means that I don’t feel bad and tearful and want to stay in bed. When I feel happy, which isn’t often, I guess I feel comfortable inside, warmish and I want to get up and do stuff and see people and go places. A lot of the time I feel like I am just functioning’, ID 12). Most participants said their positive affect was predominantly low, short-lived and difficult to elicit (‘I feel positive emotions quite infrequently and when I do feel them it’s normally because I have tried hard to feel them’, ID 4). They felt detached from it (‘I would say that I experience positive emotions <…> on a superficial level for some moments. It’s as though they are seen through a smeary glass panel, so they are there, but there is a detachment from them somehow’, ID 1) and were unable to recognize and experience positive emotions, especially without external triggers (‘Positive emotions are usually experienced in relation to external triggers, not from within me, e.g. seeing a friend’, ID 6).

Negative affect, lack of awareness and rarity of positive emotions were reported to be so extreme that they led to some participants retrospectively perceiving their brief moments of happiness as inauthentic and doubting them (‘[For me positive emotions] are infrequent and fleeting. Soon overtaken by negative emotions e.g. I feel happy then feel guilty for feeling happy. When I have had positive emotions, I am on a real ‘high’ and believe anything is possible and feel hopeful. Then negative emotions kick in and it feels like the positive ones have been fake’, ID 6); ‘If I feel happy it’s not proper happiness and my mind is never fully in the present, I am always thinking about why things aren’t perfect’, ID 8); ‘I also wonder if I ever feel true positive emotions like others do, or whether it’s simply all relative. I laugh sometimes in a way that feels uncontrollable when the scales indicate that I have lost weight, at these times I think I feel happy’, ID 2).

Another common feature in the reports was **extreme mood fluctuations** between highs and lows, and complete flatness (‘[I] feel like my emotions are completely
overwhelming (sad or happy) or I feel nothing/numb/detached’, ID 6); ‘My mood changes so frequently and cycles through so many extremes’, ID 2). These were perceived as uncontrollable and frightening by some (‘I feel quite scared of feeling strong positive (and negative...) emotions’, ID 13; ‘I can be slightly nervous about feeling joyful because I know when I am elated I tend to lose my inner control when it comes to food which I regret later. I try hard to be happy and to do things that make me happy, but it can be difficult’, ID 4). Most participants reported that their expression of both positive and negative emotions was typically subdued (‘I find it quite difficult to express any kind of emotion<...> I wish I could change this but perhaps through therapy, I will be able to’ (ID 3) or intentionally inhibited (‘If I express [emotions], I feel ashamed and afraid that later on I will suddenly realize I was doing something really shameful and hadn’t realized at the time. I feel if I tone it down then if later I do realize this then at least I won’t feel I’ve embarrassed myself so much. I suppose I replace strong emotions with more mild versions like a sense of satisfaction’, ID 13), in order to conceal their true intensity and frequency (‘I am a good actress, and can put on an air of calmness and hate to burden others with my problems’, ID 8).

**Positive Emotions: Context**

Participants identified two major sources of positive emotions: (1) spending time with family/friends and (2) achievement. Despite expectations, only two participants explicitly stated weight loss/control as something that made them happy. The majority listed being with family and friends in a ‘safe’ (food-free) context as the main source of positive emotions. One participant wrote: ‘Since I’ve been ill, I feel I have lost contact with what makes me happy <...> Therefore I am still trying to work out the context in which I experience positive emotions, but it tends to be when I am with people in a situation that doesn’t involve food’ (ID 5). Participants reported that they had no default inner sense of well-being; therefore, for them positive emotions were primarily a product of social interactions with their friends and family (‘Being sociable, and with friends and family makes me happy’, ID 13). The second most frequently listed source of happiness was achievement (at work etc.). Participants reported joy and satisfaction as a result of achieving goals and completing tasks, even small ones. (‘Feeling I have
accomplished something makes me happy – finishing a book, or a craft project or achieving a goal’, ID 13).

Inductive Thematic Analysis Findings: Summary and Conclusions
The thematic analysis showed that females with eating disorders had a disproportional negative to positive affect. They reported that their mood was predominantly negative and fluctuated uncontrollably; therefore they typically struggled with it. In addition, participants’ negative emotions were so pervasive as to interfere with their experience of positive affect. The findings that positive mood induction triggered guilt are interesting. Participants felt guilty for different reasons: for feeling happy, for being ill and for having a lot be grateful for. Regardless of the reason behind it, guilt inevitably accompanied and undermined gratitude-induced happiness. However, in the context of the task, participants were able to successfully manage their negative affect and to fully engage with the exercise.

Participants’ inability to experience positive emotions in their daily lives may be one of the factors that hinder their recovery. Participants reported that in their daily lives they generally struggled to recognize, elicit and fully experience positive affect. Previous research has shown that positive emotions are recuperative for mental and physical health, because of their effects on motivation, cognition and behaviour. Individuals with eating disorders may not be able benefit from positive emotions in this way, because they are inept at reliably inducing and maintaining them. Therefore in treatment it may be important that individuals with eating disorders are taught not only how to regulate their negative affect but also how to identify, elicit and maintain positive affect. The findings on the sources of positive emotions may be usefully applied to treatment of eating disorders. Time with family/friends and opportunities to accomplish goals could be incorporated into therapeutic interventions, in order to stimulate positive emotions (and their multiple physical, cognitive and psychological benefits) in patients with eating disorders.

First, regular interactions with family/friends in an eating-unrelated setting could be facilitated and encouraged in inpatient as well as in outpatient care environments. This
would mean less isolation, more social support and more positive emotions for the patients. Second, individuals being treated for eating disorders may benefit from being entrusted with more responsibilities, as well as more freedom and opportunities to discover and pursue their (food-unrelated) interests (especially, in the inpatient care). This is necessary for them in order to experience the sense of achievement and satisfaction. For instance, clinicians (or patients’ parents) could try giving patients little, manageable and pleasant tasks, such as to water flowers in the garden or to read a chapter in a book; this would give them a sense of achievement and increase their well-being. Both of those approaches, if implemented regularly, could increase positive emotions in sufferers of eating disorders, and may ultimately have a therapeutic effect.

Main findings (Studies 4 and 5)

Studies 4 and 5 made a number of important findings in the field of emotion regulation and eating pathology. These were as follows:

1. The tendency to experience negative affect was associated with dysfunctional emotion regulation. On the contrary, the tendency to experience positive affect was related to functional affect management.

2. Eating-disordered participants experienced negative affect (i.e. sadness, anger, fear, disgust, guilt and anxiety) intensely and frequently, and negative emotions were salient for them. They experienced positive affect rarely, if ever, and it was fleeting and easily disrupted by interfering negative affect. Participants also reported that they suffered from extreme fluctuations in affect, and were poor at eliciting and sustaining positive emotions. They were also dissatisfied with their lives.

3. Participants with eating disorders reported that the two main sources of positive emotions for them were: (1) spending time with friends and family and (2) achieving goals.

4. It was found that eating-disordered participants typically chose unhealthy emotion regulation strategies.
5. The Gratitude exercise was successful at positive mood induction and was very well-received; participants reported gaining a wide range of benefits from it on physical, cognitive and psychological levels. The results showed that participants were happier at the end of the study than they were at the beginning.

6. Following the happiness induction, participants were less likely to choose dysfunctional emotion regulation, and over the period of 4 weeks, their dysfunctional ER and eating pathology scores decreased.
CHAPTER 9. DISCUSSION OF FINDINGS AND THEIR IMPLICATIONS

The findings from the clinical and sub-clinical studies converged, supported and complemented each other. Studies 1 and 3 prepared the way - methodologically and theoretically - for the clinical studies. The sub-clinical studies showed that: 1. Disordered eating and dysfunctional emotion regulation were clearly linked, and high Global and Negative Affect Intensity were connected to both (Study 1); furthermore, eating pathology and dysfunctional affect regulation were found to be related to sub-clinical depression, anxiety and life dissatisfaction (Study 3). 2. The combination of music and the Velten mood induction procedure was very effective in inducing happiness and sadness (success rate ≥ 85%; Study 1). Positive Psychology Interventions (PPIs) were easy to implement and successfully used to induce positive mood. The Gratitude exercise was more effective (success rate = 61%) than the Positive Reminiscence task (success rate = 50%; Study 3). 3. Positive emotions led to a significant reduction in dysfunctional emotion regulation (Studies 1 and 3). Positive mood induction did not significantly affect Negative Intensity scores, but it did influence Global Affect Intensity and Positive Affect Intensity (Study 1). Positive Affect, induced using PPIs, had a significant effect on emotion regulation, increasing functional emotion regulation and life satisfaction, as well as reducing dysfunctional affect management (Study 3).

Similarly, the studies with clinical samples (Studies 4 and 5) found that: (1) the eating-disordered participants typically chose unhealthy emotion regulation strategies, and struggled with frequent and intense negative emotions; (2) the tendency to experience negative affect intensely was associated with dysfunctional emotion regulation; (3) The Gratitude exercise was successful at positive mood induction and was very well-received; participants reported multiple physical, cognitive and psychological benefits.

After the happiness intervention, participants were less likely to choose dysfunctional emotion regulation, and over the period of 4 weeks, their dysfunctional ER and eating pathology scores decreased. Introduction of the qualitative element to the methodology in Studies 4 and 5 allowed for collection of rich data on negative and positive affect as experienced by females with eating disorders. It was found that the eating-disordered participants experienced the full range of negative emotions (i.e. sadness, anger, fear,
disgust, guilt and anxiety) intensely and frequently. They experienced positive affect rarely, if ever, and it was fleeting and easily disrupted by interfering negative affect. Participants with eating disorders reported that the two main sources of positive emotions for them were: (1) spending time with friends and family and (2) achieving goals. Participants also reported that they suffered from extreme fluctuations in mood, and were poor at eliciting and sustaining positive emotions. They were also unhappy with their lives. Because Studies 4 and 5 replicated and added to the findings of Studies 1 and 3, this chapter looks mainly at the two clinical studies, and suggests possible practical applications and directions for future research.

