Differences in Emotion Reactivity between Individuals with Features of Borderline Personality Disorder and Depression

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Abstract
Depression and borderline personality disorder (BPD) are highly comorbid and are both characteristic of affective disturbance. In this study, it is hypothesized that the disorders share a common etiological factor of emotion reactivity. In addition, an investigation of specific emotional experiences that may differentiate the two symptom clusters is undertaken. A mood induction task was used to elicit emotional reactions in a sample of 121 university students. Regression analyses were conducted to examine emotion reactivity as a common factor. Unique associations between specific emotions and features of BPD, depression, and an interaction term (BPDxDep) were investigated. It was found that all models tested were significant, with the exception of joy. Features of BPD were uniquely associated with sadness, guilt, and anger, as were depressive features. BPDxDep symptoms were negatively associated with guilt. A discussion of the findings obtained and their significance in theory and in practice is undertaken.
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Chapter 1
Emotion Reactivity as the Common Factor

Borderline personality disorder (BPD) is a disorder with emotional, behavioural,
cognitive, and interpersonal consequences. To meet diagnostic criteria, individuals must
endorse five of the nine symptoms outlined by the *Diagnostic and Statistical Manual of
Mental Disorders, Fifth Edition* (DSM-5). These symptoms include fears of
abandonment, unstable relationships, identity disturbance, angry outbursts, affective
instability, self-injury, feelings of emptiness, stress related paranoia/dissociation, and/or
impulsivity (American Psychiatric Association [APA], 2013). Comorbid conditions often
accompany a diagnosis of BPD, complicating the clinical presentation. One of the most
common comorbid diagnoses is depression. In a recent epidemiological study utilizing a
community sample it was found that 12-month major depressive disorder (MDD)
comorbidity rates with lifetime BPD were 19.9%, whereas lifetime comorbidity rates
between BPD and MDD were 32.1% (Grant et al., 2008). In another study investigating
comorbidity rates in a clinical sample, 70.9% of individuals who met criteria for BPD
also met criteria for MDD (McGlashan et al., 2000). According to DSM-5, to meet
diagnostic criteria for MDD one must have had at least one major depressive episode.
During these episodes, individuals must endorse either depressed mood or loss of interest
in activities usually found pleasurable, along with five of the following symptoms:
Fluctuations in weight, disturbed sleep, psychomotor irregularities, loss of energy,
feelings of worthlessness or guilt, diminished concentration, and/or suicidal ideation
(APA, 2013). As described by the DSM-5, individuals in either diagnostic category
experience disturbances in emotional experiences. Due to similarities in symptom
endorsement, as well as high comorbidity rates, researchers have postulated that there may be common etiological factors between BPD and depression (Eaton et al., 2011; Klein & Shwartz, 2002; Zanarini, Barison, Frankenburg, Reich, & Hudson, 2009). In a study investigating different models explaining the associations between symptoms of depression and BPD, Klein and Schwartz (2002) found that the model that best explained their data included a common latent variable that underlies both depressive and borderline symptoms, along with factors that are unique to each symptom cluster. The authors concluded that it is likely that a common factor exists between these two disorders. In addition, Riso, Klein, Anderson, and Ouimette (2000) investigated first-degree probands of individuals with BPD and without a history of depression. The authors found increased risk for depression in first-degree relatives, suggesting a common etiological factor between the two disorders. However, there are differences in BPD and depression presentation. For instance, negative emotionality in BPD is often characterized by hostility, irritability, and emotional dysregulation (e.g. Pazzagli & Monti, 2000; Zanarini et al., 1998), while negative emotionality in depression is characterized by low mood, loss of motivation, and avoidance behaviour (e.g. Leventhal, 2008). While the disorders may share a common liability, it is also important to note that they are unique in certain respects. In this paper, emotion reactivity is investigated as a common factor between individuals who endorse symptoms of BPD and those who endorse symptoms of depression. In addition, an examination of how they differentially relate to specific emotional experiences is undertaken. This investigation begins by outlining different theories concerning the common factor. Then, a more detailed look into emotion reactivity as a construct related to both BPD and depression is undertaken.
Next, the specific hypotheses and the methods and results of the study are outlined.
Finally, a discussion of the obtained results is provided.

