Stress Induced Eating and HPA axis Activity in Chronic Major Depression

by

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Abstract

Individuals with depression exhibit altered psychological and physiological reactions to stress. Depressive symptoms include alterations in appetite including hyperphagia, which has been linked with obesity and poor outcome. Stress induced eating is well documented, however not within a depressed population. Social stress was induced in 14 chronically depressed participants and 11 healthy volunteers using the Trier Social Stress Task. Serial salivary cortisol was collected and a food craving challenge was administered post-stressor. No group differences in stress induced eating, or cortisol response were found. However, results indicated differences in stress perception and coping styles, between depressed and non-depressed participants. Significant associations between negative affect, stress perception, coping styles (e.g. rumination) and stress-induced eating behaviours were also found. Limited sample size, high proportion of restrained eaters may confound results; therefore future research should include larger sample size, and matched controls in order to clarify post-stress eating behaviours in depression.

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Introduction

Depressive disorders are common and present a significant public health concern with an estimated lifetime prevalence of 13.3% (Hasin et al., 2005). The World Health Organization suggests if current trends continue, by 2020 depressive disorders will be the second most common illness (Murray and Lopez, 1997). Depressive disorders often follow a chronic course due to lack of response to treatment and persistence of residual symptoms (Lam and Kennedy, 2004). Stress has been implicated in the perpetuation and maintenance of depressive symptoms. Stressful life events can precipitate the onset of a depressive illness (Brown et al., 1994) and daily hassles can perpetuate the illness (van Eck et al., 1998), while the presence of positive life events can contribute to symptom improvement (Brown, et al., 1993). The causes of depressive disorders have not been fully elucidated, however various neuroanatomical, endocrine, and neurochemical abnormalities are hypothesized to underlie the pathophysiology (Nemeroff and Vale, 2005). Symptoms of depressive disorders are numerous and varied. Among them, neurovegetative symptoms, such as fatigue, disturbances in sleep, appetite, weight and libido are the most common.

Healthy eating behaviour, though a basic necessity, is complex and can be influenced by multiple interacting factors including individual, social, and environmental ones (Polivy and Herman, 2005). Aside from the psychological factors, eating behaviour itself is important to study as it directly impacts our physical well being depending on what and how much we eat. Within the context of depressive disorders, maladaptive eating patterns have treatment implications as they could lead to comorbid eating disorders or issues such as obesity, which are associated with reduced improvement (Kloiber et al., 2007).

The following summarizes the published scientific literature on depressive illness, cognitive and physiological components of the stress response, as well as stress and emotion-induced eating behaviours. The literature on stress and depression, along with potential mediating mechanisms are

reviewed; the stress response and hypothalamic-pituitary-adrenal (HPA) axis activity in relation to eating behaviours are highlighted.

Depressive Illness

As previously noted, depression is a common mental illness frequently associated with comorbid psychiatric and medical conditions, with a high risk of morbidity (Parikh et al., 2001; Levitan et al., 1997). One-year prevalence rates are 4.2% to 5.3% (Parikh et al., 2001; Hasin et al., 2005), and occur in females approximately twice the rate than in men. Two-thirds of depressed patients have one concurrent general medical condition, while two-thirds have a comorbid psychiatric disorder (Rush, 2007). The mean age at onset is 30 years (Hasin et al., 2005) and early age of onset is associated with a more severe and recurrent forms of depression, substantial functional impairment, and greater illness burden (Zisook et al., 2007). Common presentation of symptoms includes depressed mood, loss of interest, appetite disturbances, insomnia or hypersomnia, psychomotor agitation or retardation, loss of energy/fatigue, feelings of worthlessness, concentration difficulties, and recurrent thoughts of death and/or suicide. Clusters of symptoms are categorized to form subtypes of depression. One common subtype is the atypical depression, which presents with hypersomnia, hyperphagia, weight gain, and rejection sensitivity while another is the melancholic subtype presenting with insomnia, lack of mood reactivity, appetite and weight loss (APA, 1994). Main forms of treatments for depressive disorders are pharmacological (e.g. selective serotonin reuptake inhibitors (SSRIs), serotonin norepinephrine reuptake inhibitors (SNRIs)), and psychological interventions including Cognitive Behavioural Therapy and Interpersonal Psychotherapy. While approximately 60% of individuals with depression receive treatment (Hasin et al., 2005), lack of response to treatment and significant residual symptoms contribute to a chronic course of depression. Individuals with treatment resistant depression have a very low likelihood of a sustained treatment response and as a result, have poor quality of life despite several treatment strategies (Dunner et al., 2006). Residual depressive symptoms are difficult to treat, and time to

remission is greater for those who require more trials of treatment (Rush et al., 2006), presenting a difficult problem for most patients experiencing a chronic and recurrent course.

Chronic depression is defined as the occurrence of a major depressive episode that persists for 2 years or more without symptom relief (APA, 1994). It is estimated that approximately 30-50% of those who experience an acute episode of depression will experience residual symptoms (Fawcett, 1994), which has been suggested to be a risk factor for poor outcome and impaired social functioning (Kennedy and Paykel, 2004). Often, the term chronic depression is used synonymously with treatment resistant depression and strategies to treat chronic depression are often distinct from those used for acute depression. Such strategies involve augmentation with mood stabilizing agents, atypical antipsychotics, and more recently psychostimulants (Ravindran et al., 2008). Due to its differing symptom profile and lack of response to usual antidepressant treatments, chronic depression may have distinct neurobiological mechanisms from that of acute depression (Stahl, 2001).

Depressive Illness and Eating Behaviour

The relationship between depression and eating behaviour is clinically significant. Altered appetite is a common symptom of depression, medications used to treat depression often cause weight gain, and depression commonly occurs within eating disorders (Parikh et al., 2001). Individuals with depression often exhibit hypophagia or hyperphagia; the latter is often seen in chronic forms of depression with many patients presenting with weight gain and obesity (McElroy et al., 2004). The literature on eating behaviours within depressed populations mostly focuses on comorbidities such as binge eating disorder or bulimia nervosa, as well as specific food cravings, namely carbohydrates. It is suggested that carbohydrates can relieve depressive symptoms and improve mood via increased serotonergic activity, particularly those with atypical depression (Wurtman &Wurtman, 1989; Moller, 1992). However, the relationship between negative mood and food intake is still unclear,

with more or less of specific food types having a positive or negative impact on mood (Gibson, 2006). Some have noted that individuals who are depressed tend to skip more meals which may contribute to feelings of low energy, and missed calories may be replaced by calories from snacks low in nutrition value (Fulkerson et al., 2004). Furthermore, restrained eating, defined as attempting to restrict food intake in order to maintain or lose weight, has been related to poor psychological health (Appleton & McGowan, 2006). A small number of studies have identified an association between weight gain, depression and restrained eating however; they involved small sample size (Herman and Polivy, 1976) or used self-report measure to determine the presence of depressive symptoms (Zielinski, 1978). While numerous investigations have identified contributing factors to the relationship between depression and eating, other potential contributing factors, such as stress, have not been fully explored.

Food Craving and Mood

Food cravings within depression have been an issue of particular relevance to the atypical subtype of depression. Altered monoamenergic (including 5HT and dopamine) neurotransmission, thought to underlie the pathology of depression, may also play a role in hyperphagia and carbohydrate cravings (Wurtman and Wurtman, 1989). More specifically, some have suggested that carbohydrate consumption increases the release of serotonin via insulin secretion and the plasma tryptophan ratio. As a result of feedback mechanisms, depressed individuals consume carbohydrates in an effort to make themselves feel better and to improve their mood (Wurtman and Wurtman, 1995). In addition to serotonergic system, some have suggested that carbohydrate cravings found in depressed individuals may also be linked to the opioid system (Parker and Crawford, 2007). Furthermore, it has been suggested that foods commonly subject to cravings (e.g. chocolate) have an association with the mesolimbic dopamine reward system similar to that of drugs of abuse (Parker et al., 2006).

Food cravings have been associated with psychological dysfunction, particularly depressive disorders. Investigations have found that individuals who identified themselves as having consistent food cravings and classified as binge eaters were more likely to have had an episode of major depression or a history of social phobia (Gendall et al., 1998). Furthermore, Macdiarmid and Hetherington (1995) reported that "chocolate addicts" were more depressed than individuals without a chocolate addiction. As well, in an online study assessing self-report benefits of chocolate during a depressive episode, Parker and Crawford (2007) found that chocolate craving was common among depressed respondents. Results indicate that 54.4% of respondents reported experiencing food cravings when depressed, with 44.9% specifically indicating chocolate craving. In addition, irritability and rejection sensitivity, two common symptoms of atypical depression, were found to be significant predictors of chocolate cravings. Yet despite these associations, giving in to food cravings, particularly cravings for chocolate, does not appear to improve mood. Macdiarmid and Hetherington (1995) found that depressed individuals' negative mood did not improve after chocolate intake, and furthermore, in non-depressed populations, any mood improvement occurred during food consumption only (Hetherington and Macdiarmid, 1993). In addition, others have shown that dysphoric mood persists and can even worsen post binge eating (e.g. Larson, 1982).

While the relationship between depression, food cravings, and carbohydrate consumption has been investigated, further research is warranted; particularly with those individuals who overeat, as obesity has long been associated with depression and its subtypes.

Stress And Depressive Illness

The association between stress and depressive illness is well documented. Stressful life events have been shown to precipitate the onset of a depressive episode (Paykel et al., 1978), as well, daily occurrences can also contribute to the prolongation of depressed mood. Not only can stressful experiences impact the course of illness (Hammen, 2005) but type of stressful event (e.g. romantic

loss), has been associated with certain depressive symptom presentations (Keller et al., 2007). Furthermore, alterations in the physiological response to stress have been noted in those with a depressive illness. The literature on stress perception, coping styles, and the physiological response to stress in depression will be reviewed in the following section.

Depressive Illness, Perception Of Stress, And Coping Styles

Life events, particularly negative or stressful ones, can precede the onset of a depressive episode with 50-80% of depressed individuals experiencing a major life event in 3-6 months prior to the onset of the episode (Paykel et al., 1978). Daily negative events, or otherwise referred to as hassles, both contribute to negative affect and are subject to distorted perceptions in depressive illnesses (van Eck et al., 1998). Fava et al. (1992) purports that depression can alter cognitive and affective responses to stressors, which in turn exaggerate the degree of strain and pressure derived from stressful events. Depressed individuals not only report fewer positive daily events but also appraise both positive and negative events as stressful (Peeters et al., 2003). In comparison to non-depressed individuals, those with depression report greater number of perceived stressors in the form of daily hassles and fewer uplifting events (Ravindran et al., 2002). In addition, those who were resistant to treatment and those with greater severity of illness had a higher number of perceived day-to-day stressors. Furthermore, in studies of recurrent depression, higher levels of dysfunctional attitudes and avoidant coping strategies, as well as higher levels of daily hassles predicted earlier recurrence (Bockting et al., 2006). Investigations regarding subtypes of depression revealed that higher levels of perceived stress were associated with atypical features and with anger attacks; with no significance between the two subgroups (Farabaugh et al., 2004).

Rumination as a Coping Style in Depression

Affective disturbances are associated with increased levels of rumination, cognitive distraction and emotion-focused coping (emotional expression, other-blame, self-blame, emotional containment and passive resignation) coupled with diminished problem solving and social support seeking (Matheson

and Anisman, 2003). In comparison with non-depressed individuals, depressed patients have been shown to use emotion-based coping strategies, involving emotional expression or containment, as opposed to cognitive methods of coping (Ravindran et al., 2002). One such coping style that has been extensively studied in its relationship with depression is rumination.

Rumination has long been a response style associated with not only the onset of depression but also with the persistence of depressive symptoms (e.g. Nolen-Hoeksema 1991). Ruminative thinking can be defined as uncontrollable perseverative thinking about past or present events, in particular, dwelling on negative affect (Nolen-Hoeksema, 1991). Actively inducing ruminative self-focused attention prolongs and stabilizes dysphoric mood, while actively inducing distraction attenuates and shortens it (Nolen-Hoeksema, 2004). Trait rumination has shown to be related to attentional biases for negative information in depressed participants (Donaldson et al., 2007). Furthermore, these biases have been linked with overgeneralization of negative memories, which are also thought to contribute to the persistence of depressive mood (Hermans et al., 2008; Raes et al., 2006). Within depressed populations, individuals that score high on rumination measures have significantly higher depressive symptoms and levels of hopelessness compared to depressed individuals who use distraction coping techniques (Lam et al., 2003). In addition, depressed individuals who are designated as "high ruminators", also have significantly greater scores on questionnaires measuring interpersonal problems suggesting that those who ruminate, in comparison to other coping methods (e.g. distraction), have difficulties with their personal relationships and lower social functioning (Lam et al., 2003).