When interpreting the findings, it is useful to distinguish between their practical and statistical significance. Small differences between conditions can reach statistical significance if the participant sample is large enough. Unlike statistical significance, practical (or clinical) significance is independent of sample size and addresses general meaningfulness of the results. Effect sizes and confidence intervals give a good indication of the magnitude of the difference between the conditions and the range of values. In the research presented in this thesis participant numbers were limited, and the findings of statistical significance suggest real differences between the conditions. If these differences are measurable in a relatively small sample, this means that the results are likely to be reliably replicated in larger samples, and therefore, generalizable to the population. One of the main drawbacks of this research project was limited participant numbers in clinical and subclinical groups. In future studies it may be worthwhile to recruit larger samples if there is an opportunity to do so. With regard to clinical significance, in the Studies 1, 3, 4 and 5 the effect sizes for the mood interventions as well as for the differences between experimental sub-samples (sub-clinical – controls; clinical – controls) on emotion regulation and affect intensity ranged from medium to large. This suggests that the differences between the mood induction procedures, as well as the differences between the sub-samples, are clinically meaningful and useful in practice. The findings discussed in this Chapter reached both statistical and practical significance which provides a good indication of the general validity and reliability of the results.
**Affect Intensity (AI) and Emotion Regulation (ER)**

Study 4 showed that Negative Affect Intensity was positively associated with Internal Dysfunctional ER as well as External Dysfunctional ER, and negatively with Internal Functional ER; whereas Positive Affect Intensity positively correlated with functional ER (External and Internal). In other words, participants who had a tendency to experience negative emotions intensely, were significantly more likely to use such strategies as self-harm, rumination, negative social comparison, repression, derealization (Internal Dysfunctional ER, Phillips and Power, 2007), as well as bullying, and physical and verbal assault (External Dysfunctional ER, Phillips and Power, 2007) when managing their emotions. In addition, they were significantly less likely to modify their goals, plan, put things in perspective and positively reappraise the situation in which negative emotions arose (Internal Functional ER, Phillips and Power, 2007). On the contrary, participants who typically experienced positive emotions of high intensity were more likely to use those and other healthy strategies, such as expression of feelings, advice-seeking, contact-seeking and distraction (External Functional ER, Phillips and Power, 2007). This suggests that individuals who are high in Negative Affect Intensity and low in Positive Affect Intensity may be more likely to use dysfunctional emotion regulation mechanisms. Those who are high in Positive Affect Intensity may be more inclined to choose healthy emotion management strategies.

These results suggest that positive and negative affectivity is associated with diametrically opposite choices of emotion regulation strategies, which is interesting, considering Larsen’s findings that positive and negative affect intensities are interrelated (Diener et al., 1985a). Larsen and Diener (1987) wrote that individuals who were high in Positive Affect Intensity would be the ones who are also high in Negative Affect Intensity, because general emotional reactivity (Global AI) is assumed to underlie both. Although it is clear that Positive and Negative Affect Intensity share the in-built emotional reactivity component, these two constructs refer to emotions of opposite valence (positive versus negative). Therefore, it is conceivable that there would be qualitative differences between the two types of Affect Intensity, which would produce differential effects on emotion regulation. More research is needed into the Affect Intensity construct and its relationship with emotions.
The results of Study 4 confirmed the findings of the very first study (Study 1) in showing that individuals who typically experienced intense negative emotions were more likely to use dysfunctional emotion regulation strategies (Internal and External Dysfunctional ER). This suggests that intensity of affect may be of importance in emotion regulation. For example, if a person has a tendency to experience anger intensely, they may resort to more drastic (and less healthy) measures to bring the emotion under control. Functional strategies (e.g. talking it out, positive re-appraisal) may not be effective enough to efficiently and quickly deal with and diffuse the anger, whereas dysfunctional strategies (especially External Dysfunctional ones) may be (e.g. lashing out).

The tendency to experience negative affect intensely may help to explain why an individual chooses certain regulatory strategies; this suggests that Affect Intensity needs to be included in the discourse on emotion regulation. In future research, it may be helpful to collect emotion intensity data alongside that on affect management. It is not enough to know how someone regulates their emotions, it is also important to understand the reasons behind emotion regulation choices. Dysfunctional affect management is detrimental for mental health (Phillips and Power, 2007), and in order to correct emotion dysregulation, it is necessary to understand what contributes to it. Negative Affect Intensity may be one such factor. Although this data is correlational and does not give us the direction of effect, it seems likely that emotional dysregulation is secondary to Negative Affect Intensity. Some researchers suggested that Affect Intensity is temperament-based (Larsen et al., 1986; Larsen and Diener, 1987), i.e. it is a biological tendency to be emotionally reactive. From an early age, a high Affect Intensity individual may perceive their emotions to be profound and overwhelming. If they did not learn how to respond to their emotions adaptively as children, they may struggle to functionally regulate their emotions as adults. It is likely that in such an individual, emotion dysregulation is activated by environmental triggers, for example, daily hassles and stressors. In stressful situations when the individual does not have a tried-and-tested repertoire of healthy regulatory strategies, learned from childhood, they may be more likely to become overwhelmed and steered by their negative emotions towards dysfunctional regulatory strategies (e.g. self-harm or verbal assault). This in turn, may result in psychological and physical ill-health. Studies 4 and 5 are the only
ones of their kind in examining emotion regulation in the context of Affect Intensity, and they provide the foundation to this promising line of research. Future studies should look into the origins of emotion mismanagement and regulatory choices in detail, and attempt to develop a theoretical framework for emotion dysregulation to include the Affect Intensity component.

The finding that participants who experienced more intense positive emotions were more likely to use a functional regulatory approach (Internal and External ER) suggests that individuals who are able to experience profound happiness may be better at regulating their emotions in a healthy way. This gives further support to the literature on health-benefiting and therapeutic properties of positive emotions (Danner et al., 2001; Fredrickson, 1998; Fredrickson and Losada, 2005; Fredrickson and Levenson, 1998; Fredrickson et al., 1998, 2000; Howell et al., 2007; Lyubomirsky and Dickerhoof, 2010; Lyubomirsky et al., 2006; Seligman, 2007). In addition, these findings converge with those of Study 1, where positive affectivity was significantly associated with External Functional emotion regulation. Study 4 showed that individuals with the tendency to experience positive emotions profoundly tend to choose such strategies as expression of feelings, advice-seeking, contact-seeking, exercise and distraction (External Functional ER, Phillips and Power, 2007) as well as positive re-appraisal, modification of goals, planning, putting things in perspective and concentration of a pleasant activity (Internal Functional ER, Phillips and Power, 2007).

**Emotion Profiles of eating disorders (Quantitative and Qualitative data) and Life Satisfaction**

Study 4 showed that participants in the clinical sample had significantly higher levels of baseline negative affect (Sadness, Anger, Guilt, Anxiety, Fear and Disgust) and significantly lower levels of baseline positive affect (Happiness) than controls. They also were significantly less satisfied with their lives than controls. Participants’ written reports of their positive and negative affect converged with statistical evidence. The clinical sample experienced more intense and frequent negative emotions (i.e. fear, anger, sadness, guilt, anxiety and disgust), as well as weaker and rarer positive emotions (e.g. happiness) than controls. These findings are supported by a large body of literature on the dysphoric nature of eating disorders (Barney and Irving, 2000; Brewerton et al.,
In Study 5, eating-disordered participants reported that their positive affect was infrequent, unstable, short-lived and hard to elicit. Participants struggled to recognize and trigger positive affect, and had no lasting sense of well-being. They said that positive emotions were inaccessible to them and that they perceived their experience of happiness as inauthentic. With regard to negative affect, participants reported it to be exactly the opposite: enduring, frequent, dominant and internally activated. Unlike positive affect, negative emotions felt acute and real.

Another interesting finding in Study 5 was participants reporting that they experienced extreme fluctuations from negative to positive mood to complete flatness. This suggests that unstable, imbalanced mood may be one of the core features of eating pathology. Eating disorders may develop as an attempt to bring emotions under control, or alternatively, dysphoric affect may be secondary to disordered eating. In the former case, imbalance of emotions (e.g. as a result of a psychological trauma) may precede behaviours such as bingeing and starvation, and be the catalyst for disordered eating. In the latter case, extreme fluctuations of emotions may result from poor nutritional practices and distress, associated with eating pathology. It would be interesting to investigate this further in future studies, comparing three groups: eating-disordered patients, subclinically eating-disordered sample and healthy controls. Participants can be interviewed about their positive and negative emotions, in particular, about how frequent, stable and intense those emotions are, and the data, collected from the three groups, may be analyzed in relation to participants’ severity of eating pathology.

It was found that the eating-disordered participants’ experience of positive emotions was blocked by negative affect (e.g. guilt). Participants reported that gratitude-induced positive affect was accompanied by negative emotions, in particular, feelings of guilt. They said that this made the gratitude exercise difficult for them. However, over the period of four weeks, participants became increasingly more skilled at managing the negative feelings that arose in the context of positive mood induction, and were able to experience positive affect without interference. As a consequence, participants were able to fully benefit from the Gratitude exercise. This was reflected in healthier emotion
regulation and lower eating pathology scores. Participants’ reports demonstrated how debilitating and pervasive negative affect was for them. Emotions such as fear, guilt, anger, sadness, anxiety and disgust appear to be not only more frequent, but also more intense in individuals with eating disorders.

Participants’ reports suggest that they do not experience frequent or profound positive emotions (e.g. happiness, joy). Moreover, participants reported that their default negative emotions disrupted the experience of positive affect, which greatly impaired their sense of well-being (i.e. life satisfaction). Subjective well-being is one of the key components of mental health, and inability to experience it may perpetuate psychiatric problems. These findings have significant implications for treatment of eating disorders. As part of treatment, it may be important to aim not only to minimize negative affect, but also to educate patients with eating disorders how to elicit and sustain positive emotions. Such training – delivered by means of PPIs, for example – can potentially improve efficacy of psychotherapies for eating disorders. In addition, patients should be encouraged to discuss their positive emotions and challenges, associated with eliciting and sustaining them with their clinicians.

Because positive emotions are so important for health and recovery (Danner et al., 2001; Fredrickson, 1998; Fredrickson and Losada, 2005; Fredrickson and Levenson, 1998; Fredrickson et al., 1998, 2000; Howell et al., 2007; Lyubomirsky and Dickerhoof, 2010; Lyubomirsky et al., 2006; Seligman, 2007), it would make sense to include regular positive emotion induction into treatment for eating disorders. Study 5 made an important finding, concerning the context of positive emotions. Participants with eating disorders reported that they tended to experience positive affect in two contexts: (1) when they were with their friends and family in a safe (food-free) environment, and (2) when they experienced a sense of accomplishment. This suggests that positive emotions can be reliably induced as part of treatment by encouraging those behaviours (i.e. socialization with friends/family and achievement), and interventions can be devised around them. With regards to the first context, patients in inpatient and outpatient care can be encouraged and enabled to regularly spend quality time with their loved ones. For instance, for those in inpatient care, day trips with friends may be organized (if the health of the patient permits) or alternatively, frequent visits, where the friends, family
and the patient are encouraged to concentrate on a food- and eating-unrelated activities (e.g. playing a game, watching and discussing a film).