**Common Factor: Borderline Personality Disorder and Depression**

One possible common factor between BPD and depression is the internalizing factor. Krueger has shown that the covariance observed between disorders as defined by the DSM can be encompassed by two higher-order factors, internalization and externalization. The internalizing dimension is defined by an individual’s propensity to express distress inwards, while the externalizing dimension is defined by an individual’s propensity to express distress outwards (Krueger, McGue, & Iacono, 2001). Mood and anxiety disorders have been shown to load onto the internalizing dimension, while substance use and disorders characteristic of antisocial behaviours have been shown to load onto the externalizing dimension (Krueger & Markon, 2006a). The internalizing dimension has been further subdivided into two disorder categories; disorders of distress (e.g. depression, dysthymia), and disorders of fear (e.g. panic disorder, social phobia; for a review see Krueger & Markon, 2006b). Below, evidence for this dimensional structure is presented.

According to Clark and Watson’s (1991) tripartite model, associations between personality traits and the internalizing dimension explain high comorbidity rates between mood and anxiety disorders. For instance, high co-occurrence between mood and anxiety disorders can be explained by high negative emotionality, while differences between the two can be attributed to differences on levels of extraversion (specific to depression) and differences in physiological hyperarousal (specific to anxiety). In an attempt to test the applicability of this model, Brown, Chorpita, and Barlow (1998) investigated the fit of
three different dimensional models on key features of five specific disorders – depression, generalized anxiety, panic disorder, obsessive compulsive disorder, and social phobia. The best fitting model closely resembled the tripartite model proposed by Clark and Watson (1991), such that all disorders loaded onto a higher order factor of negative affect, depression and social phobia were negatively correlated with the higher order factor of positive affect, and the latent factor autonomic arousal was most strongly related to panic disorder, and negatively correlated with generalized anxiety. It is important to note that the dimensional models referenced above focused solely on DSM-IV Axis I disorders, and usually did not include psychotic disorders (e.g. schizophrenia). In more recent investigations, the inclusion of psychotic disorders has elucidated a third factor. It has been shown that genetic and phenotypic evidence subdivides the internalizing dimension into mood, anxiety, and bipolar disorders (i.e. bipolar I, bipolar II, and cyclothymia; Watson, 2005). Additionally, Kotov and colleagues (2010) examined the dimensional structure of DSM-IV disorders, including disorders with psychotic symptoms. In their study, the internalizing and externalizing dimensions were replicated, and a new factor emerged that was termed schizophrenic. In agreement with these findings, Wright and associates (2013) also found three higher-order factors, labeled internalizing, externalizing, and psychotic experiences.

In addition to studies investigating the dimensional structure of DSM-IV Axis I psychopathology, recent work has begun to include Axis II disorders as well. Markon (2010) investigated the loadings of symptom clusters of different DSM-IV Axis I and Axis II disorders on a variety of factors. It is important to note that symptoms, not categorical diagnoses, were included in this model. Twenty symptom factors emerged
that loaded onto four higher order factors; internalizing, thought disorder, externalizing, and pathological introversion. Depressive symptomology comprised two lower order symptom factors (depression and somatoform problems), while BPD symptomology comprised four lower order facets (emotional lability, disorganized attachment, hostility, and attention seeking). Both depression and emotional lability loaded onto a common internalizing factor. However, emotional lability was also associated with the externalizing dimension. Furthermore, disorganized attachment loaded onto thought disorder, hostility loaded onto both thought disorder and externalizing, and attention seeking loaded onto the externalizing dimension. Such findings illustrate the heterogenous nature of BPD. In accordance with these findings, Roysamb and colleagues (2011) investigated the hierarchical structure of DSM-IV Axis I and II categorical diagnoses. The authors also found that both BPD and depression loaded onto an internalizing dimension. However, it should be noted that BPD also partially loaded onto an externalizing dimension and a cognitive-relational dimension, illustrating the wide range of affected areas in this diagnosis. As such, symptoms related to BPD may converge on a dimension that encompasses depression, while other symptoms are related to other disorders, including substance abuse, antisocial personality disorder, and schizotypal personality disorder. Interestingly, the authors described BPD as interspectral, stating that it loaded onto higher order factors that were comprised mostly of Axis I disorders (internalizing and externalizing factors) and those comprised mostly of Axis II disorders (cognitive-relational disturbance).