Some have made a distinction between the ruminative processes of brooding (maladaptive) and reflective pondering (Joorman et al., 2006). Depressed individuals have been shown to report higher levels of brooding than non-depressed participants. Furthermore, formerly depressed individuals also report higher brooding scores than control populations suggesting that this

component of brooding is a trait characteristic that increases vulnerability to depression (Joorman et al., 2006).

Specifically within chronic depression, rumination has been found to be particularly high in patients with dysthymic disorder (Kelly et al., 2007). Studies have shown that despite improvement of depressed mood and reduced rumination, the relation between emotion-focused coping styles and rumination persisted. The authors suggest that rumination is a potential risk factor for recurrence and may serve as a sensitive measure of depressive affect in chronic depression (Kelly et al., 2007).

The psychological components, such stress perception and coping styles, in the relationship between stress and depression have been well documented. In a parallel body of literature, the physiological response to stress in those with depression has also been extensively investigated and is reviewed in the following section.

The Physiology of the Stress Response and HPA axis

The stress response refers to the neurobiological reaction, specifically hypothalamic-pituitary-adrenal (HPA) axis activity, to a stressor. In response to psychological stimuli or stress the periventricular hypothalamus secretes corticotropin-releasing hormone (CRH) into the hypothalamo-pituitary portal circulation, which triggers the release of adrenocorticotropic hormone (ACTH) into general circulation. ACTH stimulates the release of cortisol from the adrenal cortex, which can act directly on hypothalamic neurons as well as other neurons elsewhere in the brain (Bear et al., 2007). CRH initiates events that release glucocorticoids from the adrenal cortex. As a result of the great number of physiological and behavioral effects exerted by glucocorticoids, several mechanisms have evolved to control HPA axis activation and integrate the stress response (Smith and Vale, 2006). Physiological and behavioural components of the stress response are multiple and complex and as such may be mediated by several mechanisms (Leonard, 2005; Tillbrook and Clarke, 2006).

Gender differences in HPA axis response to stress are evident within the literature. It has been reported that men respond to psychological stress with greater increases in cortisol compared to women, which is hypothesized to be associated with risk factors for different diseases. Differences in the structure of limbic regions of the brain, cognitive processing and circulating sex hormones may be involved with the difference in stress response between genders (see Kudielka and Kirschbaum, 2005 for review).

The stress response has been studied through a variety of stress induction techniques in both animals and human subjects. Methods include physical stressors, cognitive stressors, failure induction, and psychosocial stressors. Of these methods, stressors that are uncontrollable threats to the social self elicit reliable cortisol responses. The largest increases in cortisol levels were found in performance tasks that contain both a social evaluative threat and uncontrollability (Dickerson and Kemeny, 2004). One such task that contains both these factors and is often used in investigations of the stress response is called the Trier Social Stress Test (TSST). This task involves an anticipation period (10 min.) followed by a test period (10 min.) in which participants have to deliver a free speech and perform mental arithmetic in front of an audience (Kirschbaum et al., 1993). It has been shown to be an effective stressor, and a consistent method to measure the stress response. While the TSST has been modified for ease of application through the use of imaginary audience (one-way mirror) and with virtual reality technology, performing in front of a real audience has shown to produce the greatest increase in cortisol levels (Kelly et al., 2007a). Furthermore, increase in cortisol levels have been shown to be the greatest post exposure rather than during the anticipatory phase of the stress induction (Kelly et al., 2007a). Because of this, the TSST sufficiently induces stress and is a promising tool to study the stress response in an illness such as depression.

Depressive Illness and HPA Activity

Impaired regulation of the HPA system has been consistently noted in depressed individuals as evidenced by the extensive body of literature. Cortisol hypersecretion is regarded as important in the pathophysiology of major depression; however, recent studies have been inconclusive (e.g. Huber et al., 2006). Alterations in HPA axis activity is evident in many subgroups of patients with depressive illness, however, the direction of change tends to vary and may be impacted by length of depressive illness, subtype of depression, and other potential contributing factors.

Acute vs. Chronic Depressive Illness

Acute depressive illness is marked by intermittent episodes of depression and symptom remission and has been consistently associated with hypercortisolemia (e.g. Carroll et al., 2007). Within acute depression, it is proposed that HPA axis overactivity, as indicated by high levels of cortisol, are caused by an increased secretion of CRH which is not adequately suppressed by the internal negative feedback system (see Claes, 2004 for review). Explorations of HPA axis activity in acute depressive illness have found elevated total and free cortisol concentrations in plasma, urine, and cerebrospinal fluid (Carroll et al., 1976; Kling et al., 1996). Furthermore, non-suppression of cortisol has been found in response to dexamethasone suppression test (DST) and increased cortisol and ACTH concentrations after suppression by the combined dexamethasone/corticotropin test (dex/CRH) have also been observed (Holsboer, 1999; Holsboer, 2001). In addition, studies examining 24-h cortisol profiles of depressed individuals found significantly higher mean 24-h plasma cortisol levels (Halbreich et al., 1985), the cortisol nadir of depressed patients was earlier and significantly greater than non-depressed (Halbreich et al., 1985; Jarrett et al., 1983). Awakening cortisol response investigations have yielded inconsistent responses with some finding elevated levels and others a blunted effect (Huber et al., 2006). Though such inconsistencies have been found, hypercortisolemia is a fairly robust finding in acute depression and is thought to be important to the development of future pharmacological treatment for depression (Holsboer et al., 1996).

Individuals with chronic depression have also shown alterations in the stress response; however, these impairments may be different from those found in acute depression. Some investigations have found no difference in response to hormone challenges between chronically depressed individuals and non-depressed individuals (Shah et al., 1998) and reduced cortisol levels in elderly long-term depressed populations (Oldehinkel et al., 2001). Likewise, chronically depressed patients did not differ from control participants in cortisol response to dex/CRH challenge (Oshima et al., 2000) or the DST (Watson et. al., 2002). Furthermore, O'Keane and colleagues (2005) found increased response in ACTH to CRH challenge, which is in contrast to findings within acute melancholic depression. Using animal models, Parker and colleagues (2003) suggest that acute depressions are characterized by hypersecretion of CRH, ACTH, and adrenal cortisol whereas in chronic depression, enhanced adrenal responsiveness to ACTH and glucocorticoid negative feedback corresponds so that cortisol levels remain elevated while ACTH is reduced. While differences between acute and chronic depression have been noted, investigations have also found similar evidence of an overactive HPA axis and increase in CRH and cortisol concentrations between those with major depression and those with dysthymic disorder (Catalan et al., 1998). Hypercortisol secretion is a fairly robust finding in major depression, however it appears patients with chronic depression may show a different pattern and overactivity of HPA axis may not be applicable to more chronic forms of depression.

Depressive Illness subtypes

A growing body of literature suggests that subtypes of depression may also provide some explanation for the above inconsistencies. Studies investigating ACTH and cortisol dynamics in hypercortisolemic and non-hypercortisolemic depressed in-patients compared with normal volunteers found that hypercortisolemia was strongly associated with melancholic and psychotic depressive subtypes. The authors concluded that increased ACTH secretion occurs in depressed in-patients regardless of cortisolemic status, confirming central HPA axis overdrive in severe

depression (Carroll et al., 2007). In addition, findings in psychotic and melancholic depressed inpatients suggest that chronic elevation of cortisol may lead to dopaminergic, noradrenergic, and thyroid dysfuntion (Duval et al., 2006). In a review of the literature, Gold and Chrousos (2002) concluded that melancholic depression is associated with overactivity of the HPA axis and hypersecretion of releasing CRH, while there is evidence of a down-regulated HPA axis and deficiency of CRH with atypical depression. The authors suggest that this distinction accounts for the divergent symptomatology of the two subtypes. Similar evidence of hypersuppression of the HPA axis has been shown for females with atypical depression (Levitan et al., 2002) and for those with early onset, chronic depression with atypical features (Stewart et al., 2005). Though the literature is not conclusive, it appears that a distinction in HPA axis activity can be made between the subtypes of depression.

Depressive Symptom Remission and HPA axis

It has been proposed that hypercortisolemia in depression should be considered a state marker and not a trait marker as it has been shown to normalize in patients with symptom improvement (Claes, 2004). Evidence suggests that compared to individuals with residual symptoms, those who remit have a more pronounced normalization of an initially dysregulated HPA axis (Hennings et al., 2008). However, a recent study found lower serum cortisol levels and a blunted cortisol and ACTH response to a social stressor within remitted females who previously experienced recurrent depression (Ahrens et al., 2008). Researchers have suggested that interventions aimed at normalizing HPA system could prevent the development of depression. Some have suggested a potential link between alterations of the limbic-HPA system with the serotonergic hypothesis of depression and propose that chronic psychosocial stress may lead to depression depending on the psychobiological background and psychological resources; and as such, treatments targeting these components may be beneficial (Tafet and Smolovich, 2004). Furthermore, others have proposed that specific tests that assess the HPA system regulation could be used to predict clinical outcome.

Investigations found that improved HPA system regulation as measured by dex/CRH test, was associated with beneficial treatment response after 5 weeks and a higher remission rate at the end of hospitalization. Authors suggest that change in HPA system regulation assessed with repeated dex/CRH tests is a potential biomarker that may predict clinical outcome at follow-up (Ising et al., 2007).

Other Possible Contributing Factors

Other mediating factors that have been studied in relation to the stress response and system in depression include disorders of circadian time-keeping (Linkowski, 2003), comorbid disorders, early childhood adversity (Claes, 2004), and genetic polymorphisms that may increase susceptibility to depression when faced with stressful life events (Gotlib, 2007). In a study of the effects of early or recent adverse events on the stress response, depressed adolescents showed higher levels and prolonged secretion of cortisol in response to a social stress induction (Rao et al., 2008). Analyses revealed that early-life adversity and high levels of chronic stress were the best predictors of an enhanced stress response. In addition, comorbities such as anxiety may also contribute to the stress response as depressed patients with anxiety have shown an exaggerated ACTH response to a stressor compared with purely depressed patients and controls (Young et al., 2004).

As previously discussed, rumination has long been association with depression and is considered to be a vulnerability factor for depression onset and recurrence. There have been several investigations aimed at deciphering the role of rumination and the stress response, as both are associated with depressive illness. In a studies comparing individuals with high and low depression scores, participants with high scores on a self-report depression measure who were induced to ruminate showed a lower decrease in cortisol (area under the curve) over the course of the experiment than those with lower depression scores (Kuehner et al., 2007a). In non-depressed populations, McCullough et al. (2007) investigated the relationship between rumination about a recent painful interpersonal event and the stress response and found elevated cortisol levels. In

addition, co-rumination was shown to amplify stress response in non-depressed females (Byrd-Craven et al., 2008). Observed co-rumination was associated with increased cortisol immediately following a discussion of a problem in contrast to those in the control condition (without co-rumination). The authors suggest that dwelling on negative affect in particular contributed to the elevated coritsol levels (Byrd-Craven et al., 2008). In contrast, non-depressed individuals categorized into high and low ruminators, were all subjected to the TSST with no significant differences in salivary cortisol found between high and low ruminators (Young and Nolen-Hoeksema, 2001). However, the authors also included a group of individuals who were in the middle of midterm examination period and found an absence in response to the TSST and a decrease in cortisol over the course of the study (Young and Nolen-Hoeksema, 2001). These results are consistent with results found within chronic illnesses such as long-term depression and PTSD. Furthermore, laboratory investigations using negative mood induction techniques to study cognitive vulnerabilities to depression found a significant association between cortisol awakening response and self-focused rumination. Specifically, low levels of awakening cortisol have been associated with high levels of self-focused rumination (Kuehner et al., 2007).

Results from naturalistic data suggest that the stress response to negative daily events is different in depressed individuals. Correlations between negative daily events and salivary cortisol, indicated that depressed participants showed no increase in cortisol after negative events and the association between negative affect and cortisol levels were weaker in depressed participants than in healthy participants (Peeters et al., 2003).

Reactivity to Social Stress in Depressive Illness

The majority of the investigations of HPA axis alterations in depression have used biological measurements or pharmacological challenges to determine the nature of impairment. Only a small number of studies have used a social stressor such as the TSST, to examine alterations in the stress

response in depressive illness (e.g. Young et al., 2004; Rao et al., 2008). The TSST is designed to induce moderate social stress (Kirschbaum et al., 1993) and may be more salient to depressed individuals who are likely to have a heightened interpersonal sensitivity as part of their symptom presentation.

Stress and Depressive Illness Summary

The relationship between stress and depressive illness is well documented. Increased life events, stress perceptions, and the use of emotion-focused styles such as rumination are often noted in depression. Depression is also associated with dysregulation of the HPA axis activity. However the direction of change may be influenced by chronicity and symptom subtypes. Normalization of HPA activity often occurs with symptom remission, while it's persistence may predict vulnerability to relapse of depressive illness.

