Emotion Regulation and Vulnerability to Depression: A Longitudinal Test of the Diathesis-Stress Model

By

Michael Douglas Tooley

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Abstract

Maladaptive emotion regulation is an established vulnerability marker for depression. Within a diathesis-stress framework individual differences in emotion regulation constitute sensitivity to stress, such that people who are less able to effectively regulate their emotions are more likely to become depressed when stress is encountered. Markers of maladaptive emotion regulation have been examined from affective, neurological, and cognitive perspectives and, for the most part, have been examined in independent lines of research. As such, the independent and interactive contributions of maladaptive emotion regulation markers are still unknown. The current thesis addresses this gap with a longitudinal study. Emotion regulation markers and depression were assessed at the outset of the study (time one) then life stress and depression were measured three months (time two) and twelve months (time three) later. Three trait measures of emotion regulation were assessed: spontaneous emotion regulation (as indexed by startle reactivity following negative images), frontal and parietal resting EEG asymmetries, and brooding rumination. All emotion regulation markers were found to be independent markers of vulnerability to depression. The emotion regulation markers measured at time one were then tested within a diathesis stress framework to predict stress sensitivity at time two. Poorer online regulation interacted with life stress to predict depression. That is, poor online regulators were sensitive to stress at three months, whereas good online regulators were not. Stress sensitivity was tested again at time three, twelve months after the initial assessment. At this time point frontal asymmetry, parietal asymmetry and life stress interacted to predict depression. When right parietal activity was low, rightward frontal asymmetries showed more sensitivity to stress. However, people with leftward asymmetries showed less stress sensitivity. Brooding predicted depression at three

months but not at twelve months and did not interact with stress at either time point. The findings of this thesis show that, within the diathesis-stress framework, online regulation measures indicate short-term sensitivity to stress; however, EEG asymmetry measures show sensitivity to stress in the longer term.

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Chapter One

Emotion Regulation and Vulnerability to Depression

Depression is a common mental health disorder with broadly reaching individual and societal consequences. The distress experienced by people with depression affects many facets of their personal and professional lives - placing strain on relationships and reducing productivity and ability to perform at work (Stewart, Ricci, Chee, Hahn & Morganstein, 2003). According to the World Health Organization, depression is the leading cause of disability in terms of years lost to the disorder (World Health Organization, 2008). Prevalence rates indicate that approximately 16-19% of people will become depressed at some point in their lives (Bromet, Andrade, Hawang, Sampson, Alonso, de Girolamo et al., 2011; Kessler, Berglund, Chiu, Demler, Heringa, Hiripr et al., 2004; Oakley-Brown, 2006) and show that depression affects almost twice as many women as men. (Kessler, McGonagle, Swartz, Blazer & Nelson, 1993, Nolen-Hoeksema, 2001; Patten, Wang, Williams, Currie, Beck, Maxwell, & el-Guebaly, 2006). These epidemiological data highlight the importance of research aimed at understanding the causes of depression.

Factors that lead to *first* episodes of depression are pivotal to understanding this disorder. Approximately half of those who experience a first episode of depression will have another, and for many these episodes recur throughout their lives (Klein & Allmann, 2014; Mattisson, Bogren, Horstmann, Munk-Jörgensen & Nettelbladt, 2007; Moffitt, Caspi, Taylor, Kokaua, Milne, Polanczyk, & Poulton, 2010). As the number of episodes mount, the typical duration of each episode increases and the time between episodes shortens (Bolland & Keller, 2002). The triggers for depressive episodes also change; first episodes of depression are likely to be precipitated by a strong stressor, but the stress required to trigger depression decreases with each

episode (Lewinsohn, Allen, Seeley, & Gotlib, 1999; Post, 1992). Put simply, a first episode of depression drastically reduces the stress threshold needed to trigger future episodes. A major task for psychological researchers is therefore to identify factors contributing to the critical first episode.

Stress and Depression

Stressful events have a well-established role in the onset of first episodes of depression. Evidence for a stress-depression relationship has been observed using a variety of stress measures, including: daily hassles (e.g., see Lazarus, DeLongis, Folkman, & Gruen, 1985); major life events (e.g., see Monroe, Slavich & Georgiades, 2014); and chronic stress (e.g., see Klein & Allmann, 2014). However, while the stress-depression relationship is robust, the relationship is also complex and dynamic (see Monroe & Simons, 1991). Of particular importance, the experience of stress is not necessarily followed by depression (e.g. see Hammen, 2006; Monroe & Reid, 2009). Many individuals are resilient to stress, meaning they can experience high levels of stress without experiencing depression (Bonanno, 2004; Coifman & Bonanno, 2010; Hammen, 2005), whilst other individuals are sensitive to stress, and go on to experience depression after a stressful episode (Hammen, 2015). Individual stress sensitivity across the population can be conceptualised as a continuum, ranging from highly sensitive to resilient (Willner, Scheel-Krüger, & Belzung, 2013). In order to understand first depression episodes, it is necessary to determine which individual characteristics interact with stress to constitute vulnerability to depression.

The Stress Response

In order to examine factors that may interact with stress to lead to depression, it is important to understand what the stress-response is in itself. The stress response consists of a number of diverse and highly coordinated processes that are executed

across multiple levels of psychobiological functioning (McEwen, 2007). Instigating the stress response is essential under conditions of acute threat, where it drives changes in both central and periperal nervous systems to produce adaptive responses and aid survival (Theil & Dretsch, 2011). Under conditions of stress, individuals instigate automatic and emotionally motivated response tendencies that bias attention toward threating stimuli, and initiate defensive emotional responses. However, the stress response comes with physiological and psychological costs whereby, under conditions of prolonged or chronic stress, the load of maintaining the stress response begins to exhaust available resources (Arnsten, 2009; McEwen, 2007).

The brain is central to the stress response. It determines what is deemed a stressor and how an individual will respond to it. At the same time, the brain itself undergoes systemic and functional changes when stress is experienced (McEwen, 2006). The influence of stress on the central nervous system has been proposed to be a key factor in the development of depression (see Willner et al., 2013). In particular, neurological changes as a result of stress affect how emotion is processed and regulated by the brain. Prefrontal cortical regions are heavily responsible for the regulation of attention (important for identifying threatening stimuli) and emotional responses, via connections to parietal and subcortical regions respectively (Lee, Heller, van Reekum, Nelson, & Davidson, 2012; Ochsner, Ray, Cooper, Robertson, Chopra, Gabrieli, & Gross, 2004). Arnsten (2009) proposes that acute stress shifts neural processing transiently from slower more controlled processing driven by the prefrontal cortex, to rapid and reflexive processing driven by subcortical regions (e.g., the amygdala). However, chronic stress may have prolonged effects on the prefrontal cortex, resulting in long-term influences on its function that result in a focus on negative information and negative affect, and may ultimately lead to depression.

Diathesis-Stress Model

Of particular interest in the development of depression are the effects of life stress (e.g. Post, 1992), that is, the stress experienced as individuals engage in their day-to-day lives. Life stressors can range from psychosocial stressors such as the loss of an interpersonal relationship (e.g., the death of a parent or the break up of a romantic relationship), through to changes in life circumstances, such as unemployment. The diathesis-stress model considers the role of individual differences in response to stress in predicting depressive outcomes. It proposes that pre-existing traits (diatheses) make an individual more (or less) susceptible to the deleterious effects of stress (Monroe & Simons, 1991; Willner et al., 2013). In other words, diatheses moderate the stress–depression relationship, meaning some individuals are more vulnerable to depression in response to stress than others.

A wide range of factors can constitute a vulnerability within the diathesis-stress model. Research from different facets of psychology has explored possible vulnerability traits ranging from genetic (e.g. Caspi, Sugden, Moffit, Taylor, Craig, Harrington et al., 2003;), to neurological (e.g. Henriques & Davidson, 1991), to cognitive (e.g Alloy, Abramson, Whitehouse, Hogan, Panzarella, & Rose, 2006), and to interpersonal (e.g. Shahar, Joiner, Zuroff, & Blatt, 2004) factors. In this thesis, I focus on trait measures of *emotion regulation* as potential diatheses. Specifically, I will examine the moderating effects of patterns of frontal and parietal electroencephalogram (EEG) activity, spontaneous emotion regulation ability, and brooding rumination within the diathesis-stress model.

Emotion Regulation and Vulnerability to Depression

Emotion regulation is a good candidate diathesis within the diathesis-stress model. It is an important factor in determining how individuals adjust to stress (Bonanno & Burton, 2013) and atypical emotion regulation is common across psychopathologies, particularly mood disorders (Aldao, Nolen-Hoeksema & Schweizer, 2010; Gross & Munõz, 1995; Nolen-Heoksema, Wisco, & Lyubomirsky, 2008; Rottenberg, Gross, & Gotlib, 2005). Emotion regulation is the altering of the quality, intensity, duration, or type of an emotional response (Gross & Thomson, 2007; Gross, 2013) and is conceptualised as a distinct set of processes from the emotional response itself (see Koole, 2009, but see Gross & Feldman Barrett, 2011).

Emotional responses consist of three core dimensions: subjective experience, physiological responses, and behavioral responses. Emotional responses play an important role in ensuring an individual can adapt appropriately and flexibly. When an emotional response is triggered, motivation systems are activated to guide behavioural responses, and these depend on the valence of the situation. For example, in a positive situation (such as finding a food source) an individual might be guided to approach the situation. However, in a negative situation (such as the appearance of a predator) it may be more adaptive to be motivated to withdraw from the situation. The motivational priming hypothesis (Lang, 1995) argues that the neurobiological underpinnings of emotion can motivate behavior through two core systems: (1) a defensive system that motivates protective behavioral and physiological responses (e.g., withdrawal) in order to avoid harm or overcome threat, and is typically associated with negative subjective feelings (e.g., fear or disgust); and (2) an appetitive system that motivates life sustaining and reproductive (e.g., approach) related behaviors and physiological responses in order to benefit from potentially

advantageous situations, and is often associated with positive subjective feelings (e.g., enthusiasm or sexual arousal).

The multi-process model (Bradley, Codispoti, & Lang, 2006; Bradley & Lang, 2007) proposes that motivational priming (and associated emotional response) occurs across a series of distinct stages. First, emotional challenge is followed by an early orienting response, where salient perceptual features of potentially relevant emotional stimuli (high arousal stimuli, regardless of valence) capture perceptual and attentional resources, enabling further assessment of the emotional significance of the stimulus (see Bradley, Keil & Lang, 2012). During the early orienting stage, autonomic responses are also activated in preparation for potential action (Bradley, 2009; Bradley et al., 2012). If the stimulus is deemed relevant, the emotional response progresses to the next stage; alternatively if the stimulus is not deemed relevant the response can be regulated (i.e., stopped).

In the second stage of the emotional response the relevant motivational priming system (i.e., the defensive or appetitive system) is activated. The appetitive system is engaged in response to positively valenced stimuli and the defensive system is engaged in response to negatively valenced stimuli. (Bradley, Codispoti, Cuthbert, & Lang, 2001). At this stage, the emotional response may be regulated if no further processing or behavioural response is deemed necessary. However, if further behavioural response is required (i.e., approach or withdrawal), then physiological (e.g., increased heart rate) and subjective (e.g., feeling scared) changes occur in order to support the appropriate behavioural response (Bradley, Moulder, & Lang, 2005). Of particular importance, if a significant threat is detected, a stress response is activated in order to help the organism cope with the threat (Thiel & Dretsch, 2011).

The regulation of emotion can occur at any stage of the response. Emotion regulation processes vary along a continuum from highly effortful, intentional, attempts to alter the response, through to automatic, implicit, regulation processes that occur without intention (Berkman & Leibermann, 2009; Gyurak, Gross & Etkin, 2011; Mauss, Bunge, & Gross, 2007). Multiple strategies may be drawn upon to regulate a specific emotional response (Aldao & Nolen-Hoeksema, 2013) and strategies can vary depending on the environmental context (Aldao, 2013). Emotion regulation strategies differ in their effectiveness (Gross, 2013). Additionally, the regulation strategies that an individual tends to use habitually can vary between people (Aldao, Nolen-Heoksema, & Schweizer, 2010; Berkman & Leiberman, 2009). Developing adaptive emotion regulation abilities is considered a key achievement of developmental maturation (Diamond & Aspinwell, 2003).

Depression is characterised by atypical emotion regulation (Aldao, Nolen-Hoeksema, & Schweizer, 2010). The adaptive and flexible use of emotion regulation is an important buffer against stress (see Troy & Mauss, 2011), as has been shown in both cross-sectional (Moore et al., 2008; Troy, Shallcross & Mauss, 2013; Troy et al., 2010) and prospective designs (Bonanno, Papa, Lalande, Westphal & Coifman, 2004; Pakenham, 2005; Shallcross, Troy, Bolland & Mauss, 2010; Van der Veek, Kraaij & Garnefski, 2009; Vanderhasselt, Koster, Onraedt, Bruyneel, Goubert, De Raedt, 2014). Clinically depressed patients rated by a clinical interviewer as more skilled emotion regulators (assessed using the Operationalized Skills Assessment Inventory; Stenzel et al., 2010) show more symptom improvement at discharge than poorer emotion regulators (Fehlinger, Stumpenhorst, Stenzel & Rief, 2013). However, a distinction should be made between emotion regulation *capability* and emotion regulation *tendency*. Emotion regulation *capability* refers to an individual's ability to

engage in a regulatory process, while emotion regulation *tendency* refers to the strategies that a person typically or habitually uses regardless of ability (see Berkman & Leiberman, 2009). For example, an individual may be capable of using a particular emotion regulation strategy (e.g. cognitive reappraisal) when asked to do so, but may not employ this strategy habitually when they encounter emotional events in their day to day lives (e.g. Suri, Whittaker & Gross, 2014). This distinction is important as depressed people have a tendency to draw upon less effective strategies, when attempting to alleviate negative mood (Garnefski & Kraaij, 2006; Joormann & Gotlib, 2010).

Individual differences in emotion regulation and responses to stress are of particular interest in studies of depresssion. It has been proposed that it is the dysregulation of the stress response and associated negative emotions, as opposed to the stress response itself, that underlies depression (Flynn & Rudolph, 2007). Stressful events are by their nature emotional events (Sarason, Johnson, & Siegel, 1978), and physiological responses driven by the stress response are often percieved as negative emotional experiences (Thiel & Dretch, 2011). Therefore, there is good reason to consider individual differences in emotion regulation tendency when examining vulnerability to depression within the diathesis-stress framework. In this thesis I consider three markers of emotion regulation tendency: neurological traits as revealed in resting measures of cortical activity; online emotional responding and regulation as indicated in the startle eye-blink paradigm, and cognitive coping strategies as assessed through self-report of ruminative tendencies.

Measures of Trait Emotion Regulation

Regional electroencephalographic asymmetries. Prefrontal cortical function and activity within a frontal-parietal network have been strongly linked to emotion

regulation (Ochsner, Silvers, & Buhle, 2012; Lee et al., 2012), and atypical patterns of functioning across these regions has been associated with mood disorders (Johnstone, Van Reekum, Urry, Kalin, & Davidson, 2007; Willner et al., 2013). As such, measures of brain activity may provide a good index of neurological vulnerability to disorders involving poor emotion regulation (Davidson, 2004) In this thesis, I specifically focus on hemispheric differences in resting brain activity as a possible diathesis.

Hemispheric asymmetries within the alpha band (8-13Hz), as measured by electroencephalographic (EEG) activity, are of interest in the study of emotion, emotion regulation, and depression (Coan & Allen, 2004; Thibodeau, Jorgenson, & Kim, 2006). Alpha power is typically interpreted as the inverse of cognitive activity (Coan & Allen, 2004; Klimesch, 1999). Comparing alpha over a region of one hemisphere relative to the equivalent region of the opposite hemisphere provides a relative measure of left versus right activity (Allen, Coan & Nazarian, 2004; Coan & Allen, 2004). Of particular relevance to the study of depression and emotion regulation has been measurement of asymmetric activity while participants are at rest over frontal cortical and, to a lesser extent, parietal cortical areas (see Davidson, 2004; Harmon-Jones et al., 2010;). Resting frontal asymmetry has been shown to be a reliable (Hagemann, 2004) and relatively stable trait (Allen et al., 2004). Asymmetric frontal alpha appears to be generated in the dorsolateral prefrontal cortex (dlPFC; Pizzagalli, Sherwood, Henriques, & Davidson, 2005), and may reflect activity in a frontal-parietal network (see Laufs et al., 2003; Mantini, Perrucci, Dal Gratta, Romani & Corbetta, 2007) that is engaged in tasks requiring executive control and regulation of emotion (Ochsner et al., 2012).

Three interpretations have provided potential explanations of the relationship between frontal asymmetry and emotion; a valence based interpretation, a motivation based interpretation and a cognitive control based interpretation. The valence interpretation argues that leftward frontal asymmetry is associated with positive emotionality and rightward frontal asymmetry with negative emotionality (e.g. Heller, Nitschke, & Miller, 1998). Evidence for this interpretation came from findings that depression (Henriques & Davidson, 1991), remitted depression (Henriques & Davidson, 1990), and trait negative affect measures were related to rightward asymmetry and positive trait affect measures to leftward asymmetry (Tomarken et al., 1992; Tomarken & Davidson, 1994). However, this interpretation may have failed to capture the full extent of the relationship between frontal asymmetry and emotion.

The motivational direction hypothesis has gained support (see Harmon-Jones, 2010). The motivation hypothesis argues that frontal asymmetries are better interpreted in terms of motivation to approach or withdrawal from a situation. According to this interpretation leftward asymmetries are associated with approach motivation and rightward with withdrawal motivation, regardless of valence (e.g. Harmon-Jones & Allen, 1997; Sutton & Davidson, 2010). Often positive emotions are associated with approach (e.g. joy) and negative emotions with withdrawal (e.g. fear; Lang, 1995). However, Carver and Harmon-Jones (2009) point out that an approach motivation can also be negative, such as when anger (a negative emotion) provides motivation to approach a threat (e.g. to defend of one's position). In support of this hypothesis subjective measures of trait anger have been associated with more leftward frontal asymmetries (Harmon-Jones & Allen, 1998). Moreover, this relationship is independent of attitudes to anger (i.e. trait anger is not related to positive attitudes towards anger), thus the association between trait anger and leftward asymmetry is

not due to anger being experienced as a positive emotion (Harmon-Jones, 2004). As anger is a negative emotion and also motivates approach behaviours, findings that anger is associated with leftward frontal asymmetry supports the motivation hypothesis (Harmon-Jones et al., 2010).

The asymmetric inhibition model provides a third explanation of the role frontal asymmetry in emotion processes, with specific regard to emotion regulation (Grimshaw & Carmel, 2014). This model proposes that frontal asymmetry reflects an individual's ability to recruit executive control processes that govern attention systems. Bottom up processes draw attentional resources toward emotional information, due to the adaptive value of such information. However, current goals (and well being) often benefit from inhibition of such emotional distraction. Executive control processes provide top down control over attentional systems, regulating non-beneficial emotional responses and maintaining attention on the task at hand. Relative leftward frontal asymmetries are proposed to reflect an individual's ability to successfully control negative emotional information. On the other hand, relative rightward frontal asymmetries are proposed to reflect ability to control positive emotional information. Therefore, relative leftward frontal asymmetry reflects better emotion regulation ability of negative information and relative rightward frontal asymmetry poorer emotion regulation of negative information.

Relatively rightward frontal EEG asymmetries are associated with current (Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1991) and remitted (Gotlib et al., 1998; Henriques & Davidson, 1990; Stewart, Coan, Towers & Allen, 2011) depression, and with familial (Field & Diego, 2008) and genetic (Bismark et al., 2010; Feng et al., 2012; Smit, Posthuma, Boomsma, & De Geus, 2007) risk of the disorder. Most relevant to the current study, relative rightward frontal asymmetries

have been shown to predict the onset of depression over time (Mitchell & Possell, 2012; Nusslock et al., 2011; Pössel, Lo, Fritz, & Seemann, 2008). In other words, relative rightward frontal asymmetry precedes depression and is therefore not simply a marker of depression itself, but rather reflects a vulnerability to depression. In a cross-sectional study frontal asymmetry scores, measured while watching an emotional film, interacted with stress to predict depressive symptoms. Children at high risk of depression who had relative rightward frontal asymmetries showed more sensitivity to stress than those with relative leftward frontal asymmetries (Lopez-Durren, Nusslock, George & Kovacs, 2011). These studies indicate a direct predictive relationship between rightward frontal asymmetry and depression. However, no studies have prospectively tested whether resting frontal asymmetries act as a diathesis within the diathesis-stress model. If so, one might expect a stronger relationship between stress and depression in vulnerable people (with relative rightward frontal asymmetry) than in those who are more resilient (with relative leftward frontal asymmetry).

Parietal asymmetries have received less attention than frontal asymmetries but are also associated with depression. It has been argued that it is specifically right parietal cortical function that is impaired in depression, reflecting deficiencies in the processing of emotional information associated with reduced arousal (e.g., Bruder, 2003; Heller, 1993; Heller & Nitschke, 1997; Moratti, Rubio, Campo, Keil, & Ortiz, 2008). Therefore, when interpreting parietal asymmetries in this thesis, I discuss findings in terms of relative high or low right parietal activity (rather than relative leftward or rightward asymmetry scores as is conventional when discussing frontal asymmetries). Relatively low right parietal activity has been associated with current depression (Bruder, Fong, Tenke, Leite, Towey, Stewart et al., 1997; Kentgen, Tenke,

Pine, Fong, Klein, & Bruder, 2000), previous depression (Stewart et al., 2011), higher depression scores in a non-clinical sample (Blackhart, Minnix & Kline, 2006), and familial risk for depression (Bruder et al., 2012; Bruder, Tenke, Warner, & Weissman, 2007; Henriques and Davidson 1990). Additionally, in a longitudinal design, low right parietal activity measured at age six was found to predict established cognitive vulnerabilities to depression at age seven (Hayden, Shankman, Oliion, Durbin, Tenke, Bruder, & Klein, 2008).

While the majority of depression research has examined frontal and parietal asymmetries separately, the interaction between these two regions may provide a more comprehensive account of psychopathology (Heller, 1993; Heller & Nitschke, 1997). Similar to previous models, the Circumplex model argues that frontal asymmetry reflects the valence component of emotional experience, with leftward asymmetries reflecting positive experience, and rightward asymmetries reflecting negative experience. However, additionally the Circumplex model proposes that parietal asymmetry indexes the arousal component of emotional experience, with rightward parietal asymmetry (that is, high right parietal activity) indexing high arousal, and leftward asymmetry (that is, low right parietal activity) reflecting low arousal. Importantly, it is the interaction between the valence (frontal) and arousal (parietal) systems that reflects mood disorders. Depression is characterised by a pattern of negative valence and low arousal, therefore the neural correlates reflecting this pattern should be rightward frontal asymmetry and low right parietal activity (leftward parietal asymmetry).

The Circumplex model is useful as it predicts dissociated patterns of neural correlates for depression and anxiety. Anxiety is characterised by feelings of worry and feeling tense (Heller & Nitschke, 1998). Anxiety and depression are both

associated with negative emotional responses and are thus both reflected by rightward frontal asymmetries. However, anxiety is associated with high arousal emotional responses whereas depression is associated with low arousal emotional responses. Therefore, dissociable patterns of right parietal activity should distinguish these disorders, such that anxious individuals have high right parietal activity (high arousal) and depressed individuals have low right parietal activity (low arousal; Heller, 1993; Heller & Nitschke, 1997). However, it is unknown whether these characteristic patterns of neural activity exist before psychopathology is experienced, and if they do whether they are a marker of stress sensitivity. As such, the present thesis investigated the frontal asymmetry by parietal asymmetry interaction as a predictor of depression, both directly and within the diathesis-stress model. Vulnerability to depression may be characterised by a combination of rightward frontal asymmetry and low right parietal activity.

Spontaneous emotion regulation. A core aspect of depression is the failure to habitually and effectively regulate emotions as they occur and are experienced (Jazaieri, Urry, & Gross, 2013). Failing to effectively engage regulatory processes when an emotional response is experienced leads to prolonged responses, and may constitute a vulnerability to depression (Ehring, Tuschen-Caffier, Schnülle, Fischer, & Gross, 2010). To date most research examining emotion regulation in experimental contexts has assessed instructed regulation, that is, the ability to use a particular strategy when asked to do so. Most studies of this type have assessed ability to use cognitive reappraisal (the reframing of an emotional experience to be more positive or negative; see Aldao et al., 2010). The use of instructed reappraisal maintains good experimental control. However, despite evidence that cognitive reappraisal is a beneficial strategy (e.g. Troy, Wilhelm, Shallcross, & Mauss, 2010), it is not as

commonly drawn upon in real world contexts as was previously assumed (Suri et al., 2014). This may be because cognitive reappraisal ability is impaired under stress (Raio et al., 2013). Additionally, cognitive reappraisal is one of many possible regulation strategies, and individuals are likely to draw upon a variety of techniques, or use multiple techniques in conjunction, to regulate emotion in their day-to-day lives (Heiy & Chavens, 2014). Evidence that dysphoric (Quigley & Dobson, 2013) and previously depressed (Ehring et al., 2010) individuals are as capable as healthy controls of reappraisal when instructed, despite little tendency to do so spontaneously, suggests that instructed reappraisal may not be the best process to target when investigating emotion regulation as a vulnerability marker of depression. The distinction between regulation capability and tendency (see Berkman & Leiberman, 2009) highlights the utility of examining spontaneous regulation of responses to emotional challenge - that is the outcome of participant's own habitual, undirected, regulation attempts - as a measure of the use of regulation strategies in a flexible and adaptive way.