While much of the research cited above supports the notion that both BPD and depression load onto an internalizing factor, this finding does not elucidate what the
common liability between the two disorders may comprise of. One possible common etiological factor between BPD and depression is the personality trait neuroticism. Neuroticism is defined as a domain of personality that distinguishes emotional stability and adjustment with negative emotionality and maladjustment (Costa & McCrae, 1992a). Relationships between neuroticism and the internalizing dimension have been found in previous research. In fact, associations between the two constructs point to the possibility that they are one in the same. Griffith and colleagues (2010) replicated previous findings by illustrating that a single internalizing factor was a common factor to lifetime diagnoses of mood and anxiety disorders. In addition to this, Griffith et al. found a near perfect correlation between the personality trait neuroticism and this internalizing factor ($r = 0.98$). The authors warn against the interpretation that the internalizing factor and neuroticism are in fact equivalent before replication of these findings. However, it is important to note that several studies have found correlations between internalization and trait negative emotionality. For instance, Krueger and colleagues (2001) replicated this two-factor psychopathology model and illustrated that negative emotionality was positively correlated with internalization while constraint was negatively correlated with externalization. In addition, positive emotionality was negatively related to internalization only in women. It should be noted that these correlations were much smaller than the correlation observed in the above study (e.g. internalization and negative emotionality correlation in women: $r = 0.22$). In another study examining the association between internalization and neuroticism, Hettema, Neale, Myers, Prescott, and Kendler (2006) found that shared genetic factors explained high co-occurrence rates between a series of internalizing disorders (depression, generalized anxiety, panic, agoraphobia,
social phobia, animal phobia, and situational phobia), especially those genetic factors shared with the personality trait neuroticism. However, additional genetic factors explained high comorbidity rates between depression, generalized anxiety, and panic disorder that were not shared with genetic factors explaining variance in neuroticism. Such findings do not support the previous study in which neuroticism and the internalizing factor were essentially equivalent. Nevertheless, the two constructs possess substantial overlap.

Taking the above into consideration, it is possible that the common factor between depression and BPD is high trait neuroticism. The relationships between BPD and neuroticism and depression and neuroticism have been shown in several studies (Morey & Zanarini, 2000; Saklofske, Kelly, & Janzen, 1995). However, it is important to note that neuroticism is a common liability factor for many disorders, including anxiety disorders (e.g. panic disorder, social phobia; Bienvenu et al., 2004) and several personality disorders (e.g. dependent personality disorder, avoidant personality disorder; Widiger & Mullins-Sweatt, 2009). As such, its specificity in identifying commonalities between BPD and depression is low. In addition, the ways in which neuroticism is assessed varies between questionnaires. For instance, the NEO-PI-R – a widely used Big Five self-report method – assesses six facets of neuroticism. These facets are anxiety, angry hostility, depression, self-consciousness, vulnerability, and impulsiveness (Costa & McCrae, 1992b). The CPI-Big Five, on the other hand, assesses four lower order facets of neuroticism, labeled anxiety, depression, rumination, and irritability (Soto & John, 2009). As such, concerns about the ways in which neuroticism is operationalized have risen. Ormel, Rosmalen, and Farmer (2004) argue that neuroticism may be no more than a
measure of an individual’s typical degree of distress. Their proposition is based on five key arguments that are corroborated by research. First, high item overlap between neuroticism measures and symptoms of depression and anxiety point to the likelihood that neuroticism is related to these disorders simply due to overlapping content. Second, high correlations between neuroticism and distress measures suggest that they may be tapping into the same factor – degree of distress. Third, changes in levels of neuroticism across a specific passage of time suggest that most of the variance in neuroticism is mutable, suggesting that it is not measuring a temperamental, unchanging trait. Fourth, research on the genetic contribution of neuroticism suggests that it is 40% genetic, pointing to a large percentage of variance explained by environmental factors that may include life stressors that impact degree of distress. Finally, neuroticism, depression, and anxiety share a large proportion of genetic risk, suggesting that content overlap may contribute to these relationships, reifying the notion that neuroticism is a non-informative marker of psychopathology (for a review see Ormel et al., 2004). However, in a recent study testing the content overlap claim, Ulaszek and colleagues (2009) found that a general neuroticism factor explained a large portion of the variance in depressive symptomology, more so than items that were deemed as overlapping in content (e.g. often feel blue). The same result was obtained for symptoms of anxiety, suggesting that content overlap is not the primary reason for associations between neuroticism and depression and anxiety. Nevertheless, Guarino, Roger, and Olason (2007) point to the heterogeneous nature of neuroticism, as it encompasses several negative emotions amongst other symptoms (e.g. perceptions of life as stressful, poor coping strategies, somatic complaints; Costa & McCrae, 1987). In addition, the inclusion of impulsiveness
as a facet of neuroticism has only perpetuated the confusion surrounding this trait, for there is evidence to suggest that impulsivity falls within the extraversion domain (Revelle et al., 1980). While neuroticism may be a common factor between BPD and depression, its lack of specificity, its ill-defined nature, and its associations with multiple psychopathological constructs is problematic.