For those in outpatient care, spending time with family and friends may be emphasized as part of the outpatient treatment requirements. In their reports, a lot of participants said that they would enjoy a break from talking about their illness. Therefore, providing conditions for socializing in a food-free environment on a regular basis and instructing patients’ visitors to focus on topics for conversation unrelated to eating disorders could increase patients’ sense of well-being. The regular boost in positive emotions may be beneficial for their mental and physical health, and may positively influence recovery. Patients should be able to choose whom they want to spend time with; they may have difficult relationships with some friends and family members, and encouraging to spend time with them may cause distress. As was discussed in Chapter 1, patient’s dysfunctional relationships with certain family members (e.g. their mother) may contribute to development of eating pathology (Bruch, 1973; Minuchin et al., 1975, 1978; Ward and Gowers, 2003). As the primary purpose of this intervention is to induce positive affect, it may be important to not include those family members in the positive affect induction activities.

Encouraging relationships may have several important benefits; it may help: (1) to re-build social connections that may have been affected by the illness, (2) to develop social skills, and (3) to establish a reliable support network. In the future, the patient will be able to draw on these social resources if they are in a difficult situation, and may be less likely to relapse into their eating disorder. This means that spending time with friends and family may be a valuable treatment tool. Perceived isolation from family and friends (as is often the case in the inpatient setting) may be detrimental for the well-being of patients and may negatively affect their recovery. Hawkey and Cacioppo (2010) found that perceived social isolation was associated with a large number of negative psychological and physical health outcomes, such as mortality, depressive symptoms, poor physiological functioning, as well as decreased capacity to regulate emotions, cognitions and behaviours. This means that inpatient care itself may be making recovery more difficult for patients with eating disorders if patients perceive themselves to be isolated from their loved ones. Therefore, it is important to ensure that
patients (particularly, in the inpatient care) have regular contact with their families and friends. This may have significant benefits for patients’ health.

Another important source of happiness that eating-disordered participants wrote about was a sense of achievement. Participants reported that an accomplishment, however small (e.g. finishing a book), made them feel happy. It would be worthwhile to further research the concept of accomplishment and what it means to the individuals with eating disorders. It may be that achievement is so important for them because it is driven by their perfectionism and low self-esteem (Bastiani et al., 1995; Bruch, 1973; Button et al., 1996; Casper 1983; Fairburn et al., 1997, 1999; Garner et al., 1983, 1984; Gual et al., 2002; Sassaroli et al., 2008; Shafran et al., 2002). It is plausible that the sense of accomplishment has the potential to elicit positive emotions, give a boost to self-esteem, and increase one’s sense of environmental mastery. An eating-disordered patient would benefit from all of those.

Individuals with eating disorders often perceive their weight loss and restricted eating as an achievement (‘a source of perverse satisfaction’, Palmer, 2000) and as a measure of self-worth, as opposed to a disease. Eating disorders may provide a sense of accomplishment and reward, and this would make individuals reluctant to give them up. As part of treatment patients are encouraged to pursue their treatment goals (e.g. a healthy BMI), however, it is not clear whether achieving those would give them a sense of accomplishment. One can expect that if it did, a higher percentage of patients would recover from their eating disorders. Therefore, in treatment it may be important to provide opportunities for achievement that are unrelated to eating pathology. Assigned activities could be based on the preferences and interests of the individual, as well as be simple to carry out to minimize the risk of failure. For instance, inpatients may be provided with games, art projects, reading assignments and craft projects following an assessment of their interests and preferences. This can be done by asking the patients about what activities they would enjoy doing, and by using an additional measure, such as Values in Action Inventory of Strengths (VIA, Peterson and Seligman, 2004) as an indicator.

The VIA Inventory (Peterson and Seligman, 2004) allows identifying individuals’ unique strengths, for instance, love of learning (‘mastering new skills <and> topics’,
Peterson and Seligman, 2004), *kindness* (‘generosity, nurturance, care, compassion…’, Peterson and Seligman, 2004) and *leadership* (‘encouraging a group of which one is a member to get things done and at the same time maintaining good relationships within the group’, Peterson and Seligman, 2004). Developing one’s strengths is associated with increased well-being and personal growth (Park et al., 2004). Achievement-based activities for the patients in inpatient and outpatient settings can be devised to fit around their strengths. For instance, a patient whose main character strength is *love of learning*, may be given an assignment to research a topic that interests him/her. Someone who scored high on *kindness*, may be entrusted with the responsibility of looking after the family pet at home; an individual whose key strength was *leadership* may be given the task of organizing a craft project for their friends. Engaging in such activities not only would result in a sense of achievement, and positive emotions, but also would help to develop the patients’ character strengths, which may contribute to better psychological adjustment, as well as improved health and well-being. Clinicians and family could work together in developing, organizing and assigning such mood-boosting activities to their patients.

*Scheduling of pleasant activities* and *graded tasks* (e.g. encouraging maintenance of social relationships) are elements of the Well-Being Therapy (Fava and Ruini, 2004) that may be applied in clinical and non-clinical settings to induce positive emotions and increase well-being in individuals with eating disorders. These elements may be incorporated into routine psychotherapeutic treatment in order to enhance patients’ psychological well-being, which may boost the effectiveness of therapies such as CBT. To sum up, Studies 4 and 5 assessed negative affectivity in eating disorders across several basic negative emotions as well as looking at positive affect. This allowed for a comprehensive picture of emotional profiles of eating pathology. It is recommended that in clinical assessment and as part of case formulation, account should be taken not only of negative affect, but also of the patient’s positive emotions; clinicians should strive to understand the conditions for positive affect, its frequency and challenges, associated with eliciting and maintaining it. Because positive emotions are so important for health, more emphasis needs to be placed on ensuring well-being of patients in inpatient and outpatient care. In Study 5, participants reported that for them positive emotions were elicited in the contact with family and friends, as well as in achievement
scenarios. Therapies may benefit from incorporating those and other wellbeing-boosting strategies into treatment plans of patients with eating disorders.

Regulation of Affect

Study 4 found that eating-disordered participants typically chose dysfunctional emotion management strategies in order to regulate their negative emotions. They were significantly more likely to manage their emotions in a dysfunctional way (Internal and External Dysfunctional ER; Phillips and Power, 2007) and less likely to use External Functional ER (Phillips and Power, 2007) strategies than controls. In addition, they reported significantly higher Negative Affect Intensity and lower Positive Affect Intensity, as well as lower Life Satisfaction than controls. Eating pathology as measured by the EAT-26 (Garner et al., 1982) and EDE-Q facets (Fairburn and Beglin, 1994) were positively related to Internal Dysfunctional ER (Phillips and Power, 2007) and negatively to Functional ER (External and Internal, Phillips and Power, 2007).

Overall, the evidence suggests that individuals with eating disorders may tend to use dysfunctional emotion regulation strategies to deal with intense negative emotions that they consistently experience. Study 4 showed that participants with eating disorders were more likely to manage their emotions using unhealthy strategies such as: self-harm, rumination, negative social comparison, repression, de-realization (Internal Dysfunctional ER, Phillips and Power, 2007), bullying, verbal assault, physical assault, making others feel bad and lashing out (External Dysfunctional ER, Phillips and Power, 2007). They were significantly less inclined to express their feelings, distract themselves, and seek advice and contact (External Functional ER, Phillips and Power, 2007). Participants with eating disorders experienced intense negative emotions and weak positive affect, and were generally unhappy with their lives, compared to controls.

There are multiple studies suggesting that disordered eating represents an attempt at regulating enduring (i.e. frequent/continuous) negative emotions (Bydlowski et al., 2005; Fox and Power, 2007; Heatherton and Baumeister, 1991; Root, Fallon and Friedrich, 1986; Taylor et al., 1997). Study 4 findings appear to confirm that, and additionally show that the tendency to experience intense negative affect is damaging for healthy emotion regulation. The results show that not only frequency, but intensity
of emotions matters when it comes to emotion management choices. It is conceivable that the tendency to experience intense and frequent negative affect and the inability to manage it adaptively may be at the core of eating pathology. Negative Affect Intensity may even be just as important in the development of emotion dysregulation as the frequency of negative emotions.

It is plausible that an eating disordered individual is born with the tendency for intense emotions, and this affects her choice of regulatory strategies as an adult. In particular, she may struggle to regulate her emotions functionally. The mechanism for this may be as follows. If the female in our example was not taught how to recognize and effectively cope with her emotions in early childhood, she may find her intense negative affect unmanageable. It is likely that she would be easily overwhelmed by the sheer intensity of her negative emotions, and would tend to opt for the regulatory strategies that require the least effort in implementation. It is plausible that dysfunctional strategies (e.g. rumination, lashing out) are simpler and more readily available than functional ones (e.g. advice seeking, positive reappraisal), and perhaps, demand less time and effort. As evidence from the literature on Affect Intensity suggests (including that from Study 1), Negative Affect Intensity may be an aspect of innate temperament (Larsen et al., 1986; Larsen and Diener, 1987), and as such cannot be altered by psychotherapy and interventions. However, the understanding that intense as well as frequent negative affect is implicated in the emergence of emotion dysregulation and through that of eating pathology, may bring us closer to improving treatment.

Disordered eating itself may be a dysfunctional regulatory strategy (e.g. a variant of self-harm). There is evidence that anorexic, bulimic and EDNOS individuals tend to score highly on self-injury measures (Paul et al., 2002). Eating disorders (e.g. bingeing) may represent a type of self-harm, which suggests that eating pathology may be used as a way of emotion regulation. This means that disordered eating may fulfil the same functions as other types of self-harm; in other words, by purging, bingeing and self-starvation, the eating-disordered individual may attempt to reduce their acute negative affect. Self-harm may be a powerful emotion regulation strategy (Gratz, 2003; Klonsky, 2007; Mikolajczak et al., 2009); deliberate self-harm behaviour was found to effectively relieve acute negative emotions, such as anxiety and guilt (Klonsky, 2007).
According to NICE (2004), self-harm may fulfil multiple and varied functions for those who engage in it. This may apply to self-harm by disordered eating. As well as reduction of painful affect, individuals may use self-harm by disordered eating in order to punish themselves, to signal to others about their distress, and to externalize their negative affect (Gratz, 2003). Self-harm may be helpful as a short-term emotion regulation strategy, but in the long run it is maladaptive (Gratz, 2003). Therefore eating-disordered individuals should be encouraged to find alternative, more functional ways of managing their intense and frequent negative affect.