Objective measures of spontaneous regulation can be obtained in laboratory based experimental paradigms by presenting emotional challenges (e.g. emotional images) and using objective measures (e.g. psychophysiological responses) to track emotional reactivity both during and after the emotional challenge. By letting participants respond and regulate of their own volition (i.e., not requesting that particular strategies or approaches be employed) an objective measure of their tendency to habitually regulate emotions can be obtained (Davidson, 1998). Psychophysiological indicators of emotional response and spontaneous regulation are particularly useful as they are able to track the dynamics of emotional reactivity across time without requiring introspective subjective reports.

One method for measuring both emotional responses and spontaneous emotion regulation is the startle eye-blink reflex (e.g Dillion & LaBar, 2005; Driscoll, Tranel, & Anderson, 2009; Jackson, Malmstadt, Larson, & Davidson, 2000). The startle eye-blink is an innate behavioural response to a sudden and intense sensory stimulus, such as a loud noise. It is a component of an organism-wide startle reflex, which is automatic and adaptive (it protects the eyes from damage). It can be measured non-invasively by placing electrodes on the skin over the orbicularis oculi muscle, which is responsible for reflexive closure of the eyelid (Blumenthal et al., 2005). Importantly, when the parameters of the stimulus eliciting the startle (e.g. sound intensity) are held constant, blink magnitudes can be modulated by the emotional valence of an additional stimulus (e.g., an image). While it is the intense sensory stimulus that causes the blink, it is the nature of the additional foreground stimulus that modulates the magnitude of the blink. Relative to neutral stimuli, concurrent processing of unpleasant stimuli leads to larger blink magnitudes, and processing of pleasant stimuli leads to smaller blink magnitudes (Bradley, Cuthbert & Lang, 1999).

In a typical affective startle paradigm, brief (50ms), intense bursts of white-noise (~90-110dB) are used to initiate the startle eye-blink response. Stationary emotional images are frequently used to modulate the startle response. Emotional images (e.g., an attacking dog) present an emotional challenge to an individual, and are useful as they allow experimenters to have control over the stimuli, particularly in terms of content and duration of exposure. White-noise bursts are presented to *probe* the emotional response while participants view an emotional stimulus (see Bradley et al., 1999). Electromyography (EMG) electrodes are positioned over the orbicularis oculi muscles so that electrical activity, produced by action potentials within the muscle, can be recorded. The magnitude of this electrical activity reflects the response

to the noise probe, modulated (either up or down) by the emotional reactivity the participant is experiencing at the time of the probe.

Distinct patterns of startle eye-blink responses are observed depending on *when* (relative to the emotional stimulus onset) the noise probe is presented and the *arousal* and *valence* properties of the foreground image (Bradley, Codispoti & Lang, 2006; Dicther, Tomarken, & Baucom, 2002). These patterns of responding are shown in Figure 1.1. In a typical startle eye-blink paradigm participants passively view images. Patterns of emotional response across time can then be obtained by presenting the startle probe at different time points after the onset of the image. Image-probe latencies are divided into three distinct response periods to indicate emotional responses across time; early, late, and post-image startle response periods.

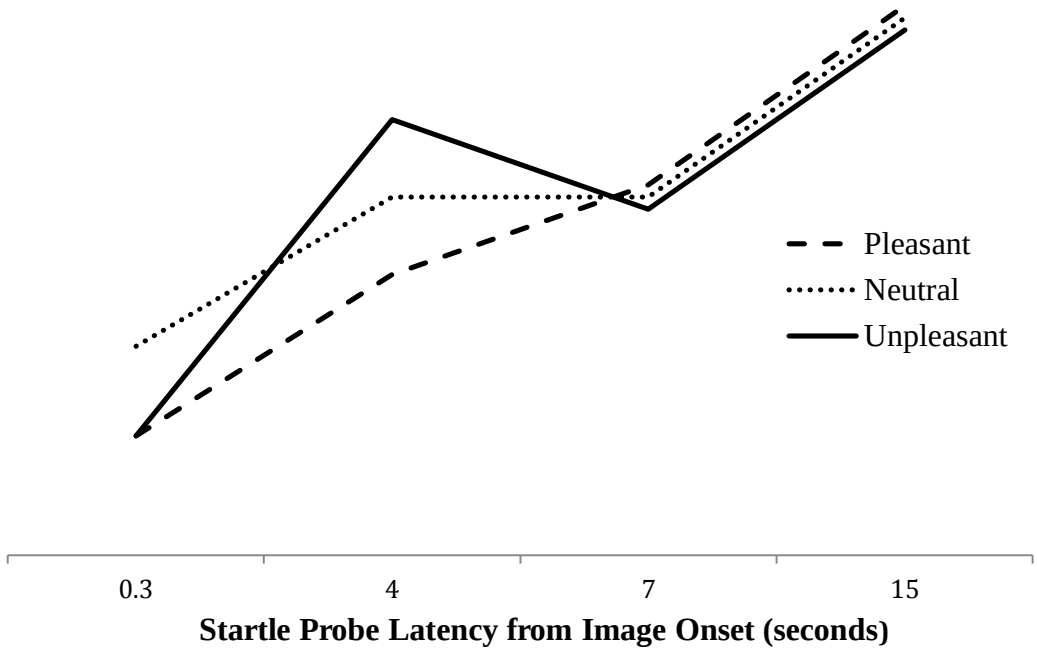

Figure 1.1 Idealised depiction of the time course of startle responses to emotional images. The typical pattern of startle responses to noise probes are represented at four latencies from onset of an emotional image (image presented from 0 to 6 seconds). The 0.3s probe depicts the orienting response and the 4s probe depicts modulation of the startle response by emotional valence. The probe at 7s (1s post-image) shows dissipation of emotional reactivity and at 15s (baseline probe) shows responding in the absence of any foreground stimuli.

The early response period is maximal approximately 300 milliseconds after stimulus onset and is considered to reflect the allocation of attentional resources to high arousal images, regardless of valence (Bradley et al., 2012). Probes presented 300ms from image onset occur during early processing of the image and produce attenuation of startle blinks (a reduction in magnitude) for high arousal (positive or negative) images relative to low arousal images (Bradley, Cuthbert, & Lang, 1993). During this early phase of processing, high arousal information is detected as potentially important and attentional resources are recruited to determine the stimuli's relevance. Responses to startle probes are attenuated for high arousal images due to the gating of sensory inputs, thus protecting attentional processing of the important emotional stimulus (see Filion, Dawson, & Schell, 1998). High arousal images produce more attenuation as they inherently contain biologically relevant information and thus engage more attentional resources, leaving fewer resources to process the startle probe. This early probe time reflects the arousal-sensitive orienting stage of the emotional response, as outlined in the multi-process model (see Bradley et al., 2012).

The late response period typically begins 1.5 seconds after stimulus onset, peaking between 3 and 4 seconds from onset, and may extend until image removal (typically 5 to 8 seconds). Responses during this period are modulated by the valence of the image and reflect activation of defensive and appetitive motivational systems (Bradley et al., 2006). Relative to neutral stimuli, blink magnitudes are attenuated by pleasant stimuli and potentiated by unpleasant stimuli (Bradley, Cutherbert, & Lang, 1999). The magnitude of these effects increases as the arousal of stimuli increases (Bradley et al., 1999; Lang, Bradley, & Cuthbert, 1990). Startle modulation at this time point is highly robust (Lang et al., 1990), and is thus used as a reliable measure

of valanced emotional responses during the motivational priming stage of responding (as outlined by the multi-process model).

The post-image startle response is measured during the period directly after an image has been removed and reflects ongoing emotional processing related to the removed stimulus. Therefore this probe point reflects whether a response has been regulated (i.e., blink magnitude does not differ by the valence of previous foreground stimulus) or is ongoing (i.e., blink magnitude is still modulated by previous foreground stimulus). This time point is used to measure emotion regulation (e.g. Jackson et al., 2003) and when no instruction to regulate is given reflects spontaneous emotion regulation. Post-image reactivity has been measured anywhere from 300ms to 6 seconds after stimulus removal (e.g. Bradley et al., 1993; Bradley et al., 2006; Ditcher, Tomarken & Baucom, 2002; Jackson, Muller, Dolski, Dalton, Nitschke, Urry et al., 2003; Larson, Nitschke, & Davidson, 2007; Larson, Ruffalo, Nietert, & Davidson, 2005; Larson, Taubitz & Robinson, 2010; Taubitz, Robinson & Larson, 2013). As images used in laboratory experiments are relatively low intensity emotional stimuli (compared to emotional stimuli encountered in the real world) the elicited emotion is relatively transient, and reactivity rapidly degrades after images are removed. For example, emotional responses to an actual threatening stimulus (e.g., coming across a snake in the wild) may be expected to go on for much longer periods of time than for images, and require more intensive regulation (Bradley et al., 1999). Therefore, post-image probes are often presented soon after image removal.

Individuals differ in the rate at which emotion is regulated and the degree to which they successfully regulate emotion (Gross, 2013). The regulation of emotion can be tapped using the startle eye-blink reflex by indexing the amount of emotional reactivity present after an emotional stimulus is removed relative to reactivity after a

neutral stimulus is removed. During the post-image period (e.g., one second post offset) more reactivity indicates less emotion regulation and less reactivity indicates more regulation. When compared to responses following a neutral stimulus, better emotion regulators would be expected to show *less* startle potentiation following the removal of a negative emotional stimulus (indicating reduced activation of defensive motivational systems) and poorer emotion regulators would be expected to show *more* potentiation (indicating ongoing activation of defensive motivation systems; e.g. Dichter et al., 2002; Jackson et al., 2003; Larson et al., 2010; Larson et al., 2007; Taubitz et al., 2013). Thus, probes at this post-image point are considered to be sensitive to individual differences in ability to regulate emotional responses.

The startle eye-blink has been used to explore the relationship between emotional reactivity (both response during image presentation and regulation after image offset) and depression. The typical arousal-based pattern of response has been reported at the early response phase for depressed individuals (Dichter et al., 2004), indicated that they show normal attention to emotional images. However, during the late response period clinically depressed individuals consistently show no startle modulation by emotional valence, as seen in controls (Allen, Trinder, & Brennan, 1999; Dichter & Tomarken, 2008; Dichter, Tomarken, Shelton & Sutton, 2004; Kavani, Gray, Checkley, Raven, Wilson, & Kumari, 2004). No studies have reported post-image startle reactivity for a clinically depressed population. Subclinical populations have also been assessed using the startle eye-blink paradigm but with mixed findings. For example, at the late emotion response period subclinical individuals (who score high on scales of depressive symptoms), have been found to show patterns of typical modulation (Larson et al., 2007), reduced modulation (Taubitz et al., 2013), and no modulation (Mneimne, McDeremut, & Powers, 2008)

by emotion valence. Post-image probes have also shown mixed findings for subclinical individuals. One study found they did not differ from controls (Larson et al., 2007) and another found sustained potentiation following unpleasant images for those with higher depressive symptoms (Taubitz et al., 2013).

Particularly relevant to the current study, markers of vulnerability to depression (as opposed to current depression) have been related to post-image startle responses (Jackson et al., 2003; Larson et al., 2010). Both of these studies show typical emotion modulation by valence at the late probe but at the post-image probe have shown that individuals possessing a neurobiological marker (rightward frontal asymmetry; Jackson et al., 2003) or a genetic marker (the G-allele of the monoamine oxidase A gene; Larson et al., 2010) of depression show sustained potentiation following unpleasant images, indicating poorer spontaneous emotion regulation for those more vulnerable to depression. No studies have examined the relationship between vulnerability and the early response probe. Additionally, no prospective research has examined a startle eye-blink index of spontaneous emotion regulation, either as a direct predictor of depression, or as a moderator of the stress-depression relationship.

Brooding rumination. Rumination is a cognitive response style to stress that involves the tendency to repeatedly focus on and mull over the negative aspects of a stressful situation or sequence of events (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). People who ruminate typically employ this strategy as an attempt to cope with negative emotional responses (Papageorgiou & Wells, 2003). However, rumination may be a maladaptive emotion regulation strategy that actually increases negative mood and disrupts potentially beneficial regulation strategies (Lyubomirsky & Tkach, 2004; Spendelow & Jose, 2012; Ward, Lyubomirsky, Sousa, & Nolen-Hoeksema, 2003). Trait rumination is typically assessed using self-report measures, and it has

been shown to be related to poorer outcomes in multiple aspects of individuals' lives (Nolen-Hoeksema et al., 2008; Siegle, Moore & Thase 2004), including less adaptive responses to stress (Nolen-Hoeksema et al., 2008), and a more chronic course of depression (Nolen-Hoeksema & Murrow, 1991).

Rumination is a robust predictor of depression (Burwell & Shirk, 2007; Ciesla & Roberts, 2007; Mezulis, Simonson, McCauley, & Vender Stoep, 2011; Treynor, Gonzalez, & Nolen-Hoeksema, 2003) and as a trait it remains relatively stable across time despite changes in depressive symptoms (Bagby, Rector, Bacchiochi, & McBride, 2004). This level of stability indicates that it is an emotion regulation trait rather than symptomatic of depression itself. Longitudinal studies have shown ruminative response styles to be both a mediator (Jose & Brown, 2008; Michl McLaughlin, Shepard, & Nolen-Hoeksema, 2013) and moderator (Abela & Hankin, 2011) of the stress depression relationship.

A common measure of rumination, the Ruminative Response Scale (Treynor et al., 2003) has been found to tap three types of rumination: depression related rumination, which was found to be highly confounded with depression; reflective rumination, which is associated with adaptive and helpful cognitive thoughts about stressful events; and brooding rumination, which is the problematic aspect of rumination. Brooding is the tendency to dwell on the self-referential negative consequences of a situation (e.g., "why me" type thoughts), while reflection is the tendency to consider and understand a situation (Treynor et al., 2003). Brooding and reflection both show a positive relationship with depression (Joormann, Dkane, & Gotlib, 2006; Treynor et al., 2003). However, a longitudinal examination has shown brooding to correlate positively with future depressive symptoms and reflecting to correlate negatively with future depression symptoms (Treynor et al., 2003).

Therefore, while reflection may relate to distress in the short term it may also aid successful problem solving in the long term. On the other hand, brooding does not appear to be related to successful problem solving (Treynor et al., 2003). Brooding rumination has also been associated with an attentional bias toward negative information, while controlling for depression (Joormann et al., 2006), indicating that brooding reflects a tendency to focus on negative events independently of the influence of depression. Brooding may also act as a diathesis, as it has been shown to moderate the stress-depression relationship, such that individuals who report a greater tendency to draw on a brooding coping style also show worse depressive symptoms when encountering stress (Bastin, Mezulis, Ahles, Raes & Bijttebier, 2014; Cox, Funasaki, Smith, & Mezulis, 2011, Jose, Kramer & Hou, 2014; but also see Paredes & Zumalde, 2014). Therefore, brooding rumination is considered to be a trait measure of emotion regulation that acts as a diathesis within the diathesis-stress model. As such, it may reflect a common underlying vulnerability to depression as measured by regional EEG activity and spontaneous emotion regulation.

Summary

In the current thesis I will test three very different trait measures of emotion regulation – regional EEG activity, spontaneous emotion regulation, and brooding rumination – within the diathesis-stress model. In Study One, I measure these emotion regulation traits in a population of young women and test whether they reflect the same or different manifestations of an underlying vulnerability to depression. As studies have largely examined these emotion regulation trait measures within independent lines of research, it remains unclear whether these are different manifestations of the same underlying vulnerability or whether they are each tapping into independent vulnerability markers for depression. In Study Two and Study Three,

I prospectively test these trait measures of emotion regulation within the diathesis-stress model at a short term (three month) follow-up for Study Two, and a longer term (twelve month) follow-up for Study Three. Stress and depression are measured at time two and again at time three and the trait measures of emotion regulation (measured at time one) are used to predict changes in depression symptoms at each following time point. This design enables the different emotion regulation markers and life stress to be tested as direct predictors of depression (i.e, do one or all of these measures predict depression at the follow-up time points). Importantly, the interaction between emotion regulation measures and life stress can also be tested at each follow-up time point. In other words, the three month and twelve month follow-up measures of life stress and depression, in conjunction with the time one measures of emotion regulation, allow trait emotion regulation to be directly tested within the diathesis-stress model. If one, two or all of the emotion regulation measures act as a diathesis within the diathesis-stress framework, emotion regulation would be expected to interact with life stress such that poorer emotion regulators would be expected to show increases in depression symptoms when they experience life stress, whereas good emotion regulators would be expected to be less sensitive to changes in depressive symptoms when they experience life stress.

Chapter Two

Study One: Relationships Among Trait Emotion Regulation Measures

The aim of Study One was to test the relationship between three different trait measures of emotion regulation; that is, the degree to which regional EEG activity (frontal and parietal asymmetries), spontaneous emotion regulation (as measured by the startle eye-blink reflex), and brooding rumination reflect different manifestations of the same underlying vulnerability to depression or are independent of one another. For example, EEG asymmetries may reflect the neural substrates that control spontaneous emotion regulation, which in turn may reflect individual ruminative coping styles. Alternatively, one or all of the proposed diatheses may reflect independent trait vulnerabilities. Very few studies have examined the relationships between pairs of these variables but, as described below, there is evidence to suggest they may be related (e.g. Jackson et al., 2003; Key, Campbell, Bacon, & Gerin, 2008; Nusslock et al., 2011; Ray, Ochsner & Cooper, 2005; Tomarken & Davidson, 1994). However, no studies have directly tested the relationship between these three trait emotion regulation measures in one study. An in-depth analysis of the relationships between these three trait markers of emotion regulation will lead to a more comprehensive understanding of vulnerability to depression and establish whether the independent lines of research that have investigated these phenomena are tapping the same or different underlying mechanisms.

Regional EEG Activity and Spontaneous Emotion Regulation

It has been proposed that more leftward frontal asymmetry is related to individual tendencies to effectively regulate negative emotional responses and more rightward asymmetry is related to ineffective regulation tendencies (for more information see Coan, Allen, & McKnight, 2006; Davidson, 2004; Gable, Mechin,

Hicks, & Adams, 2015; Grimshaw & Carmel, 2014). Jackson and colleagues (2003) tested this hypothesis empirically. In a sample of 47 people they examined the relationship between frontal EEG asymmetries and startle eye-blink measures of emotional reactivity during (at 2.5 and 4.5 seconds after picture onset) and after (1 second post-image offset) passive viewing of negative images compared to neutral images. Probes presented during picture viewing were used to measure the emotional response to the picture, and probes presented after picture offset were used to index spontaneous emotion regulation (sustained emotion reactivity after picture offset). They found that emotional reactivity following offset of negative images correlated with frontal asymmetry measures, such that more rightward frontal asymmetry scores were associated with increased emotional reactivity (i.e., more potentiation following negative images relative to neutral images) when compared to those with more leftward asymmetry. This indicated that those with a more rightward frontal asymmetry showed sustained emotional processing of negative images, and were thus less successful at spontaneously regulating their emotional response. It should be noted, however, that the correlations Jackson and colleagues (2003) found between frontal asymmetry and spontaneous emotion regulation were at frontal-pole (FP2-FP1) and frontal-central (FC4-FC3) electrode sites, but asymmetries have been most frequently related to depression at frontal-medial (F4-F3) and frontal-lateral (F7-F8) sites (see Coan & Allen, 2004). Jackson and colleagues also explored the relationship between parietal asymmetry and emotion regulation but found that these measures did not correlate. In sum, Jackson and colleagues' (2003) findings support the notion that resting frontal asymmetries reflect spontaneous emotion regulation ability, suggesting that these two trait markers may in fact be different manifestations of the same underlying vulnerability to depression (see Davidson, 1998).

Jackson and colleagues' (2003) findings have been extremely influential. They provided support to Davidson's (2004; 1998) model - that rightward frontal asymmetry may be linked to less adaptive emotion processing (and thus predict vulnerability to depression) - and has stimulated much research on frontal asymmetry and emotion related processes (e.g., Kim, Cornwell, & Kim, 2012; Miskovic, Schmidt, Georgiades, Boyle, & MacMillan, 2009; Moran, Mehta, & Kring, 2012). However, in the twelve years since their results were reported, there have been no published replications, and a search on PsychInfo shows 197 citations (as of March 2015). In order to usefully guide theory and future research, such an influential finding requires replication.

Brooding Rumination and Spontaneous Emotion Regulation

Studies examining startle reactivity following the removal of emotional image have found mixed findings (Bradley et al., 1993; Bradley et al., 2006; Dichter et al., 2002; Jackson et al., 2003; Larson et al., 2007; Larson et al., 2005; Larson et al., 2010; Taubitz et al., 2013). Ditcher and colleagues (2002) examined the time course of startle reactivity to emotional images post-image removal and found that startle responses did not differ by image (also see Bradley et al., 1993; Bradley et al., 2006). They speculated that individual differences in cognitive coping styles such as rumination might be linked to startle response measures of spontaneous emotion regulation. However, their study was not designed to examine individual differences, and no direct evidence for their proposal has yet been reported. Nonetheless, other physiological measures of sustained emotional processing have been related to rumination. For example, higher levels of rumination have been related to larger skin conductance responses following negative feedback (Rossi & Pourtois, 2014); sustained pupil dilation while viewing negative words (Siegle, Steinhauer, Carter,

Ramel & Thase, 2003); and prolonged cardiovascular recovery when asked to recall a stressful life event (Key et al., 2008). Taken together, these findings indicate that ruminative coping styles are associated with prolonged physiological recovery to emotional events, and I propose that the startle reflex may also tap into these processes. Based on this assumption, it is plausible that spontaneous emotion regulation (as indexed by the startle eye-blink) is reflective of the same underlying process as brooding rumination.

Brooding Rumination and Regional EEG Activity

Frontal EEG asymmetries have been proposed to reflect the neural substrates of ruminative response styles (Reid, Duke, & Allen, 1998). Independent studies of both brooding rumination and rightward frontal asymmetry have shown each to predict future depression (e.g. Mezulis et al., 2011; Nusslock et al., 2011). Thus, it might be expected that higher levels of brooding rumination would be related to more rightward frontal asymmetries. However, when considered in the context of theories of frontal asymmetry this relationship may not be so clear (see Reid et al., 1998). Davidson (2004; also see Harmon-Jones, 2010) proposes that rightward frontal asymmetries reflect trait tendencies to engage in withdrawal behaviours, whereas leftward frontal asymmetries reflect trait tendencies to engage in approach-related behaviours. While rumination does involve withdrawal from the current external context, and thus could potentially be reflected by rightward frontal asymmetries, it also involves internal verbal processes and is an attempt to actively cope with (approach) problems. As such, it is also conceivable that brooding rumination could be related to leftward frontal asymmetries (see Heller & Nitschke, 1998; Heller & Nitscke, 1997). Although almost no studies to date have assessed the relationship between frontal EEG and brooding rumination directly, one study found that higher

trait rumination (not brooding rumination) correlated with more rightward frontal asymmetry scores in a population with no current or previous depression diagnosis (Nusslock et al., 2011). While this one study supports the proposal that brooding rumination is related to withdrawal processes, it remains to be thoroughly explored whether trait brooding rumination is reflected in resting frontal asymmetries, and if so, whether it relates to leftward or rightward asymmetries.

Study One

The primary aim of Study One was to explore relationships among three trait measures of emotion regulation - regional EEG activity, spontaneous regulation as indexed by the startle eye-blink, and self-reported brooding rumination. Startle eye-blink probes were presented at four different time points. Two time points measured emotional responses during the image. The first was an early emotional response probe (the *early* probe), presented at 300 milliseconds from image onset and used to measure attentional allocation to images. The second was a late response probe presented at four seconds after image onset, used to measure emotional modulation by valence. A third probe, presented one second after picture offset, measured ongoing emotional reactivity as an indicator of spontaneous emotion regulation. And finally, a fourth probe was included 8 – 10 seconds after image offset to measure baseline responses to probes (i.e., startle reflexes in the absence of emotional stimuli.)

Participants were women between 18 and 24 years of age, with no history of depression (previous or current). No previous diagnosis of depression was an important selection criterion for two reasons. First, the diathesis-stress model defines vulnerability as existing *prior* to an episode of depression (Willner et al., 2013). Second, the role of stress in the onset of depression changes as the number of experienced episodes increases (Willner et al., 2013; Post, 1992). Therefore,

individuals with a history of depression are likely to display a different relationship between stress and depression than individuals without a history of depression. Young adults are in a transitional developmental period that often involves significant changes in context and social support systems (Schulenberg, Sameroff, & Cicchetti, 2004) and such changes are likely to include elevated prevalence of negative events and high levels of distress (Arnett, 1998; Schulenberg et al., 2004). Also, many young adults are likely to possess risk factors for depression (vulnerability) but are yet to experience a depressive episode (Pettit, Hartley, Lewinsohn, Seeley & Klein, 2013). These factors make young adults a good population in which to study factors that predict future depression.