Another possible common factor is the construct of emotion sensitivity. In Linehan’s (1993) biosocial model, emotion sensitivity is considered a biological vulnerability, one that interacts with an invalidating childhood environment to manifest in symptoms of BPD. This emotion vulnerability is comprised of three factors; (1) high baseline emotional arousal, (2) intense emotional responses, and (3) slow return to baseline. The first tenet, high baseline emotional arousal, is the tendency to have a heightened level of emotional intensity without provocation and on a regular basis. The second tenet, intense emotional responses, is the propensity to experience greater emotional reactivity when confronted with an emotion-provoking stimulus. The last tenet, slow return to baseline, is the tendency to remain at a heightened emotional state after reacting to a specific stimulus for a longer period of time before returning to baseline emotional levels. Both BPD and depression are characterized by emotion difficulties (Beck 1976; Linehan 1993). While individuals with depression have persistent low moods (Bylsma, Morris, & Rottenberg 2008), individuals with BPD exhibit rapid oscillations between different types of negative emotions, experiencing several emotions within the course of one day (Glaser, Van Os, Mengelers, & Myin-Germeys 2008). Staebler, Gebhard, Barnett, and Renneberg (2009) investigated differences in mood and emotion reactivity across two different time points (during crisis and 8-months later) in
individuals with either BPD or depression. Baseline mood states were similar across the two patient groups, with both exhibiting high negative and low positive emotions compared to controls. However, with respect to variations in baseline emotions across time, no differences in BPD patients were found, although other symptoms did improve between crisis and 8-month follow up. On the other hand, individuals with depression illustrated differences between these two time points, only exhibiting significantly lower positive emotions during crisis but not 8-months later, when compared to controls. In addition, individuals with depression reported less negative emotions at time two when compared to time one. In accordance with these findings, both BPD and depression groups share high negative emotionality and low positive emotionality at baseline. While baseline levels for individuals with BPD did not change across time, individuals with depression experienced less negative emotions and higher positive emotions 8-months later. It seems that the two disorders share a common factor of baseline emotionality, with individuals with depression illustrating a more phasic pattern than those with BPD. Considering these findings, it seems plausible to speculate that a common factor related to affective disturbance may exist between the two disorders. Below, a more specific review of the association between emotion sensitivity and each of the disorders is outlined.

**Emotion Sensitivity in Borderline Personality Disorder**

The literature on emotion sensitivity in BPD is quite scant. Most of the research in this field focuses on the general construct of emotion dysregulation, ignoring the three different components of Linehan’s biosocial model. Nevertheless, there is a small body of research that has concentrated its efforts on this area of study. In this section, emotion
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recognition tasks, physiological studies, and self-report measures of emotion sensitivity in BPD are reviewed.

Emotion recognition tasks comprise of three different design methods; accuracy of emotion recognition, sensitivity of emotion recognition, and biases in emotion recognition. In accuracy of emotion recognition, participants are shown faces displaying emotions and are asked to choose which emotion they believe is being displayed. This form of research is concerned with inaccuracy in reading facial cues, which may lead to problems in interpersonal relationships. In emotion recognition sensitivity research, individuals are either presented with faces that are displaying pure emotions at different intensities (e.g. 10% sad – 100% sad), or they are presented with morphed faces displaying different intensities and different mixtures of emotions (e.g. 10% sad/90% angry, 50% sad/50% angry, etc.). In this research, the magnitude of intensity required to accurately recognize an emotion is of most interest. If different populations need less emotion expression than others to identify emotions, this implies heightened emotion sensitivity. Finally, two different kinds of stimuli, morphed (i.e. emotionally ambiguous) and neutral faces, are used to illustrate biases in emotion selection (e.g. an individual chooses happy faces more often than not when presented with emotionally ambiguous or neutral facial stimuli). In this paper, the focus is on sensitivity in emotion recognition as it is most closely tied to the construct of emotion sensitivity.