Negative Affect, and, Stress Induced Eating Behaviours

Eating fulfils a biological need; however, social and cultural meanings can be deduced from food choices (Hetherington, 2002). Normal eating behaviours are complex and influenced by multiple factors such as mood, emotion, social and collective influences (Polivy & Herman, 2005). Two influences on eating behaviour that have been the focus of a large body of literature are negative affect and stress; which can both lead to overeating. Within this body of literature, the two types of eating responses that emerge are emotional eating and restrained eating. Both emotional eating (overeating in response to negative emotional arousal) (Wallis and Hetherington, 2004), and restrained eating (cognitively controlled effort to restrict food intake to control body weight) have been linked with overeating (e.g. Herman and Polivy 1975). Though these two types may overlap, i.e. an individual can be categorized as both an emotional and restrained eater (Lindeman and Stark, 2001), motivations behind the two categories appear to be distinct. In a proposed model of emotion-induced changes of eating, Macht (2008) suggests that emotional eaters eat to reduce negative

emotions while restrained eaters increase food intake because emotions impair cognitive control over their restricted eating pattern. The literature on eating in response to negative affect, stress induced eating and the associations with emotional and restrained eating will be reviewed in the following section.

Negative Affect Induced Eating– Emotional Eating

It is commonly agreed that food preference and intake, specifically overeating, is linked with mood. Mood induced eating is often used interchangeably with emotional eating, however, they are not synonymous. As previously mentioned, emotional eating is defined as overeating in response to negative emotional arousal (Wallis and Hetherington, 2004), yet it has also been identified as a coping method in response to both negative mood- and stress-induced eating. Other methods such as emotion-oriented and avoidance distraction coping have been associated with emotional eating (Spoor et al., 2007). Several factors such as food choice, sensory and neurobiological components that influence emotional eating will be discussed.

Factors Associated with Emotional Eating

Food Type

The selections of a particular food type and macronutrient intake in response to stress or negative mood have been the focus of many scientific investigations. It has been suggested that sweet, fatty foods that are low in protein provide alleviation from stress in vulnerable people via enhanced function of the serotonergic system (Gibson, 2006). Others have found that long term intake of particular macronutrients (protein and carbohydrates) were related to mood. More specifically, high proportionate protein intake over long term was found to be associated with high self-reported depression scores, whereas high proportionate carbohydrate intake was associated with low depression over long term (de Castro, 1987). In contrast to previous literature, participants

reporting more symptoms of depression ate less total carbohydrates as percentage of total energy intake and smaller quantities of sugar than less depressed participants. In addition, anxious subjects consumed greater quantities of nutrients found in protein sources; suggesting that anxiety is associated with a low carbohydrate, high fat, high protein diet (Pellegrin et al., 1998). While carbohydrate craving and increased intake have been associated with negative affect and depressive symptoms, inconsistencies have been found.

Sensory Factors

Researchers hypothesize that some individuals may be especially sensitive to the mood altering effects associated with eating sweet foods. Such individuals are more likely to consume sweet tasting foods especially under stress to ameliorate their depressed mood, which increases risk of overeating and obesity. Specifically, it has been suggested that individuals' cravings for sweet food, sensitivity to mood altering effects of sweet foods, and propensity to have an impaired control over eating sweets are directly associated with elevated hedonic reaction to more intense sweet taste (sweet liking) (Kampov-Polevoy et al., 2006). One such example that is subject to food cravings and has been associated with emotional eating is chocolate consumption. Studies investigating the effects of chocolate on mood found that eating palatable chocolate improved negative mood, and increased joy to a higher extent than eating unpalatable chocolate or eating nothing, though the effects were short-lived (Macht and Mueller, 2007). Furthermore, of those that craved chocolate, approximately 60% indicated that chocolate had the capacity to improve mood and indicated that they felt less irritated and anxious (Parker and Crawford, 2006). In addition, Wilner and colleagues (1998) found that both animals and human subjects' performance under the progressive ratio schedule for sweet rewards is increased by depressive mood induction. Specifically in human participants, depressive mood induction increased chocolate cravings and significant correlations between chocolate craving and chocolate reinforced progressive ratio performance were found. The

palatability of chocolate has allowed researchers to explore the association of sensory factors that may be related to food craving and emotional eating in response to negative affect.

Neurobiology of Emotional Eating

In a recent review article, Gibson (2006) suggests that eating a particular food, or combination, can alter mood via sensory (including hedonic) effects, associated social context, cognitive expectations, psychological distraction, changes in appetite, or nutritional modulation of brain function. Humans tend to be aroused, alert and even irritable when hungry. However, after eating a satiating meal, individuals typically become calm, lethargic and mood is more likely to be positive than negative. In addition, animal studies show that fatty foods low in protein act as part of a feedback loop via release of glucocorticoid hormones and insulin, to limit HPA axis activity during stress (Gibson, 2006). Studies investigating mood and cortisol reactivity in response to stress found that high reactors consumed more sweet foods than low reactors, across stress and rest days. Furthermore, self-reported increases in negative mood during a stressor were also significantly positively related to caloric consumption (Epel et al., 2001).

Sensitivity To Reward

Another area of investigation of the neurobiology associated with emotional eating pertains to neurotransmitter system implicated in reward and punishment systems. Sensitivity to Reward (STR) is conceptualized as a psychobiological personality trait rooted firmly in the availability of dopamine in the mesocorticolimbic ('common reward') pathways. This construct describes the ability to derive pleasure or reward from natural reinforcers like food, and from pharmacologic rewards like addictive drugs, and as such is a good candidate for studying motivational factors and

eating behaviours (Salamone et al., 2003). A full review is beyond the scope of this paper, though it will be briefly discussed, as it may be a contributing factor to emotional eating.

The most evidenced neural substrates of reward are the dopamine, opioid and benzodiazepine/GABA neurotransmitter systems: there may be a dissociation of these systems such that dopamine underlies motivational aspects of eating (wanting), whereas opioid and benzodiazepine systems may mediate hedonic evaluation of food stimuli (liking) (Gibson, 2006). In addition, specific foods such as chocolate are thought to act on serotonergic, dopamenergic and endogenous opioid and have been suggested to have the same type of effect on the reward system as drugs of abuse. However, recent reviews indicate that the mood altering effects of chocolate are fleeting and are more likely to prolong dysphoric mood rather than alleviate it (Parker et al., 2006).

STR has been positively correlated with measures of emotional overeating (Gibson, 2006) and has been studied in binge eating as well as obese populations. STR has been found to positively predict overeating and been associated with a preference for foods high in fat and sugar; and that these two behaviours predict a higher Body Mass Index (Davis et al., 2007). Studies suggest that overweight women are significantly more sensitive to reward than those of normal weight, while obese woman were more anhedonic than the overweight woman (Davis et al., 2004).

Overeating in response to negative affect, or emotional eating, may be viewed as a coping strategy. Sweet, fatty foods, low in protein have been shown to be the food of choice for emotional eaters and that these choices may be influenced by heightened sensitivity to the perceived mood altering effects of these foods, though inconsistencies have also been found. Neurobiological data suggest that HPA axis may also be involved as emotional eaters exhibit higher levels of cortisol in reaction to stress. Furthermore, STR and its suggested mechanisms may provide additional information on the role in emotional eating in those who overeat. Majority of this research has been conducted in normal populations but has important implications for depressed populations and warrants further investigation.

Stress Induced Eating

Similar to the altered eating behaviours found in depression, stress induced eating may also be bidirectional, i.e., some reduce food intake while others overeat in reaction to stress. Theories suggest overeating in response to stress may occur through a process of affect self-regulation or as a distraction technique to avoid aversive environmental stimuli. As such, attention is shifted away from negative affect or negative self-appraisal towards immediate stimulus in the environment thus disinhibiting intake in restrained eaters and promoting overeating in those susceptible to emotional eating (Wallis and Hetherington, 2004).

Laboratory studies with animals have mostly been directed toward finding physiological pathways to explain stress-induced eating. Other research focusing on human subjects investigates individual differences in learning history, attitudes, or biology and its effects of stress on eating. Individual differences such as gender, coping methods, and level of restraint can impact both the amount and type of food eaten after a stressful experience (Greeno and Wing, 1994).

Impact of Stress on Food Choice and Intake

Similar to the literature on overeating in response to negative affect, food composition (high fat/ high energy) and palatability may influence why certain foods are chosen during times of stress (Kandiah et al., 2006). Specifically, stress not only increases consumption of food but also moves consumption toward high caloric snack foods that are normally avoided (particularly sweet high fat foods like chocolate) and away from healthier foods such as fruits (Zellner et al., 2006). Daily hassles are associated with an increase in individuals' desire to eat and concomitant negative affect adds substantially to the prediction of this effect. Similar to associations found within the stress and depression literature, ruminative thinking associated with daily hassles may also contribute to stress induced eating (Kubiak et al., 2008).

In addition, it is not only the amount and nutritional content of food that is impacted by stress; it may also be the context of food intake. Evidence suggests that snack consumption may be more susceptible to stress than meals (Oliver et al., 2000) and as such, studies have focused on snack intake rather than meals (Newman et al., 2007).

Gender, Stress and Eating Behaviours

Gender is an important factor throughout the literature on stress and eating behaviours (Klein et al., 2004). The gender differences are such that some studies only focus on females (e.g. Zellner et al., 2006, Epel et al., 2001). Differences between genders have been found in not only in the amount and type of food consumed in response to stress but also in certain eating habits. More specifically, evidence suggests that women score higher on emotional eating measures than men in laboratory stress induction investigations (Oliver et al., 2000). In addition, studies have found that women ate nearly twice as much sweet food under stress than they did in the control condition (Grunberg and Straub, 1992). Furthermore, studies that focused on disinhibition of restraint in stress induced eating found that females overeat in response to a stressful experience more so than males. However, females who overeat during stressful situations may do so as a result of an eating style characterized by the inability to maintain control over self-imposed rules, and not necessarily because of dietary restraint (Weinstein et al., 1997)

Gender differences have also been found in food choice in response to stress. Zellner and colleagues (2006) conducted similar but separate studies first with females only, and then subsequently with males alone. Consistent with previous literature, they found that females consume greater overall quantities as well as greater high caloric/fat foods in response to stress. However, when investigating males only, participants in the no-stress group consumed more potato chips and more M&Ms compared to the stress group. While this is contrary to results found in females, it is also contrary to men's own self report of eating habits under stress (Zellner et al.,

2007). Due to these documented differences, it may be important to consider gender when investigating stress-induced eating behaviours.

The Stress Response and Eating Behaviours

HPA axis is involved with stress and eating regulation. Noted biological mediators in stress and eating behaviours include cortisol, glucocorticoids, insulin, leptin, neuropetide Y, and opioid signaling. In a recent review of the literature, Adam & Epel (2007), suggest a possible influence of cortisol on the reward value of food through mediators such as leptin, insulin, and neuropeptide Y. As well, the authors discuss the impact of chronic stress and on insulin, glucocorticoids, and visceral fat accumulation. These mechanisms have been explored in both laboratory and naturalistic investigations.

Laboratory Investigations

In response to a novel laboratory stressor, Epel et al., (2001) found that cortisol reactivity was related to greater caloric intake in women. Those who were categorized as high cortisol reactors ate more food then low reactors after a stressor; however, in the control condition (days without stress), low reactors tended to eat more than the high reactors. Furthermore, studies investigating stress response using the Trier Social Stress Test found that those who responded with high cortisol were likely to consume more calories after the stressor, particularly high fat food (Kirschbaum et al., 1993).

In addition, investigations focusing on other mediators involved with food intake have provided information on the relationship between the stress response and eating behaviours. For example, cortisol response has been positively correlated with blood glucose changes. Men who fasted were assigned to eat one of four food conditions: a) glucose, b) protein, c) fat, or d) just water, before exposure to a social stress task. The authors showed that both absolute and net cortisol increases were greater in the glucose group leading the authors to suggest a central mechanism

responsible for regulation of energy balance and HPA axis activation rather than peripheral mechanisms (Gonzalez-Bono et al., 2002).

Naturalistic Investigations

Investigations of stress induced eating in naturalistic settings often include measurement of daily stressors and snack food intake. Some of shown a relationship between daily stressors and increased intake of snack food; however, only for high cortisol reactors who reported greater levels of anxiety (Newman et al., 2007). In the same study, significant positive associations between hassle number and snack intake, as well as hassle intensity and snack intake were found. Furthermore, eating style variables (restraint, emotional eating) were significantly associated with snack intake for both low and high reactors, however the association was stronger with high reactors.