Women were exclusively recruited because depression manifests differently for men and women (Hankin & Abramson, 2001; Nolen-Hoeksema, 2001) and thus vulnerability factors are likely to differ. Also, prevalence of depression is twice as high for women than men, so examining diatheses in women only should increase the likelihood of seeing changes in depression symptoms across time. Selecting young women maintained a more homogeneous sample and therefore maximized power to detect predicted relationships. Additionally, rumination is a more prevalent cognitive coping style for young women than for men (Jose & Brown, 2008; Nolen-Hoeksema, 2001; Johnson & Whisman, 2013; Treynor, Gonzalez, & Nolen-Hoeksema, 2003), and frontal EEG asymmetries have been observed to be a reliable predictor of depression for women (Thibodeau et al., 2006), but this relationship may be less robust for men (Stewart, Bismark, Towers, Coan, & Allen, 2010; Stewart et al., 2011). Therefore, while the question of vulnerability to depression in men is important, it was beyond the scope of the current study.

Hypotheses

Startle modulation.

1. The early probe: The early probe time point was included as an exploratory measure to investigate whether early attentional allocation to emotional images (in addition to spontaneous emotion regulation) relates to established factors vulnerability to depression (i.e., regional EEG activity and brooding rumination). Responses to early startle probes reflect allocation of attention to high-arousal emotional material; more attenuation of the startle eye-blink indicates increased attentional allocation to the foreground stimulus (Bradley et al 2003). Therefore, it was expected that, relative to neutral responses, startle responses would be attenuated to both positive and negative high arousal images.

***2. The late probe*:** Late emotion response probes measure differential emotional response by image valence and served as a manipulation check to ensure that emotional modulation of the startle eye-blink was attained by the images. In line with typical emotional modulation of the startle response (Bradley et al., 2006), it was expected that startle responses at the late probe time point would show, relative to neutral images, potentiation in the presence of unpleasant images and attenuation in the presence of pleasant images.

***3. The post-image probe*:** Post-image startle responses were not expected to differ by image valence at the group level, as emotional responding was expected to have dissipated by this point. However, individual differences in post-image potentiation indexes spontaneous emotion regulation. Therefore, individual differences in post-image potentiation were expected to interact with the other proposed trait measures of emotion regulation, all thought to indicate vulnerability to depression. Predictions for this time point are outlined below in the section labeled trait measures of emotion regulation.

Trait measures of emotion regulation.

Regional EEG activity and spontaneous emotion regulation: If spontaneous emotion regulation and patterns of regional EEG activity are different manifestations of the same underlying process, then regional EEG activity should predict spontaneous regulation. However, if these are independent vulnerability markers no such relationship should be observed. Davidson's (1998) model proposes that rightward frontal asymmetry reflects vulnerability to depression. Similarly, larger post-image emotion reactivity is proposed to reflect vulnerability to depression (Davidson, 1998). Thus, in line with the findings of Jackson and colleagues (2003), it was predicted that frontal EEG asymmetries would correlate with post-image emotional reactivity such that individuals with a more rightward asymmetry would show more emotional reactivity (more potentiation relative to neutral images) after negative images were removed than individuals with a more leftward asymmetry.

Recent evidence indicates that rather than examining frontal asymmetries in isolation, looking at the interaction between frontal and parietal asymmetry may provide a more comprehensive measure of emotional processing (Grimshaw, Foster, & Corballis, 2014). According to Heller's Circumplex model (1993) low right parietal activity and rightward frontal asymmetry are the underlying EEG manifestations reflecting vulnerability to depression. Thus, parietal and frontal EEG measures may interact in the current study such that low right parietal activity and rightward frontal asymmetry predict more startle potentiation after negative images, whereas low right parietal activity and leftward frontal asymmetry predict less startle potentiation.

Brooding rumination and spontaneous emotion regulation. If post-image emotion reactivity is tapping into ongoing ruminative processes, as proposed by Ditcher and colleagues (2002), higher brooding rumination scores are expected to

positively correlate with sustained emotion reactivity as indicated by enhanced post-image reactivity.

Brooding rumination and EEG activity. Predictions for the relationship between regional EEG activity and brooding rumination are uncertain. If verbal and approach behaviours are reflected in brooding rumination (Reid et al., 1998), then greater brooding rumination is expected to relate to leftward frontal asymmetry. However, if brooding rumination reflects an individual's withdrawal from his or her current context, brooding rumination should relate to rightward frontal asymmetry.

Method

Participants

The study was approved by the Human Ethics Committee of the School of Psychology, Victoria University of Wellington. One hundred and fifty four undergraduate women participated in the first experimental session. All were aged 18 to 24 years and reported that they were right-handed with no history of neurological disorder. Participants who reported a previous diagnosis of depression or anxiety (n = 34) were removed. A further five participants were removed due to equipment failure or experimenter error. This attrition resulted in a total of 115 participants (M_{AGE} = 18.90 years; SD_{AGE} = 1.33) for Study One analysis.

Stimuli and Apparatus

Seventy-two images (24 pleasant, 24 neutral and 24 unpleasant), covering a variety of emotion categories, were selected from the International Affective Picture System (IAPS) based on standardised female ratings for arousal and valence (Lang, Bradley, & Cuthbert, 2008; see Appendix A). Standard arousal ratings differed significantly between pleasant (M = 6.33, SD = .61) and neutral (M = 2.55, SD = .35) images, $t(46) = 26.20$, $p < .001$; between unpleasant (M = 7.26, SD = .27) and neutral

images, $t(46) = 51.83$, $p < .001$; and between pleasant and unpleasant images, $t(46) = -6.80$, $p < .001$[1]. Standard valence ratings differed between pleasant (M = 7.55, SD = .52) and neutral (M = 4.98, SD = .15) images, $t(46) = 23.14$, $p < .001$, between unpleasant (M = 1.43, SD = .21) and neutral images, $t(46) = -65.03$, $p < .001$, and between pleasant and unpleasant images, $t(46) = 52.93$, $p < .001$.

All stimuli and questionnaires were presented using Psychology Software Tools' E-prime version 1.1 (Schneider, Eschman, & Zuccolotto, 2002) running on a Dell Optiplex 760 computer with a Dell 1908FPb 19" LCD monitor (1024 x 768 pixels, 60Hz refresh rate). Acoustic startle probes were presented using Sony MDR-V150 headphones. These probes comprised 50ms of white noise with near instantaneous rise time and a sound pressure level of 95dB.

Questionnaire Measures

All questionnaires were adapted for presentation by E-prime and were presented on the same computer used for stimulus presentation. Participants completed the following questionnaires:

Beck Depression Inventory-II (BDI-II). The (BDI-II; Beck, Steer, & Brown, 1996) was used to assess current symptoms of depression (see Appendix B). The BDI-II consists of 21 items with four response alternatives and participants endorse the alternative that best describes how they have felt during the previous two weeks. Each alternative carries a value between 0 and 3, reflecting progressively more depression symptomology, for example, item 1: "(0) I do not feel sad; (1) I feel sad; (2) I am sad all the time and I can't snap out of it; (3) I am so sad and unhappy that I can't stand it". Responses are summed to provide a total inventory score between 0-

[1] Positive images were included to provide a manipulation check for modulation of the startle effect by valence and were not of interest in the current thesis with regards to measures of emotion regulation. For this reason images were not equated for arousal

63. The BDI is a widely used measure of depression symptomology for both clinical assessment and research purposes (Hill and Lambert, 2004). It has good reliability and validity in psychiatric and non-psychiatric populations (Beck, Steer, & Garbin, 1988) and also in a population of young adults attending university (Storch, Roberti, & Roth, 2004).

Ruminitive Response Scale (RRS). Brooding rumination was measured using the brooding subscale from the Ruminative Response Scale (Nolen-Hoeksema & Morrow, 1991). The RRS is a 22 item scale (see Appendix C) in which participants report the typical thoughts they experience when they are sad or depressed. Ratings are made for each item on a 4-point Likert scale, from 1 (almost never) to 4 (almost always), to indicate the degree to which they endorse each item. The brooding subscale (Treynor et al., 2003) consists of five of these items related to maladaptive thoughts about the desired situations (e.g. "When you feel down sad or depressed do you think "what am I doing to deserve this"). Total scores for the brooding subscale range from 5 to 20 with higher scores indicating higher levels of brooding

Procedure

Participants were shown around the EEG laboratory, and briefed on the procedure, before they provided written informed consent. Following this, the EEG cap and EMG electrodes were fitted. The experimental session consisted of three phases completed consecutively and always in the same order. First, eight minutes of resting EEG was recorded. Second, the startle eye-blink paradigm was presented in which participants passively viewed images on the monitor while the startle probes were presented over headphones. This was directly followed by a 'washout' period during which participants rested for five minutes while listening to relaxing music. Third, they completed the depression BDI and RRS questionnaires. The entire

experiment was completed in a dimly lit, sound-attenuated, and electrically-shielded chamber. Participants sat in a comfortable chair approximately one meter from the monitor. Instructions were given to remain still and avoid excessive blinking or eye movements during all EEG and EMG recording phases.

Once participants were comfortable, the eight minutes of resting EEG was recorded. This phase was divided into four blocks of two minutes, two with eyes open (O) and two with eyes closed (C), the order of which was counterbalanced across participants (either O-C-C-O or C-O-O-C). Participants then performed a passive picture-viewing task while EMG was recorded. They were told that emotional images would appear on the screen and that they should watch these images for the entire time they were present, while ignoring the noises that would come through the headphones. Each trial (see Figure 2.1 for a schematic of the trial procedure) started with the onset of an IAPS image (6 second duration), which filled the entire monitor. A blank screen was presented after each image for a variable ISI between 12 and 14 seconds. Startle probes were presented at one of four possible time points during each trial: after 300 milliseconds, 4 seconds, 7 seconds (1 second after image offset) or 14-16 seconds (8-10 seconds from image offset). The 14-16 second probe time was included to provide a baseline measure of startle responding (i.e., startle responses when no stimulus was present) and also served to reduce predictability of probe presentation with each image. A total of six startle probes were presented for each image category at each time point, and image valence and startle probe time were randomised from trial to trial. After the startle paradigm was complete participants were instructed to close their eyes and relax while listening to 5 minutes of relaxing music: the first 5 minutes of Beethoven's Piano Concerto No. 5 "Emperor" II. *Adagio un poco moto*. This phase was designed to provide some time for potential arousal

effects induced by the startle experiment to dissipate. Participants then completed the questionnaires.

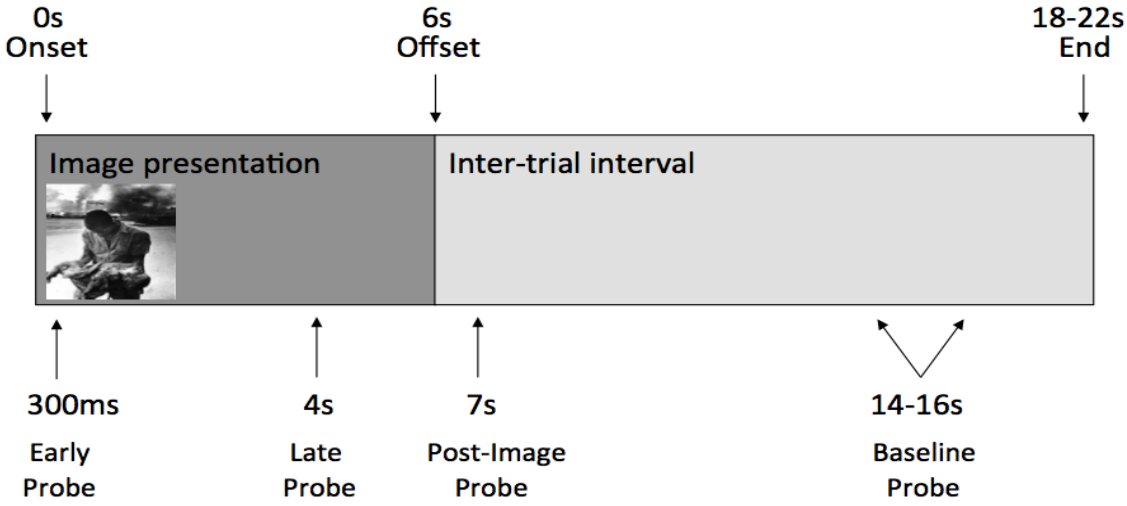

Figure 2.1. Trial schematic for the startle paradigm. Each trial started with the presentation of an image which remained on screen for 6 seconds. This was followed by an inter-trial interval of 12 to 16 seconds. A startle probe appeared at one of the four possible time points for each trial.

Physiological data recording, reduction and analysis

Electroencephalographic (EEG) activity was continuously recorded using a lycra Quik-Cap (Compumedics NeuroMedical Supplies) embedded with 30 Ag/AgCl electrodes (FP1, FP2, F7, F3, FZ, F4, F8, FT7, FC3, FCZ, FC4, FT8, T7, C3, CZ, C4, T8, TP7, CP3, CPZ, CP4, TP8, P7, P3, PZ, P4, P8, O1, OZ, and O2) arranged according to the 10-20 system. All channels were referenced online to physically linked mastoids (M1+M2). Each channel was sampled at 500Hz, using BrainAMP amplifiers and recorded using BrainVision Recorder software (BrainProducts GmBH, Gilching, Germany). Vertical and horizontal eye movements were measured via electro-oculogram (EOG) channels derived from electrodes placed above and below the left eye and lateral to each eye. All electrode impedances were below 10KΩ.

EEG data were processed offline using BrainVision Analyzer software (BrainProducts GmBH, Gilching, Germany). EEG signals were filtered using a notch

filter at 50Hz, a high pass filter at 0.01Hz and a low pass filter at 30Hz. Each two minute block was divided into 1.024 second epochs with 50% overlap, using a Hamming window of 10%. Raw power for each electrode was subjected to a Fast Fourier Transform (FFT) then averaged across all epochs. Alpha power (8-13Hz) was then extracted. Epochs including blinks were not removed as blinks have been shown to have minimal influence on asymmetries derived from alpha power (Hagemann, 2004); this allowed the number of epochs to remain similar across all participants. Frontal and parietal asymmetry scores were derived from ln(F4)-ln(F3) and ln(P4)-ln(P3), respectively (see Allen et al., 2004). For the frontal asymmetry index, more positive scores reflect a leftward frontal asymmetry and more negative scores reflect a rightward frontal asymmetry. Although positive parietal asymmetry values similarly reflect leftward asymmetry and negative values reflect rightward asymmetry, for theoretical reasons it can be useful to recast parietal asymmetry in terms of relative right parietal activity (see Heller & Nitschke, 1997). Thus negative parietal asymmetry scores reflect relatively high rightward parietal activity and positive scores reflect relatively low right parietal activity.

Electromyographic (EMG) recording of startle eye-blinks was conducted according to the guidelines for human startle eye-blink set out by Blumenthal and colleagues (2005). Two 4mm (internal diameter) electrodes (Biopac Systems, Inc.) were placed over the orbicularis oculi muscle under the right eye. The first electrode was placed directly below the forward gazing pupil with the second placed approximately 10mm lateral to the first. Electrode impedances were kept below 10kΩ. The EMG signal was filtered online with a high pass at 0.016Hz and a low pass at 1000Hz and amplified using BrainAMP amplifiers, then sampled at rate of 2500Hz, in order to obtain clear resolution of the EMG signal and avoid aliasing effects. The

signal was then recorded with BrainVision Recorder software (BrainProducts GmBH, Gilching, Germany).

Further offline processing of the eye-blink EMG data was based on criteria set out by Bradley and colleagues (2006). The EMG signal was filtered with a digital band pass filter between 90Hz and 250Hz and a mains notch filter at 50Hz. The signal was then rectified and smoothed using a moving average filter with a time constant of 123ms. Each blink was baseline zeroed across the interval covering 50ms before probe onset**.** Eye-blink magnitudes were scored as the peak voltages exceeding five standard deviations above baseline activity that occurred between 21ms and 180ms from probe onset. Trials where no blink was detected were scored as a zero response trial. Blinks were removed as artifacts if there was excessive noise in the EMG signal, movement during the baseline period, or if the blink criterion threshold was met before 20ms from probe onset (indicating a premature blink too early to be the result of the startle probe). Blinks with magnitudes more than three standard deviations above a participant's mean were excluded. Due to large individual differences in magnitude, all blinks were standardized by z-transformation within participants then converted to T-scores. T-scores produce a mean of 50 and standard deviation of 10 for each participant. Thus, scores over 50 represent blinks larger than the participant's mean and values below represent blinks smaller than the participant's mean.

Results

The aim of this study was to test the relationships between the proposed trait measures of emotion regulation. However, in order to check that I achieved manipulation of emotional reactivity by emotional images, I first examined the time course of emotion reactivity to images, as indexed by startle eye-blink modulation. At the early probe attenuation was expected to high arousal images, regardless of

valence. The expected pattern of emotion reactivity at the late probe was potentiation to unpleasant images and attenuation to pleasant images, relative to neutral images. It was expected that (at the group level) emotion reactivity would have dissipated by one-second post-image offset. Therefore, no modulation by valence was expected at the post-image probe or at the baseline probe. After this manipulation check I tested whether vulnerability markers of depression were related to current depressive symptoms in this non-depressed sample. I then tested the key hypotheses of this study, - whether relationships exist among trait measures of emotion regulation (spontaneous emotion regulation, regional EEG activity, and brooding rumination).

Startle Eye-Blink

Standardised startle response magnitudes to probes were tested using a 4 (probe time: early, late, post-image, baseline) x 3 (valence: unpleasant, neutral, pleasant) repeated measures analysis of variance (ANOVA). Greenhouse-Geisser corrections for violations of sphericity were used where necessary. Figure 2.2 plots standardised magnitudes of startle responses during the three image trial types across the four time points. A significant main effect of probe time, $F(3, 289) = 45.16$, $p < .001$, $\eta_p^2 = .284$, and a marginally significant main effect of valence, $F(2, 228) = 2.73$, $p = .067$, $\eta_p^2 = .023$, were qualified by a probe time by valence interaction, $F(6, 586) = 2.26$, $p = .046$, $\eta_p^2 = .019$. The main effect of probe time revealed the typical attenuation of startle probes relative to stimulus onset, with a graded increase over time (e.g. Bradley et al., 2006). To further explore the interaction of valence and probe time, effects of image valence on startle magnitude at each time point (early, late, and post-image probes) were analysed in separate one-way ANOVAs.

Attentional allocation. At the early probe the expected pattern of startle modulation is attenuation to both unpleasant and pleasant high arousal images relative

to neutral images. This pattern of response was not observed in this study. The follow up one-way ANOVA for the early probe time point showed a marginal *linear* effect of valence $F(2, 228) = 2.456$, $p = .086$. Follow up t-tests showed that startle magnitudes trended towards being larger for unpleasant images (M = 48.26, SD = 5.042) than neutral (M = 47.37, SD = 2.89), $t(114) = 1.83$, p = .070, $d = .217$, and pleasant images (M = 47.29, SD = 3.97), $t(114) = 1.76$, $p = .081$, $d = .214$. Responses to pleasant and neutral images did not differ, $t(114) = .185$, $p = .854$. This pattern of potentiation to unpleasant images differs from the pattern of attenuation to both pleasant and unpleasant images that is typically seen 300ms from image onset. This suggests that these early responses did not tap attentional orienting to high arousal images as expected.

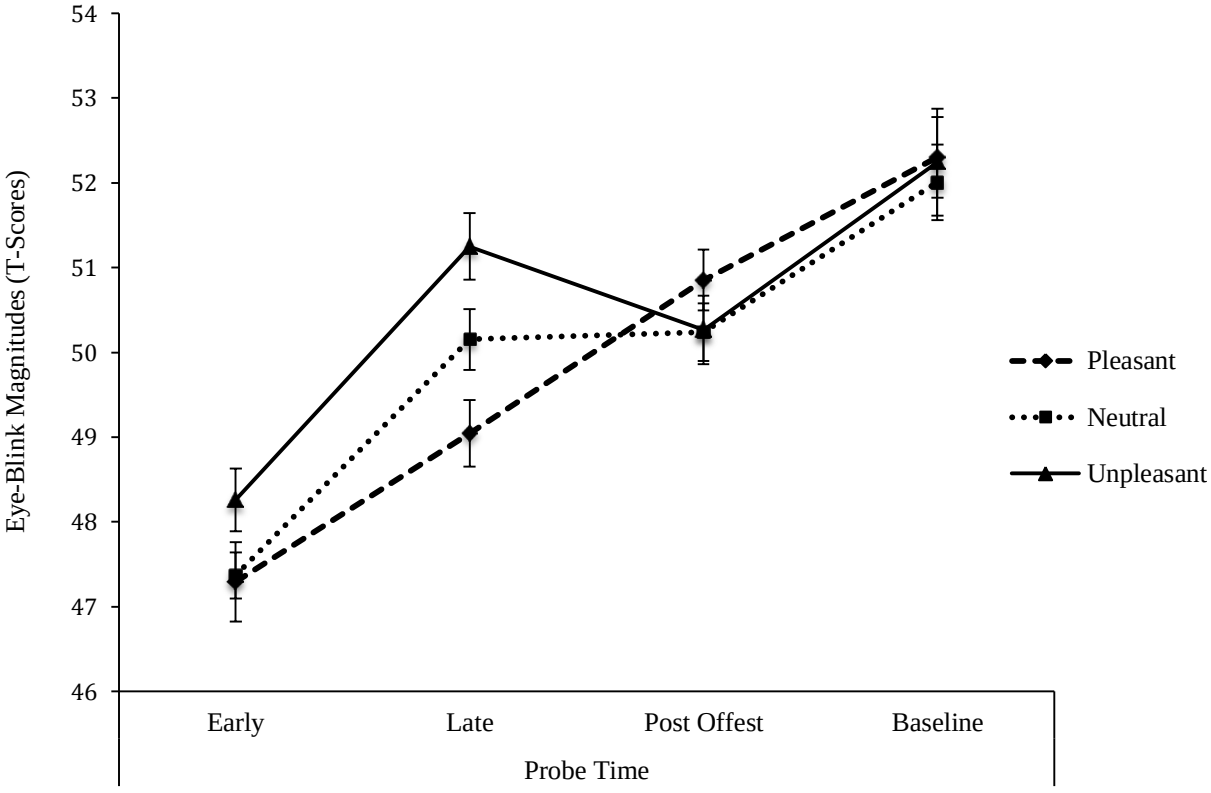

Figure 2.2. Standardised blink reflexes to startle probes during and after viewing emotional images.

The late probe (manipulation check). Startle responses during the late phase of emotion image viewing typically show potentiation of eye-blinks to unpleasant images and attenuation to pleasant images. The follow up one-way ANOVA for the late time point showed a significant linear effect of valence $F(2, 228) = 7.50$, $p = .001$. Follow-up t-tests showed that startle responses to unpleasant images (M = 51.25, SD = 4.21) were larger than those to pleasant images (M = 49.04, SD = 4.189), $t(114) = 3.96$, $p < .001$, $d = .526$, and marginally larger than those to neutral images (M = 50.15, SD = 3.86), $t(114) = 1.88$, $p = .062$, $d = .272$. Also, responses for neutral images were marginally larger than responses for pleasant images, $t(114) = 1.95$, $p = .054$, $d = .276$. Results are in line with typical startle modulation by image valence at the late probe and show that modulation of the startle reflex by emotional images was achieved, with potentiation observed for unpleasant images and attenuation for pleasant images, relative to neutral images.

Spontaneous emotion regulation. No effects of valence were expected at this post-image probe time as it was proposed that, while individual differences in post-image probe responses would reflect spontaneous emotion regulation, at the group level, emotional responses would have dissipated. The follow up one-way ANOVA confirmed that no differences in startle responses existed for the post-image probe, $F(2, 228) = .845$, $p = .431$. These results indicate that at the group level, the valence effects of the emotional images had dissipated after image offset.

Baseline probe. No effect of valence was expected for the baseline probe. A follow up one-way ANOVA for responses at the baseline probe found no differences in startle responses at this time point, $F(2, 228) = .089$, $p = .915$. This finding indicates that valence of the preceding image had no effect on startle responses for probes presented at the baseline time point.

Summary of startle eye-blink findings. Analysis of startle eye-blink responses revealed typical potentiation by unpleasant images and attenuation by pleasant images at the late probe time point, thought to reflect priming of defensive and appetitive responding respectively. The post-image probe showed that this priming had dissipated one second after the image was removed, thought to reflect emotion regulation. However, the typical effect was not observed in the early time point (attenuation to both unpleasant and pleasant images), indicating that the early probe did not successfully tap attentional orienting to the emotional images as intended.

Startle modulation metric. Although the startle eye-blink paradigm revealed expected effects of emotional reactivity and regulation at a group level, individual differences in these processes are of key importance for understanding vulnerability to depression. For the purposes of correlating emotional modulation of startle responses with other variables, an unpleasant reactivity measure was calculated. Startle eye-blink responses to neutral images were subtracted from responses to negative images at the early, late and post-image time points. For this reactivity metric, positive values reflect potentiation of the startle response and negative values reflect attenuation. All following analyses involving startle eye-blink responses use this emotion reactivity metric.

Trait Measures of Emotion Regulation and Depressive Symptoms

All three trait measures of emotion regulation (EEG asymmetry, online emotion regulation, and self-reported brooding) are hypothesised vulnerabilities to depression. Therefore, I first tested whether they were related to depressive symptoms in this non-clinical population. BDI-II scores evidenced a strong positive correlation with brooding rumination ($r(113) = .664$, $p < .001$). However, depressive symptoms did

not correlate with post-image reactivity ($r(113) = -.040$, $p = .671$), frontal asymmetry ($r(113) = .147$, $p = .117$), or parietal asymmetry ($r(113) = .001$, $p = .995$).