Emotion sensitivity recognition tasks in BPD populations have resulted in contradictory findings. The following studies all investigated emotion sensitivity in six emotions: Sadness, happiness, surprise, anger, fear, and disgust. Lynch and colleagues (2006) found that individuals with BPD showed heightened sensitivity to overall
emotions, such that they correctly identified facial affect at an earlier stage of emotional expression than healthy controls. An investigation of differences across specific emotional expressions revealed that individuals with BPD identified anger and happiness significantly earlier than healthy controls, illustrating heightened sensitivity to these two emotions specifically. In another study, Jovev et al. (2011) found no differences between individuals with subsyndromal BPD (i.e. 3 or more criteria endorsed) and healthy controls on emotion sensitivity. The authors did, however, obtain moderate effect sizes when differences in rate of identification of specific emotions were examined, such that individuals with BPD symptoms identified disgust and fear at later stages than healthy controls. This suggests that individuals with BPD symptoms are less sensitive to expression of disgust and fear. Domes and colleagues (2008) also found no significant differences between individuals with BPD and healthy controls on emotional detection threshold. In addition, no differences on specific emotions were found in their study. Finally, Robin and others (2012) investigated differences in emotion sensitivity between adolescents with BPD and healthy controls. The authors found that adolescents with BPD identified anger and happiness at significantly later stages than did the comparison group. These results stand in direct opposition to results obtained by Lynch and colleagues (2006) above. Discrepant findings may be due to differences in sample selection. For instance, studies investigating adolescent and subsyndromal populations found that their BPD sample was less sensitive in identifying specific emotions when compared to controls. Jovev et al. (2011) suggest that emotion sensitivity may develop later in the disorder, or is only found in severe BPD. In addition, differences in methodology employed may lead to the influence of extraneous factors (e.g. unlimited vs. limited
responding and guessing) that are impacting the obtained findings. Such contradictory findings are problematic when attempting to interpret emotion sensitivity in BPD. In addition, facial recognition research focuses on the ability to identify emotions in others, not in the self. It is important to note that any conclusions about emotion sensitivity within the individual from facial recognition tasks are based on the assumption that sensitivity within the self influences how emotions in others are viewed. The focus of this particular study is on emotion sensitivity within the individual. As such, the above results are difficult to interpret within this framework.

While it is difficult to draw conclusions about emotion sensitivity in BPD based on emotion recognition tasks, physiological measures of emotion sensitivity aim to address this question through biological measures. However, physiological measures assessing certain tenets of the emotion sensitivity model have also yielded some contradictory results. Studies assessing emotion reactivity using skin conductance (SC) measures have found evidence of decreased reactivity (Herpertz, Kunert, Schwenger, & Sass, 1999; Herpertz et al., 2000), while another study found trends towards increased reactivity, although significance levels were not reached (Schmahl et al., 2004). Additional studies do find a relationship between BPD and heightened emotion reactivity, with increases in heart rate (Ebner-Priemer et al., 2007), a larger startle response (Ebner-Priemer et al., 2005), and decreases in respiratory sinus arrhythmia (SRA), which is indicative of greater emotion reactivity (Austin, Riniolo, & Porges, 2007). Kuo and Linehan (2009) investigated baseline emotion sensitivity in individuals with BPD, as well as emotion reactivity after mood induction using physiological measures. Both RSA and skin conductance response (SCR) were used to assess baseline sensitivity, with SCR also
being used to examine emotion reactivity after a mood induction task. Findings showed that individuals with BPD possessed higher baseline emotion sensitivity, indicated by low RSA measures and high SCR measures before any mood induction. Following mood induction, however, SCR levels of individuals with BPD rose at the same level as SCR levels in the control group. As such, no evidence for emotion reactivity was found. Self-report measures of felt emotion reactivity corroborated SCR measures after induction. While this study provides preliminary evidence for heightened baseline sensitivity, it does not support previous findings of higher emotion reactivity after mood induction.

Behavioural measures of certain tenets of the emotion sensitivity model seem to produce the most consistent results in investigating the relationship between BPD and emotion sensitivity. In a study examining emotion reactivity to everyday life events, Glaser et al. (2008) used an experience sampling method to assess reactions to everyday events of individuals with either BPD or psychosis, compared with controls. Participants were beeped for six consecutive days, ten times each day at which point they were instructed to report their current thoughts, appraisals, and moods. Findings showed that individuals with BPD reported higher subjective stress and higher emotion reactivity to everyday life events, when compared to individuals with psychosis, as well as controls. This finding was particularly interesting considering previous research showing that individuals with psychosis experience everyday life events as more stressful than controls (Myin-Germeys et al., 2003). As such, when compared to a population with known heightened sensitivity to life events, individuals with BPD reported even higher stresses and emotional reactions. Furthermore, in a study investigating predictors of borderline features in middle childhood, Crick, Murray-Close, and Woods (2005) found that
emotion sensitivity was predictive of borderline features when fourth, fifth, and sixth
grade children were assessed throughout one full school year. Such findings point to a
possible biological predisposition to emotion sensitivity, as was suggested in Linehan’s
(1993) biosocial model.