**The Gratitude Intervention: Effectiveness**

The Gratitude intervention was successful in inducing positive mood in 75% (Study 4) of cases when it was induced once, and on average in 82% of cases when it was induced over the period of 4 weeks (Study 5). This intervention was rated by the clinical sample as very beneficial on several levels: physical (e.g. increased energy), psychological (e.g. increased well-being) and cognitive (e.g. positive thinking and enhanced awareness). Participants reported that after the gratitude exercise they experienced an increase in subjective well-being, reduction in anxiety, feeling reassured, positive focus, optimism and physical energy. They said the exercise raised their awareness of the good in their lives, instilled a more balanced perspective, enhanced their appreciation of life, and gave them a renewed sense of purpose. These are important benefits that were achieved as a result of positive affect induction, carried out over a period of one month. One can expect that if the Gratitude exercise (or another comparatively effective PPI) is administered for several months, sustained gains in well-being and psychological health would be achieved. It is worth investigating this further with a larger sample and a longer mood manipulation period. As Table 28 (p.198) shows, in Study 5 the baseline pre-induction happiness levels rose by 2 points from week 1 to week 4. This means that the happiness induction consistently increased the level of positive affect over the period of 4 weeks.

However, participants reported that the Gratitude exercise and positive cognitive and emotional changes, elicited by it, often resulted in feelings of guilt. Guilt is a negative emotion, and it is interesting that it arose out of positive affect in order to block it. This
means that in individuals with eating disorders positive emotions may be tainted and undermined by negative ones. Therefore, in psychotherapy, eating-disordered patients may require help and training in how to elicit, manage and sustain positive affect. When applying PPIs such as the Gratitude task, it is important to remember that negative affect may interfere with the experience of positive emotions in individuals with eating disorders. Future studies should examine the concept of guilt as an accompanying factor of gratitude-induced happiness in eating disorders in more detail.

**Effects of happiness on emotion regulation and disordered eating**

Study 4 showed that the one-off Gratitude intervention led to a significant reduction in Internal Dysfunctional scores (in the control sample only) and in External Dysfunctional scores (in both clinical and control samples). The one-off Gratitude intervention was successful in reducing dysfunctional emotion regulation scores in the control sample. In other words, participants were significantly less likely to choose such strategies as self-harm, rumination, negative social comparison, repression, de-realization (Internal Dysfunctional ER, Phillips and Power, 2007), bullying, verbal assault, lashing out at objects, physical assault and making others feel bad (External Dysfunctional ER, Phillips and Power, 2007), following happiness induction. However, in the clinical sample mood induction only positively affected external dysfunctional scores. This means that after the gratitude task, eating-disordered participants were less likely to choose bullying, verbal assault, physical assault, making others feel bad and lashing out at objects as their regulatory strategies (External Dysfunctional ER, Phillips and Power, 2007). Positive emotions had no significant effect on life satisfaction, Internal and External Functional scores. Compared to neutral mood, induced positive affect did not make participants feel better about their lives, or more inclined to use functional ER strategies, such as expression of feelings, advice-seeking, contact-seeking (External Functional ER, Phillips and Power, 2007), positive reappraisal, modification of goals, planning and putting things in perspective (Internal Functional ER; Phillips and Power, 2007).

However, Study 5 showed that there was a significant gradual decline in dysfunctional (Internal and External Dysfunctional ER) and an increase in functional emotion regulation (Internal and External Functional ER) in the happiness condition over the
course of one month. This means that longitudinal administration of the gratitude intervention improved functionality of emotion regulation in the sample with eating disorders. Furthermore, over the course of one month, eating pathology significantly decreased in both happiness and neutral conditions. Decrease in Restraint and overall eating pathology (the overall EDE-Q score) reached statistical significance. The one-off Gratitude exercise was successful in reducing dysfunctional emotion regulation (both Internal and External) in the control group, and externally dysfunctional emotion regulation in the clinical sample (Study 4). This suggests that one-off gratitude-induced happiness may have a different effect on emotion regulation in those with and without eating disorders. In healthy individuals it led to a global improvement in emotion regulation, whereas in patients with eating disorders it was less successful, only improving one type of regulatory strategies. This may be because eating-disordered participants failed to fully benefit from the one-off happiness induction because of the guilt component, associated with the Gratitude exercise. In the process of positive mood induction, participants had to deal with the feelings of guilt and this took their attention away from the purpose of the exercise, making it less effective. This would explain the difference between the groups. However, in the longitudinal study over a period of one month gratitude-induced happiness led to a gradual reduction in dysfunctional and increase in functional emotion regulation strategies in the clinical sample, compared to eating-disordered controls who did the neutral task. This means that positive emotions induced over a period of one month were effective in improving emotion regulation in patients with eating disorders. Higher effectiveness of the longitudinal happiness induction may be explained in terms of increased practice of the Gratitude task. It is plausible that participants became more skilled at managing the guilt arising in the context of the exercise, and were able to benefit from the positive affect induction increasingly more over time. Another finding was that eating pathology decreased in both positive and neutral conditions over the period of one month. This suggests that positive affect may be an important factor in treating eating pathology. It is likely that this lack of difference between the experimental and control groups can be attributable to insufficient participant numbers in the conditions.

These findings are of significance because they have clinical implications. The results suggest that psychotherapeutic treatment of eating disorders would benefit from
incorporating elements of Positive Psychology Interventions, aimed at increasing positive emotions. Other PPIs may also be implemented as part of positive emotion training in individuals with eating disorders. An exercise book of PPIs that contains multiple positive affect-boosting tasks can be put together, and eating-disordered patients may be asked to fill in the book once a week every week, and afterwards discuss how it went with their clinician. Perhaps, this PPI manual can also be used by patients with other disorders, e.g., depression, OCD, where emotion dysregulation is present.

*Effectiveness of Mood Induction Methods (Music + the Velten Mood Induction procedure, Gratitude exercise and Positive Reminiscence task)*

As can be seen on Figure 41, out of the three mood manipulation methods that had been used in this research, music + the Velten Mood Induction procedure was shown to be the most successful in inducing positive mood (85% success rate). It was followed by the Gratitude exercise, which effectively induced happiness in between 61% and 86% of participants (mean success rate = 77%). The Positive Reminiscence task was the least effective of the three, and successfully elicited positive affect in 50% of participants. It is interesting that the Music + Velten method was the most effective; perhaps, this can be attributed to the fact that it involved a combination of two approaches: uplifting music (Music mood induction) plus positive statements (the Velten Mood Induction procedure; Velten, 1968). This combination is very time-efficient because participants are using two different modalities at the same time: sight (the Velten Mood Induction procedure) and hearing (Music mood manipulation); this method also has the added benefit of preventing boredom and tiredness because there is nothing repetitive about it and virtually no effort is required on the part of the participants (mere exposure would suffice).

PPIs on the other hand cannot be used simultaneously. However there is evidence for the *shotgun* effect (Sin and Lyubomirsky, 2009), when using several mood-boosting interventions one after another tends to lead to a bigger improvement in mood than when only one method is used (Sin and Lyubomirsky, 2009; Seligman et al., 2005). This means that if the Positive Reminiscence task and the Gratitude exercise are combined into one positive affect induction method, it may boost the effectiveness of
both. However, in a hypothetical study that combines Positive Reminiscence task and the Gratitude exercise, the participants would have to complete one task before starting the other. This requires extra time, and may also affect participants’ motivation because the two tasks are similar (e.g. both involve writing and require engagement of cognitive processes, such as memory). In future studies it would be interesting to see whether combining a PPI (e.g. the Gratitude exercise) with music would improve its effectiveness. If this is indeed shown to be the case, it may be possible to develop a mood induction procedure that is effective in a 100% of cases. This would have important implications for the research on the effects of positive emotions, as well as the use of positive affect as a palliative treatment in psychotherapeutic settings.

Figure 41: Effectiveness of three methods of mood induction (Positive Reminiscence, Gratitude Exercise and Music + the Velten Mood Induction Procedure)

![Figure 41: Effectiveness of three methods of mood induction (Positive Reminiscence, Gratitude Exercise and Music + the Velten Mood Induction Procedure)](image)

In studies where clinical samples are readily available and there are no geographical obstacles to collecting data in person, the Music + Velten Procedure is recommended; it is the most effective positive mood induction method out of the three that were used in the present research project. More research into the effectiveness of mood induction methods is needed, and the current study provides a stepping stone towards that. The
data, presented on Figure 41, were not statistically analyzed for significant differences, because this was not the purpose of this thesis. In the future it may be useful to compare positive mood induction methods from experimental psychology, such as the Music and Velten procedure, with PPIs from positive psychology, such as the Gratitude task, within the same study. If it is definitively shown that Music + the Velten procedure is more universally effective than PPIs, there is no reason why it cannot be used in positive psychology research to induce positive affect, in addition to or instead of the PPIs.

**Main findings**

Research in this thesis adds to the literature on regulation of emotion in eating disorders. The results show that eating pathology was associated with dysfunctional emotion regulation, high Global and Negative Affect Intensity (Study 1), as well as depression, anxiety and low life satisfaction (Study 3). It was found that eating-disordered participants typically chose unhealthy emotion regulation strategies (Studies 4 and 5). This means that individuals with eating pathology may be more likely to use the full spectrum of unhealthy regulatory strategies, such as: self-harm, rumination, negative social comparison, repression, de-realization (Internal Dysfunctional ER, Phillips and Power, 2007), as well as bullying, verbal assault, making others feel bad, lashing out and physical assault (External Dysfunctional ER, Phillips and Power, 2007). Furthermore, they were found to experience highly intense negative emotions and to be emotionally reactive, which may have contributed to the choice of dysfunctional regulatory strategies. As Study 4 found, the tendency to experience intense negative affect was associated with dysfunctional emotion regulation. On the contrary, the tendency to experience positive affect was related to functional affect management (Study 4).