Restrained Eating

As previously described, restrained eating is defined as a voluntary cognitively controlled effort to restrict food intake to control body weight and has been linked with overeating (e.g. Herman and Polivy 1975). It has been suggested that restraint is one of the best predictors of stress-induced eating and that restrained eaters characteristically exhibited a hyperphagic response to stress (e.g. Zellner et al., 2006) and therefore an important component in the stress-induced eating literature. A full review of the components of restrained eating is beyond the scope of this paper, however, given its importance in stress induced eating, a brief overview of some of the characteristics of restrained eating is provided.

Many facets of restrained eating and conditions under which restrained eaters will relinquish control have been investigated and identified. For example, Heatherton and colleagues (1989) found that restrained eaters were unresponsive to internal hunger state and exhibited an overreliance on external cognitive cues. In comparison to unrestrained eaters, restrained eaters were more likely to behave in accordance with placebo messages and ate more when given "hungry" messages than

when given "full" messages with incongruent hunger ratings. In addition, restrained eaters are sensitive to foods that would be perceived as "unhealthy" as they displayed disinhibition when anticipating the consumption of what was considered to be forbidden food type despite the caloric value (high or low) (Knight and Boland, 1989). Furthermore, high caloric content can provoke greater anxiety in restrained eaters (Mills and Palandra, 2007).

Restrained eaters have been shown to experience more food cravings and are more likely to eat the craved food compared with unrestrained eaters. Individuals who were categorized as restrained eaters and who were deprived of chocolate spent the least time doing an anagram task before a "taste-rating task" in which they expected to be able to consume chocolate foods (Polivy et al., 2005). As well, individuals who restrain their eating also show a highly specific response to exposure to food cues. Restrained eaters were shown to be more responsive than unrestrained eaters to pre-eating exposure to smell and thought cues, and ate significantly more after such cues (Fedoroff et al., 2003).

In addition to external food cues, restrained eaters also respond to body weight information. McFarlane et al. (1998) found that restrained eaters overindulge and relinquish dietary restraint when led to believe they were 5 lb heavier than their actual weight. In the same study, restrained eaters also reported lower self-esteem, less positive moods, and more negative moods than did restrained eaters who were provided with accurate body weight information or who were led to believe they were 5lbs under their actual weight (McFarlane et al., 1998).

Stress and Restrained Eating

As previously mentioned, restrained eaters tend to eat more in response to stress compared to non-restrained eaters who tend to refrain from eating during periods of stress (e.g. Herman and Polivy, 1975). Studies indicate that most people who reported overeating when stressed were categorized as restrained eaters (Greeno and Wing, 1994). In addition, and consistent with previous literature, gender differences are also apparent in restrained eating, as the percentage of restrained eaters

among stress overeaters is higher for women than for men (Greeno and Wing, 1994; Zellner, 2006). Type of stress, such as a threat to ego (Heatherton et al., 1991) or cognitive load (Ward and Mann, 2000) may influence disinhibition in restrained eaters as well. In addition to laboratory investigations, naturalistic studies have also shown that the hyperphagic response to stress may be characteristic of restrained eaters. In a study examining workload stress, the authors found that not only did restrained eaters consume more food overall, they specifically ate more sweet and fatty foods under high-work-stress circumstances. Furthermore, the observed hyperphagic response was also found to be associated with stress perception. Individuals who consumed more food in response to stress also had a greater increase in perceived stress between the low-and high-workload sessions, suggesting an emotional component to the hyperphagic response (Wardle et al., 2000).

Some contradicting results have been found in restrained eaters in response to stress. A few studies have found no significant difference in total amount of consumption between restrained and non-restrained eaters under stress, though restrained eaters consumed more of a certain food type compared to those not under stress (Shapiro et al., 2005). Others found no proof of the disinhibition effect and that a tendency to overeat did not predict the amount of food eaten after stress (Ouwens et al., 2007). Some researchers have suggested that it is unlikely that restraint is responsible for stress-induced eating. According to some researchers, behavioral and physiological data indicate that restrained eating may be a proxy risk factor for vulnerability to weight gain rather than a cause of stress induced eating (Lowe and Kral, 2006).

Specifically focusing on the stress response, a growing body of literature suggests that restrained eating is associated with cortisol levels. Increased levels of urinary and salivary cortisol have been found in those who were categorized as restrained eaters (McLean et al., 2001; Anderson et al., 2002). Furthermore, a significant relationship between cognitive dietary restraint and elevated cortisol levels has been found (Putterman and Linden, 2006). Some have hypothesized that the fact

that restrained eaters struggle to restrict food intake may lead to stress and a corresponding increase in cortisol (Anderson et al., 2002), however this relationship has yet to be determined.

Emotional and Stress Induced Eating Summary

The literature of stress induced eating suggests that individuals with emotional eating patterns and/or restrained eating patterns tend to overeat in response to stress. There are significant similarities in choice and amount of food between stress induced - and negative affect induced eating. Restrained eating is proposed to be a good predictor of eating in response to stress. Increased levels of urinary and salivary cortisol are associated with restrained eating patterns.

Stress, Depression, And Eating Behaviours

Depressed individuals exhibit an altered stress response as evidenced by HPA axis activity, as well, is a population that is associated with obesity. Hypercortosolemia has been evidenced, with some inconsistencies, in individuals with major depression. Furthermore, high cortisol reactions have also been evidenced in individuals who over eat in response to stress. However, the role of stress and its mechanisms has not been fully explored in depressed populations.

Within the existing literature, perceived stress and depression was found to influence food choice within college students. Naturalistic findings indicated that an increase in perceived stress was associated with a decrease in fresh fruit intake and an increase intake of ready to eat food and snack food. Similarly, increases in depression measures were associated with a decrease in fresh fruit intake and an increase in ready to eat food and fast food intake (Liu et al., 2007).

In addition to the HPA axis, other mechanisms of interest include the role of leptin in depression, given its role in food intake and energy balance. Altered leptin serum levels have been evidenced in depression, though the results have been inconsistent thus far (Yang et al., 2007). In a study investigating those with depression with atypical features, serum leptin levels were higher in patients with atypical depression than controls but not for those with typical depressive disorder,

however, BMI did not significantly differ between the depressive groups (Gecici et al., 2005). A recent article postulates that due to the interaction between cytokines, the HPA axis, and leptin, cytokines may alter appetite and energy balance in depression through these mechanisms and within the subtypes of depression (Andreasson et al., 2007).

Other than to identify symptomatology or a comorbid disorder, eating behaviours in the context of stress induction have not been well studied within depressive illnesses. The role of the HPA axis has been examined in the stress response in both normal and depressed populations, as well as in those with differing eating behaviours. However, potential associations between HPA axis activity and stress induced eating within depression have not yet been investigated. Furthermore, coping strategies, and perceptions of stress have been explored in relation to stress within a clinically depressed population, however the association with eating behaviours has not.

Rationale

From the review of the literature, the following salient information relating to the interaction of depressive illness, stress response and eating behaviours are noted:

- Increased stress perceptions and emotion-focused coping styles appear to be common in those with depressive illnesses
- Dysregulation of the HPA axis has been noted in depressive illnesses. Hypercortisolemia is often associated with acute depression, while reduced cortisol levels has been associated with chronic depression, though inconsistencies exist.
- Sweet, fatty foods, low in protein have been shown to be the food of choice for both stress induced eating and emotional eaters. These choices may be influenced by heightened sensitivity to the perceived mood altering effects of these foods.

- High cortisol reactions have also been evidenced in individuals who overeat in response to stress and emotional eaters. STR and its suggested mechanisms may provide additional information on the role in emotional eating in those who overeat.
- Restrained eating pattern appears to be a robust predictor of overeating in response to stress, and has been correlated with cortisol reaction levels

Several studies have measured eating behaviours in response to stress through a variety of ways (e.g. total amount of food consumed, caloric/fat intake, type of food, and cravings) within a laboratory setting (e.g. Oliver et al., 2000) using a social stress induction such as the TSST. However, these studies have only been conducted in non-clinical populations. Furthermore, the same social stress induction has been used to investigate HPA axis activity in depressed populations (Burke et al., 2005), though without a focus on eating behaviours. Given that individuals with depression have an altered reaction to stress both psychologically and physiologically, and irregular eating patterns, implementing a stress induction and measuring eating behaviours as well as HPA axis activity may help elucidate the role of stress on eating behaviours within a depressed population.

Aim

The aim of this study is to explore the effect of stress induction on eating behaviour in subjects with chronic depression (compared to normal controls) and to determine possible neurobiological and behavioural correlates in this population.

Hypotheses

It is expected that depressed individuals will report a greater perception of day to day stressors as compared to normal controls and these subjects will show a greater degree of emotional eating and emotion-focused coping styles. Furthermore, these subjects will exhibit a) increased food cravings and eating behaviours following stress induction; b) higher cortisol response to the stress induction task, (but gender differences may occur); c) greater intake of sweet high fat food post stress

induction, outside the experimental setting in comparison to controls as evidenced by food choices and amount indicated in food records.

With respect to the normal control population, it is expected that individuals with restrained eating patterns will overeat in response to a social stress challenge compared to those without restrained eating patterns. Furthermore, those with restrained eating patterns within the normal control population will exhibit a higher cortisol response to the stress induction compared to subjects without restrained eating patterns.

Methods

Overall Design

Stress-induced eating behaviours and physiological response to a social stressor were measured and compared between participants with chronic Major Depressive Disorder and healthy volunteers. Measures assessing depression symptoms and eating behaviours were collected, as well; information on stress perception, daily hassles and uplifts was obtained. Participants were exposed to a social stress challenge (Trier Social Stress Test). Serial salivary cortisol samples were collected and subsequent food intake was measured.

Participants

Fourteen participants who met diagnostic criteria for chronic Major Depressive Disorder, and 11 volunteers with no history of psychiatric disorders were recruited from an ongoing study "Individual differences in stress responsivity in adults with chronic depression: A socio-biological approach" within the Mood and Anxiety Program at the Centre for Addition and Mental Health. Depressed participants were included in the study if they met the following criteria: 25-55 years of age, current DSM-IV diagnosis of Major Depressive Disorder for the past two years, physically healthy, and able to provide informed consent. In addition, depressed individuals who met the following criteria were excluded from the study: current substance abuse or dependence; have received psychotherapy within the past months; been treated with reversible inhibitors of monoamine oxidase inhibitors (MAOI) or tricyclic antidepressants within past 3 months; had electroconvulsive therapy within past 6 months; depot neuroleptics within the past 6 months; acute risk of suicidality; history of psychotic symptoms; diagnosis of Bipolar I & II, or another Axis I disorder as the primary diagnosis including anorexia nervosa and/or bulimia nervosa; a diagnosis of a major medical illnesses (asthma, heart disease, crohn's, rheumatoid arthritis, diabetes, Hep C, etc.); medications such as antibiotics and most steroid medications; however, women on birth

control pills are eligible; currently pregnant; major recent life stressors (deaths, divorce, job loss, moving etc.).

Male or female participants between 25-55 years of age and without a history of psychiatric disorders were recruited for the comparison group. Individuals were required to be physically healthy and able to provide consent in order to be included in the study. Participants were excluded from the study if they met any of the following criteria: current or past psychiatric disorder; psychiatric medications; any significant current substance abuse/dependence (with the exception of nicotine); major medical illnesses (asthma, heart disease, crohn's, rheumatoid arthritis, diabetes, Hep C, etc.; unstable hypertension or hypothyroidism; medications such as antibiotics and most steroid medications (however, women on birth control pills are eligible); currently pregnant; major recent life stressors (deaths, divorce, job loss, moving etc.).

Measures

Hamilton Depression Rating Scale 29-item (HAMD-29) (Hamilton, 1967): This clinician-rated scale measured depressive symptom severity. Scores were obtained for the first 17-items, representing the core symptoms of depression, as well as 29 –item scores which includes items specific to subtypes of depression (e.g. atypical). All raters were required to successfully complete training specific to this measure.

Uplifts and Hassles Scale (Kanner et al., 1981): 53-item self-report scale that contains two scales and measured the presence of uplifts and hassles in an individuals' daily life. Participants were asked to provide a rating on a 4-point likert scale (0- "None/not applicable" to 3-"A great deal") for a number of variables found in daily life (e.g. children, spouse, job, etc) based on whether it was an uplift or a hassle for that particular day. Separate scores were obtained for hassles and uplifts.

Coping Strategy Scale (CSS) (Beckham and Adams, 1984): 46-item scale that assessed various coping strategies in response to problems or stress with a 4-point likert scale (0"Never" – 3 "Frequently"). This scale measured the following 13 coping strategies: Problem Solving, Cognitive Restraint, Active, Avoidant, Rumination, Humour, Social Support, Emotional Expression, Blame Others, Self-Blame, Emotional Control, Passivity, Religiosity. A separate score was obtained for each coping strategy.

Perceived Stress Scale (PSS) (Cohen, Kamarack, and Mermelstein, 1983): 14-item scale that measured the degree to which life situations are perceived as stressful. Participants were asked to indicate the frequency at which they experience various thoughts/feelings related to stress on a 5-point likert scale (0 - "never" to 4 "very often").