In addition to these findings, more specific investigations of the relationship
between BPD and particular emotions have taken place. Gratz, Rosenthal, Tull, Lejuez,
and Gunderson (2010) investigated emotion reactivity to a general stressor, compared
with receiving a negative evaluation. While no differences were found between BPD
outpatients and a no personality disorder (no-PD) group in reactivity to the general
stressor, individuals with BPD exhibited heightened emotion reactivity to negative
evaluations, with a slower return to baseline. Specifically, individuals with BPD endorsed
higher feelings of shame after receiving a negative evaluation. This study illustrates a
context and emotion specific response in patients with BPD when compared to no-PD
patients. Furthermore, Rusch and colleagues (2007) found that women with BPD
endorsed higher levels of shame, guilt, state-shame, and anxiety when compared to
women with social phobia and healthy controls. After adjusting for levels of shame, the
relationship between BPD and guilt was no longer significant, while the relationship
between shame and BPD remained significant after adjusting for levels of guilt. This
suggests that individuals with BPD are more characteristic of shame-proneness than of
guilt-proneness, in comparison to individuals with social phobia and healthy controls. In
accordance with these findings, associations between BPD and anxiety disorders have
been established. Silverman, Frankenburg, Reich, Fitzmaurice, and Zanarini (2012)
compared BPD patients and a DSM-IV Axis II group on anxiety disorder comorbidity in
a 10-year longitudinal study. The authors found that BPD patients were almost two times more likely to endorse symptoms of any anxiety disorder compared to the Axis II group. While both groups experienced significant decreases in anxiety symptom endorsement during the study, the BPD group remained significantly higher throughout the 10-year course of the study. In another study, Gratz, Tull, and Gunderson (2008) investigated the specificity of anxiety sensitivity in distinguishing individuals with BPD from DSM-IV Axis I diagnoses. The authors found that individuals with BPD endorsed higher ratings of anxiety sensitivity when compared with their no-PD group, and that this relationship was fully mediated by experiential avoidance (i.e. avoidance of unwanted internal experiences/emotions). In addition, this relationship remained significant after controlling for negative affect, impulsivity, and emotion intensity. Another common emotion related to BPD is anger. In accordance with the DSM-5, one of the symptoms that may be endorsed for a BPD diagnosis includes angry outbursts (APA, 2013). Furthermore, Baer and Sauer (2011) found that both depressive and anger rumination were significantly related to borderline features. The relationship between negative affect and BPD features was reduced when anger rumination was included in the model, suggesting that associations between BPD features and anger may be stronger than depressive rumination. In addition, Jacob and colleagues (2008) conducted a mood induction study with a specific focus on anger. Surprisingly, individuals with BPD did not report heightened reactions to anger in comparison to healthy controls. However, they did experience prolonged feelings of anger (i.e. a slower return to baseline emotional anger) after mood induction in comparison to controls. The authors suggest that individuals with BPD may not experience more anger, but that they have difficulty regulating this
emotion, contributing to cognitive and interpersonal impairments experienced by individuals with BPD. Furthermore, Koenigsberg and colleagues (2002) found that endorsement of anger lability significantly distinguished individuals with BPD than those with other personality disorders. The above studies illustrate that symptoms of emotion dysregulation in BPD may be related to specific forms of negative affect.

Overall, research on emotion sensitivity in BPD has resulted in contradictory findings. Studies investigating heightened sensitivity using cognitive, physiological, and self-report measures have yet to come to a consensus. While different study designs may have resulted in these discrepancies, it is also possible that heightened sensitivity is more emotion specific, as illustrated in the findings of Gratz and associates (2010) and as evidence by the aforementioned relationships between BPD and specific emotional experiences.

**Emotion Sensitivity in Depression**

As was previously mentioned, depression is characterized by low mood for most of the day, nearly every day. Considering that this is a hallmark feature of the disorder, it is imperative that a distinction between moods and emotions is made before discussing the relevant literature on emotion sensitivity in depression. According to Rottenberg (2005), moods can be defined as “diffuse, slow-moving feeling states that are weakly tied to specific objects or situations” (p. 167). In addition, consequences of moods can be observed in feeling states and cognition, with effects lasting hours to days. In contrast, Rottenberg (2005) defines emotions as “quick-moving reactions that occur when organisms encounter meaningful stimuli that call for adaptive responses” (p. 167). Such emotional reactions last seconds to minutes, and usually involve behavioural and
physiological changes. Taking into account these differences, a theoretical consideration of the effects of mood on emotions is necessary.