The eating-disordered participants experienced negative affect (i.e. sadness, anger, fear, disgust, guilt and anxiety) intensely and frequently, and negative emotions were salient for them. They experienced positive affect rarely, if ever, and it was fleeting and easily disrupted by interfering negative affect. The participants also reported that they suffered from extreme fluctuations in affect, and were poor at eliciting and sustaining positive emotions. They were also dissatisfied with their lives (Studies 4 and 5). The participants
with eating disorders reported that the two main sources of positive emotions for them were: (1) spending time with friends and family and (2) achieving goals (Study 5). Overall, dysfunctional emotion regulation was associated with anxiety, depression, low life satisfaction (Study 3), as well as high Negative Affect Intensity and Global Affect Intensity (Study 1). This means that individuals who use dysfunctional regulatory strategies are more likely to be anxious, depressed, unhappy, and to have the tendency to experience intense emotions. The combination of Music and the Velten Mood Induction procedure were very effective in inducing happiness and sadness (success rate \( \geq 85\% \)). Positive emotions led to a reduction in dysfunctional emotion regulation. Mood induction did not significantly affect Negative Intensity scores, but it did influence Global Affect Intensity and Positive Affect Intensity (Study 1). In both the clinical and subclinical samples, PPIs were easy to implement and successfully used to induce positive mood. The Gratitude exercise was more effective (success rate = 61\%) than the Positive Reminiscence task (success rate = 50\%). Positive Affect, induced using PPIs, had a significant effect on emotion regulation, increasing functional ER and life satisfaction, as well as reducing dysfunctional affect management (Study 3). Studies 4 and 5 made a number of interesting findings in the field of emotion regulation and eating pathology. The Gratitude exercise was successful at positive mood induction and was very well-received; participants reported gaining a wide range of benefits from it on physical, cognitive and psychological levels. Furthermore, there was a gradual sustainable increase in baseline happiness over the period of four weeks, and participants were happier at the end of the study (Week 4) than they were at the beginning (Week 1). Following the happiness induction, participants were less likely to choose dysfunctional emotion regulation, and over the period of 4 weeks, their dysfunctional ER and eating pathology scores decreased.

The findings that experimentally-induced positive emotions have the potential to improve emotion regulation, increase well-being and reduce eating pathology have important implications for treatment of eating disorders: (1) Elements of positive psychology interventions and other well-being boosting activities may benefit treatment, if incorporated into therapies for eating pathology alongside interventions targeting negative affect. This would allow for a comprehensive treatment of affect (positive and negative); (2) clinicians should pay more attention to positive affect, and
challenges, associated with eliciting and sustaining it. In a therapeutic setting, patients may need to be educated how to elicit and maintain positive emotions in themselves; (3) the tendency to experience intense negative affect needs to be taken into account when explaining emotion regulation choices in eating-disordered patients, as well as healthy individuals. This may require administration of the emotion regulation measures alongside Affect Intensity scales. This is a promising line of research, and more studies are needed in order to elucidate the relationship between affect and its regulation in eating disorders.
REFERENCES


Buckholdt, K., Parra, G. and Jobe-Shields (2010). Emotion dysregulation as a mechanism through which parental magnification of sadness increases risk for binge eating and limited control of eating behaviours. *Eating Behaviours, 11*, 122-126;


Eisenberg, N. and Okun, M. (1996). The relations of dispositional regulation and emotionality to elders’ empathy-related responding and affect while volunteering. *Journal of Personality, 64*, 157-183;


Lyman, B. (1982). The nutritional values and food group characteristics of foods preferred during various emotions. *Journal of Psychology*, 112, 121-127;


Snaith, R. (2003). The Hospital Anxiety and Depression Scale. Health and Quality of Life Outcomes, 1 (29), 1-4;


APPENDICES

Appendix 1: Regulation of Emotion Questionnaire (REQ; Phillips and Power, 2007)

We all experience lots of different feelings or emotions. For example, different things in our lives make us feel happy, sad, angry and so on… The following questions ask you to think about how often you do certain things in response to your emotions. You do not have to think about specific emotions but just how often you generally do the things listed below. Please tick the box corresponding to the answer that fits best. We all respond to our emotions in different ways so there are no right or wrong answers.

<table>
<thead>
<tr>
<th>In general how do you respond to your emotions?</th>
<th>Never</th>
<th>Seldom</th>
<th>Often</th>
<th>Very Often</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I talk to someone about how I feel</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. I take my feelings out on others verbally (e.g. shouting, arguing)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. I seek physical contact from friends or family (e.g. a hug, hold hands)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. I review (rethink) my thoughts or beliefs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. I harm or punish myself in some way</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. I do something energetic (e.g. play sport, go for a walk)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. I dwell on my thoughts and feelings (e.g. It goes round and round in my head and I can’t stop it)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Never</td>
<td>Seldom</td>
<td>Often</td>
<td>Very Often</td>
</tr>
<tr>
<td>---</td>
<td>------------------------------------------------------------------</td>
<td>-------</td>
<td>--------</td>
<td>-------</td>
<td>------------</td>
</tr>
<tr>
<td>8</td>
<td>I ask others for advice</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td>o</td>
</tr>
<tr>
<td>9</td>
<td>I review (rethink) my goals or plans</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td>o</td>
</tr>
<tr>
<td>10</td>
<td>I take my feelings out on others physically (e.g. fighting, lashing out)</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td>o</td>
</tr>
<tr>
<td>11</td>
<td>I put the situation into perspective</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td>o</td>
</tr>
<tr>
<td>12</td>
<td>I concentrate on a pleasant activity</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td>o</td>
</tr>
<tr>
<td>13</td>
<td>I try to make others feel bad (e.g. being rude, ignoring them)</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td>o</td>
</tr>
<tr>
<td>14</td>
<td>I think about people better off and make myself feel worse</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td>o</td>
</tr>
<tr>
<td>15</td>
<td>I keep the feeling locked up inside</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td>o</td>
</tr>
<tr>
<td>16</td>
<td>I plan what I could do better next time</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td>o</td>
</tr>
<tr>
<td>17</td>
<td>I bully other people (e.g. saying nasty things to them, hitting them)</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td>o</td>
</tr>
<tr>
<td>18</td>
<td>I take my feelings out on objects around me (e.g. deliberately causing damage to my house, school etc.)</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td>o</td>
</tr>
<tr>
<td>19</td>
<td>Things feel unreal (e.g. I feel strange, things around me feel strange, I daydream)</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td>o</td>
</tr>
<tr>
<td>20</td>
<td>I telephone friends or family</td>
<td>o</td>
<td>o</td>
<td>o</td>
<td>o</td>
</tr>
<tr>
<td></td>
<td>Never</td>
<td>Seldom</td>
<td>Often</td>
<td>Very Often</td>
<td>Always</td>
</tr>
<tr>
<td>-------</td>
<td>-------</td>
<td>--------</td>
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<td>------------</td>
<td>--------</td>
</tr>
<tr>
<td>21.</td>
<td>〇</td>
<td>〇</td>
<td>〇</td>
<td>〇</td>
<td>〇</td>
</tr>
<tr>
<td></td>
<td>I go out and do something nice (e.g. cinema, shopping, go for a meal, meet people)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix 2: Affect Intensity Measure Questionnaire (Larsen, 1984)

The following questions refer to emotional reactions to typical life-events. Please indicate how YOU react to these events by placing a number from the following scale in the blank space preceding each item. Please base your answers on how YOU react, not on how you think others react or how you think a person should react.

e.g. 1. ___ 6__ When I accomplish something difficult I feel delighted or elated.

Never – 1
Almost Never – 2
Occasionally - 3
Usually - 4
Almost Always - 5
Always – 6

1. _____ When I accomplish something difficult I feel delighted or elated.
2. _____ When I feel happy it is a strong type of exuberance.
3. _____ I enjoy being with other people very much.
4. _____ I feel pretty bad when I tell a lie.
5. _____ When I solve a small personal problem, I feel euphoric.
6. _____ My emotions tend to be more intense than those of most people.
7. _____ My happy moods are so strong that I feel like I'm in heaven.
8. _____ I get overly enthusiastic.
9. _____ If I complete a task I thought was impossible, I am ecstatic.
10. _____ My heart races at the anticipation of some exciting event.
11. _____ Sad movies deeply touch me.
12. _____ When I'm happy it's a feeling of being untroubled and content rather than being zestful and aroused.
13. _____ When I talk in front of a group for the first time my voice gets shaky and my heart races.
14. _____ When something good happens, I'm usually much more jubilant than others.
15. _____ My friends might say I'm emotional.
16. _____ The memories I like the most are of those times when I felt content and peaceful rather than zestful and enthusiastic.
17. _____ The sight of someone who is hurt badly affects me strongly.
18. _____ When I'm feeling well it's easy for me to go from being in a good mood to being really joyful.
19. _____ "Calm and cool" could easily describe me.
20. _____ When I'm happy I feel like I'm bursting with joy.
21. _____ Seeing a picture of some violent car accident in a newspaper makes me feel sick to my stomach.
22. _____ When I'm happy I feel very energetic.
23. _____ When I receive a reward I become overjoyed.
24. _____ When I succeed at something, my reaction is calm and contentment.
25. _____ When I do something wrong I have strong feelings of shame and guilt.
26. _____ I can remain calm even on the most trying days.
27. _____ When things are going good I feel 'on top of the world'.
28. _____ When I get angry it's easy for me to still be rational and not overreact.
29. _____ When I know I have done something very well, I feel relaxed and content rather than excited and elated.
30. _____ When I do feel anxiety it is normally very strong.
31. _____ My negative moods are mild in intensity.
32. _____ When I am excited over something I want to share my feelings with everyone.
33. _____ When I feel happiness, it is a quiet type of contentment.
34. _____ My friends would probably say I'm a tense or 'high-strung' person.
35. _____ When I'm happy I bubble over with energy.
36. _____ When I feel guilty, this emotion is quite strong.
37. _____ I would characterize my happy moods as closer to contentment than joy.
38. _____ When someone compliments me, I get so happy I could 'burst'.
39. _____ When I am nervous I get shaky all over.
40. _____ When I am happy the feeling is more like contentment and inner calm than one of exhilaration and excitement.
Appendix 3: Satisfaction with Life Scale (Diener, 1985)

Please use one of the following numbers from 1 to 7 to indicate how much you agree or disagree with the following statements.

7 = Strongly Agree  
6 = Agree  
5 = Slightly agree  
4 = Neither agree, nor disagree  
3 = Slightly disagree  
2 = Disagree  
1 = Strongly disagree

e.g. 1.____6____In most ways my life is close to my ideal

1.________In most ways my life is close to my ideal  
2.________The conditions of my life are excellent  
3.________I am satisfied with my life  
4.________So far I have got the important things I want in my life  
5.________In I could start my life over, I would change almost nothing
Appendix 4: Eating Disorder Examination Questionnaire (EDE-Q; Fairburn and Beglin, 1994)

The following questions are concerned with the PAST FOUR WEEKS ONLY (28 days). Please read each question carefully and circle the appropriate number on the right. Please try to answer all the questions.