Relationships Between Trait Vulnerability Markers

This section tests the hypotheses regarding relationships among trait emotion regulation measures. As all hypotheses predict that vulnerability is related to individual variability in reactivity to *unpleasant* emotional images, I did not examine startle reactivity to pleasant images any further. Additionally, in order to reduce the large number of potential correlations between variables and minimise the possibility of type 1 error, analyses focus on specific predictions, as outlined in the hypotheses. Although I report correlations with all three measures of startle reactivity (early, late, and post-image time points), hypothesis testing focused on the post-image time point, as that most clearly reflects emotion regulation (as opposed to emotional responding). Descriptive statistics for all study variables are presented in Table 2.1 and the correlations between study variables are presented in Table 2.2.

Regional EEG Activity and Spontaneous Emotion Regulation. Contrary to Jackson and colleagues' (2003) findings, no correlation was observed between frontal asymmetry and post-image emotion reactivity following unpleasant images ($r(113) = -.099$, $p = .292$). Because Jackson and colleagues reported their relationship between emotion reactivity and frontal asymmetry at frontal-pole and frontal-central sites (unlike the frontal sites used in this study), exploratory correlations were also computed for these sites. No correlations with post-image emotion reactivity were found at either frontal-pole ($r(113) = -.044$, $p = .639$) or frontal-central ($r(113) = -.082$, $p = .384$) sites. Thus, in this much larger sample of young women with no previous diagnosis of depression, no support was found for the hypothesis that frontal asymmetry, in isolation, relates to spontaneous emotion regulation.

Table 2.1.

Descriptive statistics for time-one variables

Variable Name	Mean	SD	Minimum	Maximum
Frontal Asymmetry	-.0292	.0942	-.2732	.1871
Parietal Asymmetry	.0567	.3189	-0.7137	1.294
Early Pleasant Reactivity	-.0757	4.390	-10.89	13.04
Early Unpleasant Reactivity	.8922	5.226	-12.06	21.84
Late Pleasant Reactivity	-1.109	6.099	-23.39	14.61
Late Unpleasant Reactivity	1.097	6.249	-15.27	16.22
Post-Image Pleasant Reactivity	.0615	5.167	-16.69	16.44
Post-Image Unpleasant Reactivity	.0029	5.464	-11.15	15.85
Brooding Rumination	9.278	3.074	5	18
Beck Depression Inventory	7.061	5.326	0	21

A non-predicted significant negative correlation was observed (see Figure 2.3) between parietal asymmetry and post-image reactivity following unpleasant images ($r(113) = -.197$, $p = .035$), such that individuals with greater right parietal activity (as indexed by more negative parietal asymmetry scores) showed more emotion reactivity following offset of unpleasant images than those with lower right parietal activity. This is an interesting finding as parietal asymmetry has been related to depression (e.g. Bruder et al., 1997; Kentgen et al., 2000; Stewart et al., 2011) but no relationship between emotion regulation and parietal asymmetry has been established. However, this relationship is the opposite of what might be expected given that low right parietal activity and larger post-image startle potentiation are proposed to both be vulnerability markers of depression.

Table 2.2
Correlations Among Study One Variables

	Variable	1	2	3	4	5	6	7	8	9	10
1	Frontal Asymmetry	1									
2	Parietal Asymmetry	-.268**	1								
3	Early Probe Pleasant	.152	-.090	1							
4	Early Probe Unpleasant	.066	.012	.256*	1						
5	Late Probe Pleasant	.062	-.054	.105	-.017	1					
6	Late Probe Unpleasant	-.036	-.058	.179	.001	.531**	1				
7	Post-Image Probe Pleasant	-.080	.040	-.044	-.161	.004	.014	1			
8	Post-Image Probe Unpleasant	-.099	-.197*	-.166	-.012	-.007	.135	.261**	1		
9	Brooding Rumination	.022	.089	-.124	.053	-.162	.134	-.083	-.006	1	
10	Beck Depression Inventory	.147	.001	.037	-.005	-.104	.039	-.049	-.040	.664**	1

NB: ** $p < .01$ * $p < .05$

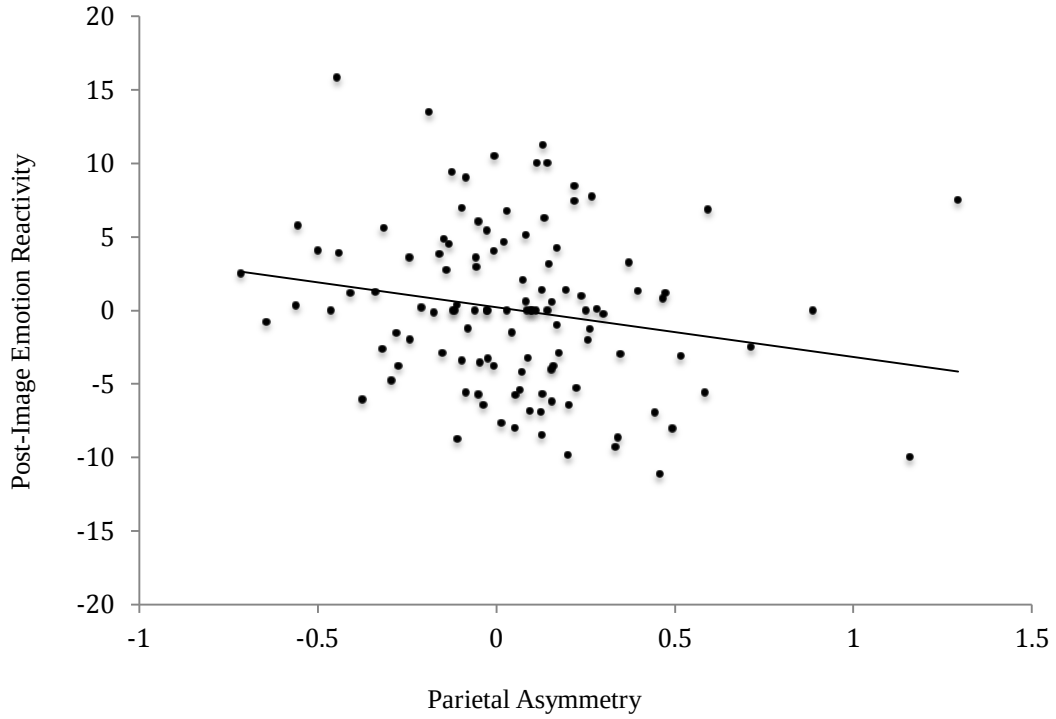

Figure 2.3. Correlation between parietal asymmetry and emotion reactivity to unpleasant images at the post image probe

Frontal by parietal interactions were then tested as predictors of spontaneous emotion regulation using a hierarchical linear regression analysis (see Table 2.3). Frontal and parietal asymmetries were entered at Stage 1 and the frontal by parietal interaction term was entered at Stage 2. Frontal and parietal activity did not interact to predict responses to the post-image probe (β = -.066, p = .592). The correlation between parietal asymmetry and the post-image probe (reported above) was also observed in this regression analysis (β = -.241, p = .015).

The only significant finding for the post-image probe (regulation) time point was that right parietal activity directly predicted emotion reactivity (as reported above). Importantly, frontal asymmetry and right parietal activity did not interact to predict post-image probe reactivity. This result suggests that regional EEG measures of frontal and parietal activity that, according to Heller (1993), reflect a marker for

depression, do not reflect a different manifestation of the same underlying process as spontaneous emotion regulation as measured by post-image probe reactivity in the startle paradigm. However, parietal asymmetry does appear to be related to spontaneous emotion regulation.

Table 2.3
Frontal and parietal asymmetries predicting unpleasant emotion reactivity at the post image probe.

	Predictor	R^2	ΔR^2	β	t	*p*
Step 1		.064				.025
	Frontal Asymmetry			-.164	-1.726	.087
	Parietal Asymmetry			-.241	-2.540	.015
Step 2		.066	.002			.592
	Frontal Asymmetry			-.156	-1.617	.109
	Parietal Asymmetry			-.282	-2.314	.023
	Frontal Asymmetry x Parietal Asymmetry			-.066	-.537	.592

Brooding Rumination and Spontaneous Emotion Regulation. Brooding rumination did not correlate with unpleasant emotional reactivity at the post-image probe ($r(113) = .006$, $p = .951$). This indicates that brooding rumination does not reflect the same underlying vulnerability trait as spontaneous regulation.

Brooding rumination and regional EEG activity. Brooding rumination also showed no relationship with frontal asymmetry ($r(113) = .022$, $p = .816$) or parietal asymmetry ($r(113) = .089$, $p = .342$). This indicates that brooding rumination does not reflect the same underlying vulnerability trait as regional EEG activity.

Brooding rumination summary. Taken together these findings indicate that brooding rumination appears to reflect a unique manifestation of vulnerability to

depression. In other words, it is a different vulnerability trait marker than spontaneous regulation or regional EEG activity.

Discussion

The aim of Study One was to test whether three trait markers of emotion regulation (spontaneous emotion regulation, regional EEG activity, and brooding rumination) are common manifestations of the same vulnerability to depression or whether each operates as an independent marker of vulnerability. Spontaneous regulation was measured using a startle reactivity index of emotional reactivity after the offset of an emotional image. Two additional measurements of emotional reactivity were obtained during the image, and a baseline measurement of startle magnitude in the absence of emotional stimuli was also used. Frontal asymmetry and right parietal activity were indexed using regional measures of EEG activity, measured as the inverse of alpha power. Finally, brooding rumination was indexed using self-reported scores on the Ruminative Response Scale (Nolen-Hoeksema et al., 2008). The core finding was that none of the hypothesised relationships between these trait measures of emotion regulation were observed, suggesting that spontaneous regulation of emotion, regional EEG markers of vulnerability, and self-reported brooding rumination each reflect independent markers of vulnerability to depression. However, an unpredicted relationship was observed between spontaneous emotion regulation and parietal asymmetry, such that high right parietal activity was associated with poorer emotion regulation.

Startle Eye-Blink

The expected effect of emotion reactivity at the late probe was observed, indicating that modulation of emotional reactivity by valence of the emotional images was achieved. However, emotion reactivity at the early probe time point was

inconsistent with the pattern of startle attenuation typically observed at this point of processing high arousal stimuli (Bradley et al., 1993). Attenuation of the startle in the presence of high arousal images (regardless of valence) is proposed to reflect sensory gating that protects the processing of the foreground image from interruption by the probe (Filion et al., 1998). In the current study, no attenuation was observed to pleasant images. Also, rather than attenuation to unpleasant images a trend for potentiation to unpleasant images was observed. This indicates that rather than reflecting the protection of early attentional information, responses to the early probe indexed very early emotional reactivity to (specifically) images with an unpleasant valence. This is likely to reflect early activation of defensive motivational systems – the same system that is proposed to potentiate startle at the later time point (Bradley et al., 2001). Importantly, these findings indicate the early probe did not tap early attentional processes with this sample as intended, making it impossible to interpret the early probe responses as attentional.

Patterns of emotional responding across time to startle probes are well documented (e.g. Bradley et al., 2006, Bradley et al., 1999), and significant potentiation of the startle by unpleasant images has been observed very early in emotional responding (e.g. by 500ms; Bradley et al., 2006). Early processing of emotional information is affected by individual differences. For example, Sass, Heller, Stewart, Silton, Edgar, Fisher and Miller (2010) showed that individuals with high levels of anxiety (as measured by anxious arousal and anxious apprehension levels) had a larger emotional response at very early stages of processing measured using event related potentials. Additionally, Li, Zinbarg and Pallar (2007) found that negative information was processed more quickly by individuals scoring high on trait anxiety than those with low trait anxiety scores. Therefore, it is conceivable that

potentiation to unpleasant images at the early probe, as observed in the current study, is driven by individuals with high levels of anxiety. However, as the current study did not record anxiety this hypothesis cannot be tested in this study.

Spontaneous Emotion Regulation and Regional EEG Activity

Based on Jackson and colleagues' (2003) study, it was hypothesized that rightward frontal asymmetry would relate to greater post-image emotional reactivity. No evidence for this relationship was observed. Jackson's findings have been used as indirect support for Davidson's (2004; 1998) theory that rightward frontal asymmetry reflects less adaptive emotional regulation and is a vulnerability marker for depression. Inconsistent with this proposal, current findings indicate that spontaneous emotion regulation and frontal asymmetries may be independent markers of vulnerability.

The methodology of the current study closely matched that of Jackson and colleagues' (2003), although with a much larger sample. It should be acknowledged that either Jackson's or the current results may represent a spurious finding. As such, my findings beg further investigation and replication. One disparity that may account for the current failure to replicate Jackson is the use of a different participant sample. First, Jackson recruited older men and women (57 to 60 years old) whereas the current study recruited younger women (18 to 24 years old). Regional EEG activity measurements have been shown to be less consistent in older, relative to younger, populations (Duffy, Albert, McAnulty & Garvey, 1984; Duffy, McAnulty & Albert, 1993). Similarly, older adults produce the typical valence modulation of startle eye-blink responses to emotional images less consistently than younger populations (e.g. Feng, Courtney, Mather, Dawson, & Davison, 2011). Older populations have also been found to habituate more quickly and produce smaller magnitude blinks (Ford &

Pfefferbaum, 1991; Ludewig, Ludewig, Seitz, Obrist, Geyer, & Vollenweider, 2003). Taken together, it is possible that my study is tapping into different processes, or different stages of processing to Jackson and colleagues', leading to a disparity in our results. For example, Jackson's measurement at the post-image offset time point could be tapping into a delayed emotional response, rather than a sustained emotional response. However, if this were the case it might expected that startle eye-blink modulation during picture viewing would not have been found, which they report. Importantly, whilst it is unclear how the nature of age differences may account for the present failure to replicate Jackson, it is a consideration to bear in mind, especially given that Jackson and colleagues' findings have been generalized to younger populations as support for resting prefrontal activity as a mediator of spontaneous emotion regulation (e.g. Kim et al., 2012; Lopez-Durren et al., 2011).

A second disparity between the population used in Jackson and colleagues' (2003) study and the current study, is that whereas Jackson did not report controlling for previous or current experience of depression, the present study controlled for these variables; participants reported no history of depression and were not currently depressed (mean BDI score = 7.06; see Table 2.1). Frontal EEG asymmetries show a relationship with current (Gotlib et al., 1998; Henriques & Davidson, 1991) and previous (Gotlib et al., 1998; Henriques & Davidson, 1990; Stewart et al., 2011) depression. Further, there is a considerable body of evidence showing that patterns of startle responding differ for depressed (e.g. Dichter et al., 2004; Dichter et al., 2008; Moran et al., 2012) and dysphoric individuals (Mneimne et al., 2008; Taubitz et al., 2012), compared to never depressed controls. It is possible that the findings of Jackson and colleagues are driven by people within their sample who had either previously, or were at the time, experiencing depression (or high levels of depressive

symptoms). If this were the case, then the present failure to replicate Jackson and colleagues is not surprising as these participants were not present within the current sample.

Taken together, it is possible that the specific sample used within my study accounts for the failure to replicate Jackson and colleagues' (2003). I chose to recruit young women as they represent a more homogenous sample in regards to vulnerability to depression, as well as emotional responding and regulation. My sample was also larger and therefore had greater statistical power than the original study. Due to these factors, my sample should provide clearer insight into the relationship between spontaneous emotion regulation and regional EEG markers of vulnerability to depression. However, it may be that in making the sample more homogenous, I excluded the individuals with the very characteristics that were in fact driving Jackson and colleagues' original effect.

While the expected correlation between frontal asymmetry and spontaneous emotion regulation was not found, an unexpected (though weak) relationship between parietal asymmetry and spontaneous regulation of emotion was observed. Higher right parietal activity (i.e., rightward parietal asymmetry) was related to poorer spontaneous emotion regulation (more post-image potentiation). This finding is the opposite to what would be predicted based on proposals that depression is associated with low right parietal activity and poorer emotion regulation (e.g. Bruder et al., 2007; Davidson, 1998). The parietal cortex is important in the orienting of attention to salient (e.g. emotional) stimuli and also in the voluntary shifting of attention (Corbetta & Shulman, 2002). Further, the parietal cortex is associated with arousal responses – with higher rightward activity reflecting increased arousal experience (Anders, Lotze, Erb, Grodd, & Birmaumer, 2004). It could be that individuals with a rightward

parietal asymmetry had a stronger arousal response to images (they were more salient) culminating in sustained attention to emotion. As such, emotional responses took longer to regulate as reflected by the sustained potentiated startle response. A number of factors could produce a pronounced arousal experience. One possibility is anxious-arousal (Heller & Nitschke, 1998). It is possible that the relationship between high right parietal activity and poorer emotion regulation relates to higher levels of anxious arousal. However, this is just speculation, as I did not measure anxiety. It is also possible that higher levels of arousal were caused by an environmental factor, potentially something as simple as caffeine intake (e.g. drinking coffee), which is thought to increase right parietal activity by increasing arousal (e.g. Stewart et al., 2011).

Brooding Rumination, Spontaneous Regulation, and Regional EEG Activity.

Brooding rumination did not correlate with spontaneous emotion regulation or patterns of regional EEG activation, suggesting that it is an independent marker of depression vulnerability. Interestingly, brooding rumination was the only trait measure of emotion regulation in the current study to correlate with current depressive symptoms – a finding that has frequently been reported in the literature (Burwell & Shirk, 2007; Mezulis et al., 2007; Treynor et al., 2003). This suggests that brooding rumination may be reflective of current non-clinical depressive symptomology.

Summary

The first study was designed to test the degree to which three measures of emotion regulation reflect different manifestations of the same underlying vulnerability traits of depression, or are independent factors that reflect separate vulnerabilities. Of particular interest, no support was found for frontal EEG

asymmetries alone, or in conjunction with parietal activity, predicting spontaneous regulation as indexed by the startle eye-blink. However, results do suggest that there is a relationship between parietal asymmetry and spontaneous emotion regulation. Second, the early probe did not index attentional processes as expected but instead appeared to index early defensive activation to negative images. Although these results could possibly be interpreted within the context of anxiety, the current study was limited by the fact that no direct measures of anxiety were included. Importantly, the core finding was that spontaneous emotion regulation, frontal asymmetry, and brooding rumination do not appear to reflect common underlying vulnerability to depression but rather are independent vulnerability factors. This suggests that future studies should examine these factors as independent markers of vulnerability with differing manifestations rather than as common predictors of depression.

Chapter Three

Study Two: The Diathesis Stress Model at Three Months

The diathesis-stress model proposes that pre-existing differences in ability to regulate negative emotions constitute a vulnerability to depression. People less capable of regulating negative emotions are more susceptible to the detrimental effects of stress. Stress has a well established positive relationship with depression. For the vast majority of individuals who experience depression, a significant stressor precedes the first episode (Post, 1992; Willner et al., 2012). However, most individuals who experience stressful events do not go on to experience depression (Bonanno, 2004; Coifman & Bonanno, 2010; Hammen, 2005). The diathesis-stress model accounts for such individual differences in responses to stress by proposing that some individual traits mark sensitivity to stress, and others mark resilience to stress (Monroe & Simons, 1991; Willner et al., 2013). These trait markers of sensitivity are a diathesis as they predispose individuals to become depressed *if* a significant stressor is experienced. For an individual trait to be considered a vulnerability to depression within the diathesis-stress framework it first must be present before a depressive episode is experienced, and second must interact with stress to predict changes in depressive symptoms. Trait measures of emotion regulation have been proposed to reflect an individual's sensitivity or resilience to stress (Bonanno & Burton, 2013) and thus make a good candidate as a diathesis to stress.

Study One showed that three trait measures of emotion regulation – spontaneous emotion regulation, frontal asymmetry, and brooding rumination – are independent vulnerability markers that do *not* reflect different manifestations of the same underlying vulnerability to depression. These trait measures of emotion

regulation appear to operate independently in the development of depression and can therefore be considered as independent diatheses - measurement of one emotion regulation marker cannot be generalized to reflect the other markers. As such it is important to establish if one, two or all of these indices of trait emotion regulation predict depressive symptoms over time, either directly or by interacting with stress within a diathesis-stress framework. The aim of Study Two was to test trait markers of emotion regulation as both direct predictors of depression, and as factors that interact with stress to predict depression, as described by the diathesis-stress framework. This follow-up study was conducted three months after the original experimental session reported in Study One, and is the first of two follow-up studies. An additional follow-up at twelve months (Study Three) is reported in chapter four.

As described in Study One, the three measures of emotion regulation used were: spontaneous emotion regulation, as indexed by post-image startle reactivity; frontal and parietal asymmetry, as indexed by regional EEG measures of cortical activity; and a self-report measure of brooding rumination. Although some studies have reported a relationship between the proposed diatheses and depression, very few have done so in a prospective study, and even fewer have tested them within the diathesis-stress framework.

Regional EEG Activity

Three studies have prospectively examined resting EEG activity as a predictor of depression (Mitchell & Possel, 2012; Nusslock et al., 2011; Pössel et al., 2008). Pössel and colleagues (2008) recorded frontal asymmetry and parietal asymmetry, and used these measures to predict self-reported depressive symptoms twelve months later. Their sample of 80 adolescents (35 women) was aged between 13 and 15 years old and had no previous or current diagnosis of depression. Consistent with the

hypothesis that frontal asymmetry is a direct predictor of depression, rightward frontal asymmetry predicted increased depressive symptoms at twelve months. However, they did not measure life stress and so could not examine regional EEG measures within the diathesis-stress framework. Unexpectedly, they found high (rather than low) right parietal activity also predicted depression, independent of frontal asymmetry. They suggested that anxious arousal (the feeling component aspect of anxiety) might mediate the reported relationship between high right parietal activity and depression. Although they controlled for anxiety, their measurement mainly captured anxious apprehension (the worry component of anxiety) rather than anxious arousal. As such, using this measure as a covariate may not have removed variance due to anxious arousal. The results of Pössel and colleagues suggest that frontal asymmetry shows promise as a candidate diathesis but the role of parietal asymmetry is less clear.

Mitchell and Pössel (2012) also examined frontal asymmetry as a predictor of depressive symptoms. Their sample consisted of 41 adolescent boys (mean age = 13.91) with no previous or current diagnosis of depression. Consistent with results of Pössel and colleagues (2008) they found that rightward frontal asymmetry directly predicted an increase in self-reported depressive symptoms twelve months later. However, once again this relationship was not tested within a diathesis-stress framework. Parietal asymmetry was not measured as a predictor.

Nusslock and colleagues (2011) conducted a comprehensive longitudinal study examining the value of frontal asymmetry and cognitive response styles in predicting first diagnoses of depression. Rightward frontal asymmetry was found to relate to more negative coping styles, and importantly predicted first diagnosis of depression over a three year follow-up period. Nusslock and colleagues did not report parietal

asymmetry measures. Taken together the results of Pössel and colleagues (2008), Mitchell and Pössel (2012), and Nusslock and colleagues (2011) provide consistent evidence that rightward frontal asymmetry predicts depression, although null effects have also been reported (Blackhart et al., 2006). However, none of these studies tested frontal asymmetry within the diathesis-stress framework - that is by determining whether asymmetry predicted the response to stress. The role of parietal asymmetry also remains unclear.

Recent evidence suggests that parietal asymmetry may also play a role in depression (Bruder et al., 1997; Bruder et al., 2012; Pössel et al., 2008) and emotional processing more broadly (Grimshaw et al., 2014). However, only one study (Pössel et al., 2008) has reported parietal asymmetry as a predictor of depression in a longitudinal design. Further, while Pössel and colleagues used parietal asymmetry as well as frontal asymmetry to predict depression across time, they did not examine the interaction between these two regional EEG markers. Heller's circumplex model (1993) proposes that it is the interaction between frontal asymmetry and parietal asymmetry that predicts depression. More specifically, *low* right parietal activity in conjunction with rightward frontal asymmetry is hypothesised to reflect depression. Conversely, *high* right parietal activity in conjunction with rightward frontal asymmetry is hypothesised to reflect anxious arousal. However, there is little empirical evidence on whether parietal asymmetry (alone, or in conjunction with frontal asymmetry) directly predicts depression, or predicts depression within a diathesis-stress framework.

Brooding Rumination

Brooding rumination reliably correlates with current depression (e.g., Nolen-Hoeksema et al., 2008; Siegle et al., 2004; Treynor et al. 2003) and has been shown in

a number of prospective studies to predict depression (Bastin, Bijttebier, Raes & Vasey, 2014; Burwell & Shirk, 2007; Bastin et al., 2014; Gibb, Grassia, Stone & Uhrlass, 2013; Jose & Weir, 2013; O'Conner, O'Conner & Marshall, 2007; Paredes & Zumalde, 2014;). Importantly, brooding rumination has also been shown to function as a diathesis within the diathesis-stress framework (Bastin et al., 2014; Cox et al., 2011; but also see Paedes & Zumalde, 2014). For example, Cox and colleagues (2011) measured brooding in 111 adolescents (80 women; mean age = 16.4 years), and assessed stress and depressive symptoms 8 and 12 weeks later. Brooding rumination was found to moderate the stress-depression relationship such that high levels of brooding exacerbated the effect of stress on depressive symptoms. They found no direct relationship between brooding and depressive symptoms. Similar results were reported in Bastin and colleagues (2014) in a sample of 368 adolescents (232 women; 9-15 years) across a twelve month follow-up period. Taken together, these studies indicate that brooding rumination reflects sensitivity to stress.