The mood-facilitation hypothesis suggests that individuals experiencing a specific mood (e.g. depressed mood) are more likely to experience a mood-congruent emotional reaction (e.g. sadness). It is speculated that these reactions are intense and facilitated by current feeling states. Three major views have spawned from this concept: (1) negative potentiation (Beck, 1976), higher emotion reactivity to negative stimuli, facilitated by depressed mood; (2) positive attenuation, decreased emotion reactivity to positive stimuli, due to depressed mood; and (3) emotion context insensitivity (ECI; Rottenberg, 2005), emotion reactivity is reduced to all stimuli, regardless of emotional valence. While the first two views support one another, the third view stands in complete opposition to the other theories. A recent meta-analysis addressing discrepant results and investigating these divergent theories found that the ECI view was most supported, such that reactivity was reduced in both positive ($d = -.53$) and negative ($d = -.25$) stimuli (Bylsma et al., 2008). This seemingly aberrant finding was explained by mood flattening – individuals with severe depression experience constriction in emotions, with impairments in a variety of emotions. While such findings seem widely supported, they also seem counterintuitive. High comorbidity rates between BPD and depression contradict this emotional flattening. Although both groups often describe feelings of emptiness, individuals endorsing symptoms of BPD usually experience a myriad of strong emotional experiences in a given day.

The authors of the meta-analysis computed two effect sizes; $d = -.53$ for positive stimuli, and $d = -.25$ for negative stimuli (Bylsma et al., 2008). These reported effects are
considered moderate and small, respectively (Cohen, 1988). Put in other terms, the magnitude of difference between individuals with depression and comparison groups is not large. This may be an indication of heterogeneity in patient samples. While some individuals with depression may experience this extreme emotional flattening, others may endorse low mood with high sensitivity to emotional experiences. These two disparate experiences are closely related to melancholic (lack of reactivity to usually pleasurable stimuli) and atypical (mood reactivity) subtypes of depression (APA, 2013). An example of this can also be illustrated in a paper examining emotion sensitivity in bipolar patients. Gray and colleagues (2006) were interested in examining differences in emotion sensitivity in patients with bipolar disorder, during a depressed phase or during a manic phase. Findings showed that no differences in sensitivity were apparent in individuals experiencing mania. However, during depressed moods, individuals with bipolar disorder were more sensitive to emotions of anger and disgust and less sensitive to happiness in a morphed facial expression task, when compared to healthy controls. Furthermore, currently depressed individuals were generally more sensitive to the emotional expressions presented in the study, with the exception of happiness. While major depressive episodes during bipolar disorder are characterized as the same as those occurring during depression, this may point to yet another subtype that leads to distinct relationships between depression and emotion sensitivity. Research examining subtypes of depression and their differential relations to emotion sensitivity is necessary.

Differences in mild, moderate and severe depression may also explain these findings. In analog and adolescent samples, evidence for emotion sensitivity has been found. Golin, Hartman, Klatt, Munz, and Wolfgang (1977) assessed heightened
sensitivity to negative/positive evaluations in a sample of college students. Students endorsing depressive symptomology reacted more strongly to negative evaluations, when compared with non-depressed college students. Lewinsohn, Lobitz, and Wilson (1973) also used an analog sample to assess reactivity to aversive stimuli (i.e. electric shock) in depressed students. A greater autonomic response was found during presentation of aversive stimuli, but not before or after exposure, when compared with psychiatric and healthy controls. Furthermore, individuals with severely flattened affect are associated with more severe forms of depression, and longer episodes of depression (Rottenberg, Kash, Gross, & Gotlib, 2002). Emotional flattening can be interpreted as an adaptive symptom – a system that is overloaded with negative emotions may require blunted affect to free up some cognitive reserves. As such, individuals with mild to moderate depression, or analog samples, may not experience this emotional numbness. Unfortunately, few studies have examined this, and it is recommended that future research investigate differences in emotion sensitivity across depression severity.

Interestingly, most of the research focusing on emotion recognition in depression illustrates biases toward identification of negative emotions. Although some studies employ morphing tasks, impairments in recognition of subtle expressions are illustrated through negative biases. Surguladze and colleagues (2004) assessed accuracy of emotion recognition in patients with depression, using two different facial stimuli (sad and happy). Emotions were displayed at 50% or 100% intensity, and were presented for either 100 ms or 2000 ms. Overall, patients were less accurate in identifying facial expressions. There was a tendency away from labeling happy faces as happy; however, they were not identified as sad. In addition, sad faces presented at 100 ms were less accurately
perceived in the depression group. General impairments in identification may explain these findings, as well as overall cognitive deficits congruent with symptoms of depression. In a study examining biases in emotion recognition of neutral faces, Leppanen, Milders, Bell, Terrier, and Hietanen (2004) found that individuals with depression were slower at identifying neutral faces and attributed both sadness and happiness to faces with no expression. Happiness was usually identified when such patients were in remission. While these findings suggest cognitive biases, they could also be interpreted in an emotion sensitivity context. Individuals with depression either during an episode or in remission may be projecting feeling states onto neutral faces during this task. If this was the case, individuals more attuned to their emotional states may also exhibit these biases, illustrating sensitivity to internal emotions. Nevertheless, this specific hypothesis was not tested, and such speculation should be considered with caution.