<table>
<thead>
<tr>
<th>ON HOW MANY DAYS OUT OF THE PAST 28 DAYS ........</th>
<th>No days</th>
<th>1-5 days</th>
<th>6-12 days</th>
<th>13-15 days</th>
<th>16-22 days</th>
<th>23-27 days</th>
<th>Every day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Have you been deliberately trying to limit the amount of food you eat to influence your shape or weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>2. Have you gone for long periods of time (8 hours or more) without eating anything in order to influence your shape or weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>3. Have you tried to avoid eating any foods which you like in order to influence your shape or weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>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?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>5. Have you wanted your stomach to be empty?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>6. Has thinking about food or its calorie content made it much more difficult to concentrate on things you are interested in; for example, read, watch TV, or follow a conversation?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>7. Have you been afraid of losing control over eating?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>8. Have you had episodes of binge eating?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>9. Have you eaten in secret? (Do not count binges.)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>10. Have you definitely wanted your stomach to be flat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>11. Has thinking about shape or weight made it more difficult to concentrate on things you are interested in; for example, read, watch TV or follow a conversation?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>12. Have you had a definite fear that you might gain weight or become fat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>13. Have you felt fat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>14. Have you had a strong desire to lose weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>
OVER THE PAST FOUR WEEKS (28 DAYS)

<table>
<thead>
<tr>
<th>Question</th>
<th>Options</th>
</tr>
</thead>
</table>
| 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) | 0 – None of the times
1 – A few of the times
2 – Less than half the times
3 – Half the times
4 – More than half the times
5 – Most of the times
6 – Every time |
| 16. Over the past four weeks (28 days), 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? (Please circle YES or NO and put appropriate number in box) | YES
NO |
| 17. How many such episodes have you had over the past four weeks?       | YES
NO |
| 18. During how many of these episodes of overeating did you have a sense of having lost control over your eating? | YES
NO |
| 19. 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? | YES
NO |
| 20. How many such episodes have you had over the past four weeks?       | YES
NO |
| 21. Over the past four weeks have you made yourself sick (vomit) as a means of controlling your shape or weight? | YES
NO |
| 22. How many times have you done this over the past four weeks?         | YES
NO |
| 23. Have you taken laxatives as a means of controlling your shape or weight? | YES
NO |
| 24. How many times have you done this over the past four weeks?         | YES
NO |
| 25. Have you taken diuretics (water tablets) as a means of controlling your shape or weight? | YES
NO |
| 26. How many times have you done this over the past four weeks?         | YES
NO |
| 27. Have you exercised hard as a means of controlling your shape or weight? | YES
NO |
| 28. How many times have you done this over the past four weeks?         | YES
NO |
OVER THE PAST FOUR WEEKS (28 DAYS)  
(PLEASE CIRCLE THE NUMBER WHICH BEST DESCRIBES YOUR BEHAVIOUR)  

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>NOT AT ALL</th>
<th>SLIGHTLY</th>
<th>MODERATELY</th>
<th>MARKEDLY</th>
</tr>
</thead>
<tbody>
<tr>
<td>29. Has your weight influenced how you think about (judge) yourself as a person?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>30. Has your shape influenced how you think about (judge) yourself as a person?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>31. How much would it upset you if you had to weigh yourself once a week for the next four weeks?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>32. How dissatisfied have you felt about your weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>33. How dissatisfied have you felt about your shape?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>34. How concerned have you been about other people seeing you eat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>35. How uncomfortable have you felt seeing your body; for example, in the mirror, in shop window reflections, while undressing or taking a bath or shower?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>36. How uncomfortable have you felt about others seeing your body: for example, in communal changing rooms, when swimming or wearing tight clothes?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
Appendix 5: Eating Attitudes Test (EAT-26; Garner et al., 1982)

Please respond to each of the following statements:

Scale:
Always
Usually
Often
Sometimes
Rarely
Never

1. Am terrified about being overweight
2. Avoid eating when I am hungry
3. Find myself preoccupied with food
4. Have gone on eating binges where I feel that I may not be able to stop
5. Cut my food into small pieces
6. Aware of the calorie content of foods that I eat
7. Particularly avoid foods with a high carbohydrate content (i.e. bread, rice, potatoes, etc.)
8. Feel that others would prefer if I ate more
9. Vomit after I have eaten
10. Feel extremely guilty after eating
11. Am preoccupied with a desire to be thinner
12. Think about burning up calories when I exercise
13. Other people think that I am too thin
14. Am preoccupied with the thought of having fat on my body
15. Take longer than others to eat my meals
16. Avoid foods with sugar in them
17. Eat diet foods
18. Feel that food controls my life
19. Display self-control around food
20. Feel that others pressure me to eat
21. Give too much time and thought to food
22. Feel uncomfortable after eating sweets
23. Engage in dieting behaviour
24. Like my stomach to be empty
25. Enjoy trying new rich foods
26. Have the impulse to vomit after meals

Please respond to each of the following questions:

1) Have you gone on eating binges where you feel that you may not be able to stop? (Eating much more than most people would eat under the same circumstances)
   No ___ Yes ___ How many times in the last 6 months? ________

2) Have you ever made yourself sick (vomited) to control your weight or shape?
   No ___ Yes ___ How many times in the last 6 months? ________

3) Have you ever used laxatives, diet pills or diuretics (water pills) to control your weight or shape?
   No ___ Yes ___ How many times in the last 6 months? ________

4) Have you ever been treated for an eating disorder? No ___ Yes ___ When? ________

5) Have you recently thought of or attempted suicide? No ___ Yes ___ When? ________

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Appendix 6: The SCOFF Questionnaire (Morgan et al., 1999)

Please circle as appropriate

1. Do you make yourself sick because you feel uncomfortably full?
   Yes
   No

2. Do you worry you have lost control over how much you eat?
   Yes
   No

3. Have you recently lost more than one stone in a 3 month period?
   Yes
   No

4. Do you believe yourself to be fat when others say you are too thin?
   Yes
   No

5. Would you say that food dominates your life?
   Yes
   No
Appendix 7: Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983)

Please choose one response from the four given for each question. Try to give an immediate response and do not think too long about your answers.

A I feel tense or ‘wound up’:
Most of the time  3
A lot of the time  2
From time to time, occasionally  1
Not at all  0

D I still enjoy the things I used to enjoy:
Definitely as much  0
Not quite so much  1
Only a little  2
Hardly at all  3

A I get a sort of frightened feeling as if something awful is about to happen:
Very definitely and quite badly  3
Yes, but not too badly  2
A little, but it doesn’t worry me  1
Not at all  0

D I can laugh and see the funny side of things:
As much as I always could  0
Not quite so much now  1
Definitely not so much now  2
Not at all  3

A Worrying thoughts go through my mind:
A great deal of the time  3
A lot of the time  2
From time to time, but not too often  1
Only occasionally  0

D I feel cheerful:
Not at all  3
Not often  2
Sometimes  1
Most of the time  0

A I can sit at ease and feel relaxed:
Definitely  0
Usually  1
Not Often  2
Not at all  3

D I feel as if I am slowed down:
Nearly all the time  3
Very often  2
Sometimes  1
Not at all  0

A I get a sort of frightened feeling like 'butterflies' in the stomach:
Not at all  0
Occasionally  1
Quite Often  2
Very Often  3

D I have lost interest in my appearance:
Definitely
I don't take as much care as I should
I may not take quite as much care
I take just as much care as ever

A
I feel restless as I have to be on the move:
Very much indeed
Quite a lot
Not very much
Not at all

D
I look forward with enjoyment to things:
As much as I ever did
Rather less than I used to
Definitely less than I used to
Hardly at all

A
I get sudden feelings of panic:
Very often indeed
Quite often
Not very often
Not at all

D
I can enjoy a good book or radio or TV program:
Often
Sometimes
Not often
Very seldom
Appendix 8: The Velten Mood Induction Procedure (Velten, 1968)

Happiness Items

I feel good today.
I have complete confidence in myself.
I feel light-hearted.
If your attitude is good, then things are good, and my attitude is good.
I’ve certainly got energy and self-confidence to spare.
Sometimes it feels good to get away from the noise by going to a park.
I usually feel at ease when I meet new people.
I’m pleased that most people are so friendly to me.
I feel cheerful and lively.
My parents are pretty proud of me most of the time.
I can just imagine myself on a warm summer day on the beach – the surf gently rolling in, gulls calling out, the salt in the air, the warm sun on my body.
I have a fresh outlook on life. I’m secure in my optimism.
There should be opportunity for a lot of good times coming.
My judgement about most things is sound.
This is one of those days when I can grind out work with practically no effort at all.
I feel enthusiastic and confident now.
I feel a calm acceptance of everyone.
I like to imagine myself high up on a mountain top, fresh air, so quiet.
I feel that no matter what happens, I can make the best of it and be happy.
I feel laid-back and content.
I know I can achieve the goals I set.
I know that in the future I won’t let so-called “problems” get me down.
I’m optimistic that I can get along very well with most people that I meet.
I’m feeling amazingly good today!
I have a sense of power and vigour.
Things look good. Things look great!
I feel that many of my friendships will stick with me in the future.
I can find the good in almost anything.
I feel so happy and playful today.
I feel free and unencumbered.
Things will be better and better today.
I can almost imagine a sea breeze blowing through my hair.
When I want to I can make friends extremely easily.
I’m full of energy and ambition.
My favourite tune keeps going through my head.
Life is firmly in my control.
This is great - I really do feel good.
I feel like bursting with laughter – I wish somebody would tell me a joke and give me an excuse!
This is one of those days when I’m ready to go.
I feel great!!!

Neutral Items

Nothing can be burned that has already been burned once.
Japan was elected to the United Nations almost fourteen years after Pearl Harbour.
We have two kinds of nouns denoting physical things: individual and mass nouns.
Saturn is sometimes in conjunction beyond the sun from the Earth, and is not visible.
There is a large rose-growing centre near Reading, Berkshire.
Milk contains calcium.
The term Union Jack possibly dates from Queen Anne’s time (reigned 1702-1714).
The Orient Express travels between Paris and Istanbul.
There isn’t a scientific explanation for every U.F.O. sighting.
The Hope Diamond was shipped from South America to London through the regular mail service.
Slang is a constantly changing part of the language.
In 2001 over 55 million cigarettes were smoked in the UK.
99.1% of Alaska is owned by the US government.
West Samoa gained its independence in 1965.
Potter wrote numerous satires on social cynicism.

The lowest recorded temperature in the Antarctic was measured at –84 degrees Celsius.

The eyes and nose of a frog are on top of its head, enabling it to breathe and see when most of its body is under the water.

In 1965, Elizabeth made the first state visit by a British monarch to Germany in 56 years.

Light travels at 186,000 miles a second.

The town of Tidikelt in the Sahara Desert once went ten years without rainfall.

The Chinese language has many dialects, including Mandarin and Cantonese.