Scale of Sensitivity to Reward and Punishment (SPSRQ) (Torrubia et al., 1995): 48 yes-no response items that contained two independent scales; one that measured sensitivity to reward and the other measured sensitivity to punishment.

Positive and Negative Affect Scale (PANAS) (Watson and Clark, 1988): 20-item self-report measure containing two scales; one of positive emotions, and one of negative emotions. Participants indicated to what extent they felt the various emotions on a 5-point likert scale (1-"Very slightly or not at all" to 5- "Extremely"). Separate scores for positive and negative affect were obtained.

Dutch Eating Behaviour Questionnaire-Revised (DEBQ) (Van Strien et al., 1986): 33-item self-report scale that measured emotional, external, and restraint eating. Participants were asked to assess the accuracy of each item in relation to their own eating behaviour on a 5-point likert scale (1-"Never" to 5 "Very Often"). Scores for these three types of eating were generated.

The Restraint Scale (Herman and Polivy, 1980): 11-item self-report scale that measured the degree of restrained eating, as well as disinhibition, over-eating, and weight fluctuations.

General Food Craving Questionnaire – Trait and State (GFCQ) (Cepeda-Benito et al., 2000): The Trait measure is a 21-item self-report scale that assessed stable features of food cravings across various dimensions. Participants were required to provide a rating across a 5-point likert scale (1 – "Never" to 5-"Always") in response to statements referring to food craving. The State measure is a 15-item self-report scale that assessed food cravings but in a manner that is sensitive to contextual, psychological, and physiological state changes. Separate scores for trait and state food cravings were obtained.

Food Records: Participants recorded food intake (upon wakening until sleep) for a period of three consecutive days, beginning on the day of the stress induction. They were asked to provide information on the food type, amount consumed, and time of day. Participants were provided with verbal and written instructions, along with a sample of a completed food record for reference.

Trier Social Stress Test (Kirschbaum et al, 1993): Based on the procedures developed by Kirschbaum et al. (1993), social stress was induced by placing participants in a novel situation in which they believed they were being socially evaluated. The task required participants to make a 5-minute speech and to complete a 5-minute arithmetic challenge (counting backwards in 13's from 1022) in front of a panel of three confederates. Participants were informed that the panel members were behavioral analysts who would be assessing for signs of anxiety. To heighten the feeling of evaluation, participants were also led to believe that both video and audio recordings were being obtained through a microphone and video camera for assessment of anxiety symptoms.

Salivary Cortisol Samples: Serial salivary cortisol samples were obtained before and after the stress induction (Trier Social Stress Test). Participants were instructed to lightly chew on cotton salivettes (Sarstedt, Montreal, Quebec) for approximately 45 seconds. The samples were stored in – 80 degrees Celsius until they were analyzed.

Procedure

Screening Procedures (Visit 1)

All participants underwent the Structured Clinical Interview for DSM-IV, Axis I Disorders (SCID-I) (First et al., 1990) to confirm a current diagnosis of Major Depression Disorder for the past two years, and in the case of controls, the absence of any psychiatric disorders. In addition, the SCID-I was used to determine subtype of depressive episode i.e., atypical, melancholic, or neither. Demographic information, medical history, physical exam, psychiatric history and current treatment information were obtained. Height and weight were measured in order to calculate Body Mass Index (BMI). Vitals (blood pressure, heart rate) were obtained in order to exclude individuals with potential cardiac issues or health risks. Participants were also seen by a physician to confirm study eligibility criteria and physical health.

Trained raters administered the HAMD-29 and the following self-report measures were given to the participant for completion: DEBQ, Hassles and Uplifts, CSS, PSS, SPSRQ, the Restraint Scale, and GFCQ – Trait version.

Experimental Day (Visit 2)

Stress Induction: Trier Social Stress Test and Serial Salivary Cortisol Samples

On a separate day, participants returned for the stress induction. They were not provided with any specific information prior to the experimental day but were told they would experience a mild social stressor. To control for diurnal cortisol secretion, all experiments were conducted after 1400h and experiments were scheduled at either 1400h or 1600h. Upon arrival, participants were lead to a separate room where they remained until the stress induction procedures. Salivary cortisol samples, and vital signs (blood pressure and heart rate) were taken upon arrival, 15 minutes after arrival and immediately preceding the stress induction (T0).

Ten minutes before the "task", participants were brought into the experimental room and given instructions with three panel members present. In the experimental room there was a video

and audio recording equipment, a microphone on a stand, and a long table behind which three confederates in white lab coats were seated. Participants were asked to pretend that they were applying for a job and to prepare a speech explaining why they would be the best candidate for that job. In addition, participants were informed that they would have to perform a second task but that those instructions will be given immediately following their speech. The participants were then given the opportunity to ask questions and then were led back to the other room to begin speech preparations.

After a period of 10 minutes of preparation time, participants were brought back into the experimental room. At the same time, one panel member turned on the video camera, and a second panel member initiated the audio recording. Participants were given instructions to step behind the microphone and to begin their speech. If participants finished before the time limit of five minutes, panel members remained silent for 20 seconds and then if the participant had not continued with the speech, asked questions from a scripted list. After the speech task, participants were then given the instructions for arithmetic challenge. If participants make an error, a panel member informed them of the error and instructed them to begin again. This continued until the time limit (5 minutes) was completed. Participants were then instructed to return to the other room and await further instructions.

At this time, the experimenter instructed the participant to rest while serial cortisol samples were obtained. Samples were collected at the following time points after the stress induction: 10 minutes (T10), 30 minutes (T30), 50 minutes (T50), and 70 minutes (T70). Vitals were obtained immediately following, 10 minutes, and 30 minutes post-stress induction. The experimenter kept conversation to a minimum and deferred any questions until the end of the experiment. Participants were debriefed after completion of the food craving challenge.

Cue Elicit Craving Challenge (CEC):

Based on procedures used in Davis et al., 2007a, participants were asked to indicate their 'favourite snack food' (that did not require cooking or to be kept frozen) prior to the experimental day. If participants indicated a healthy snack, the experimenter probed to elicit a food that was considered enjoyable and "forbidden". The participants were not given any indication as to why they were being asked to provide this information. The weight of the snack food was measured prior to its presentation.

Seventy minutes post-stress induction, after all cortisol samples have been collected, the experimenter entered the room and administered the CEC. Participants were presented with their favourite snack food and asked three questions. They were instructed to try and answer the questions without concern of caloric content or time of day. Participants were asked the following questions and to provide answers on a scale of 1-10 (1=not a lot to 10=a lot): 1) How hungry does it make you feel to see your favourite snack? 2) How much would you like to eat some of this snack – even just a small portion? At this point, participants were asked to eat a small portion of the snack. After they were finished, the experimenter then asked the final question "Now that you've had a taste of your favourite snack, how strong is your desire to have some more?". The participants were then asked to complete the GFCQ – State version. Participants were free to continue eating their snack for the remainder of the visit but were not specifically instructed to do so. If a participant asked if they could continue to eat, the experimenter stated, "the snack is yours to eat, you may do whatever you'd like."

Food Record Procedures:

Participants were asked to complete a food record for three consecutive days starting on the day of experiment. They were asked to provide information for all foods consumed from the time they awoke until the time they went to bed on each of the days. They were asked to provide details such as time consumed, amount, as well as caloric and fat information whenever possible. The

experimenter instructed the participants on how to complete the food records and provided written instructions along with a completed sample food record as reference.

In addition to the food records, participants were asked to complete a PANAS for each of the three days that they complete a food records.

At the end of the CEC, participants were informed that the experiment was finished and were fully debriefed. At this time, the experimenter asked questions to determine if the stress induction was successful, and to assess whether or not participants had discovered the true purpose of the study. Participants were also given the opportunity to ask questions and provided with contact information should they feel any ill effects of participating in the stress protocol. At the very end of the study visit, just prior to leaving, any remaining portion of the snack food was weighed.

Statistical Analyses

Independent-samples t-tests were used to detect group differences in all study measures. Simple correlations and linear regressions were used to explore associations between psychological and behavioural data.

Salivary cortisol levels were determined, in duplicate, by RIA using radioimmunoassay kits (ICN Biomedical Inc Costa Mesa, CA.). The intra-and extra-assay variability was less than 10%. Mixed measure ANOVAs were used to examine group differences in cortisol levels throughout stress induction. In addition, area under the curve (AUC) was calculated using the AUC_G formula by Pruessner et al., (2003), as well, peak percentage change in cortisol was calculated (maximum post-stress cortisol measure – T0 / T0 x100). One-way ANOVAs were used to detect group differences in AUC and peak percentage change. Simple correlations and linear regressions were used to explore associations between psychological measures and physiological data.

Results

Twenty-six individuals (females = 20, males =6) consented to participate in this study. All those who were approached agreed to participate, however, one male participant failed to maintain scheduled study appointments and was withdrawn from the study. Fourteen participants (females = 11, males = 3) were included in the depressed group, while the control group consisted of 11 participants (females = 9, males = 2).

Demographic Data

The mean age of participants was 38.64 (*SD*=8.84), with a range of 26 years to 51 years. The mean age of depressed individuals was 41.86 (*SD*=7.19) and 34.90 (*SD*=7.69) for those in the control group. The average weight and BMI for the sample are presented in Table 1. No significant differences in weight ($t(23)=-.55$, $p=.59$) and BMI ($t(23)=-.71$, $p=.49$) between the depressed and control groups were found.

Fifty-two percent of the sample (n=13) was classified as having a normal weight (BMI of 18.5 – 24.9), while 20% (n=5) were classified as being overweight (BMI of 25.0-29.0). Twelve percent of participants were considered Obese class I (BMI of 30.0-34.9), and 12% were categorized as Obese class II (BMI of 35.0-39.9) with one participant as Obese class III (BMI of >=40). Chi-square analysis indicated that there were no differences in proportion of BMI classification between depressed and control participants $\chi 2$ (1, N=24)=3.23, $p=.52$.

Depression Measures

All individuals within the patient group met DSM-IV-TR criteria for major depressive disorder for a period of >= 2 years. In addition, 64% (n=9) met diagnostic criteria for atypical subtype of depression, 22% (n=2) met for melancholic depression, and 33% (n=3) were not categorized as either subtype. Fifty-seven percent (n=8) met diagnostic criteria for a comorbid anxiety disorder. None of the participants met diagnostic criteria for any eating disorder, including binge eating disorder. Furthermore, 78% (n=11) were receiving medication for the treatment of depression at the

time of study participation. The mean HAMD-17 item score for the depressed group was 19.43 (*SD*=3.94) and the HAMD-29 average score was 32.07 (*SD*=7.83), with no significant differences between the diagnostic subtypes, as well as no significant differences between those with and without a comorbid disorder.

Stress and Coping Measures

Independent-samples t-tests were used to test group differences in perception of stress (PSS), daily uplifts and hassles, and coping strategies (CSS). Mean PSS score was significantly higher for depressed individuals (*M*= 34.92, *SD*=9.14) than for those in the control group (*M*=25.18, *SD*=8.45) (t (20) = 2.64, p=.01, effect size d=1.18). Additionally, daily hassles and uplift scores significantly differed between the two experimental groups. Scores measuring daily uplifts were significantly lower for depressed participants (*M*=30.09, *SD*=15.42), than for those in the control group (*M*=48.00, *SD*=22.50) (t (19)=-2.15, p=.04, effect size d=.98). However, contrary to results from previous studies (e.g. Ravindran et al., 2002), there were no differences in reported daily hassles between depressed individuals (*M*=43.50, *SD*=15.93) and controls (*M*=44.40, SD=21.30). Analysis of the CSS indicated that control participants had higher scores than depressed on the problem solving subscale (t (23)=-2.36, p=.027, with an effect size of d=.99) and the active subscale (t (23) = -3.66, p=.001, with an effect size of d=1.53), indicating a greater use of "non-emotional" coping strategies.

In addition to the stress and coping measures, results from the sensitivity to reward and punishment scale (SPSRQ) revealed differences between depressed and control participants. Depressed individuals exhibited a greater sensitivity to punishment (*M*=13.77, *SD*=5.34) than controls (*M*=7.55, *SD*=5.24) (t (22) = 2.87, p=.01, with an effect size of d=1.53). However, no significant group differences were found in sensitivity to reward scores.

Eating Measures

Independent-samples t-tests were used to detect differences in eating behaviour between experimental groups. There were no differences between the depression and control groups for all subscales (emotional, external, and restraint) of the DEBQ. In addition, there were no differences found in trait food cravings (GFCQ-Trait) between depressed and control participants.