Spontaneous Emotion Regulation

No studies have tested whether a startle index of online and spontaneous emotion regulation predicts depressive symptoms across time either directly, or within a diathesis-stress framework. My studies address this gap in the literature and include spontaneous emotion regulation (as indexed by post-image startle reactivity) as a predictor of depression alongside regional EEG markers and brooding rumination.

Study Two

The aim of this study was to test trait emotion regulation markers - spontaneous emotion regulation, regional EEG activity, and brooding rumination - within the diathesis-stress model in a short term longitudinal design. The proposed diatheses and depressive symptoms were measured at time one (reported in Study One). Three

months later (time two) life stress and depressive symptoms were measured using an online survey. Life stress was operationalized as the number of self-reported life events (as experienced in one's day-to-day life) across the three months prior, weighted by the degree of distress subjectively experienced for each event.

In Study Two I also addressed one of the major limitations of Study One – that because the research questions focused on depression, no measures of anxiety were collected. Anxiety, in particular anxious arousal, has been proposed to moderate the relationship between parietal asymmetry and depression (Stewart et al., 2011). Anxious arousal is a dimension of anxiety that is characterised by increased physiological arousal and feelings of tension in the body. Low right parietal activity alone is considered to reflect depression in the absence of anxiety, whereas high right parietal activity is proposed to reflect either anxiety alone or depression comorbid with anxious arousal (Heller & Nitschke, 1998). Therefore, to ensure that any observed relationship between parietal asymmetry and depression could not be accounted for by anxious-arousal, I tested whether anxious arousal moderated the relationship between parietal asymmetry and depression.

Hypotheses

Life Stress. It is well established that stress predicts depression (see Hammen, 2005). It was therefore expected that life stress would directly predict increases in depression at time two.

Spontaneous emotion regulation. If poor online regulation of emotion is a diathesis to depression then emotion reactivity at the post-image probe at time one would be expected to interact with life stress to predict depression at time two, such that poorer regulators (those more reactive to the post-image probe) would show a

stronger relationship between stress and depression, than good regulators (those less emotionally reactive to the post-image probe).

Regional EEG activity. In order to accurately assess the role of regional EEG activity within the diathesis-stress framework, anxious arousal was measured. Stewart and colleagues (2011) postulated that right parietal activity might moderate the relationship between anxious arousal and depression. This proposal is based on Heller's Circumplex model (Heller, 1993; Heller & Nitschke, 1998), which argues that high right parietal activity is related to anxious arousal comorbid with depression, whereas low right parietal activity is related solely to depression. It was predicted that for individuals with high right parietal activity at time one, higher levels of anxious arousal would be associated with higher levels of depression at time two; however for individuals with low right parietal activity, it was expected there would be no relationship between anxious arousal and depression.

Frontal asymmetry. On the basis of previous studies (Mitchell & Possel, 2012; Nusslock et al., 2011; Pössel et al., 2008) frontal asymmetry was expected to directly predict depression at time two such that individuals with more rightward asymmetry scores at time one would show greater increases in depressive symptoms at time two. If frontal asymmetry acts as a diathesis within the diathesis-stress framework, then frontal asymmetry should interact with life stress to predict depression at time two. Specifically, when levels of life stress are high individuals with relative rightward asymmetries are more likely to show increases in depression symptoms, whereas relative leftward individuals are less likely to show increased depression symptoms. Conversely, when life stress is low depression symptoms should not increase, regardless of frontal asymmetry.

Parietal asymmetry. Parietal asymmetry has been proposed to relate to depression, such that low right parietal activity at time one should predict depression (or increased depressive symptoms) at time two (Bruder et al., 1997). However, Pössel and colleagues (2008) found that *high* right parietal activity predicted increased depression. There is a high degree of comorbidity between depression and anxious arousal (Mineka, Watson, & Clark, 1998) and Pössel and colleagues proposed that their parietal asymmetry – depression relationship was driven by anxious arousal. Therefore, investigations of parietal asymmetry and depression should control for anxious arousal (see Stewart et al., 2011). As such, anxious arousal was controlled for in all analyses that examined parietal asymmetry. Under these conditions it was predicted that (consistent with Bruder et al., 1997) low right parietal activity at time one would predict depression at time two. Furthermore, if parietal asymmetry acts as a diathesis within the diathesis-stress framework, it was expected that right parietal activity would interact with life stress to predict time two depression, such that those with low right parietal activity would show a strong relationship between stress and depression, whereas those with high right parietal activity would show a weak relationship between stress and depression.

Frontal by parietal interaction. Based on indirect empirical findings, Heller (1993) suggested that a conjoint measure of frontal and parietal asymmetries may both directly, as well as via an interaction with life stress, predict depression at time two. Based on Heller's Circumplex model, it was expected that, because anxious arousal was controlled for, individuals with high right parietal activity at time one would *not* show an interaction between frontal asymmetry and life stress in predicting depression. However, it was expected that individuals with low right parietal activity would show an interaction between frontal asymmetry and life stress that would

predict depression at time two; specifically individuals with a rightward frontal asymmetry would show increased depressive symptoms when stress is high, but those with a leftward frontal asymmetry would be more resilient to stress and thus show less depression under high stress conditions, The same pattern of EEG activity may also predict depression directly (i.e., not through life stress).

Brooding rumination. Brooding rumination reflects a maladaptive style of emotion regulation and therefore it was predicted that brooding scores at time one would interact with life stress to predict increases in depression at time two. Consistent with Cox and colleagues (2011) and Bastin and colleagues (2014), individuals scoring higher on the brooding scale were expected to experience increases in depression when life stress is high, whereas individuals scoring lower on brooding were expected to show less increase in depression symptoms in response to life stress.

Method

Participants

Seventy-six individuals chose to complete the Study Two survey, out of the 115 participants from Study One who were invited. All were female, right handed, and reported no history of previous depression or neurological disorder at time one. They were aged between 18-24 years (M = 19.03, SD = 1.395) when entering the study three months previously. Group comparisons (reported below) were made to test for differences between those who responded to the three month follow-up questionnaire and those who did not.

Questionnaire Measures

All questionnaires were adapted for online use in Survey Monkey (surveymonkey.com). Participants completed the following questionnaires:

BDI-II (Beck et al., 1996). The Beck Depression Inventory-II (BDI-II) was used to assess current symptoms of depression. See Study One for a detailed description.

Life Event Questionnaire (Norbeck, 1984). The life event questionnaire (see Appendix D is an eighty-two item questionnaire that asks responders to indicate if they have experienced each of the listed events in the last three months and if so, how stressful they found the event from "0 = not stressful" to "3 = highly stressful" (e.g. "have you experienced a separation with partner or spouse due to conflict"). This measure has been specifically designed for use with a population of young women to measure events they are likely to encounter and that are typically experienced as stressful by this cohort. Life stress can take many forms and attempts have been made to classify different forms of life stress (for a review see Monroe & Reid, 1991).This measure shows good test-retest reliability and construct validity (see Norbeck, 1984).

Mini Mood and Anxiety Symptom Questionnaire (mini-MASQ; Clark & Watson, 1995). Anxious arousal was measured using a subscale of the mini MASQ (See Appendix E). The Mini MASQ is a 26-item scale that assesses symptoms of depression and anxiety. The anxious arousal subscale consists of 10 of these items. Participants respond on a 5-point Likert scale as to how they have been feeling over the past week, ranging from "1 = not at all" to "5 = extremely" (e.g "have you felt your hands were cold or sweaty"). Total scores of anxious arousal range from 10 to 50, with high scores indicating higher levels of anxious arousal.

Procedure

Participants were emailed an invitation to participate. In the email they were provided with a link to the survey and a unique identification number. The survey was presented on Survey Monkey (www.surveymonkey.com). When participants followed

the link they were led to a page explaining the experiment. The participant provided informed consent by indicating that they had read this information and agreed to participate. The questionnaires were then presented in the order of: Life event questionnaire, BDI-II, and the mini-MASQ. Participants were sent a movie voucher as thanks for their participation. All questionnaire responses were scored and subscales were calculated as per the requirements of each questionnaire. To test the hypotheses derived from the diathesis-stress model, the following scores were used from each scale: The subjective stress score was used from the life event questionnaire, the total depression score from the BDI-II, and the anxious arousal scale from the MASQ. Each participant's scores were then matched to their data from Study One.

Results

As only a proportion (66%) of the original sample collected at time 1 responded to the survey at time 2, it was important to establish whether time two responders accurately reflect the wider sample collected at time one. In order to assess this, differences in time one variables between responders and non-responders (at time two) were analysed. Groups did not differ on frontal asymmetry, $t(113) = -.686$, $p = .494$; parietal asymmetry, $t(113) = -.555$, $p = .580$; BDI-II at time one, $t(113) = 1.435$, $p = .154$; brooding rumination, $t(113) = 1.100$, $p = .274$; or emotional reactivity at the post-image, $t(113) = 1.042$, $p = .300$ probe time. Taken together, these results suggest that responders at time two reflect the larger sample collected at time one. Descriptive statistics for all Study Two predictors and outcome variables are presented in Table 3.1 for both responders and non-responders. The diathesis-stress model was tested at the three month follow-up time point using hierarchical linear regression analysis. Depression scores at time two were entered as the dependent variable and the

depression scores at time one were entered into each analysis at step one in order to covary time one depression, and therefore assess changes in depression over time. For all analyses examining the role of parietal asymmetry, anxious arousal was statistically controlled for by adding it to at step one of the regression model. Correlations between Study Two variables are presented in Table 3.2.

Table 3.1
Descriptive Statistics for Study Two Variables for both Responders and Non-Responders

Variable Name	Time Two Responders (N = 76)		Time Two Non-Responders (N = 39)	
	Mean	SD	Mean	SD
Frontal Asymmetry	-.0248	.0893	-0.0376	0.1036
Parietal Asymmetry	.0685	.3110	0.0335	0.3366
Post-Image Emotion Reactivity	-.0803	5.718	0.8517	4.928
Brooding Rumination	9.053	3.089	9.718	3.034
Beck Depression Inventory (Time 1)	6.553	5.198	8.051	5.501
Beck Depression Inventory (Time 2)	10.00	8.168	-	-
Life Stress (Time 2)	21.54	15.24	-	-
Anxious Arousal (Time 2)	14.80	5.568	-	-

Life Stress and Changes in Depression

Depression. A paired samples t-test was conducted to compare BDI-II scores at time one with time two. BDI-II scores at time two (M = 10.00, SD = 8.168) were significantly higher than at time one (M = 6.55, SD = 5.198), $t(75) = 5.498$, $p < .001$, $d = .504$.

Life Stress and depression. A two-step hierarchical linear regression was conducted to test life stress as a predictor of changes in depression between time one and time two (see Table 3.3). Depression at time two was entered as the dependent measure. Depression at time one was entered at step one of the regression as a covariate. Life stress was then entered at step two as a predictor of changes in

depression. As predicted, this model (R^2 = .609, p = .005, F (2,73) = 56.968, p < .001) showed that both depression (β = .701, p < . 001) at time one and life stress (β = .216, p = .005) at time two significantly predicted depression at time two, accounting for a total of 60% (adjusted R^2) of the variance. The addition of life stress significantly improved the models prediction (ΔR^2 = .044, p = .005). It should also be noted that life stress at time two positively correlated with depression scores at time one (r(74) = .24, p = .038), indicating that high depression scores at time one may have lead to more experience of stress at time two.

Table 3.2
Correlations Among Study Two Variables

	Variable	1	2	5	6	7	8	9	10
1	Frontal Asymmetry	1							
2	Parietal Asymmetry	-.287**	1						
5	Post-Image Emotion Reactivity	.031	-.323**	1					
6	Brooding Rumination	.071	.154	.020	1				
7	Beck Depression Inventory (Time 1)	.121	.055	-.097	.741**	1			
8	Beck Depression Inventory (Time 2)	.150	.033	.028	.684**	.752**	1		
9	Life Stress (Time 2)	.071	-.019	-.078	.224	.238**	.383**	1	
10	Anxious Arousal (Time 2)	.144	-.152	.063	.186	.178	.434**	.187	1

NB: ** p < .01 * p < .05

Tests of the Diathesis-Stress Model

Spontaneous emotion regulation. A three-step hierarchical linear regression was conducted to test the direct relationship between life stress and spontaneous emotion regulation (i.e., emotion reactivity at the post-image time point) on time two depression, and the diathesis-stress interaction between spontaneous emotion regulation and life stress *predicting* depression at time two. Depression at time one

was entered at step one, the predictors (life stress, and post-image emotion reactivity) were entered at step two, and the interaction between life stress post-image emotion reactivity was entered at step three. Step 1 shows the relationship between depression at time 1 and depression at time two. Step 2 adds in stress and post-image reactivity as direct predictors of depression, over and above that accounted for by depression at time one (that is, whether they predict changes in depression). Step 3 adds the interaction term for spontaneous emotion regulation and life stress. If significant, this indicates that stress and startle reactivity at the post-image probe interact to predict depression, which would suggest that spontaneous emotion regulation acted as a diathesis.

Table 3.3
Hierarchical linear regression model with depression (time 2) as the outcome variable and life-stress (time 2) as the predictor.

	Predictor	Adj. R^2	R^2	ΔR^2	β	t	*p*
Step 1		.560	.566				<.001
	Beck Depression Inventory Time 1				.752	9.815	<.001
Step 2		.599	.609	.044			.005
	Beck Depression Inventory Time 1				.701	9.303	<.001
	Life Stress Time 2				.216	2.865	.005

NB. Adj. R^2 = Adjusted R^2

The results of the regression model are reported in Table 3.4. Step three of the model was a significant predictor (R^2 = .64, p = .050, F (4,71) = 31.870, p < .001) explaining 62% (adjusted R^2) of the variance. As expected, life stress (β = .293, p = .001) predicted changes in depression, with more life stress predicting more depression at time two. In support of spontaneous emotion regulation acting as a diathesis in the diathesis-stress framework, addition of the post-image reactivity by life stress interaction a stage three significantly improved the models prediction (ΔR^2

= .020, β = .276, p = .050). This interaction was plotted using ModGraph (Jose, 2013) and is presented in Figure 3.1. ModGraph plots the relationship between two variables at three different levels of a third moderation variable (the mean, one standard deviation above the mean, and one standard deviation below the mean). Under conditions of low stress, post-image reactivity did not relate to depression at time two. However, as stress increased, larger post-image reactivity exacerbated the relationship between stress and depression, with more reactive individuals (i.e., poorer spontaneous regulators) showing more sensitivity to stress - leading to increased depressive outcomes - than less reactive individuals at the post-image probe (i.e., better spontaneous regulators).

Table 3.4

Hierarchical linear regression model with depression (time 2) as the outcome variable. The predictors were life stress and emotion reactivity at the post-image time point and the interaction between post image reactivity and life stress.

	Predictor	Adj. R^2	R^2	ΔR^2	β	t	*p*
Step 1		.560	.566				<.001
	Beck Depression Inventory (Time 1)				.752	9.815	<.001
Step 2		.607	.622	.057			.007
	Beck Depression Inventory (Time 1)				.710	9.489	<.001
	Life Stress (Time 2)				.222	2.978	.004
	Post-Image Emotion Reactivity				.114	1.561	.123
Step 3		.622	.642	.020			.050
	Beck Depression Inventory (Time 1)				.688	9.276	<.001
	Life Stress (Time 2)				.293	3.603	.001
	Post image Unpleasant Reactivity				-.110	-.826	.412
	Post-Image Emotion Reactivity x Life Stress (Time 2)				.276	1.993	.050

NB. Adj. R^2 = Adjusted R^2

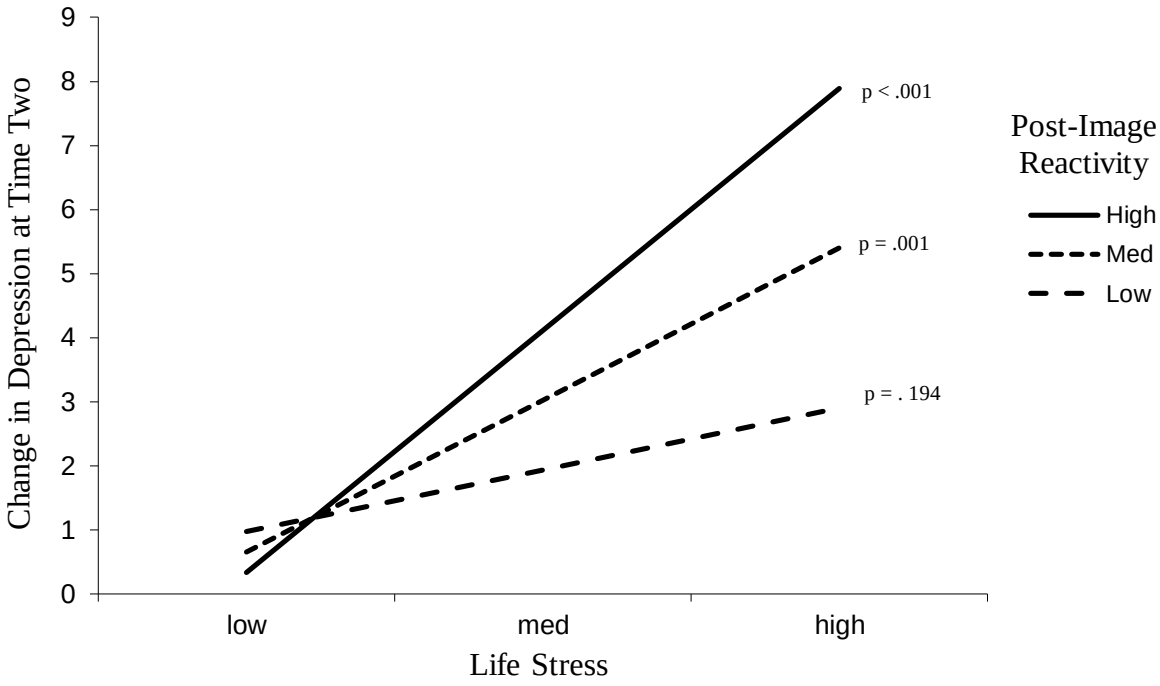

Figure 3.1 Moderation by spontaneous emotion regulation of the relationship between stress and depression (see Table 3.4).

Regional EEG activity.

Parietal asymmetry and anxious arousal. Anxious arousal has been proposed to moderate the relationship between parietal asymmetry and depression (Heller & Nitschke, 1998; Stewart et al., 2011). Therefore, the role of parietal asymmetry at time one and anxious arousal at time two on depression at time two was tested using a three-step hierarchical linear regression. Depression at time two was the dependent variable. Depression at time one was entered at step one as a covariate in order to assess changes in depression across the three month period. Parietal asymmetry and anxious arousal were added at step two and the interaction between parietal asymmetry and anxious arousal was entered at step three.

Step three of this model significantly predicted changes in depression (R^2 = .688, p = .014, F (4,71) = 39.211, p < .001), explaining a total of 67% (adjusted R^2) of the variance, as shown in Table 3.5. The addition of the interaction between anxious arousal and parietal asymmetry at step three significantly improved the models

prediction (ΔR^2 = .028, β = -.513, p = .014), indicating that the relationship between parietal asymmetry (at time one) and depression (at time two) depended on levels of anxious arousal (at time two). This interaction was plotted using ModGraph (Jose, 2013) and is presented in Figure 3.2. The relationship between *low* right parietal activity and depression was independent of anxious arousal, but the relationship between *high* right parietal activity and depression depended on levels of anxious arousal. For individuals with high right parietal activity, high levels of anxious arousal were associated with increases in depression whereas low levels of anxous arousal were not. This finding supports the relationship proposed by Heller and Nitschke (1998) and Stewart and colleagues (2011).

Table 3.5

Hierarchical linear regression model with depression as the outcome variable and anxious arousal and parietal asymmetry as predictors.

	Predictor	Adj. R^2	R^2	ΔR^2	β	t	*p*
Step 1		.560	.566				<.001
	Beck Depression Inventory (Time 1)				.752	9.82	<.001
Step 2		.647	.661	.095			<.001
	Beck Depression Inventory (Time 1)				.698	10.001	<.001
	Anxious Arousal (Time 2)				.317	4.494	<.001
	Parietal Asymmetry				.046	.655	.515
Step 3		.671	.688	.028			.014
	Beck Depression Inventory (Time 1)				.712	10.336	<.001
	Anxious Arousal (Time 2)				.313	4.592	<.001
	Parietal Asymmetry				.530	2.591	.012
	Parietal Asymmetry x Anxious Arousal (Time 2)				-.513	-2.507	.014

NB. Adj. R^2 = Adjusted R^2

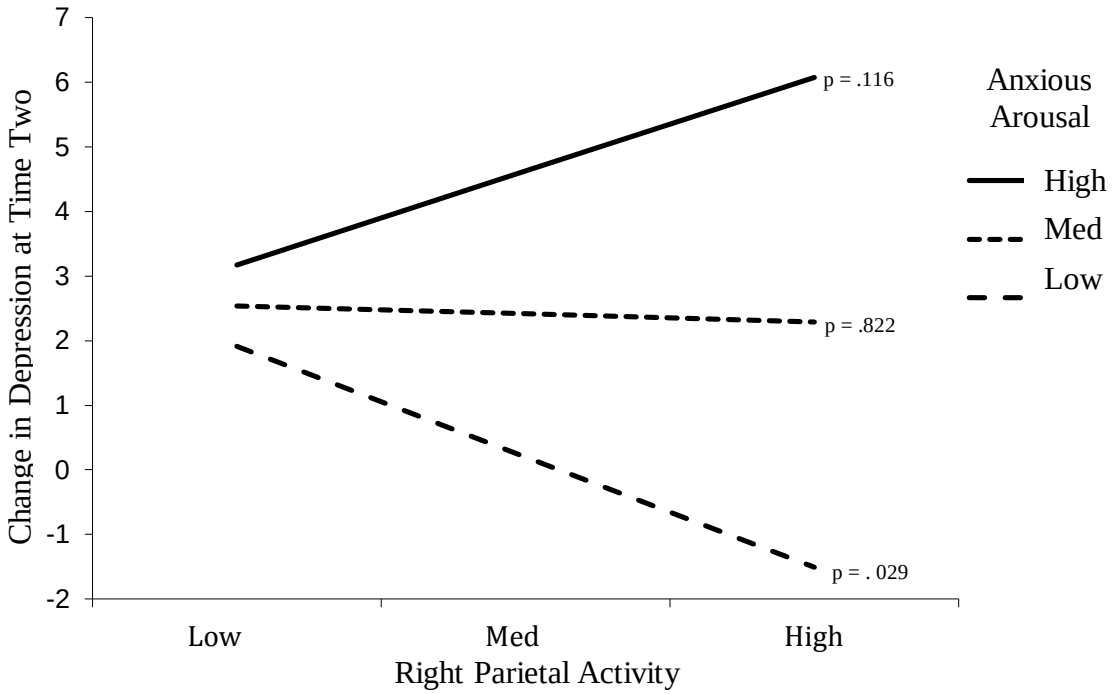

Figure 3.2. Moderation by anxious arousal of the relationship between right parietal

Frontal and parietal asymmetry. Frontal and parietal EEG asymmetries and their interaction were examined within the diathesis-stress framework (see Table 3.6). Due to the relationship between anxious arousal and parietal asymmetry reported above, hierarchical regression analyses included anxious arousal at step one as a covariate. This step removes the variance associated with anxious arousal from depression scores, allowing for a more direct test of the relationship between asymmetry measures and depressive symptoms. A four-step hierarchical regression model was tested. Depression and anxious arousal were entered into the model at step one as covariates. The three predictors (life stress, frontal asymmetry, and parietal asymmetry) were entered at step two. Each of the three two-way interactions (frontal x parietal, frontal x life stress, and parietal x life stress) were entered at step three and the three-way interaction of life stress x frontal asymmetry x parietal asymmetry was entered at step four. This model failed to reach significance for both the three-way interactions and the two-way interactions, indicating that regional EEG asymmetries did not predict either depression or sensitivity to stress at the three month follow-up.

Table 3.6

Hierarchical linear regression model with depression (time two) as the outcome variable and life stress, frontal asymmetry and right parietal activity and their interactions as predictors.