Descartes came up with the theory of coordinate geometry by looking at a fly walking across a tiled ceiling.

Grizzly bears often walk in the same footprints they made the previous day.

English is the most common second language.

Unlike most African nations, Ethiopia was never a European colony.

Nerve signals may travel through nerve or muscle fibres at speeds as high as 200 miles per hour.

The Roman Emperor, Caligula, appointed his favourite horse, Incitatus, a consul of Rome.

Some 99% of the world’s chromium is found in South Africa and Zimbabwe.

Charles DeGaulle was elected President of France in 1965.

The lowest point in the US is Death Valley, at 282 feet below sea level.

Illinois produces more corn than any other state.

The Plague is Albert Camus’ best-known novel.

At one time, the Eastern and Western hemispheres were a single landmass.

In hockey, a “butterfly” is a goaltending style in which the goalie keeps his knees together and feet slightly apart.

The first CD pressed in the US for commercial release was Bruce Springsteen’s “Born in the USA”.

An estimated total of 210,000 battle deaths occurred during the Civil War.

A dragonfly flaps its wings 20 to 40 times a second, bees and houseflies 200 times, some mosquitoes 600 times, and a tiny gnat 1,000 times.

Spain was settled by Iberians, Basques, and Celts, and was conquered by the Romans in 200 BC.

Winds ten times stronger than a hurricane on Earth blow around Saturn’s equator.

The average fox weighs 14 pounds.
Sadness Items

I feel a little low today

I get the feeling people who are friendly to me are just being “nice” and don’t really like me.

I’m afraid the economic situation for my generation looks pretty bleak.

I can remember times when everybody but me seemed happy and full of energy.

Often I have found myself staring into the distance, my mind a blank, when I definitely should have been working.

People annoy me; I wish I could be by myself.

I’ve had important decisions to make in the past, and I’ve sometimes made the wrong ones.

There have been days when I felt confused and everything went miserably wrong and I was powerless to stop it.

No matter how hard I try, I just can’t help feeling that things are going to get worse and worse.

I’ve had daydreams in which I kept reliving past mistakes – sometimes I wish I could start over again.

I’m ashamed that I’ve caused my parents needless worry.

Just when I think things are going to get better, something else goes wrong.

The world is so full of suffering and unhappiness that no matter how hard I try, sometimes it really gets me down.

At times I’ve felt so tired and discouraged that I went to sleep rather than face important problems.

My life is so tiresome – the same old thing day after day depresses me.

I couldn’t remember things right now if I had to.

I just can’t make up my mind; it’s so hard to make simple decisions.

I’ve doubted that I’m a worthwhile person.

It often seems that no matter how hard I try, things still go wrong.

I’ve noticed that no one seems to really understand or care when I complain or feel unhappy.

I’m uncertain about my future

I’m discouraged and unhappy about myself.

Things are worse now than when I was younger.

The way I feel now, the future looks boring and hopeless.

Some very important decisions are almost impossible for me to make.

I feel horribly guilty about how I’ve treated my parents at times
Things are easier and better for other people than me. I feel like there is no use in trying.
It takes too much effort to convince people of anything; there’s no point in trying.
Often people make me very upset. I don’t like to be around them.
I fail in communicating with people about my problems.
I’ve felt so alone before that I could have cried.
My thoughts are so slow and downcast I don’t want to think or talk.
I’ve lain awake at night worrying so long that I hated myself.
I have the feeling that I just can’t reach people.
I just don’t care about anything. Life just isn’t any fun.
I have too many bad things in my life.
It’s so discouraging the way people don’t really listen to me.
Everything seems utterly futile and empty.
I’m haunted with thoughts about myself and how I come across to others.
All of the unhappiness of my past is taking possession of me.
Appendix 9: The Emotional Assessment Scale (EAS; Carlson et al., 1989)

For each word on this questionnaire, please place a slash somewhere on the appropriate line to indicate how you are feeling at the moment.

*e.g. Surprised*

Least possible____________________________________Most possible

**Surprised**

Least possible____________________________________Most possible

**Afraid**

Least possible____________________________________Most possible

**Disgusted**

Least possible____________________________________Most possible

**Angry**

Least possible____________________________________Most possible

**Guilty**

Least possible____________________________________Most possible

**Anxious**

Least possible____________________________________Most possible

**Sad**

Least possible____________________________________Most possible

**Delighted**

Least possible____________________________________Most possible

**Scared**

Least possible____________________________________Most possible

**Astonished**

Least possible____________________________________Most possible
Repulsed
Least possible__________________________________________Most possible

Mad
Least possible__________________________________________Most possible

Ashamed
Least possible__________________________________________Most possible

Worried
Least possible__________________________________________Most possible

Disturbed
Least possible__________________________________________Most possible

Joyful
Least possible__________________________________________Most possible

Frightened
Least possible__________________________________________Most possible

Amazed
Least possible__________________________________________Most possible

Sickened
Least possible__________________________________________Most possible

Annoyed
Least possible__________________________________________Most possible

Humiliated
Least possible__________________________________________Most possible
Nervous
Least possible__________________________________________Most possible

Hopeless
Least possible__________________________________________Most possible

Happy
Least possible__________________________________________Most possible
Appendix 10: Creative Behaviour Inventory (Hocevar, 1980)

This is an inventory, not a test. The inventory is simply a list of activities and accomplishments that are commonly considered to be creative. For each item, circle the answer that best describes the frequency of the behaviour in your adolescent and adult life. Be sure to answer every question.

Scale:

Never

Once or Twice

3-5 Times

More than 5 times

1. Received an award for acting
2. Worked as an editor for a school or university literary publication
3. Worked as an editor for a newspaper or similar organization
4. Constructed something that required scientific knowledge such as a radio, telescope, scientific apparatus, etc. (excluding school or university course work)
5. Painted an original picture (excluding school or university course work)
6. Entered a speech contest
7. Designed and made your own greeting cards
8. Gave a recital
9. Presented an original mathematics paper to a professional or special interest group
10. Founded a literary magazine or similar publication
11. Made a craft out of metal (excluding school or university coursework)
12. Made candles
13. Knitted or crocheted something (excluding school or university coursework)
14. Put on a puppet show
15. Made your own holiday decorations
16. Built a hanging mobile (excluding school or university coursework)
17. Received an award for performance in modern dance or ballet
18. Received an award for performance in popular dance
19. Had a mathematics paper published
20. Made a sculpture (excluding school or university coursework)
21. Had original music published or publically performed
22. Had a piece of literature (e.g., poem, short stories, etc.) published in a school or university publication
23. Developed an experimental design (excluding school or university work)
24. Wrote poems (excluding school or university course work)
25. Wrote a play
26. Entered a project or a paper into a science contest
27. Received an award for an artistic accomplishment
28. Received an award for making a craft
29. Made a craft out of plastic, plexiglass, stained glass, or a similar material (excluding school or university coursework)
30. Made cartoons
31. Made a leather craft (excluding school or university course work)
32. Made a ceramic craft (excluding school or university course work)
33. Wrote music for one instrument (excluding school or university course work)
34. Wrote music for several instruments (excluding school or university course work)
35. Designed and made a piece of clothing (excluding school or university course work)
36. Cooked an original dish
37. Prepared an original floral arrangement
38. Applied math in an original way to solve a practical problem
39. Wrote an original computer program (excluding school or university course work)
40. Drew a picture for aesthetic reasons (excluding school or university course work)
41. Wrote the lyrics to a song (excluding school or university course work)
42. Choreographed a dance (excluding school or university course work)
43. Wrote a short story (excluding school or university course work)
44. Applied math in an original way to solve a practical problem
45. Wrote a clever or humorous letter
46. Directed or organized a political group
47.Entered a mathematical paper or project into a contest
48. Planned and kept a garden
49. Entered a contest as a musician
50. Started but did not finish a novel (excluding school or university course work)
51. Made a musical instrument
52. Started but did not finish a novel (excluding school or university course work)
53. Helped design a float
54. Entered a contest as a singer
55. Entered a contest as a musician
56. Designed and constructed a craft out of wood (excluding school or university course work)
57. Planned and directed a school or community event
58. Won an award for some achievement in literature
59. Entered a contest as a musician
60. Won an award for some achievement in literature
61. Entered a mathematical paper or project into a contest
62. Had a scientific paper published
63. Entered a contest as a singer
64. Entered a contest as a musician
65. Planted and directed a school or community event
66. Planned and directed a school or community event
67. Planned and directed a school or community event
68. Planned and directed a school or community event
69. Planned and directed a school or community event
70. Planned and directed a school or community event
71. Planned and directed a school or community event
72. Planned and directed a school or community event
73. Planned and directed a school or community event
74. Planned and directed a school or community event
75. Planned and directed a school or community event
76. Planned and directed a school or community event
77. Planned and directed a school or community event
78. Planned and directed a school or community event
79. Wrote a play which was given a public performance
80. Directed or managed a dramatic production
81. Designed and made a costume
82. Made up magic tricks
83. Played an instrument (percussion, including piano) with a reasonable degree of proficiency
84. Played an instrument (string) with a reasonable degree of proficiency
85. Played an instrument (brass) with a reasonable degree of proficiency
86. Played an instrument (wind) with a reasonable degree of proficiency
87. Participated in a drama workshop, club or similar organization (excluding school or university course work)
88. Participated in a craft workshop, club or similar organization (excluding school or university course work)
89. Participated in a writer’s workshop, club or similar organization (excluding school or university course work)
90. Participated in a dance workshop, club or similar organization (excluding school or university course work).
Appendix 11: The Informed Consent Form

Please complete and sign the form below to show that you have read and understood the Information Sheet and that you agree to take part in the study.

CONSENT FORM

Please circle as appropriate

Have you read the Information sheet?............................................................YES/NO

Have you received enough information about the study?...............................YES/NO

Do you understand that all the information you provide will be kept anonymous and confidential?.............................................................................................YES/NO

Do you understand that you do not need to take part unless you want to and if you do take part, you are free to withdraw:
- at any time
- without giving any reason for doing so
- and without any consequence to you? .................................YES/NO

Do you agree to take part in the study?............................................................YES/NO

Signed………………………………………………………………………………….

Your name (in block capitals)………………………………………………………….

Your age (years)………………………………………………….

Your email address………………………………………………………….

Your home address…………………………………………………………………………………

……………………………………………………………………………………………………

Your phone number……………………………………………………………………

Date………………………………..

THIS FORM WILL BE NOT BE KEPT WITH ANY ANSWERS THAT YOU GIVE

Thank you.
Appendix 12: Information Sheet for the Eating-Disordered Sample (Study 4 and 5)

I would like to invite you to take part in my research project looking at positive emotions in individuals with the diagnoses of Anorexia Nervosa, Bulimia Nervosa and EDNOS.