Restraint scale scores did not significantly differ between groups. The mean restraint score for depressed individuals was 17.57 (*SD*=6.22), and 17.30 (*SD*=7.09) for control participants. Seventy-five percent (n=18) of the total sample was considered to be a restrained eater (score of >=15 for female and >=12 for males), with 71% (n=10) of depressed individuals and 80% (n=8) of control participants considered restrained eaters. Chi square analysis indicated that there were no significant differences in proportion of restrained eaters between depressed and control participants, $\chi 2$ (1, N=23)=.23, p=.63. Furthermore, there were no differences in proportion of restrained eaters between the depressive subtypes, $\chi 2$ (1, N=23)=1.75, p=.42.

Simple correlations revealed no significant association between age and restraint scale scores. In addition, no significant relation between depression symptom rating scale and restraint scale scores was found. There were no significant correlations between HAMD-17 item, HAMD-atypical subscale, and HAMD-29 item scores and restraint scale scores. However, there was a significant association between sensitivity to punishment scores and restraint scores. Simple correlations indicated a significant positive association between restraint scores and sensitivity to punishment scores for the sample (r=.44, p=.04). In addition, regression analysis indicated that sensitivity to punishment scores was predictive of restraint scores, b=.49, $t(21)$=2.23, p=.04 and explained a significant proportion of the variance, R^2 = .19, $F(1,21)$=4.96, p=.04.

Stress Induction

Positive and negative affect measures administered before and after the stress induction were analyzed for group differences. Independent samples t-tests revealed significant differences

between depressed and control participants indicating that control participants reported greater positive affect (M=34.56, SD=6.65) than depressed participants (M=18.50, SD=8.06) ($t(18)$=-4.70, p=<.001, with an effect size of d=2.22), before the stress induction. Surprisingly, differences between depressed (M=21.67, SD=12.14) and non-depressed (M=13.56, SD=3.78) participants in negative affect scores of the PANAS before stress induction did not reach statistical significance ($t(17)$=1.89, p=.09).

Post-stress induction differences between the depressed and non-depressed participants on both negative and positive subscales of the PANAS were found. Depressed participants had a significantly higher mean negative affect score (M=25.44, SD=7.43) than controls (M=15.20, SD=3.71) ($t(16)$=3.65, p<.01, with an effect size of d=1.83) and reported significantly lower mean score (M=19.33, SD=4.36) than control participants (M=34.22, SD=8.42) ($t(16)$=-4.71, p<.01 with an effect size of d=2.36) on positive affect subscale post-stress induction. In addition, paired-sample t-tests were conducted separately for each group to detect within-subject affect differences pre and post stress induction. There were no significant differences in affect before and after stress induction for either depressed and control groups. Induction of stress did not appear to impact affect for all participants.

Cue Elicit Craving Challenge (CEC)

Ratings for the three questions of the CEC challenge were summed together to create a total score (Davis et al., 2007). Analyses were conducted both on total and individual item scores to detect group differences in response to stress. Mean CEC total and individual item scores are presented in Table 2. Independent-samples t-tests did not reveal any significant differences between depressed and control participants for individual items or total scores of the CEC.

The Trier Social Stress Test was scheduled at 1400h and 1600h and as such, potential differences in hunger levels could have impacted hunger ratings measured within the CEC. Independent-samples t-tests were employed to detect differences between the two time points of

stress induction and indicated that there were no differences in CEC item or total scores based on time of experiment.

Mood Measures and CEC

Simple correlations were used to examine associations between mood measures and CEC scores. There were no significant associations between HAMD-17 total scores and CEC total scores, as well as HAMD-29 total scores and CEC total scores. However, when items from the CEC challenge were analyzed individually, a significant positive correlation was found between degree of hunger (CEC Item 1) and HAMD-17 total scores for the sample (r=.41, p=.05) as well as for HAMD-29 scores (r=.41, p=.05). Furthermore, linear regression analysis indicated an association between HAMD-17 scores and degree of hunger (CEC Item 1) b=.117, $t(21)$=2.031, p=.055, R^2 = .164, $F(1,21)$=4.125, p=.05, as well as HAMD-29 scores, b=.071, $t(21)$=2.037, p=.055 R^2=.165, $F(1,21)$=4.147, p=.055, that approached significance. No significant associations were found between HAMD (17 and 29 item) scores and the other CEC items. It appears that most associations occurred between HAMD scores and degree of hunger (CEC Item 1) scores suggesting that as depression scores increase so do hunger ratings post-stress.

Negative affect scores from the PANAS were correlated with CEC total and individual item scores. A positive correlation between post-stress negative affect scores and CEC total score approached significance (r=.46, p=.056), and linear regression analysis with CEC total score as the dependent measure, indicated an association that approached significance b=.279, $t(16)$=2.07, p=.055, R^2=.211, $F(1,16)$ =4.28, p=.055. No other associations between negative affect scores and CEC scores reached significance.

In order to determine whether or not negative affect prior to the stress induction was associated with CEC scores, correlation analysis of pre-stress negative affect (PANAS) and CEC scores were conducted. No significant associations between pre-stress negative affect scores and CEC scores were found. However, an inverse association that approached significance was found

between pre-stress negative affect scores of the PANAS and desire to eat after tasting snack (CEC Item 3), r=-.44, p=.07. These findings suggest that pre-stress negative affect does not appear to predict post-stress hunger in the same manner as post-stress negative affect. However, higher pre-stress negative affect may be associated with a lower desire to eat after tasting a snack.

Correlations were also conducted with pre and post-stress positive affect scores of the PANAS scale and CEC scores to explore associations between positive affect and CEC scores. No associations between positive affect scores and CEC scores reached significance.

Coping/Stress Measures and CEC

Relations between perception of stress, coping strategy style, daily uplifts and hassles, and sensitivity to reward/punishment were explored using simple correlations and linear regression. Perception of stress scores were significantly positively associated with degree of hunger (CEC Item 1) (r=.45, p=.04), and was found to be predictive of degree of hunger (CEC Item 1) scores, b=.115, $t(19)$=2.190, p=.041, and accounted for a significant proportion of the variance R^2 = .202, $F(1,21)$=4.479, p=.041. Furthermore, a significant negative correlation was found between CSS – Problem solving scores and degree of hunger (CEC Item 1) (r=-.48, p=.02). Problem solving scores were also found to predict degree of hunger, b=-1.923, $t(21)$=-2.509, p=.02 and accounted for a significant proportion of the variance R^2 =.231, $F(1,22)$=6.296, p=.02). There was also a positive association between CSS - Rumination scores and degree of hunger (CEC Item 1) scores that approached significance, r=.39, p=.07. No significant associations between CSS subscale and CEC scores were found. It appears that higher perception of stress scores and higher rumination scores are related to higher hunger ratings, while greater use of problem solving is associated with lower hunger ratings.

Eating Measures and CEC

Relations between eating behaviours and CEC scores were explored through correlation analysis of DEBQ, Restraint Scale, and GFCQ-Trait/State and CEC scores. DEBQ emotional eating scores

were found to predict degree of hunger (CEC Item 1) scores b=1.317, $t(19)$=2.205, p=.040 and accounted for a significant proportion of the variance in degree of hunger, R^2=.204, $F(2, 19)$=4.862, p=.04). While no other associations reached significance, a number of marginally significant relations were found. Positive correlations between DEBQ emotional eating subscale scores and desire to eat before tasting snack (CEC Item 2) (r=.37, p=.09); DEBQ restrained eating subscale scores and degree of hunger (CEC Item 1) (r=.41, p=.06); and CEC total scores and DEBQ external eating subscale scores (r=.39, p=.08) approached significance.

The findings from eating measures scores and post-stress CEC scores indicate that higher emotional eating scores predicted hunger ratings. Furthermore, greater levels of both external and restraint eating (as measured by the DEBQ) may be associated with greater CEC total scores and higher hunger ratings respectively.

Food Consumption During Experiment

The amount of food consumed (grams) during the experiment was calculated by subtracting the difference between the two time points when food was weighed (before CEC challenge and immediately before participant left). The nutritional content (caloric, fat, protein, and carbohydrates) of the food was analyzed using ESHA Food Processor SQL software (version 10.3.0) and was reviewed by a registered dietician. The mean weight of food consumed during the experiment was 22.64g (SD=20.61) for depressed participants and 12.48g (SD=17.324) for control participants. Numerically, the depressed group consumed a greater quantity of food during the experiment; however, an independent-samples t-test indicated that there was no significant difference between the groups. In addition, the mean caloric value of the food consumed during the experiment was 98.99kcal (SD=88.232) for depressed individuals and 59.24 (SD=85.270) for controls. Again, an independent-samples t-test indicated that this difference between groups did not reach statistical significance. Furthermore, nutritional composition of the snack, absolute or percentage there were no significant differences in protein, fat, and carbohydrates consumption

during the experiment between depressed and control participants. As well, there was also no difference in quantity consumed during the CEC for those who went through the stress experiment at 1400h compared to those who were scheduled for the 1600h time point ($t(21)=-.662$, $p=.521$).

Affect and Food Consumption during Experiment

Simple correlations and linear regression[1] were employed to examine associations between both positive and negative affect and quantity of food consumed during the experiment. Pre-stress positive affect scores were negatively associated with amount consumed after the stress induction ($r=-.477$, $p=.023$), and predicted the same, $b=-.929$, $t(16)=-2.170$, $p=.045$ ($R^2=.227$, $F(1,16)=4.709$, $p=.045$). However, no significant correlations were found between pre-stress negative PANAS scores and amount of food consumed post-stress induction. Post-stress negative PANAS scores were found to have a positive association with, and predicted the amount of food consumed after stress, $b=1.339$, $t(16)=2.204$, $p=.043$, and accounted for a significant proportion of the variance ($R^2=.233$, $F(1,16)=4.857$, $p=.043$). It appears that pre-stress positive affect and post-stress negative affect predicted the amount of food consumed after the stress induction.

Stress/Coping Measures and Food Consumption During Experiment

Correlation analyses were conducted to examine any associations between coping styles, perception of stress, sensitivity to reward/punishment, and quantity of food consumed during the experiment. Rumination coping style subscale scores of the CSS were found to have a positive association with the amount of food consumed post-stress induction ($r=.41$, $p=.02$). Furthermore, linear regression revealed that rumination coping scores were predictive of amount of food consumed post-stress, $b=10.28$, $t(21)=2.08$, $p=.05$ and that this accounted for a significant proportion of the various $R^2=.17$, $F(1, 21)=4.31$, $p=.05$. In addition, PSS scores were positively correlated with ($r=.49$, $p=.01$) and found to be predictive of amount consumed post stress $b=.965$, $t(19)=2.449$, $p=.024$ ($R^2= .240$, $F(1,19)=6.00$, $p=.024$). Interestingly, a significant positive association between sensitivity to punishment scores and amount of food consumed post-stress was found ($r=.39$, $p=.04$). It appears

that as rumination coping style and perception of stress increases so does the amount of food consumed after stress induction; and that sensitivity to punishment may potentially be associated as well.

Eating measures and Food Consumption during Experiment

Measures of eating behaviour were correlated with amount of food consumed during the experiment to identify potential associations. Results indicated that GFCQ-Trait scores were significantly positively correlated with amount consumed post-stress induction (r=.632, p=.004). A linear regression of GFCQ-Trait scores, with the group variable included in the model, were found to predict amount of food consumed post stress revealed a significant association b=.793, $t(13)$=3.221, p=.007, and accounted for a significant proportion of the variance R^2=.514, $F(2,13)$=6.888, p=.009. However, no associations were found between GFCQ-State scores and amount of food consumed post stress. In addition, external eating scores (DEBQ) were significantly associated with the amount food consumed post – stress induction (r=.712, p<.001). A linear regression model of group and DEBQ external eating scores indicated that external eating scores significantly predicted the amount of food consumed after the stress induction b=24.406, $t(18)$=4.068, p=.001, and accounted for a significant proportion of the variance, R^2=.509, $F(2,18)$=9.329, p=.002. Food cravings, as a trait, along with external eating scores predicted food consumption post-stress in that as food craving trait scores and external eating scores increased, so did the quantity of food consumed after the stress induction.

Food Record Analysis

All data collected from the food records were analyzed using ESHA Food Processor Software SQL (version 10.3.0) and was reviewed by a registered dietician. Separate analyses were conducted for food records obtained on the day of the stress induction and for average intake over 3 consecutive days post-stress induction.

Food Records – Day of Experiment

Food record data for the day of the experiment was analyzed and caloric intake (kcal), protein, carbohydrate, and fat consumption (gram) were compared between depressed and control groups using independent-samples t-tests. Upon recommendation of a registered dietician, one participant was excluded due to the initiation of a highly restrictive diet the week of the experiment and food record collection. Mean intake for the day of the experiment is presented in Table 3. No statistically significant differences in nutritional intake were found between depressed and non-depressed participants for the day of the experiment.