	Predictor	Adj. R^2	R^2	ΔR^2	β	*t*	*p*
Step 1		.649	.659				<.001
	Anxious Arousal (Time 2)				.309	4.465	<.001
	Beck Depression Inventory (Time 1)				.702	10.13	<.001
Step 2		.667	.689	.031			.085
	Anxious Arousal (Time 2)				.290	4.167	<.001
	Beck Depression Inventory (Time 1)				.657	9.401	<.001
	Life Stress (Time 2)				.172	2.480	.016
	Frontal Asymmetry				.033	.470	.640
	Parietal Asymmetry				.056	.797	.428
Step 3		.670	.705	.016			.309
	Anxious Arousal (Time 2)				.258	3.608	.001
	Beck Depression Inventory (Time 1)				.654	9.272	<.001
	Life Stress (Time 2)				.236	3.000	.004
	Frontal Asymmetry				.164	1.378	.173
	Parietal Asymmetry				.212	1.287	.202
	Frontal Asymmetry x Life Stress (Time 2)				-.136	-1.185	.240
	Parietal Asymmetry x Life Stress (Time 2)				-.213	-1.407	.164
	Frontal Asymmetry x Parietal Asymmetry				-.066	-.773	.442
Step 4		.674	.713	.007			.199
	Anxious Arousal (Time 2)				.249	3.481	.001
	Beck Depression Inventory (Time 1)				.654	9.318	<.001
	Life Stress (Time 2)				.240	3.072	.003
	Frontal Asymmetry				.108	.857	.395
	Parietal Asymmetry				.345	1.785	.079
	Frontal Asymmetry x Life Stress (Time 2)				-.070	-.559	.578
	Parietal Asymmetry x Life Stress (Time 2)				-.346	-1.898	.062
	Frontal Asymmetry x Parietal Asymmetry				.143	.785	.435
	Frontal x Parietal x Life Stress				-.211	-1.296	.199

NB. Adj. R^2 = Adjusted R^2

Brooding rumination. Brooding rumination was tested within the diathesis-stress framework using a three-step hierarchical regression model. Depression at time one was entered into the model at step one. Life stress and brooding rumination were entered at step two and the brooding by life stress interaction was entered at step three (see Table 3.7). Step three of the model failed to reach significance indicating that life stress and brooding rumination did not interact to predict depression at time two. However, step two of the model explained 63% (adjusted R^2) of the variance (R^2 = .643, p = .001, F (3,72) = 42.641, p < .001). Life stress (β = .203, p = .007) and brooding rumination (β = .260, p = .016) directly predicted depression at time two. This finding indicates that while brooding predicted depression three months later, it did not act as a diathesis within the diathesis-stress framework.

Table 3.7

Hierarchical linear regression model with depression (time two) as the outcome variable and brooding, life stress and the brooding x life stress interaction

	Predictor	Adj. R^2	R^2	ΔR^2	β	t	*p*
Step 1		.560	.566				<.001
	Beck Depression Inventory (Time 1)				.752	9.815	<.001
Step 2		.625	.640	.074			.001
	Beck Depression Inventory (Time 1)				.511	4.818	<.001
	Life Stress (Time 2)				.203	2.778	.007
	Brooding Rumination				.260	2.464	.016
Step 3		.623	.643	.003			.424
	Beck Depression Inventory (Time 1)				.500	4.665	.<001
	Life Stress (Time 2)				.385	1.616	.111
	Brooding Rumination				.360	2.206	.031
	Brooding Rumination x Life Stress				-.228	-.804	.424

NB. Adj. R^2 = Adjusted R^2

Discussion

Study Two tested whether three trait measures of emotion regulation predicted depression within the diathesis-stress framework. The measures of emotion regulation recorded at time one were spontaneous emotion regulation as measured by startle modulation, regional EEG activity, and brooding rumination. At a three month follow-up (time two), stress and depression were recorded. Time one measures of emotion regulation were then tested to examine whether they acted as a diathesis (sensitivity to stress) to predict depression. It was predicted for each of the trait measures of emotion regulation that, should they act as a diathesis to depression, they would interact with life stress to predict depression at time two. The core findings were that: (1) spontaneous emotion regulation acted as a diathesis within the diathesis-stress framework; (2) regional EEG activity did not predict depression either directly, or within the diathesis-stress framework; and (3) brooding rumination predicted depression directly, but not within the diathesis-stress framework.

Depression symptoms at time one significantly predicted depression symptoms at time two, showing that these individuals manifested moderate stability of depressive symptoms over three months. Additionally, life stress significantly predicted increases in depression at time two (when controlling for time one depression). Of note was the finding that depressive symptoms showed a marked increase between time one to time two. My data does not speak to why this may be, however, the population used (young women starting their first year of university) were selected as they were engaged in a developmental period of change and thus likely to encounter experiences that may have been particularly stressful (Schulenberg et al., 2004). The positive correlation observed between time one depressive symptoms and time two life stress indicates that more depressed individuals may go

on to experience (or at least report) more life stress. It should be noted that life stresses are not simply random events; some people may engage in behaviours that make them more or less likely to experience stress, or to feel stressed by those experiences. Depression may also cause people to remember events as more stressful. Thus depression and stress may be interconnected across these two time points. These findings do indicate that life stress was an important factor in the perpetuation of depressive symptoms in the current sample, but also show that the issue of causality may be more complex than is suggested by the simple prediction of stress to depression indicated here.

Spontaneous Emotion Regulation

Spontaneous emotion regulation was proposed to act as a diathesis to depression. Indeed, spontaneous emotion regulation, as indexed by the post-image startle eye-blink, acted as a diathesis within the diathesis-stress framework. Startle responses to the negative post-image probes interacted with life stress to predict increases in depression at time two. As can be seen in Figure 3.1, poorer emotion regulators were more sensitive to stress, and showed larger increases in depression when stress was experienced. However, good emotion regulators were more resilient in the face of stress and did not show significant increases in depression, regardless of life stress. This finding is important as, to the best of my knowledge, this is the first longitudinal study to test this relationship between spontaneous emotion regulation, stress, and depression, and supports theoretical models of emotion regulation operating as a vulnerability marker in the diathesis stress model (e.g. Davidson 1998; Gross, 2013). These findings also show that startle measures of online spontaneous regulation can predict depression, providing an objective and useful alternative to instructed emotion regulation paradigms.

Regional EEG Activity

Frontal and parietal asymmetries did not directly predict depression, nor did they interact with stress to predict depression. As such, these particular regional EEG markers do not seem to act as a diathesis in the diathesis-stress model; at least at a three month follow-up time point. Although this result is inconsistent with previous reports of frontal asymmetry predicting depression, an important distinction is that the present study uses a three month follow-up period, whereas previous longitudinal studies have used longer follow-up periods (twelve months: Pössel et al., 2008, Mitchel & Pössel, 2012; and 3 years: Nussock et al., 2011). Therefore, it may be that regional EEG activity is only predictive over a longer period of time, when ample opportunity to experience life stress is allowed for. On the other hand, failure to find a predictive relationship between frontal asymmetry and depression across a twelve month period has also been reported (e.g. Blackhart et al., 2006).

Brooding Rumination

Brooding rumination was a direct predictor of depression at the short-term three month follow-up. This result replicates a number of findings (Burwell & Shirk 2007; Mezulis et al. 2011) showing that brooding predicts depression. This finding is particularly interesting given that brooding rumination was the only proposed diathesis that directly predicted depression in the current study. It may be that measures of brooding rumination tap, to some degree, into current depression symptomology, as well as reflecting a trait vulnerability to depression. This perspective is further supported by the findings of Study One where measures of brooding strongly correlated with current depression ($r(74) = .66$). However, as brooding rumination at time two was not measured, the extent to which brooding and depression scores covary across time could not be analysed.

Whilst brooding directly predicted depression, it did not act as a diathesis within this diathesis-stress model. As discussed in regards to regional EEG activity, it may be that a three month follow-up did not allow for sufficient stress to be experienced to reveal a diathesis in this variable. However, Cox and colleagues (2011) found brooding rumination acted as a diathesis at three months. Further, when controlling for BDI-II scores at time one, stress significantly predicted BDI-II scores at time two in the current study. These results suggest that in the three month period between time one and time two the levels of stress experienced did affect depression scores (i.e., three months was a sufficient time period to experience a certain amount of life stress, and for the stress to predict more depressive symptoms). This finding suggests that there was enough variance in life stress to detect changes in depression, however there may not have been enough to detect changes in depression based on the interaction between brooding and life stress.

Although previous findings of brooding interacting with stress to predict depression at a three month period were not replicated, it should be noted that the current sample consisted of a 76 young adults (aged 18 – 24) whereas past studies investigating brooding rumination within the diathesis-stress model have examined the relationship in adolescents (Bastin et al., 2014; Cox et al., 2011) and in larger samples. However, Paredes and Zumalde (2014) reported results consistent with the current study – brooding directly predicted depression, but was not a diathesis – in a large population of adolescents. Based on the mixed patterns of findings, it appears that the relationship between brooding rumination and stress to predict depression is inconsistent, indicating that other factors (such as developmental period) may moderate how brooding rumination operates within the diathesis-stress model.

Summary

The current study aimed to test whether three trait measures of emotion regulation acted as diatheses within the diathesis-stress framework to predict depression, or predicted depression directly, at a three month follow-up. Brooding rumination was the only measure that directly predicted increases in depressive scores. Spontaneous regulation of emotion, as indexed by the startle eye blink, was the only proposed trait that acted as a diathesis within the diathesis-stress framework (poorer regulators were more sensitive to stress, and showed increasing depressive symptoms with higher stress levels). However, it could be that a longer term follow-up, that allows for more life stress to occur, may untangle whether regional EEG measures (frontal and parietal asymmetries) and brooding rumination also act as diatheses to depression.

Chapter Four

Study Three: The Diathesis Stress Model at Twelve Months

In Study three, I tested the three proposed diatheses – spontaneous emotion regulation, regional EEG activity, and brooding rumination – as predictors of depression within the diathesis-stress model at a twelve month follow-up. In Study Two the diathesis-stress model was tested in a three month follow-up, and revealed that spontaneous emotion regulation, as measured by the startle eye-blink response, acted as a diathesis while the other proposed trait vulnerabilities did not act as diatheses. However, it is possible that some diatheses are only apparent across a longer period, when enough time has elapsed for sufficient stress to be experienced and depressive symptoms to manifest. To test this possibility the current study replicated the design of Study Two but tested the proposed traits within the diathesis-stress model as predictors of depression at twelve months from time one.

Study Two found that regional EEG activity did not act within a diathesis-stress framework to predict depression, nor did it directly predict depression. The latter finding is inconsistent with evidence suggesting that frontal asymmetry predicts depression (Mitchell & Pössel, 2012; Nusslock et al., 2011; Pössel et al., 2008;). This disparity in findings may be due to differences in the follow-up period; two previous studies have used a twelve month follow-up (Mitchell & Pössel, 2012; Pössel et al., 2008), and one study has used a three year follow-up period (Nusslock et al., 2011). In light of the failure to replicate findings from these studies at a considerably shorter three month follow-up (reported in Study Two), a longer period is warranted.

Prospective studies examining the relationship between brooding rumination and depression, both directly and within the diathesis-stress framework, have yielded mixed findings. Bastin and colleagues (2014) found that over a period of twelve

months brooding rumination directly predicted depression and operated as a diathesis within the diathesis-stress framework. Similarly, Cox and colleagues (2011) found that brooding operated as a diathesis in a three month follow-up period, but reported that brooding did not directly predict depression. In a six month follow-up study, Praedes and Zumalde (2014) failed to find that brooding rumination moderated the stress depression relationship, though they did find that brooding directly predicted depression. Taken together, it is unclear how brooding rumination relates to depression over time.

No previous studies have examined spontaneous emotion regulation, as indexed by the startle reflex, as a predictor of depression over time, directly or within the diathesis-stress framework. However, spontaneous emotion regulation was the only proposed trait marker of vulnerability to depression that acted as a diathesis within the diathesis-stress framework at the three month follow-up. As such, this marker appears to reflect sensitivity to stress, at least over a relatively short (three month) period of time. However, the reliability of this marker as a diathesis within the diathesis-stress framework to predict depression over a longer period of time is still unknown.

Study Three

The aim of Study Three was to the test trait emotion regulation markers – spontaneous emotion regulation, regional EEG activity, and brooding rumination – within the diathesis-stress framework across a twelve month period. The same design was used as for Study Two but at 12 (rather than three) months. The proposed diatheses were measured at time one (as reported in Study One) and stress and depression were recorded twelve months later, using an online questionnaire. Participants from time one were contacted at twelve months with an invitation to complete an online survey. A number of participants from time one responded at both

time two and time three, however, a significant proportion of time three participants did not complete the time two survey. For this reason a follow-up investigation across all three time points was not possible. Rather, Study Three complemented Study Two as an additional follow-up study of the participants from Study One.

Hypotheses

Spontaneous emotion regulation. No previous studies have investigated startle measures of spontaneous emotion regulation in a twelve month follow-up design. However, based on the results of Study Two the same hypotheses were adopted as proposed and supported in Study Two, that spontaneous emotion regulation (as measured by startle reactivity to the post-image probe) at time one would interact with stress to predict depression at time three.

Regional EEG activity.

Frontal asymmetry. Based on the same reasoning presented in Study Two, frontal asymmetry was expected to directly predict depressive symptoms. No studies have tested resting frontal asymmetries across twelve months within the diathesis-stress framework. However, if frontal asymmetry acts as a diathesis within the diathesis-stress framework then frontal asymmetry should interact with life stress to predict depression in the manner hypothesised in Study Two.

Parietal asymmetry. As described in Study Two, it was expected that parietal asymmetry would predict depression directly, such that lower right parietal activity would predict increases in depressive symptoms at twelve months. It is unknown whether right parietal activity operates as a diathesis within the diathesis-stress model. However, if it does then individuals with low right parietal activity may be more sensitive to stress and thus would show increases in depression when life stress is experienced. Anxious arousal has been proposed to moderate the relationship between

parietal asymmetry and depression (see Heller & Nitschke, 1998; Stewart et al. 2011) such that higher right parietal activity would be observed in depression that is comorbid with anxious arousal. This relationship was observed in Study Two and therefore anxious arousal was statistically controlled for in all analyses that included parietal asymmetry as a predictor of depression.

Frontal by parietal interaction. No studies have reported the relationship between frontal and parietal asymmetries as direct predictors of depression or as interacting with stress to predict depression. Based on the relationships proposed by Heller's Circumplex Model (1993), it may be that individuals with low right parietal activity and a rightward frontal asymmetry show increased depression at the twelve month follow-up. Further, low right parietal activity and rightward frontal asymmetry may act as a diathesis and interact with stress to predict depression. In contrast, individuals with low right parietal activity and leftward frontal asymmetries are expected to be resilient to stress and thus show less increases in depressive symptoms when stress is experienced.

Brooding rumination. Longitudinal studies examining the relationship between brooding rumination and depression, both directly and within the diathesis-stress framework within a twelve month follow-up period have shown mixed findings (Bastin et al., 2014; Cox et al., 2011; Praedes & Zumalde, 2014). Based on the findings of Study Two, brooding rumination was expected to directly predict depression. Further, if brooding rumination acts as a diathesis, then brooding rumination at time one should interact with stress to predict depression at time three, in the manner described in Study Two.

Method

The method of Study Three mirrored that of Study Two with the exception that the participants were invited to complete the follow-up survey twelve months from time one, rather than three months.

Participants

Sixty three individuals chose to participate in Study Three. Of the 115 individuals who participated in Study One 113 were invited complete Study Three as two individuals asked to be removed from the database at Study Two. Of these, 46 had also participated in the three month follow-up. Thus, although there was some overlap between the two samples, 30 people participated at 3 months but not at twelve months, and 17 people participated at twelve months but not at three months. All participants were female, right handed, and reported no history of previous depression or neurological disorder at time one. They were aged 19 to 24 years ($M = 19.84$, $SD = 1.32$).

Questionnaire Measures

The same measures were used as those reported in Study Two. These were the BDI-II (Beck et al., 1996), the Life Event Questionnaire – modified for use with female respondents (Nordbeck, 1984), and the anxious arousal subscale of the mini-MASQ (Clark & Watson, 1995).

Procedure

The procedure was identical to that of Study Two. The participants from Study One were emailed twelve months from time one with an invitation to participate in the follow-up survey. A link to the survey was included in the email. Individuals who chose to participate followed the link to the online survey, which was hosted on surveymonkey.com. Informed consent was obtained before completing the survey.

Questionnaires were completed in the following order: Life Event Questionnaire; BDI-II; and mini-MASQ. Participants were sent a movie voucher to thank them for their participation.

Results

The sample comprised 55% of the participants from time one. Differences were assessed between time one variables for participants who responded at time three (responders) and those who did not (non-responders). No differences were found for: parietal asymmetry, $t(113) = -.457$, $p = .649$; BDI-II at time one, $t(113) = 1.014$, $p = .313$; brooding rumination, $t(113) = .885$, $p = .378$; and emotional reactivity at the post-image startle probe, $t(113) = -.032$, $p = .974$. However, time three responders and did differ from non-responders in average frontal asymmetry scores, $t(113) = 2.649$, $p = .009$, $d = .489$, such that those who replied to the time three follow-up survey (M = -.049, SD = .093) had, on average, more rightward asymmetry scores than those who did not reply (M = -.004, SD = .091). This difference needs to be considered when interpreting the findings of Study Three and is discussed below. Descriptive statistics for all Study Three variables are reported in Table 4.1 for both responders and non-responders.

The correlations between Study Three variables are presented in Table 4.2. Brooding at time one positively correlated with anxious arousal at time three ($r(74) = .289$, $p = .027$), indicating that individuals who brooded more at time one also reported more anxious arousal at time three. Life stress positively correlated with depression at time three ($r(74) = .406$, $p = .001$), indicating that those participants who were experiencing more life stress at time three also reported more depressive symptoms at time three. In line with the findings of Study Two, anxious arousal at time three showed a strong positive correlation with depression at time three ($r(74) =$

.548, $p < .001$), indicating that individuals who reported experiencing more anxious arousal also reported more depressive symptoms. Anxious arousal also showed a strong positive correlation with life stress ($r(74) = .454$, $p < .001$), indicating that individuals experiencing more anxious arousal also experienced more life stress.

Table 4.1
Descriptive Statistics for Study Three Variables for both Responders and Non-Responders.

Variable Name	Time Two Responders (N = 63)		Time Two Non-Responders (N = 52)	
	Mean	SD	Mean	SD
Frontal Asymmetry	-.0497	.0927	-0.0042	.0906
Parietal Asymmetry	.0690	.3598	.0416	.6237
Post-Image Emotion Reactivity	.1556	5.761	-0.0132	5.909
Brooding Rumination	9.048	2.825	9.558	3.357
Beck Depression Inventory (Time 1)	6.603	5.701	7.615	4.831
Beck Depression Inventory (Time 3)	8.508	8.232	-	-
Life Stress (Time 3)	20.48	15.043	-	-
Anxious Arousal (Time 3)	13.63	5.401	-	-

Changes in Depression

A paired samples t-test was conducted to compare BDI-II scores at time one with those at time three. BDI-II scores at time three (M = 8.51, SD = 8.23) were significantly higher than at time one (M = 6.60, SD = 5.70), $t(75) = 2.13$, $p < .038$, $d = .504$. These results show that higher levels of depression were experienced in the sample at time three compared to time one.

Life Stress and Depression

Life stress was tested as a predictor of changes in depression between time one and time three (see Table 4.3.). Depression at time three was entered as the dependent measure. Depression at time one was entered at step one as a covariate, and life stress

Table 4.2

Correlations Among Study Three Variables

	Variable	1	2	3	4	5	6	7	8
1	Frontal Asymmetry	1							
2	Parietal Asymmetry	-.274**	1						
3	Post-image Emotion Reactivity	-.046	-.251*	1					
4	Brooding Rumination	.107	.110	-.108	1				
5	Beck Depression Inventory (Time 1)	.139	-.032	-.281	.671**	1			
6	Beck Depression Inventory (Time 3)	.145	-.052	-.166	264*	.529**	1		
7	Life Stress (Time 3)	.044	-.177	-.001	.112	.329**	.406**	1	
8	Anxious Arousal (Time 3)	.165	-.012	-.187	289*	.481**	.548**	.454**	1

NB: ** $p < .01$ * $p < .05$

was entered at step two. This model ($R^2 = .340$, $p = .022$, F (2,60) = 15.646, $p < .001$) accounted for 32% (adjusted R^2) of that variance and showed that both depression at time one (β=.443, $p < .001$) and life stress at time three ($\beta = .260$, $p = .022$) significantly predicted depression at time three.

Table 4.3

Hierarchical linear regression model with depression (time 3) as the outcome variable and life-stress (time 2) as the predictor.

	Predictor	Adj. R^2	R^2	ΔR^2	β	t	*p*
Step 1		.268	.280				<.001
	Beck Depression Inventory (Time 1)				.529	4.866	<.001
Step 2		.318	.340	.061			.022
	Beck Depression Inventory (Time 1)				.443	3.992	<.001
	Life Stress (Time 3)				.260	2.346	.022

Tests of the Diathesis-Stress Model

Spontaneous emotion regulation. A three-step hierarchical linear regression was used to test the relationship between spontaneous emotion regulation and life stress on depression. Depression at time three was entered as the outcome variable. Depression at time one was entered at step one of the regression as a covariate. Life stress and post-image emotional reactivity was entered at step two. The interaction between spontaneous emotion regulation and life stress was entered at step three to test whether spontaneous emotion regulation acted as a diathesis at the twelve month follow-up. Table 4.4 shows the results of this model. The model failed to reach significance, indicating that emotion reactivity at the post-image probe did not predict depression twelve months later either directly or within the diathesis-stress framework.

Table 4.4
Hierarchical linear regression model predicting depression at time three with life stress and emotion reactivity at the post-image probe, and the interaction between post-image reactivity and life stress.

	Predictor	Adj. R^2	R^2	ΔR^2	β	t	*p*
Step 1		.268	.280				<.001
	Beck Depression Inventory (Time 1)				.529	4.866	<.001
Step 2		.312	.345	.066			.060
	Beck Depression Inventory (Time 1)				.425	3.710	<.001
	Life Stress (Time 3)				.266	2.380	.021
	Post image Unpleasant Reactivity				-.073	-.676	.501
Step 3		.333	.376	.031			.096
	Beck Depression Inventory (Time 1)				.440	3.886	<.001
	Life Stress (Time 3)				.304	2.704	.009
	Post image Unpleasant Reactivity				-.324	-1.776	.081
	Post image Unpleasant Reactivity x Life Stress (Time 3)				.312	1.694	.096

NB. Adj. R^2 = Adjusted R^2

Regional EEG activity. A four-step hierarchical linear regression was run to test the relationship between frontal asymmetry, parietal asymmetry, and life stress in predicting depression. Depression at time three was entered as the dependent variable. Depression at time one and anxious arousal at time three were entered as covariates at step one. The main effects of life stress, frontal asymmetry, and parietal asymmetry were entered at step two. Three two-way interactions were then entered at step three, frontal asymmetry x parietal asymmetry, frontal asymmetry x life stress, and parietal asymmetry x life stress. Finally, the three-way interaction was entered at step four.

The results of this model are presented in Table 4.5. Step four of the model (R^2 = .537, p = .002, F (9,53) = 6.842, p < .001) explained 46% (adjusted R^2) of the variance. Addition of the three-way interaction at step four between life stress, frontal asymmetry, and parietal asymmetry improved the models prediction (ΔR^2 = .092, β = -.838, p = .002). Significant two-way interactions were also observed at step four of this model for parietal asymmetry x life stress (β = -.558, p = .007), frontal asymmetry x life stress (β = -.489, p = .026), and frontal asymmetry x parietal asymmetry (β = .692, p = .038). Step one of this model was also significant (R^2 = .392, p < .001, F (2,60) = 19.344, p < .001), showing that depression at time one (β = .384, p = .004) and anxious arousal at time three (β = .342, p = .001) were both direct predictors of depression at time three. Step two and step three of this model failed to significantly predict depression.

All of the main effects and two-way interactions observed in step 4 are subsumed within the three-way interaction; therefore, the three-way interaction was probed further. To deconstruct the three-way interaction, the sample was median split by right parietal asymmetry. The interaction between frontal asymmetry and life stress was then examined independently for the high and low right parietal activity

Table 4.5

Hierarchical linear regression model predicting depression at time three with life stress, frontal asymmetry, and right parietal activity, and their interactions as predictors

	Predictor	Adj. R^2	R^2	ΔR^2	β	t	p
Step 1		.372	.392				<.001
	Anxious Arousal (Time 3)				.342	3.330	.001
	Beck Depression Inventory (Time 1)				.384	2.964	.004
Step 2		.359	.411	.019			.608
	Anxious Arousal (Time 3)				.319	2.504	.015
	Beck Depression Inventory (Time 1)				.318	2.702	.009
	Life Stress (Time 3)				.152	1.282	.205
	Frontal Asymmetry				.036	.335	.739
	Parietal Asymmetry				-.005	-.048	.962
Step 3		.363	.445	.034			.356
	Anxious Arousal (Time 3)				.351	2.279	.009
	Beck Depression Inventory (Time 1)				.358	2.979	.004
	Life Stress (Time 3)				.083	.602	.550
	Frontal Asymmetry				.234	1.146	.257
	Parietal Asymmetry				.025	.099	.922
	Frontal Asymmetry x Life Stress (Time 3)				-.217	-1.022	.311
	Parietal Asymmetry x Life Stress (Time 3)				-.191	-1.082	.284
	Frontal Asymmetry x Parietal Asymmetry				-.188	-.961	.341
Step 4		.459	.537	.092			.002
	Anxious Arousal (Time 3)				.243	1.968	.054
	Beck Depression Inventory (Time 1)				.470	4.053	<.001
	Life Stress (Time 3)				.029	.224	.823
	Frontal Asymmetry				.296	1.563	.124
	Parietal Asymmetry				.519	1.853	.070
	Frontal Asymmetry x Life Stress (Time 3)				-.489	-2.294	.026
	Parietal Asymmetry x Life Stress (Time 3)				-.558	-2.821	.007
	Frontal Asymmetry x Parietal Asymmetry				.692	2.127	.038
	Frontal x Parietal x Life Stress				-.838	-3.252	.002

NB. Adj. R^2 = Adjusted R^2

Table 4.6

Hierarchical linear regression models predicting depression at time three with life stress, frontal asymmetry, and their interactions for both high and low right parietal activity groups

	Predictor	Adj. R^2	R^2	ΔR^2	β	t	p
		High Right Parietal Activity					
Step 1		.371	.411				<.001
	Anxious Arousal (Time 3)				.471	3.060	.005
	Beck Depression Inventory (Time 1)				.292	1.897	.068
Step 2		.442	.514	.102			.076
	Anxious Arousal (Time 3)				.254	1.483	.150
	Beck Depression Inventory (Time 1)				.248	1.699	.101
	Life Stress (Time 3)				.369	2.279	.031
	Frontal Asymmetry				.176	1.228	.230
Step 3		.474	.559	.045			.116
	Anxious Arousal (Time 3)				.114	.612	.546
	Beck Depression Inventory (Time 1)				.241	1.695	.102
	Life Stress (Time 3)				.539	2.856	.008
	Frontal Asymmetry				-.160	1.228	.230
	Frontal Asymmetry x Life Stress (Time 3)				.462	1.626	.116
		Low Right Parietal Activity					
Step 1		.348	.391				.001
	Anxious Arousal (Time 3)				.332	1.911	.066
	Beck Depression Inventory (Time 1)				.383	2.203	.036
Step 2		.299	.393	.001			.969
	Anxious Arousal (Time 3)				.332	1.742	.093
	Beck Depression Inventory (Time 1)				.387	2.086	.047
	Life Stress (Time 3)				.009	.051	.959
	Frontal Asymmetry				-.039	-.248	.806
Step 3		.444	.537	.144			.010
	Anxious Arousal (Time 3)				.244	1.416	.169
	Beck Depression Inventory (Time 1)				.572	3.212	.004
	Life Stress (Time 3)				-.290	-1.526	.140
	Frontal Asymmetry				.526	2.142	.042
	Frontal Asymmetry x Life Stress (Time 3)				-.734	-2.790	.010

NB. Adj. R^2 = Adjusted R^2

groups using two separate three-step hierarchical linear regressions. As before, anxious arousal at time three and depression at time one were entered as covariates at step one. The direct predictors, life stress and frontal asymmetry, were entered at step two and the interaction between life stress and frontal asymmetry was entered at step three.