The following information is to help you decide whether you would like to take part. It is arranged in the form of questions and answers, and gives details of what the research project involves.

If having read this information sheet, you still have some questions, please do not hesitate to get in touch with me (my contact details are at the end of this Information Sheet) and I will do my best to help you with your query.

Please keep this Information Sheet for your records.

Questions, answered in this Information Sheet:
Q 1: What is the purpose of the study and who is conducting it?
Q 2: What will I have to do?
Q 3: What are the possible advantages of taking part?
Q 4: What are the possible disadvantages of taking part?
Q 5: What if something goes wrong?
Q 6: What if I don’t want to carry on with the study?
Q 7: Will the information I provide (personal details as well as answers on questionnaires) be kept confidential?
Q 8: What will happen to the results of the study?
Q 9: Who is organizing the research?
Q 10: Who has reviewed the study?
Q 11: What if I want to get independent advice and further information?
Q 12: How do I take part?

Q 1: What is the purpose of the study and who is conducting it?

- This research project is part of the PhD in Clinical Psychology that I am doing. The main aim is to look at how individuals with eating disorders regulate their emotions, and at the role of positive emotions on their health over time.

- About me:

My name is Augustina Skoropadskaya. I graduated from the University of Edinburgh with an MA Psychology (Hons.), and stayed on to do a PhD in Clinical Psychology at the School of Health in Social Science. I am interested in how individuals with eating disorders regulate their emotions, and how we can maximize effectiveness of psychotherapeutic treatments. I am currently at the end of my second year.

Q 2: What will I have to do?

The study has two separate parts. You can participate in both, or just the part of your choosing.

- **PART 1:** In the first part you will be asked to fill out a battery of questionnaires on your emotions and eating behaviours, and do a short writing task in between. The task would require you to spend some time thinking about something or recalling something, and then writing about it. The task will be one of simple writing exercises, to which you will be randomly assigned. Stamped return envelopes will be provided.

  Total time: approximately 30 mins.

- **PART 2:** Participation involves filling out a set of questionnaires at 3 time intervals (at the start of Part 2, 2 weeks in and 4 weeks in), and completing a short writing task on a weekly basis for 4 weeks. The task again will be one of simple writing exercises, to which you will be randomly assigned.
For instance, if you fill out the first set of questionnaires on Monday, 5th July, this means that you will be doing the exercises for the following 28 days, and Sunday, 1st of August will be the last day. In this example, you would fill out your second set of questionnaires on Sunday, 18th June and the third – on Sunday, 1st of August. Over the course of the month, you would have filled out 3 sets of questionnaires, and done 4 writing tasks.

I may contact you by email from time to time to check on how you are getting on with the exercises and the questionnaires. I will also send you reminders about the writing exercise every week, and post the questionnaire sets to you every 2 weeks. Stamped return envelopes will be provided. You are welcome to contact me any time with regard to anything to do with the research project and your participation.

Total time: approximately 15-25 minutes per week over 4 weeks.

Q 3: What are the possible advantages of taking part?
There is evidence to suggest that taking part in a study looking at positive emotions may benefit your physical and mental health. Participation does not take a lot of time, it is easy and the study is designed so that you enjoy taking part as much as possible. Negative outcomes are extremely unlikely. This is a new exciting line of research, and you get the opportunity to advance clinical psychology by making your contribution.

Q 4: What are the possible disadvantages of taking part?
Some participants may find that dedicating around 25 minutes per week over 4 weeks is too much of a demand on their time or health. This is completely understandable, and if that’s the case for you but you still would like to be involved, I would be happy for you to do just one part of the study.

Q 5: What if something goes wrong?
If during the course of the study you feel unwell, you can contact your GP, your doctor and myself for advice. You can also phone B-EAT on 0300 123 3355, or log on to www.b-eat.co.uk. If you have a complaint, please get in touch with me in the first instance. I will try to resolve the issue to the best of my ability.

Q 6: What if I don’t want to carry on with the study?
If you wish to drop out of the study, you are free to do so at any time with no consequences to you. I would be grateful if you could inform me of your decision if you do decide to drop out; this is so that I do not send you any further correspondence. We can also stop the study temporarily, for instance, to give you a chance to get better, and then start again at a later date, if you wish to do so.

Q 7: Will the information I provide (personal details as well as answers on questionnaires) be kept confidential?
Yes, all the information that you’ll provide will be kept anonymous and confidential, and only my project supervisor and I will have access to it.

Q 8: What will happen to the results of the study?
Results will be written up and submitted as part of my PhD thesis. Please note that you will not be personally identified in the thesis, and it will not be possible to link you to the study.

Q 9: Who is organizing the research?
The research project is organized by the University of Edinburgh.

Q 10: Who has reviewed the study?
This study has been reviewed and approved by the School of Health in Social Science Ethics Committee (University of Edinburgh).

Q 11: What if I want to get independent advice and further information?
Independent Advice:
You can talk to your GP, your clinician or obtain independent advice by phoning B-EAT (eating disorders charity) on 0300 123 3355, or by going to www.b-eat.co.uk.

Specific information about this research project:
Please feel free to get in touch with me by phoning ************, or emailing ************. I will be happy to answer any questions, and I sincerely hope that you will decide to take part in my research project.

Q 12: How do I take part?
If having read this information sheet, you decide to take part in my research project, please EMAIL ME WITH YOUR NAME, AGE, DIAGNOSIS AND POSTAL ADDRESS FOR CORRESPONDENCE at ************.

I look forward to hearing from you.

Kind regards,
Augustina

THANK YOU
Appendix 13: Data screening statistics for Study 1

Key:
BM = Before Mood Induction
AM = After Mood Induction
InDys = Internal Dysfunctional emotion regulation
ExDys = External Dysfunctional emotion regulation
ExFun = External Functional emotion regulation
InFun = Internal Functional emotion regulation
Positive AI = Positive Affect Intensity
Negative AI = Negative Affect Intensity
Global AI = Global Affect Intensity

Table 1: Skewness, Kurtosis and Z-values for Emotion Regulation, Affect Intensity, Anxiety and Depression variables (before mood induction)

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Table 2: Skewness, Kurtosis and Z-values for Emotion Regulation and Affect Intensity variables (after mood induction)

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Figure 1: Error bars for Emotion Regulation sub-scales (Internal Dysfunctional, Internal Functional, External Dysfunctional and External Functional emotion regulation) in controls and participants with subclinical eating disorders.
Figure 2: Error bars for affect intensity (Global, Positive and Negative) in controls and participants with subclinical eating disorders.
Appendix 14: Data screening statistics for Study 2

Key:

BM = Before Mood Induction
AM = After Mood Induction
InDys = Internal Dysfunctional emotion regulation
ExDys = External Dysfunctional emotion regulation
ExFun = External Functional emotion regulation
InFun = Internal Functional emotion regulation
Positive AI = Positive Affect Intensity
Negative AI = Negative Affect Intensity
Global AI = Global Affect Intensity

Table 3: Skewness, Kurtosis and Z-values for Online Creativity and Creative Accomplishments in Six Domains

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Table 4: Skewness, Kurtosis and Z-values for Anxiety and Depression variables

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Table 5: Skewness, Kurtosis and Z-values for Emotion Regulation and Affect Intensity variables

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Appendix 15: Data screening statistics for Study 3

Key:
BM = Before Mood Induction
AM = After Mood Induction
InDys = Internal Dysfunctional emotion regulation
ExDys = External Dysfunctional emotion regulation
ExFun = External Functional emotion regulation
InFun = Internal Functional emotion regulation
Positive AI = Positive Affect Intensity
Negative AI = Negative Affect Intensity
Global AI = Global Affect Intensity

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<th>Table 6: Skewness, Kurtosis and Z-values for Emotion Regulation, Life Satisfaction, Anxiety and Depression variables (before mood induction)</th>
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Table 7: Skewness, Kurtosis and Z-values for Emotion Regulation, Life Satisfaction, Anxiety and Depression variables (after mood induction)

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<th>Ex Fun AM</th>
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Figure 3: Error bars for emotion regulation sub-scales (Internal Dysfunctional, Internal Functional, External Dysfunctional and External Functional emotion regulation) in controls and participants with subclinical eating disorders.
Figure 4: Error bars for life satisfaction, subclinical anxiety and subclinical depression in controls and participants with subclinical eating disorders.
Appendix 16: Data screening statistics for Study 4

Key:
BM = Before Mood Induction
AM = After Mood Induction
InDys = Internal Dysfunctional emotion regulation
ExDys = External Dysfunctional emotion regulation
ExFun = External Functional emotion regulation
InFun = Internal Functional emotion regulation
Positive AI = Positive Affect Intensity
Negative AI = Negative Affect Intensity
Global AI = Global Affect Intensity

Table 8: Skewness, Kurtosis and Z-values for Emotion Regulation, Affect Intensity and Life Satisfaction variables (before mood induction)

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Table 9: Skewness, Kurtosis and Z-values for Emotion Regulation and Life Satisfaction variables (after mood induction)

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Figure 5: Error bars for Emotion Regulation sub-scales (Internal Dysfunctional, Internal Functional, External Dysfunctional and External Functional emotion regulation) in controls and participants with eating disorders.
Figure 6: Error bars for affect intensity (Global, Positive and Negative) and life satisfaction in controls and participants with eating disorders.
Figure 7: Error bars for eight baseline emotions in controls and participants with eating disorders
Appendix 17: Data screening statistics for Study 5

Key:
BM = Before Mood Induction
AM = After Mood Induction
InDys = Internal Dysfunctional emotion regulation
ExDys = External Dysfunctional emotion regulation
ExFun = External Functional emotion regulation
InFun = Internal Functional emotion regulation
Positive AI = Positive Affect Intensity
Negative AI = Negative Affect Intensity
Global AI = Global Affect Intensity

Table 10: Skewness, Kurtosis and Z-values for Emotion Regulation, Affect Intensity and Life Satisfaction variables (Week 1)

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Table 11: Skewness, Kurtosis and Z-values for Emotion Regulation variables (Week 2)

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Table 12: Skewness, Kurtosis and Z-values for Emotion Regulation and Life Satisfaction variables (Week 4)

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Figure 8: Error bars for Emotion Regulation sub-scales (Internal Dysfunctional, Internal Functional, External Dysfunctional and External Functional emotion regulation) participants with eating disorders.
Figure 9: Error bars for affect intensity (Global, Positive and Negative) and life satisfaction in participants with eating disorders.