Depression /Affect Measures and Food Intake – Day of the Experiment

No significant associations were found between total HAMD-17 scores or total HAMD-29 scores and nutritional categories of food intake the day of the experiment. However, negative affect scores (PANAS) for the day of the experiment predicted carbohydrate intake for the same day, b=-6.601, $t(19)$=-2.132, p=.046 and explained a significant proportion of the variance (R^2=.193, $F(1,19)$=4.545, p=.046), though contrary to expectation, it was an inverse association. This suggests that increased negative affect scores are associated with decreased carbohydrate intake.

Stress/Coping Measures and Food Intake – Day of Experiment

The only association found to nearly approach significance between stress/coping measures and nutritional categories of food intake on the day of the experiment was for fat intake. CSS– Avoidant subscale scores were positively associated with and predicted (along with group) fat intake for the experimental day b=17.129, $t(20)$=2.072, p=.051 and explained a significant proportion of variance of fat intake, R^2=.256, $F(2, 20)$=3.441, p=.052.

Eating Measures and Food Intake – Day of the Experiment

Linear regression analysis indicated that DEBQ restraint scores significantly predicted protein intake for the day of the experiment, b=-28.301, $t(18)$=4.516, p<.0001 and along with group, accounted for a significant proportion of the variance, R^2=.283, $F(2,18)$=3.759, p=.043. As well, DEBQ restraint

scores predicted carbohydrate intake on the day of the experiment, b=-67.505, $t(19)$=4.586, p<.0001 and accounted for a significant proportion of the variance in carbohydrate intake, R^2 = .212, $F(1,19)$ 5.112, p=.036. In addition, Restraint Scale scores were negatively associated with and predicted carbohydrate intake, b=-6.562, $t(21)$=-2.115, p=.047, and also explained a significant proportion of the variance in carbohydrate intake (R^2=.176, $F(1,21)$ = 4.475, p=.047). Restraint eating, as measured by both the DEBQ and Restraint Scale were found to be negatively associated with protein and carbohydrate intake the day of the experiment, however, no other associations with caloric or fat intake were found.

Food Records – Three-Day Average

Averages for 3-day food intake were calculated and caloric intake, protein, carbohydrates and fat consumption were compared between groups using independent-samples t-tests. Mean food intake for the three day period is presented in Table 4. There were no significant differences found in caloric intake, amount of protein, carbohydrate, and fat intake over a 3-day period between depressed and control participants. In addition, no significant associations were found between weight or BMI and average 3-day intake for all nutritional categories.

Positive and negative affect scales of the PANAS that were completed over the same 3-day period as the food records were averaged over the three days. The 3-day mean negative affect scores of the PANAS scores for depressed participants were 19.67 (SD=6.14), and 14.25 (SD=3.35) for controls; while the average 3-day positive affect scores were 19.30 (SD=5.23) and 33.14 (SD=7.66) for depressed and non-depressed respectively. Independent-samples t-tests indicated there were significant differences for both positive ($t(17)$=2.518, p=.022, with an effect size d=1.12) and negative scores ($t(16)$=-4.849, p<.001, with an effect size d=2.30) between depressed and control participants.

Depression/Mood Measures and Food Records – Three-Day Average

Simple correlations and linear regressions were employed to explore associations between 3-day intake of calories, protein, carbohydrates, fat, and study questionnaire data (e.g. HAMD scores, stress perception measures, eating behaviours). No significant associations were found between HAMD scores and food intake averaged over three days. However, negative affect scores of the PANAS were found to significantly predict 3-day average protein intake, b=-2.040, $t(17)$=6.093, p<.0001 and account for a significant proportion of the variance, R^2=.203, $F(1,17)$=4.339, p=.05. No other significant associations were found for mood measures and 3-day nutritional intake.

Eating Measures and Food Records – Three-day Average

Analysis of 3-day average of caloric intake and study measures did not reveal significant associations with the exception of DEBQ restraint scores. Three-day average of caloric intake was predicted by DEBQ restraint scores, b=-352.372, $t(17)$=-2.290, p=.035 and accounted for a significant proportion of the variance, R^2=.236, $F(1,17)$=5.244, p=.035, within the study sample. Both Restraint Scale scores, b=-7.778, $t(21)$=-2.979, p=.007 (R^2=.297, $F(1,21)$=8.876, p=.007) , and DEBQ restraint scales scores, b=-66.840, $t(19)$=-2.609, p=.017 (R^2=.264, $F(1,19)$=6.808, p=.017), were found to be negatively associated with and to predict 3-day average of carbohydrate intake. Restraint scores (as measured by the Restraint Scale and DEBQ) predict caloric and carbohydrate intake in that as restraint scores increased, caloric and carbohydrate intake decreased.

Salivary Cortisol

A mixed design ANOVA was used to test group differences in cortisol levels before and after the stress induction. A significant main effect of time was found, $F(4, 80) = 6.768$, p<.0001, η^2 =.253, however, no significant main effect of group or time by group interaction were found. Cortisol levels changed significantly across the experiment but there were no differences in levels between

depressed and control participants. See Figure 1 for mean cortisol levels pre and post stress induction by group.

In addition, one-way ANOVA did not reveal significant differences between the depressed and control groups. Furthermore, simple correlations were conducted to determine any association between AUC and study measures such as stress perception, coping styles, and eating behaviours. No significant relations were found between AUC and any of the study measures.

As well, peak percentage change in cortisol response was calculated and compared between groups. One-way ANOVA did not reveal any significant differences between depressed and non-depressed participants. However, correlations revealed significant associations between peak percentage change and three study measures for the entire sample. First, a significant negative association between degree of hunger (CEC Item 1) and peak percentage change was found (r=-.50. p=.02), and peak percentage change was found to predict degree of hunger (b=-.017, t(20)=-2.57, p=.02, R^2=.249, F(1,20)=6.620, p=.02). A greater peak percentage change in cortisol response was associated with lower hunger scores (CEC Item 1). Second, positive affect scores (PANAS) post-stress induction were positively related to peak percentage change in cortisol levels (r=.54, p=.02) and were found to significantly predict peak percentage change in cortisol response (b=4.421, t(16)=2.54, p=.02, R2=.29, F(1,16)=6.45, p=.02). And third, a significant negative association was found between sensitivity to punishment and peak percentage change in cortisol response (r=-.47, p=.03) and sensitivity to punishment scores were found to predict peak percentage change in cortisol (b=-5.68, t(19)=-2.33, p=.03, R^2=.22, F(1,19)=5.44, p=.03). Higher sensitivity to punishment scores were associated with lower percentage change in cortisol levels after stress induction.

No significant differences in salivary cortisol between depressed and non-depressed participants in response to stress were found. Consistent with these findings, no significant group differences in stress-induced eating behaviours were found between depressed and non-depressed participants as well.

Discussion

Altered stress perception, coping strategies and HPA axis activity have been noted in depressed individuals. As such, it was predicted that depressed individuals, in comparison with a control group, would exhibit an altered response to stress and subsequently consume a greater amount of food post stressor. Contrary to predictions, there were no group differences in eating behaviours between depressed and non-depressed participants post-stress induction. Also in contradiction to hypotheses, there were no differences found within food choice and intake (calories, protein, carbohydrate, and fat) between depressed and control participants. Furthermore, the sample contained a large proportion of restrained eaters with no difference between depressed and non-depressed participants, limiting the interpretation of results based on this variable.

Mood and Stress Measures

Consistent with previous research, depressed participants reported higher level of perceived stress than control participants. Furthermore, non-depressed individuals endorsed the use of active coping strategies (e.g. problem solving) more so than depressed participants, however, no differences in emotion-focused coping strategies (e.g. rumination) were found between groups. Previous studies indicate that depressed individuals tend to use emotion-focused coping strategies in response to stress (e.g. Kelly et al., 2007), while in the present study, no group differences for emotion-focused subscales of the CSS were found. Also inconsistent with previous findings, there were no differences in reported hassles between depressed and non-depressed participants; though previous studies have included both acute and chronically depressed participants (e.g. Ravindran et al., 2002). However, depressed participants reported fewer uplifts than control participants. In addition, depressed individuals reported a greater sensitivity to punishment than non-depressed but there was no difference between groups in sensitivity to reward scores. Other studies have suggested that

those with depression are less sensitive to reward due to symptoms of anhedonia (Shankman et al., 2007); however, the results of this study are inconsistent with these previous findings.

As expected, depressed participants scored lower on positive affect scales and higher on negative affect scales than the control participants after stress induction. Surprisingly, differences in pre-stress negative affect scores between the two groups did not reach significance suggesting a similar level of negative affect for depressed and controls before stress was induced. However, depressed participants' positive affect scores were less than control participants before the stress induction. Furthermore, affect appeared to have remained stable for both groups throughout the stress induction protocol. Despite these results, participants were asked during the debriefing phase whether or not they felt stressed during the experiment. Only one participant indicated that he did not feel stressed and was excluded from the analyses.

It is possible that the PANAS was not sensitive enough to detect state affect in response to stress. The PANAS was designed as a trait measure but has since been shown to be effective as a state measure (Watson et al., 1994). It was administered both immediately preceding and following the stress induction, which amounts to a total time period of approximately 20 minutes. Memory for scale items may also have had an impact on PANAS scores as it was administered twice within such a short period of time. It is unclear whether or not this measure sufficiently captures affect changes within this study design. Furthermore, other studies that have measured affect before and after stress have also reported inconsistent findings, with some indicating that stress has a negative impact on affect, and others finding no difference (Burke et al., 2005). For the purposes of this study, the PANAS was administered in part as a manipulation check of stress induction, so regardless of how stress impacted affect, all participants who did not experience stress were removed from data analyses.

Eating Measures

Contrary to predictions there were no differences in any reported eating behaviours between the two groups. Depressed and non-depressed participants did not report differences in food cravings (both state and trait), emotional eating, external eating, and restrained eating (as measured by two scales). Given that depressed individuals report more negative mood and appetite disturbances, it was surprising that no differences in emotional eating and food craving measures were found between the two groups.

A large proportion of the sample (75%) was categorized as restrained eaters as measured by the Restraint Scale. Previous studies indicate an association between weight fluctuation and restrained eating (Heatherton et al., 1991). The fact that this sample had a mean weight that was considered overweight (according to BMI categories) and that the restraint scores were slightly above the scores that categorizes restrained eating may account for this disproportionate sample. Another contributing factor may be age. The minimum age requirement for this study was 25 years of age; therefore representing an older population than an undergraduate sample that is most often used to study restrained eating (e.g. Heatherton et al., 1989;). However, studies that have investigated restrained eating in populations older than undergraduate samples suggest that younger and older populations share psychological predictors of restrained eating behaviour (e.g. poor body image) (Paa and Larson, 1997). It is possible that age may contribute to restrained eating given that reduced metabolisms can contribute to increase in weight with age, and therefore, restrained eating behaviours may increase as well. Nonetheless, in this sample, age was not found to predict restrained eating scores. It is possible that this sample was biased due to the large proportion of restrained eaters.

Cue Elicit Craving Challenge

No differences in mean scores of CEC items between depressed and non-depressed participants were found contrary to predictions. It is interesting to note that all participants indicated a desire to

eat (range 5-10) after tasting a snack, as well, that only three participants indicated that they did not feel hungry after stress. This may be reflective of simply providing only palatable food (i.e. favourite snack food) and not offering other options of food. In other words, because all participants were presented with their favourite snack and not alternative food options, the CEC appeared to be successful in eliciting cravings in all participants, potentially masking any group differences.

Several additional factors may explain the lack of group differences in CEC scores as well. While it has been shown that food cravings can be manipulated using laboratory procedures and that reactivity to food cues can be readily elicited in the laboratory, this has been associated with binge eating (Sobik et al., 2005). To our knowledge, the CEC has not previously been administered with depressed patients and is used primarily to study eating disorders (Davis et al., 2004). Since binge eating and depression have different symptoms and proposed biological underpinnings (Davis et al., 2007), it is possible that the craving challenge was not appropriate for depressed participants. While a percentage of depressed participants reported hyperphagia and specific cravings, perhaps the CEC may not be specific or sensitive enough to elicit food cravings from depressed participants. Binge eating and depression often overlap; however, this sample did not contain any participants that met diagnostic criteria for binge eating. In this respect, this study could be considered a pilot investigation, as it was not known if the CEC would elicit cravings in depressed participants.

Other important variables that must be considered are the potential social factors involved with eating in the presence of other people. Due to the circumstances of this experiment, the participants were not left alone to eat. Participants were asked to "take a bite" in front of the experimenter and that may have inhibited participants from reporting level of hunger, desire to eat, and actual consumption of food in fear of social judgment or comparison. Furthermore, given that there was a high proportion of restrained eaters within the sample, participants may have been that much more sensitive to social comparison or judgment (Herman et al., 2003; Leone et al., 2007). Perhaps, if participants were left alone to eat, they would have felt less inhibited and eating

behaviour measures collected post stressor would have been more reflective of participants' stress-induced eating habits.