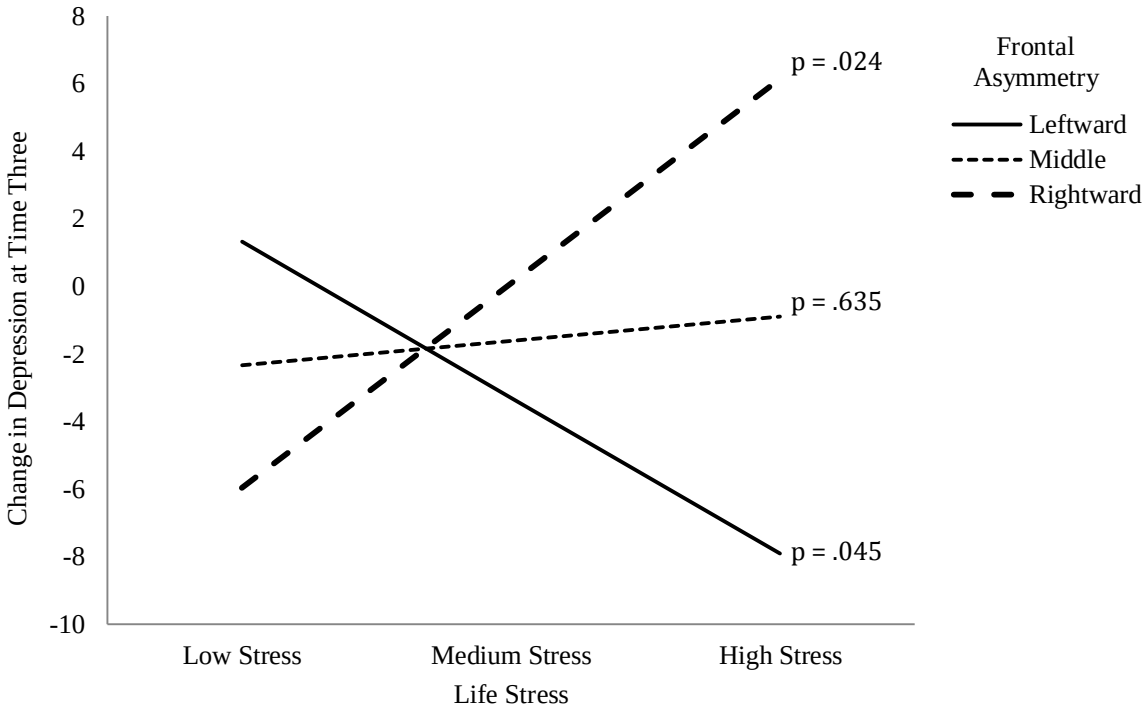

Figure 4.1 Moderation by frontal asymmetry of the relationship between stress and depression for individuals with low right parietal activity (see Table 4.5)

The results of these two models are presented in Table 4.6. Step three of the model for the high right parietal group failed to reach significance indicating that frontal asymmetry and life stress did not interact to predict depression for the high right parietal group. However, for the low right parietal group step three of the model did reach significance (R^2 = .537, p = .010, F (5,25) = 5.798, p = .001), explaining 44% (adjusted R^2) of the variance. This model yielded a significant interaction between frontal asymmetry and life stress that improved the models prediction at step three (ΔR^2 = .144, β = -.734, p = .010) to predict depression. This interaction was

plotted using ModGraph (Jose, 2013) and is presented in Figure 4.1. As predicted by the Circumplex Model (Heller, 1993), for individuals with *low* right parietal activity, frontal asymmetry interacted with life stress to predict increases in depression twelve months later. Those individuals who displayed relative rightward frontal asymmetries showed sensitivity to stress, such that as stress increased these women showed increased depressive symptoms over time. In contrast, individuals with relative leftward frontal asymmetries displayed resilience to stress and in fact manifested a decrease in depressive symptoms over time as stress increased.

Table 4.7
Hierarchical linear regression model predicting depression at time two with life stress, brooding, and their interaction.

	Predictor	Adj. R^2	R^2	ΔR^2	β	t	*p*
Step 1		.268	.280				<.001
	Beck Depression Inventory (Time 1)				.529	4.866	<.001
Step 2		.315	.348	.068			.053
	Beck Depression Inventory (Time 1)				.529	3.571	.001
	Life Stress (Time 2)				.247	2.198	.032
	Brooding Rumination				-.122	-.849	.399
Step 3		.309	.353	.005			.500
	Beck Depression Inventory (Time 1)				.528	3.496	.001
	Life Stress (Time 2)				.520	1.244	.218
	Brooding Rumination				-.010	-.047	.963
	Brooding Rumination x Life Stress				-.315	-.678	.500

NB. Adj. R^2 = Adjusted R^2

Brooding rumination. Brooding rumination was also tested at twelve months within the diathesis-stress framework using a hierarchical linear regression. Depression at time one was entered into the model at step one. Life stress and brooding rumination were entered at step two. The brooding x life stress interaction

term was entered at step three. This model (see Table 4.7) did not reach significance at step three showing that brooding rumination did not interact with life stress to predict depression at time three. Step two of the model was marginally significant (R^2 = .348, p = .053, F (3,59) = 10.502, p = .001), but this result was driven by depression at time one (β =.529, p = .001) and life stress (β =.247, p = .032) predicting depression at time three. Brooding rumination did not directly predict depression at time three in this model (β = -.122, p = .399).

Discussion

Study Three assessed three trait measures of emotion regulation as potential diatheses in the diathesis-stress model. The measures of trait emotion regulation recorded at time one were tested as predictors of depression one year later. The important findings of this study were: (1) regional EEG activity acted as a diathesis, such that individuals with low right parietal activity and rightward frontal asymmetry were more sensitive to stress; (2) spontaneous emotion regulation did not act as a diathesis at twelve months, contrary to the findings of the three month follow-up; and (3) brooding rumination did not act as a diathesis at twelve months, consistent with the findings of the three month follow-up.

Spontaneous Emotion Regulation

Spontaneous emotion regulation (post-image startle reactivity) did not act as a diathesis within the diathesis-stress model at the twelve month follow-up, whereas it did act as a diathesis at the three month follow-up reported in Study Two. This difference indicates that online regulatory processes may be more informative in regards to depression for shorter time periods. Alternatively, the very large increase in depressive symptoms at time two may reflect the notion, proposed earlier, that as this population of first year at university students are likely to be experiencing a number

life changes (Schulenberg et al., 2004) the three month follow-up time point may be tapping into the initial response (maybe shock) these experiences; first time away from home and previous social support, failing an assessment, trying to make new friends. On the other hand, the one year follow-up may provide a more stable measure of stress and depression, as individuals are more likely to have settled into the routine of university and because the one year follow-up is at a similar period of the academic year to their initial assessment. These hypotheses cannot be tested in the current study but may be a useful direction for future research.

Regional EEG Activity

Patterns of regional EEG activity predicted sensitivity to stress. It was found that individuals with low right parietal activity and rightward frontal asymmetry reported increased depressive symptoms when they experienced stress in their lives. However, individuals with low right parietal activity and leftward frontal asymmetry actually showed lower depressive symptoms when stress increased. Individuals with high right parietal activation showed no relationship between frontal asymmetry and life stress. Although Pössel and colleagues (2008) found that *high* right parietal activity predicted depression twelve months later, they had not predicted this result and suggested that anxious arousal might have mediated this relationship. In line with this possibility, the current study controlled for anxious arousal and found no relationship between parietal asymmetry and depression. This result seems to provide indirect support for a modified version of Heller's Circumplex model (Heller & Nitschke, 1998); when the effects of anxious arousal are accounted for, high right parietal activity is not related to depression. However, the current study did not set out to test the relationship between parietal asymmetry and anxious arousal – we did not measure anxious arousal at time one. As such, future studies should aim to clarify the

relationship between parietal asymmetry and anxious arousal, as well as the role of anxious arousal in the relationship between parietal asymmetry and depression.

One possible explanation for why such a pattern of regional EEG activity might predispose an individual to develop depression is that regional EEG activity reflects cognitive processing styles that bias the processing of emotional information in less adaptive ways. In support of this hypothesis, many studies report that emotional processing biases are key factors in the development and maintenance of depression (see De Raedt & Koster, 2010). Furthermore, this same frontal x parietal interaction was observed in a task measuring attention disengagement from angry faces (Grimshaw et al., 2014). In this experiment individuals with low right parietal activity and rightward frontal asymmetry were slow to disengage from threatening information (angry faces), a cognitive bias that is associated with depression (De Raedt & Koster, 2010). While this characterized pattern of frontal-parietal activity did not directly predict depression in the current study, it did predict depression within the diathesis-stress framework. As such, low right parietal activity in conjunction with a rightward frontal asymmetry seems to act as a diathesis. Interestingly, when only the low right parietal group was considered relative rightward frontal asymmetry predicted depression both directly and within a diathesis-stress framework.

A caveat to the current findings is that the population of individuals who responded to the follow-up survey at time three differed systematically in frontal asymmetry from the portion of the sample who did not respond. The sample included in this study had, on average, a more rightward frontal asymmetry. This bias limits the extent to which the present results can be compared to the results of time two (the three month follow-up), which found no relationship between regional EEG activity and depression. It may be that because this sample included more people with

rightward asymmetry, it was better able to pick up the relationship between frontal asymmetry and stress sensitivity. Although this difference in frontal asymmetry scores is important to bear in mind, it does not completely undermine the current results. Current findings were predicted on the basis of, and are to some extent consistent with, theoretical explanations of frontal asymmetry.

Brooding Rumination

Brooding rumination did not act as a diathesis of stress sensitivity, in line with findings from the short term follow-up period. Additionally, brooding rumination failed to predict depression directly, which was the case at the three month follow-up as well. Previous studies have found brooding to be either a direct predictor of depression or a diathesis within the diathesis-stress model (e.g. Bastin et al., 2014; Cox et al., 2011, Jose et al., 2014; but also see Paredes & Zumalde, 2014). However, all of the prospective studies that have examined the relationship between brooding and depression have used younger, adolescent populations. It is conceivable that brooding rumination is a less robust predictor of depression across time in later developmental periods. It should again be noted that the current sample (of 63) is considerably smaller than other longitudinal studies of brooding rumination, and as such may lack the necessary power to show such relationships across a long period of time.

Summary

The current study was designed to test three diatheses within the diathesis-stress model at a twelve month follow-up period. It was found that regional EEG activity predicted stress sensitivity, such that individuals with low right parietal activity and rightward frontal asymmetry were more likely to show increases in depressive symptoms (than individuals with other patterns of regional EEG activity) when they

experienced stressful events. In contrast to the findings of Study Two, spontaneous emotion regulation did not act as a diathesis within the diathesis-stress framework. It was also found that brooding rumination did not reflect a diathesis for depression. Interestingly, none of the proposed diatheses directly predicted depression at time three.

Chapter Five

General Discussion

In this thesis I examined three trait measures of emotion regulation proposed to be vulnerability markers of depression. At time one, three emotion regulation measures were assessed: frontal and parietal activity as indexed by regional EEG recordings, spontaneous emotion regulation as measured by modulation of the startle eye-blink reflex, and brooding rumination as measured by self report. For the most part, these three measures had been researched independently of one another, and so it has not been clear whether these markers reflect different manifestations of the same underlying vulnerability to depression, or are independent vulnerability markers. In Study One relationships between these markers were assessed, and results indicated that they represent unique vulnerability markers of depression.

In Study Two (three month follow-up) and Study Three (twelve month follow-up), I tested whether these emotion regulation measures prospectively predicted depression directly, and within the diathesis-stress framework. Consistent with the commonly observed stress-depression relationship (e.g., Hammen, 2015), highlighting the key role of stress in depression, higher levels of life stress directly predicted increased depression symptoms, both three months and twelve months after the initial experimental session. Importantly, the core findings of these follow up studies in regards to the trait emotion regulation measures were: 1) a frontal by parietal interaction acted as a diathesis within the diathesis-stress model to predict depression at 12 months; 2) spontaneous emotion regulation acted as a diathesis within the diathesis-stress model to predict depression at three months; and 3) brooding rumination was the only trait that directly predicted depression, and this was only true for the three month follow up time point.

Frontal Asymmetry

Frontal asymmetry did not relate to current depression symptoms at time-one. This is not unexpected. Vulnerability markers to depression are stable traits that are present prior to the experience of depression (Ingram, Atchley, & Segal, 2011). They do not necessarily relate to current depressive symptoms in a healthy population, but rather are expected to predict later development of depression. Therefore, the lack of a relationship between frontal asymmetry (a vulnerability) and depression at time one is not unexpected and is consistent with previous research (Nusslock et al., 2011; Mitchell & Pössel, 2012).

A particularly surprising, and more important, finding across my studies is that frontal asymmetry did not predict depression directly, or within the diathesis-stress model at time two or time three. This does not support the idea that frontal asymmetry is a vulnerability marker for depression, which is particularly notable given that other prospective studies have found that frontal asymmetry directly predicts depression (Mitchell & Pössel, 2011; Nuslock et al., 2011; Pössel et al., 2008). Differences in three methodological factors could explain the discrepancy between my findings and the findings of previous studies. These factors include the measure used to assess depression, the sample population, and the length of the follow-up periods.

The current thesis used the BDI-II to assess depression, a sample of young healthy women (aged 18 to 24), and follow-up at two later time points (three months and twelve months). These factors differ somewhat to previous studies. Nusslock et al. (2011) used depression diagnosis, rather than changes in self reported depressive symptoms, as the follow up measure of depression. It is possible that frontal asymmetry is sensitive to larger scale shifts in depression, rather than more subtle changes in depressive symptoms, as measured by the BDI-II. However, inconsistent

with this idea, Pössel and colleagues (2008) and Mitchell and Pössel (2012) found that frontal asymmetry predicted these more subtle changes in depressive symptoms, measured using the self-rating questionnaire for depressive disorders (Döpfner & Lehmkuhl, 2000, cited in Pössel et al., 2008 and Mitchell & Pössel, 2012). It could be that the relationship between frontal asymmetry and depression can not be captured using the BDI-II, however, this is unlikely given that the BDI-II is a reliable measurement of depressive symptoms (Beck et al., 1988).

While Pössel and colleagues (2008) and Mitchell and Pössel (2012) did use a self-report measure of depression symptoms to capture more subtle changes in depression, they also used a younger sample (adolescents) than the present study – which could explain why the results of my study differ to theirs. It is possible that frontal asymmetry is a more sensitive measure, or shifts more readily, in younger populations. Furthermore, it is possible that adolescents fluctuate more in depressive symptoms across a twelve month period – the length of the follow up used by both Pössel et al., (2008), Mitchell & Possel (2012) and the present study. Indeed, with a sample of young adults of comparable age to the present study, Nusslock and colleagues (2011) used a longer three year follow up. It could be that the present study did not find that frontal asymmetry predicted depression across the one year follow-up as frontal asymmetry is not a sensitive vulnerability measure across this period for young adults (18-24 years old). Perhaps if a longer follow-up time point (e.g., three years) was used, a relationship may emerge. In line with this possibility, Blackhart and colleagues (2006) also found that frontal asymmetry did not predict changes in depression at a twelve month follow up in a population of young adults (18-25 years old). Together, the findings of Blackhart and collegues and my study

suggests that frontal asymmetry is not a vulnerability marker for depression in young adults across a one-year period.

Due to inconsistencies in findings with regards to the relationship between resting EEG and depression, it has more recently been suggested that resting measures are not the best way to index frontal asymmetry (for a review see Coan et al., 2006). Resting frontal EEG measures came from a dispositional model, which suggests that resting asymmetries represent a global tendency to approach or withdraw from situations, which in turn represents a vulnerability to depression (e.g. Davidson, 1998; 2004). While the dispositional model has shown some promise, inconsistent findings have led to the proposal of the response capability model (Coan et al., 2006) - a modified version of the dispositional model. The capability model suggests that patterns of frontal asymmetry measured in one situation (e.g., at rest) will not necessarily generalize to other situations (e.g., the presence of a stressor). The model argues that frontal asymmetry patterns in the presence of an emotional challenge reflects an individual's ability to recruit prefrontal mechanisms in aid of regulating emotional responses and ultimately reflects the degree to which they are vulnerable or resilient to depression in the face of stress.

Recent investigations of the capability model suggest that frontal asymmetries recorded during an emotional challenge are more sensitive to individual differences in current and past depression diagnosis, compared to resting frontal asymmetry (Stewart, Coan, Towers, & Allen, 2014). Frontal asymmetry during emotional challenge was more strongly related to depression status than resting frontal asymmetry. Individuals with a history of depression showed more rightward asymmetry during the emotion challenge, whereas those with no history of depression showed a more leftward asymmetry (Stewart et al., 2014) Additionally, frontal

asymmetry measured during a stressor (threat of shock) was a predictor of emotion regulation, such that more leftward asymmetry predicted more successful regulation, while resting frontal asymmetry failed to account for individual differences in emotion regulation (Goodman, Rietschel, Lo, Costanzo, & Hatfied, 2013). Additionally, in a test of the diathesis-stress model in children with a familial risk of depression, frontal asymmetries during an emotional challenge (emotional film clips) moderated the relationship between stressful events and internalizing symptoms, such that high risk children showed a more rightward asymmetry than low risk peers while watching emotional films (Lopez-Duran et al., 2011). Additionally, Stewart and colleagues (2014) found that frontal asymmetry during emotional challenge is reliable across multiple EEG reference schemes, whereas resting EEG measures were found to be much less reliable across different EEG reference schemes. Together these findings indicate that frontal EEG asymmetries measured during emotional challenge may provide a more sensitive, and reliable, measure of vulnerability to depression.

Two interesting questions arise from the capability model in relation to the findings of the current thesis. First, is frontal asymmetry under emotional challenge related to spontaneous emotion regulation? In other words, would emotion elicited frontal asymmetry reflect an underlying neural index of spontaneous emotion regulation as measured by the startle paradigm? Second, does frontal asymmetry under emotional challenge prospectively predict depression within the diathesis-stress model of depression? This second question is particularly interesting given that vulnerability to depression is considered a latent characteristic (see Ingram et al., 2011), which may not be detectable unless an emotional challenge or stressor is experienced.

Frontal Asymmetry by Parietal Asymmetry

While resting frontal asymmetry alone did not act as a vulnerability marker for depression, it was found that it did predict depression through stress, but only if right parietal activity was also considered. For individuals with *low* right parietal activity, frontal asymmetry predicted depression, both directly and within the diathesis-stress framework, such that more relative rightward frontal asymmetries predicted increases in depression and sensitivity to stress. Those with more leftward frontal asymmetry were resilient to depression and less sensitive to stress. However, for individuals with *high* right parietal activity there was no relationship between frontal asymmetry, stress and future depression symptoms. These findings are partially consistent with Heller's (1993) Circumplex model of depression. Heller suggests that right parietal activity reflects arousal (high activity = high arousal; low activity = low arousal), and that frontal asymmetry reflects valence (leftward = positive; rightward = negative). Within this model, Heller argues that the combination of low right parietal activity (low arousal) and rightward frontal asymmetry (negative valence) reflects depression, while the combination of high right parietal activity (high arousal) and rightward frontal asymmetry (negative valence) reflects anxiety.

The present thesis extends Heller's (1993) model. While Heller primarily focused on the relationship between patterns of frontal by parietal activity and current depression, the present thesis examined how frontal by parietal activity acts as a vulnerability to depression, rather than a current indicator of depression. Results show that trait patterns of activity are present *before* depression occurs, and rather than predicting depression directly, frontal by parietal EEG patterns act a diathesis within the diathesis-stress model. In other words, low right parietal activity and rightward frontal asymmetry reflect stress sensitivity – that is, they affect how individuals respond to stress, which in turn influences depressive outcomes. However, this pattern

of frontal by parietal asymmetries appears to be a relatively distal predictor given that frontal by parietal activity interacted with stress at twelve months but not at three months. Interestingly, frontal and parietal activity did not relate to current depressive symptoms (reported in Study One). This is not necessarily at odds with Heller's model as the present study included only healthy participants, and it is possible that the direct relationship only manifests for clinically depressed individuals. Importantly, frontal by parietal activity is shown here to be a trait emotion regulation marker that acts as a diathesis within the diathesis-stress framework.

The Circumplex model's distinction between depression and anxiety may also help to reconcile the lack of a direct predicted relationship between frontal asymmetry and depression (as described above). Heller and Nitschke (1998) propose that parietal asymmetry dissociates depression and anxiety, such that individuals with depression alone have *low* levels of right parietal activity but individuals with anxiety or anxiety comorbid with depression have *high* levels of right parietal activity. If the current sample all had low levels of anxiety (i.e., all showed low right parietal activity), then rightward frontal asymmetry may have predicted depression. However, if a significant proportion of my sample had higher levels of anxiety, then a direct frontal-asymmetry relationship should not be observed – in fact this relationship should be moderated by parietal asymmetry, which is what was found. However, as no measure of anxiety was included at time one, whether levels of anxiety account for my findings cannot be assessed.

Regional EEG Activity and Attentional Bias

In the current thesis I found that EEG activity predicted depression but did not relate to brooding rumination (the cognitive process measured), which raises the question of what cognitive processes regional EEG measures are tapping? One

possibility, argued to link cognitive and neurobiological vulnerability to depression is diminished attentional control. Diminished attentional control, observed in depression, has been proposed to lead to a bias towards processing negative information by the attentional system (Mathews & MacLeod, 2005). De Raedt and Koster (2010) propose that exposure to stress over time (particularly during early childhood) affects the development of regulatory systems associated with the Dorsolateral Prefrontal Cortex. This leads to reduced control over attentional processes and less ability to control emotional processes under stress. These include reduced ability to inhibit (regulate) negative emotional responses or to orient attention away from negative information. This inability to adaptively control negative information processing then leads to sustained emotional responses to stress and many of the cognitive deficits observed in depression. For example, reduced ability to shift attention from negative events means negative information is maintained for longer in working memory (Joormann & Gotlib, 2008), which drives negative schemas and negative self-beliefs, and increases rumination.

De Raedt and Koster's (2010) attentional control framework may be a useful means of interpreting the results of this thesis. This model provides a useful integration of evidence from cognitive and biological lines of research and highlights the importance of attention in the etiology and presentation of depression. A key factor of De Raedt and Koster's (2010) model is the interaction between frontal control systems and parietal attentional systems. The present thesis showed that combined rightward frontal and low right parietal EEG activity was a vulnerability factor that predicted sensitivity to stress twelve months later. Within the perspective of De Raedt and Koster's model it could be considered that rightward frontal and low right parietal activity are indexing poor attentional control that results in a bias toward

negative emotional material. It may be that this negative attention bias underlies the observed link between regional EEG activity and depressive symptoms twelve months later.

A recent study supports the possibility of a link between frontal and parietal EEG activity and an attentional bias toward negative information. Using a dot-probe task (see MacLeod, Mathews, & Tata, 1986), Grimshaw and colleagues (2014) assessed whether regional EEG activity was related to attentional bias to threat (angry faces). They presented non-informative pairs of picture cues to either side of fixation, followed immediately by a probe stimulus in the location of one of the cues. The pairs of images comprised angry and neutral or happy and neutral faces. Faster responses to probes that appeared in the location previously occupied by an emotional stimulus are thought to index sustained attention to the emotional stimulus (MacLeod et al., 1986). They found that women with rightward frontal asymmetry and low right parietal activity showed an attentional bias to negative (threatening) information. In contrast, women with leftward frontal asymmetry showed no attentional bias, that is, they showed effective attentional control in the presence of threat. In light of the findings of the current thesis, this suggests that rightward frontal asymmetry and low right parietal acitivity indexes both a negative attentional bias and sensitivity to stress over time. Negative attentional biases have been shown to predict depression within the diathesis-stress framework (Beevers & Carver, 2003). It can therefore be hypothesised that rightward frontal asymmetry and low right parietal activity reflects an underlying bias to negative information and that this attentional bias in turn reflects a vulnerability to depression within the diathesis-stress framework.

Although the relationship between frontal (and to some extent parietal) asymmetries and depression have been reported for a number of decades (see Allen

Coan & Allen, 2004; Thibodeau et al., 2006), research has only recently started to address the mechanisms that underlie this relationship. As such, currently there is no direct evidence in support of the hypothesis that frontal and parietal regional EEG activity reflects attentional mechanisms involved in vulnerability to depression. To address this gap, studies are required that simultaneously assess both regional EEG activity and attentional biases within a diathesis-stress framework. While still in its infancy, this line of research could be very valuable for untangling the roles of regional EEG activity and attentional control in vulnerability to depression.

Spontaneous Emotion Regulation

Spontaneous emotion regulation, as indexed by the startle eye-blink, predicted depression at three-months within the diathesis-stress model, consistent with evidence using self-report measures of trait emotion regulation (e.g. Garnefski, Kraaij, & Spinhoven, 2001). This is an important finding as, while habitual regulation processes (such as spontaneous regulation) have been proposed to act as a diathesis to depression (Davidson, 1998), this is the first study to show that an objective measure of habitual emotion regulation (startle indices of online emotion processing) prospectively predicts depression within the diathesis-stress model. Spontaneous emotion regulation did not directly predict depression, indicating that it reflects sensitivity to stress specifically.

Interestingly, patterns of findings suggest that frontal by parietal activity and spontaneous emotion regulation act as independent diatheses. Spontaneous emotion regulation acted as a diathesis only at the three-month follow up, while frontal by parietal activity acted as a diathesis only at the twelve-month follow up. This is not entirely surprising given that in Study One these trait markers of emotion regulation were found to be different manifestations of vulnerability to depression (i.e., no

relationship was observed between frontal by parietal activity and spontaneous emotion regulation). As such, spontaneous emotion regulation appears to be a more proximal vulnerability marker and frontal by parietal activity a relatively more distal marker of stress sensitivity.

The independent nature of frontal by parietal activity and spontaneous emotion regulation does not necessarily mean that frontal by parietal activity does not reflect spontaneous emotion regulation. While the startle eye-blink measure captures variation in effectiveness of emotion regulation strategies, it does not capture *how* the emotional response is regulated. In other words, a variety of strategies could be employed to regulate an emotional response but the startle methodology used is only sensitive to the outcome of this emotion regulation and not the specific strategy employed. For example, one individual may employ an attentional redeployment strategy to regulate their emotional response and another may employ a cognitive reappraisal strategy (to equal or differing effect). It is possible that frontal by parietal activity reflects only some types of emotion regulation strategies. If this were the case, then variation in strategies used by participants in the current study may have masked any relationship between regional EEG activity and emotion regulation. Therefore, any relationship between regional EEG activity and emotion regulation would only be detectable when specific strategies are used.

In light of the capability model and the latent characteristic of vulnerability to depression, an alternative interpretation of the findings of Study Two can be proposed. Spontaneous emotion regulation at time one predicted sensitivity to stress at time two and was interpreted as indexing a proximal vulnerability marker within the diathesis stress framework. However, it could be that the startle measure of spontaneous regulation was tapping an already active (i.e., no longer latent)

vulnerability, instigated in response to stress at time one. In this scenario poorer spontaneous emotion regulation may be indexing currently experienced stress that is yet to be expressed as depressive symptoms. However, these symptoms may have been detectable at the short (three month) follow up. This question cannot be addressed in the current thesis as no measure of life stress was recorded at time one, thus stress at time one could not be controlled for. However, this hypothesis gives an alternative explanation for why poorer spontaneous emotion regulation interacted with stress to predict depression at time two.

Brooding Rumination

Brooding rumination was the only marker that did not act as a diathesis within the diathesis-stress model, suggesting that brooding rumination is not a marker of stress sensitivity. Although this finding is inconsistent with previous studies (Bastin et al., 2014 and Cox et al., 2011; but see Paredes & Zamalde, 2014), it could be that brooding is a diathesis for a subtype of stress that was not captured in the current thesis. The relationship between brooding, stress and depression has been found to be stressor dependent (e.g., Cox et al., 2011). For example, Cox et al., (2011) separated life stress into different categories (e.g., interpersonal and non-interpersonal stress). Importantly, they found that brooding interacted with some categories of stress to predict depression, but not others. The current study suggests that brooding rumination does not interact with a global measure of life stress to predict depression, but it is possible it interacts with subcategories of life stress that were not measured.

While brooding rumination was not a trait marker of stress sensitivity, it was the only trait emotion regulation marker that directly predicted depression across time – although only at the three-month follow up. Interestingly, one previous study also found that brooding did not act as a stress sensitivity marker, but did directly predict

depression across time (Paredes & Zumalde, 2014). Further, a number of other studies have shown that brooding rumination directly predicts depression (e.g., Burwell & Shirk 2007; Mezulis et al., 2011). Brooding rumination was also the only marker that related to current depressive symptoms, as measured in Study One. One possible interpretation of this data is that brooding rumination and depressive symptoms are both independent but co-occurring responses to stress. If we accept this hypothesis, then increases in depressive symptoms will be accompanied by increases in brooding (as found in Study One) when both are in response to some form of stress. Further, brooding has been proposed to be a response to depressive symptoms whereby an individual attempts to cope with feelings of depression (Lyubomirsky & Tkach, 2004). Brooding is a maladaptive coping strategy that acts to maintain depression (Joormann et al., 2006; Treynor et al., 2003), as well as increasing depression symptoms (as found in my Study Two). However, these ideas are speculative. The current study did not measure levels of life stress at time one, nor did it measure brooding at the follow up time points. As such, I can not thoroughly examine the degree to which depression and brooding co-vary over time.

Summary

An important implication of the current thesis is that frontal and parietal EEG activity, spontaneous emotion regulation and brooding rumination reflect independent markers of emotion regulation. Additionally, these three markers operate differently across time to predict depression. Brooding rumination seems particularly distinct from the other two markers as it directly predicted depression, but did not interact with stress to predict depression. Findings suggest that spontaneous emotion regulation and parietal by frontal EEG activity are markers of stress sensitivity. Spontaneous emotion regulation may act as a proximal marker of stress sensitivity,

while frontal by parietal activity may be a comparatively more distal marker of stress sensitivity. A limitation that should be kept in mind when interpreting results of the follow up studies is that different subsets of participants responded to the three-month and twelve-month follow-up surveys. Importantly, I could not conduct a time series analysis, meaning each follow up has to be considered in isolation. Thus when comparing the effects of different trait markers at each time point, I do so in different samples. Further, samples differed in a measure of interest - frontal asymmetry scores - at time two and time three. Although this does not undermine my results, ideally research should clarify the time courses of different emotion regulation measures in a consistent sample.

My thesis builds on the idea that emotion regulation is important for understanding vulnerability to depression. Findings highlight that emotion regulation is not one uniform construct but consists of number of processes that may contribute independently to depression vulnerability. Considering multiple subtypes of emotion regulation will allow for more fine-grained insight into how depression develops across time, and the role of stress in depression. One important future research direction, is to clarify what cognitive processes are reflected by measures of regional EEG activity. Attentional control is a good candidate as a cognitive process that bridges the relationship between regional EEG activity and vulnerability to depression. Additionally, the use of refined measures of regional EEG activity (as described by the capability model) is likely to provide a more reliable measure of vulnerability to depression. My research provides a necessary step towards identifying emotion regulation measures that are critical for understanding vulnerability to depression.

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Appendix A

IAPS Images

Standardised ratings for images from the International Affective Picture System (IAPS) used during the startle eye-blink paradigm.

Valence Category	IAPS Reference	Valence Mean	Arousal Mean
Neutral	7175	4.95	1.87
Neutral	7004	5.15	1.94
Neutral	7010	4.92	1.97
Neutral	7000	5.06	2.15
Neutral	7020	4.94	2.19
Neutral	7491	4.79	2.24
Neutral	7950	5.17	2.27
Neutral	7031	4.80	2.36
Neutral	2190	4.90	2.50
Neutral	2840	4.90	2.55
Neutral	7217	5.00	2.55
Neutral	7150	4.69	2.56
Neutral	7006	5.09	2.58
Neutral	7080	5.10	2.67
Neutral	7100	5.20	2.73
Neutral	7035	5.15	2.75
Neutral	5510	5.10	2.87
Neutral	6150	5.00	2.89
Neutral	7040	4.66	2.90
Neutral	7050	5.04	2.90
Neutral	7235	5.06	2.94
Neutral	7034	4.91	2.96
Neutral	7233	5.15	2.96
Neutral	7025	4.79	2.98
Pleasant	1440	8.43	4.47
Pleasant	1710	8.59	5.31
Pleasant	8210	7.60	6.00
Pleasant	4680	6.91	6.07
Pleasant	7502	8.15	6.07
Pleasant	4575	7.61	6.12
Pleasant	4608	6.66	6.14
Pleasant	8190	8.08	6.16
Pleasant	8161	6.86	6.22
Pleasant	8400	6.78	6.24
Pleasant	8080	7.73	6.25
Pleasant	2216	7.85	6.29
Pleasant	4572	7.52	6.30
Pleasant	4660	7.22	6.31
Pleasant	8200	7.86	6.37
Pleasant	8034	7.19	6.38
Pleasant	8496	7.94	6.38
Pleasant	5629	7.15	6.52
Pleasant	8180	6.86	6.63
Pleasant	8490	7.44	6.97
Pleasant	8370	7.86	6.98
Pleasant	5621	7.80	7.00
Pleasant	8030	7.35	7.38
Pleasant	8185	7.75	7.42
Unpleasant	6560	1.78	6.86
Unpleasant	9921	1.58	6.87
Unpleasant	2730	1.80	6.93
Unpleasant	9252	1.53	6.93
Unpleasant	3140	1.50	6.94
Unpleasant	3110	1.47	6.98
Unpleasant	3100	1.35	7.02
Unpleasant	3071	1.69	7.10
Unpleasant	3030	1.51	7.13
Unpleasant	6540	1.86	7.14
Unpleasant	3053	1.15	7.15
Unpleasant	3102	1.22	7.15
Unpleasant	6313	1.61	7.27
Unpleasant	3064	1.15	7.30
Unpleasant	3060	1.66	7.34
Unpleasant	3130	1.26	7.39
Unpleasant	3266	1.26	7.43
Unpleasant	3010	1.29	7.44
Unpleasant	3120	1.33	7.49
Unpleasant	6350	1.44	7.52
Unpleasant	9410	1.20	7.54
Unpleasant	3170	1.20	7.55
Unpleasant	3080	1.33	7.61
Unpleasant	3000	1.21	7.77

Appendix B

Beck Depression Inventory - II

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the **one statement** in each group that best describes the way you have been feeling during the **past two weeks, including today**. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness

0 I do not feel sad.
1 I feel sad much of the time.
2 I am sad all the time.
3 I am so sad or unhappy that I can't stand it.

2. Pessimism

0 I am not discouraged about my future.
1 I feel more discouraged about my future than I used to be.
2 I do not expect things to work out for me.
3 I feel my future is hopeless and will only get worse.

3. Past Failure

0 I do not feel like a failure.
1 I have failed more than I should have.
2 As I look back, I see a lot of failures.
3 I feel I am a total failure as a person.

4. Loss of Pleasure

0 I get as much pleasure as I ever did from the things I enjoy.
1 I don't enjoy things as much as I used to.
2 I get very little pleasure from the things I used to enjoy.
3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings

0 I don't feel particularly guilty.
1 I feel guilty over many things I have done or should have done.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.

6. Punishment Feelings

0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.

7. Self-Dislike

0 I feel the same about myself as ever.
1 I have lost confidence in myself.
2 I am disappointed in myself.
3 I dislike myself.

8. Self-Criticalness

0 I don't criticize or blame myself more than usual.
1 I am more critical of myself than I used to be.
2 I criticize myself for all of my faults.
3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

10. Crying

0 I don't cry anymore than I used to.
1 I cry more than I used to.
2 I cry over every little thing.
3 I feel like crying, but I can't.

11. Agitation

0 I am no more restless or wound up than usual.
1 I feel more restless or wound up than usual.
2 I am so restless or agitated that it's hard to stay still.
3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

0 I have not lost interest in other people or activities.
1 I am less interested in other people or things than before.
2 I have lost most of my interest in other people or things.
3 It's hard to get interested in anything.

13. Indecisiveness

0 I make decisions about as well as ever.
1 I find it more difficult to make decisions than usual.
2 I have much greater difficulty in making decisions than I used to.
3 I have trouble making any decisions.

14. Worthlessness

0 I do not feel I am worthless.
1 I don't consider myself as worthwhile and useful as I used to.
2 I feel more worthless as compared to other people.
3 I feel utterly worthless.

15. Loss of Energy

0 I have as much energy as ever.
1 I have less energy than I used to have.
2 I don't have enough energy to do very much.
3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

0 I have not experienced any change in my sleeping pattern.
1a I sleep somewhat more than usual.
1b I sleep somewhat less than usual.
2a I sleep a lot more than usual.
2b I sleep a lot less than usual.
3a I sleep most of the day.
3b I wake up 1–2 hours early and can't get back to sleep.

17. Irritability

0 I am no more irritable than usual.
1 I am more irritable than usual.
2 I am much more irritable than usual.
3 I am irritable all the time.

18. Changes in Appetite

0 I have not experienced any change in my appetite.
1a My appetite is somewhat less than usual.
1b My appetite is somewhat greater than usual.
2a My appetite is much less than before.
2b My appetite is much greater than usual.
3a I have no appetite at all.
3b I crave food all the time.

19. Concentration Difficulty

0 I can concentrate as well as ever.
1 I can't concentrate as well as usual.
2 It's hard to keep my mind on anything for very long.
3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

0 I am no more tired or fatigued than usual.
1 I get more tired or fatigued more easily than usual.
2 I am too tired or fatigued to do a lot of the things I used to do.
3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

(Beck, 1996)

Appendix C

Ruminative Response Scale & subscales

Rumination Scale

People think and do many different things when they feel depressed. Please read each of the items below and indicate whether you almost never, sometimes, often, or almost always think or do each one when you feel down, sad, or depressed. Please indicate what you *generally* do, not what you think you should do.

1 almost never 2 sometimes 3 often 4 almost always

1. think about how alone you feel
2. think "I won't be able to do my job if I don't snap out of this"
3. think about your feelings of fatigue and achiness
4. think about how hard it is to concentrate
5. think "What am I doing to deserve this?"
6. think about how passive and unmotivated you feel.
7. analyze recent events to try to understand why you are depressed
8. think about how you don't seem to feel anything anymore
9. think "Why can't I get going?"
10. think "Why do I always react this way?"
11. go away by yourself and think about why you feel this way
12. write down what you are thinking about and analyze it
13. think about a recent situation, wishing it had gone better
14. think "I won't be able to concentrate if I keep feeling this way."
15. think "Why do I have problems other people don't have?"
16. think "Why can't I handle things better?"
17. think about how sad you feel.
18. think about all your shortcomings, failings, faults, mistakes
19. think about how you don't feel up to doing anything
20. analyze your personality to try to understand why you are depressed
21. go someplace alone to think about your feelings
22. think about how angry you are with yourself

NB. The Brooding Rumination subscale comprises items 5, 10, 13, 15, 16 (Treynor et al., 2003)

Appendix D

The Life Events Questionnaire

Instructions

Listed below are a number of events, which may bring about changes in the lives of those who experience them.

Circle the events that have occurred in your life during the past year and circle whether these were Good or Bad.

Show how much the event affected your life by circling the appropriate number, which corresponds with the statement (0 = no effect, 1 = some effect, 2 = moderate effect, 3 = great effect).

If you have not experienced a particular event in the past year, leave it blank.

Please go through the entire list before you begin to get an idea of the type of event you will be asked to rate.

Event	Type of Effect		Effect of Event on Your Life			
A. HEALTH			No effect	Some effect	Moderate effect	Great effect
1. major personal illness or injury	Good	Bad	0	1	2	3
2. major change in eating habits	Good	Bad	0	1	2	3
3. major change in sleeping habits	Good	Bad	0	1	2	3
4. major change in usual type and/or amount of recreation	Good	Bad	0	1	2	3
5. major dental work	Good	Bad	0	1	2	3
6. (female) pregnancy	Good	Bad	0	1	2	3
7. (female) miscarriage or abortion	Good	Bad	0	1	2	3
8. (female) started menopause	Good	Bad	0	1	2	3
9. major difficulties with birth control pills or devices	Good	Bad	0	1	2	3
B. WORK			No effect	Some effect	Moderate effect	Great effect
10. difficulty finding a job	Good	Bad	0	1	2	3
11. beginning work outside the home	Good	Bad	0	1	2	3
12. changing to a new type of work	Good	Bad	0	1	2	3
13. changing your work hours or conditions	Good	Bad	0	1	2	3
14. change in your responsibilities at work	Good	Bad	0	1	2	3

Event	Type of Effect		Effect of Event on Your Life			
15. troubles at work with your employer or co-workers	Good	Bad	0	1	2	3
16. major business readjustment	Good	Bad	0	1	2	3
17. being fired or laid off from work	Good	Bad	0	1	2	3
18. retirement from work	Good	Bad	0	1	2	3
19. taking courses by mail or studying at home to help you in your work	Good	Bad	0	1	2	3
C. SCHOOL			No effect	Some effect	Moderate effect	Great effect
20. beginning or ceasing school, college, or training program	Good	Bad	0	1	2	3
21. change of school, college, or training program	Good	Bad	0	1	2	3
22. change in career goal or academic major	Good	Bad	0	1	2	3
23. problem in school, college , or training program	Good	Bad	0	1	2	3
D. RESIDENCE			No effect	Some effect	Moderate effect	Great effect
24. difficulty finding housing	Good	Bad	0	1	2	3
25. changing residence within the same town or city	Good	Bad	0	1	2	3
26. moving to a different town, city, state, or country	Good	Bad	0	1	2	3
27. major change in your life conditions (home improvements or a decline in your home or neighborhood)	Good	Bad	0	1	2	3
E. LOVE AND MARRIAGE			No effect	Some effect	Moderate effect	Great effect
28. began a new, close, personal relationship	Good	Bad	0	1	2	3
29. became engaged	Good	Bad	0	1	2	3
30. girlfriend or boyfriend problems	Good	Bad	0	1	2	3

Event	Type of Effect		Effect of Event on Your Life			
31. breaking up with a girlfriend or boyfriend or breaking an engagement	Good	Bad	0	1	2	3
32. (male) wife or girlfriend's pregnancy	Good	Bad	0	1	2	3
33. (male) wife or girlfriend having a miscarriage or abortion	Good	Bad	0	1	2	3
34. getting married (or beginning to live with someone)	Good	Bad	0	1	2	3
35. a change in closeness with your partner	Good	Bad	0	1	2	3
36. infidelity	Good	Bad	0	1	2	3
37. trouble with in-laws	Good	Bad	0	1	2	3
38. separation from spouse or partner due to conflict	Good	Bad	0	1	2	3
39. separation from spouse or partner due to work, travel, etc.	Good	Bad	0	1	2	3
40. reconciliation with spouse or partner	Good	Bad	0	1	2	3
41. divorce	Good	Bad	0	1	2	3
42. change in your spouse or partner's work outside the home (beginning work, ceasing work, changing jobs, retirement, etc.	Good	Bad	0	1	2	3
F. FAMILY AND CLOSE FRIENDS			No effect	Some effect	Moderate effect	Great effect
43. gain of a new family member (through birth, adoption, relative moving in, etc)	Good	Bad	0	1	2	3
44. child or family member leaving home (due to marriage, to attend college, or for some other reason)	Good	Bad	0	1	2	3
45. major change in the health or behavior of a family member or close friend (illness, accidents, drug or disciplinary problems, etc.)	Good	Bad	0	1	2	3
46. death of spouse or partner	Good	Bad	0	1	2	3
47. death of a child	Good	Bad	0	1	2	3

Event	Type of Effect		Effect of Event on Your Life			
48. death of family member or close friend	Good	Bad	0	1	2	3
49. birth of a grandchild	Good	Bad	0	1	2	3
50. change in marital status of your parents	Good	Bad	0	1	2	3
G. PARENTING			No effect	Some effect	Moderate effect	Great effect
51. change in child care arrangements	Good	Bad	0	1	2	3
52. conflicts with spouse or partner about parenting	Good	Bad	0	1	2	3
53. conflicts with child's grandparents (or other important person) about parenting	Good	Bad	0	1	2	3
54. taking on full responsibility for parenting as a single parent	Good	Bad	0	1	2	3
55. custody battles with former spouse or partner	Good	Bad	0	1	2	3
H. PERSONAL OR SOCIAL			No effect	Some effect	Moderate effect	Great effect
56. major personal achievement	Good	Bad	0	1	2	3
57. major decision regarding your immediate future	Good	Bad	0	1	2	3
58. change in your personal habits (your dress, life-style, hobbies, etc.)	Good	Bad	0	1	2	3
59. change in your religious beliefs	Good	Bad	0	1	2	3
60. change in your political beliefs	Good	Bad	0	1	2	3
61. loss or damage of personal property	Good	Bad	0	1	2	3
62. took a vacation	Good	Bad	0	1	2	3
63. took a trip other than a vacation	Good	Bad	0	1	2	3
64. change in family get-togethers	Good	Bad	0	1	2	3
65. change in your social activities (clubs, movies, visiting)	Good	Bad	0	1	2	3
66. made new friends	Good	Bad	0	1	2	3
67. broke up with a friend	Good	Bad	0	1	2	3
68. acquired or lost a pet	Good	Bad	0	1	2	3

Event	Type of Effect		Effect of Event on Your Life			
I. FINANCIAL			No effect	Some effect	Moderate effect	Great effect
69. major change in finances (increased or decreased income)	Good	Bad	0	1	2	3
70. took on a moderate purchase, such as TV, car, freezer, etc.	Good	Bad	0	1	2	3
71. took on a major purchase or a mortgage loan, such as a home, business, property, etc.	Good	Bad	0	1	2	3
72. experienced a foreclosure on a mortgage or loan	Good	Bad	0	1	2	3
73. credit rating difficulties	Good	Bad	0	1	2	3
J. CRIME AND LEGAL MATTERS			No effect	Some effect	Moderate effect	Great effect
74. being robbed or victim of identity theft	Good	Bad	0	1	2	3
75. being a victim of a violent act (rape, assault, etc.)	Good	Bad	0	1	2	3
76. involved in an accident	Good	Bad	0	1	2	3
77. involved in a law suit	Good	Bad	0	1	2	3
78. involved in a minor violation of the law (traffic tickets, disturbing the peace, etc)	Good	Bad	0	1	2	3
79. legal troubles resulting in your being arrested or held in jail	Good	Bad	0	1	2	3
K. OTHER- Other recent experiences which have had an impact on your life. List and rate.						
80. _____	Good	Bad	0	1	2	3
81. _____	Good	Bad	0	1	2	3
82. _____	Good	Bad	0	1	2	3

(Norbeck, 1984)
NB. This questionnaire was adapted for use in an online survey (ww.surveymonkey.com) and only "Bad" Events were measured.

Appendix E
Mood and Anxiety Questionnaire mini (MASQ-mini)

ID# _____

Mini-MASQ

Below is a list of feelings, sensations, problems, and experiences that people sometimes have. Read each item and then fill in the blank with the number that best describes how much you have felt or experienced things this way during the past week, including today. Use this scale when answering::

1	2	3	4	5
not at all	a little bit	moderately	quite a bit	extremely

_____ 1. Felt really happy
_____ 2. Felt tense or "high strung"
_____ 3. Felt depressed
_____ 4. Was short of breath
_____ 5. Felt withdrawn from other people
_____ 6. Felt dizzy or lightheaded
_____ 7. Felt hopeless
_____ 8. Hands were cold or sweaty
_____ 9. Felt like I had a lot to look forward to
_____ 10. Hands were shaky
_____ 11. Felt like nothing was very enjoyable
_____ 12. Felt keyed up, "on edge"
_____ 13. Felt worthless
_____ 14. Had trouble swallowing
_____ 15. Felt like I had a lot of interesting things to do
_____ 16. Had hot or cold spells
_____ 17. Felt like a failure
_____ 18. Felt like I was choking
_____ 19. Felt really lively, "up"
_____ 20. Felt uneasy
_____ 21. Felt discouraged
_____ 22. Muscles twitched or trembled
_____ 23. Felt like I had a lot of energy
_____ 24. Was trembling or shaking
_____ 25. Felt like I was having a lot of fun
_____ 26. Had a very dry mouth

(Clark & Watson 1995)
NB. The anxious arousal subscale comprises of items 4, 6, 8, 10, 14, 16, 18, 22, 24 & 26.