Mood and CEC

HAMD scores (both 17 and 29 item) were positively associated with CEC 1 item suggesting that higher depression scores are associated with higher hunger ratings post stress. Furthermore, negative affect during the stress experimental procedures was also related to hunger ratings post stress (CEC Item 1). Associations between post stress negative affect scores and total CEC scores approached significance, while higher negative affect scores significantly predicted greater food consumption after the stress induction. Conversely, greater positive affect scores were linked with lower quantity of food consumed post stress. These findings are consistent with emotional eating hypotheses suggesting that negative affect can increase food consumption.

Interestingly, associations between stress perception, coping styles and CEC scores were found. Higher perceived stress scores were associated with higher hunger ratings post stress and were found to predict amount of food consumed during the experiment. Furthermore, higher rumination as a coping method was associated greater food consumption and positive associations between rumination and degree of hunger approached significance. Higher problem solving coping strategy scores were associated with lower hunger ratings post stress. Emotion-focused coping strategies were associated with greater hunger and food consumption while more active coping style was associated with less hunger. Again, this is consistent with previous studies investigating the role of rumination in stress and eating behaviours (Kubiak, 2008).

It is interesting to note that most of the significant relations with mood, stress perception, and coping style occur with CEC Item 1 (hunger rating) but not with the other two items assessing desire to eat (before and after tasting the snack). Perhaps this is again a reflection of social factors, in that it may be easier to endorse hunger rather than actual eating behaviours. Though these factors

were also associated with food consumption, however, participants were not informed that snack food would be weighed until the very end of the experiment.

Food Record Data

When the nutritional information from food records was analyzed, there were no group differences found in caloric, protein, carbohydrate, and in fact, intake between depressed and non-depressed for either the day of the experiment or 3-day average intake. It is possible that three days were not sufficient to provide accurate information about macronutrient composition of participants' diets as some suggest that seven days provides a more accurate depiction of individuals' eating habits (Basiotis et al., 1987). As well, the fact that the sample contained a high proportion of restrained eaters may impact the data in that participants may be too concerned with appearances and therefore did not fully document all that was consumed (Maurer et al., 2006).

Mood and Food Record Data

Firstly, there were no significant correlations between depressive symptoms or affect measures for caloric or fat intake for either the day of the experiment or 3-day average. However, higher negative affect averaged over 3-days was related to lower protein intake for the same time period. Contrary to prediction, higher negative affect scores predicted lower carbohydrate intake on the day of the experiment. This is inconsistent with certain depressive symptoms, i.e., carbohydrate cravings, as well, previous studies investigating negative affect in nonclinical populations; which have both been associated with greater carbohydrate intake (Gibson et al., 2006). In addition, this is also inconsistent with studies investigating stress and macronutrient intake, which suggest that stress and negative affect is associated with higher intake of carbohydrates and fat (Epel et al, 2001). However, it is consistent with other investigations that found long-term high protein intake was associated with high self-reported depression scores, and that high carbohydrate intake was associated with low depression scores (de Castro, 1987). As well, anxious individuals have been shown to consume low carbohydrate, high fat, high protein diet (Pellegrin et al., 1998), which given

the comorbidity of anxiety in chronic depression, may accurately reflect the depressed group within this sample.

Coping/Stress Measures and Food Record Data

Few associations were found with stress/coping measures and food record data. The only statistically significant finding was that avoidant coping style scores were found to predict greater fat intake on the day of the experiment. This is consistent with some studies that have identified a relation between avoidant distraction as a coping strategy and emotional eating (Spoor et al., 2007). Furthermore, it is interesting to note that all significant associations between coping and/or stress measures and eating behaviour only occurred during the actual experiment (i.e. CEC items and amount of food consumed during the experiment). It is possible that the influence of stress perception and coping styles on eating behaviour occurs immediately following the stressor and may not be long lasting.

Eating Behaviour Measures and Food Record Data

Restrained eating, as measured by both the Restraint Scale and the DEBQ- restraint subscale, was significantly related to nutritional intake. Three-day average caloric intake was predicted by DEBQ restraint scores, however this was not the case for caloric intake on the day of the experiment. Higher restraint scores (both RS and DEBQ) were associated with lower carbohydrate intake the day of the experiment. As well, higher DEBQ restraint scores were related to lower average protein intake over 3 days. Similarly, greater restrained eating was also associated with lower carbohydrate intake and was found to predict carbohydrate intake over three days. It is not surprising that restrained scores were associated with macronutrient intake as previous studies indicate that dieters consider certain macronutrients, such as carbohydrates, to be forbidden foods (Knight and Boland, 1989).

Cortisol Data

While no group differences in levels of cortisol, AUC, and peak percentage change in response to stress were found; it is consistent with the stress-induced eating behaviour data of this investigation; which also did not reveal any differences between depressed and non-depressed participants. However, correlation analyses revealed associations between peak percentage change and hunger ratings, positive affect, and sensitivity to punishment. A greater peak percentage change in cortisol response was associated with lower hunger scores (CEC Item 1), which is somewhat inconsistent with previous findings that suggest that individuals with a greater cortisol reaction tend to consume more (Oliver, 2000). Also, unexpectedly, higher positive affect scores were found to predict greater peak percentage change in response to stress. Furthermore, higher sensitivity to punishment was associated with lower peak percentage change in response to stress. However, strong caution should be exercised when interpreting these results. A few extreme values within the peak percentage change calculations were found, and therefore may be contributing to these findings. A larger sample size could help confirm these relations with peak percentage change.

Limitations

First it should be noted that depressed participants were not individually matched to control participants. Eligible participants were enrolled in the study with the intention of retrospectively matching controls with depressed participants however, due to time constraints, this did not occur for all participants. Furthermore, since there were a high proportion of restrained eaters within the sample and that restrained eating plays an important role in stress induced eating, perhaps future investigations should control for this variable and match participants according to restrained eating habits.

Second, the results of the experiment should be interpreted with some caution due to the small sample size. Correlation and regression analyses mostly applied to the entire sample and not

specifically to the experimental group. When the group variable was added to the regression model, the significant association tended to disappear; therefore a larger sample size could help clarify experimental group contribution. Furthermore, a larger sample size would allow covariates such as gender, age, depression subtypes to be included in the analyses.

Third, a delay between stress induction and food consumption may also impact the results of the study. The rationale for this delay was two-fold. First, the consumption of food impacts cortisol levels and therefore if participants were allowed to eat it would interfere with the natural stress response (Hansen et al., 2008). Secondly, if participants found comfort in food, as suggested by emotional eating theories, the opportunity to eat may have soothed the participants and again, interfered with the natural stress response curve. In addition, given that depressed individuals tend to ruminate (Nolen-Hoeksema, 1990), it was reasoned that depressed participants would ruminate post-stress and that a delay in measurement of eating behaviour after stress would be appropriate. Furthermore, rumination has been linked with higher levels of cortisol providing further support for this current design. Recent studies have shown that when participants ruminated about a recent painful interpersonal transgression, salivary cortisol levels increased (McCullough et al., 2007).

Another limitation may involve the population of depressed participants studied in this experiment. Differences in HPA activity between acute and chronically depressed individuals have been noted (e.g. Watson, 2006). Since cortisol does impact eating behaviours, logically one may expect differences in eating behaviours of acutely and chronically depressed based on HPA axis activity. Furthermore, studies involving healthy populations indicate that those who demonstrate a high cortisol reaction in response to stress tend to eat more, therefore one may expect the acutely depressed, shown to exhibit hypercortisolemia (e.g. Holsboer, 2001), to do the same. The few studies that have been conducted so far show that individuals with chronic depression may exhibit a blunted response to stress, and therefore this may extend into eating behaviours as well, though we

had predicted the opposite. It may be helpful to include an acutely depressed group for comparison purposes.

Most of the research focusing on stress induced eating and mood do not involve clinically depressed individuals per se but instead use self-report depression and/or mood to document level of negative mood. Others have used euthymic populations and induced negative mood to study its impact on eating behaviours. A major strength of this current investigation is the careful selection of patients with chronic depression and documentation of comorbidities. Furthermore, perception of stress and various coping styles may play a role in stress-induced eating, however, a larger sample size is needed to determine if depressive illness contributes to this relation as well.

Given that a chronic course of depression, as well as weight gain and obesity are associated with poor treatment outcome, it is important to identify potential influencing factors of eating behaviours in depression as it may translate into the management of care for this population.

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Footnote

[1] All simple correlations conducted with quantity of food (grams) consumed during the experiment as the dependent variable were one-tailed. All participants were asked to consume a portion of food during the experiment, and none refused, therefore quantity of food consumption during the experiment could only vary in one direction (i.e. increase from zero).

Table 1 Mean weight and BMI scores for participants

Group	Weight (kg)		BMI	
	Mean	SD	Mean	SD
Overall	75.24	18.87	26.92	6.07
Depressed	73.37	16.88	26.15	5.28
Female	71.61	18.41	26.24	5.96
Male	79.78	8.86	25.18	0.38
Control	77.62	21.75	27.90	7.142
Female	72.50	20.02	26.39	6.39
Male	100.65	15.08	34.71	8.38

Table 2 Mean Cue Elicit Craving Challenge Scores by Group

Cue Elicit Craving (CEC) Items	Depressed		Control	
	Mean	*SD*	*Mean*	*SD*
CEC Total	21.92	4.05	21.20	5.37
CEC Item 1: "How hungry does it make you feel knowing that this (snack) is yours to eat?	6.85	2.15	5.40	2.80
CEC Item 2: "How much would you like to eat some of this snack, even just a small portion?	7.69	2.29	7.50	2.76
CEC Item 3: "Now that you've had a taste of your favourite snack, how strong is your desire to have some more?"	7.38	1.85	8.30	1.42

Table 3 Food Intake the Day of Experiment by Group

Nutritional Composition	Depressed		Control	
	Mean	*SD*	*Mean*	*SD*
Calories (kcal)	1680.01	409.48	1736.54	588.56
Protein (gram)	63.12	30.52	62.01	25.34
Carbohydrates (gram)	227.23	65.78	230.35	85.84
Fat (gram)	56.88	20.32	69.07	37.27

Table 4 Three-Day Average Food Intake by Group

Nutritional Composition	Depressed		Control	
	Mean	*SD*	*Mean*	*SD*
Calories (kcal)	1785.52	415.25	1634.51	354.07
Protein (gram)	68.73	28.72	72.85	13.55
Carbohydrates (gram)	226.49	67.05	195.03	66.53
Fat (gram)	65.32	17.38	66.50	24.98

Figure 1 Mean Cortisol Levels During Stress Induction by Group

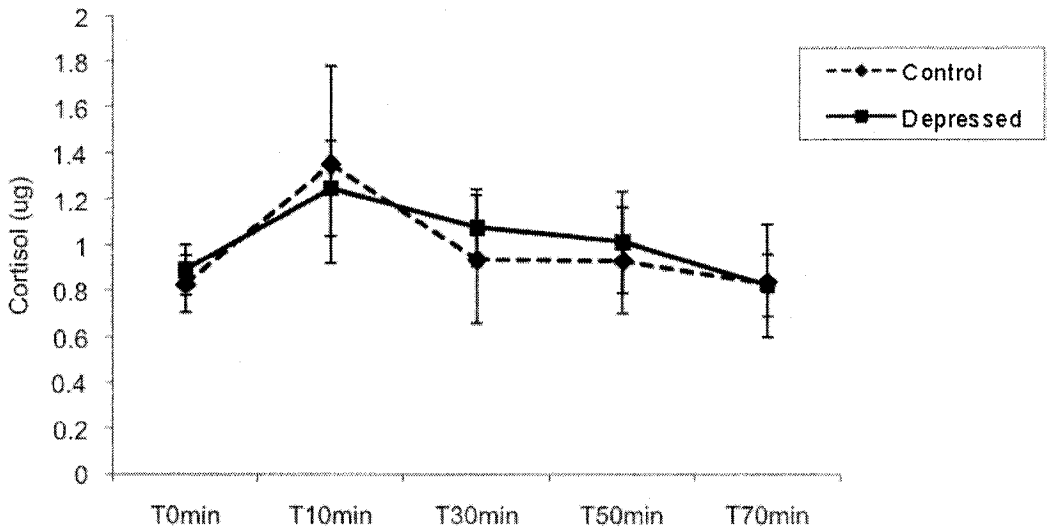

Figure 1 represents the mean cortisol levels obtained at various time points throughout the stress induction procedures for both the depressed and control group. The highest mean cortisol level occurred at T10, which was obtained 10 minutes post-stressor, then decreased throughout the rest period, returning to baseline levels when the final sample was obtained 70 minutes post-stressor (T70). No significant group differences in cortisol were found throughout the stress induction procedures.