While there is a paucity of research on emotion sensitivity in depression, studies examining associations between depression and specific emotions are abundant. Depressed mood is one of two necessary criteria for receiving a diagnosis of depression. In addition to this, guilt is another emotion that has been implicated in depression, in accordance with DSM-5 diagnostic criteria (APA, 2013). In a recent meta-analysis, associations between depressive symptoms and two specific forms of guilt – contextual maladaptive guilt (i.e. overwhelming feelings of responsibility over uncontrolled events) and generalized guilt (i.e. general feeling of guilt not associated with a specific event) – were found. It should also be noted that associations between depressive symptomology and shame were significant in this meta-analysis as well (Kim, Thibodeau, & Jorgensen,
In another study, patients with depression were compared with healthy controls on levels of guilt across a variety of situations. The authors found that patients with depression reported significantly higher levels of guilt across most situations. In addition, a family history of depression was related to even higher levels of guilt in patients with depression (Jarrett & Weissenburger, 1990).

**Proposed Study**

As has been illustrated, research on emotion sensitivity in BPD and depression is limited by heterogeneity in patient samples and heterogeneity in methodology. This field of research contains a considerable number of contradictory findings, making interpretations difficult. Furthermore, overreliance on emotion recognition tasks decreases the external validity of these findings; it is unlikely that emotion sensitivity is limited to the cognitive process of recognizing emotional expressions. As previously mentioned, facial recognition tasks focus on emotions in others, not in the self. As such, any conclusions drawn about internal emotion sensitivity are speculative. Therefore, more research is needed to enhance the body of literature in this field, as well as generalize findings to real life events. One way of doing this is to examine emotion sensitivity through a mood-induction behavioural task; one that elicits emotional reactions through memories of past events and assesses a range of specific emotions. The current study attempts to address the three questions: (1) is intensity of emotional reactivity related to borderline and depressive symptomology, (2) after mood induction, does the unique contribution of borderline symptoms correlate differently than the unique contribution of depressive symptoms in terms of specific emotional experiences, and (3) how do individuals who endorse both symptom clusters differ on emotion reactivity?
In this study, mood induction is used to elicit emotional reactions based on recollections of specific experiences. The current study focuses on the second tenet of the emotion sensitivity model – emotion reactivity. While it is likely that all three tenets are related to BPD and depression, this is beyond the scope of the current investigation. It is hypothesized that emotion reactivity to negative affect would be related to both borderline and depressive symptomology, representing a common factor between the two symptom clusters. In addition, it is hypothesized that decreased levels of positive affect would also be related to both borderline and depressive symptomology. This hypothesis is based on research implicating emotion reactivity in BPD and in individuals with mild to moderate depression.

It is also hypothesized that specific emotional experiences will be differentially related to borderline and depressive symptoms. It is expected that positive correlations exist between borderline traits and angry, anxious, and shameful reactions, in accordance with studies cited above, and negative correlations with joy. It is also expected that positive correlations between depressive symptoms and sad and guilty feelings will be observed, in accordance with previous findings and current diagnostic criteria, while we predict negative associations between joy and depressive symptomatology.

Finally, consideration of individuals who endorse both BPD and depressive symptoms is warranted. Since it is hypothesized that BPD and depression share a common factor of emotion reactivity, the authors speculate that individuals who endorse both symptom clusters will not only show associations with the emotions being investigated, but that they would show greater emotion reactivity such that this relationship will persist even when adjusting for the unique associations between
Reactivity and Borderline and Depressive Symptoms and Specific Emotional Experiences. As such, individuals endorsing both symptom clusters will have even greater reactivity, for it is presumed that they would have more pronounced impairments and associations with the common liability of emotion reactivity. This multiplicative effect hypothesis is consistent with findings that individuals with comorbid conditions usually exhibit poorer prognosis and greater impairments (Bunce & Coccaro, 1999; Soloff, Lynch, Kelly, Malone, & Mann, 2000).

Chapter 2
Methods and Results

Methods

Participants and Recruitment

Students were recruited through the Introduction to Psychology subject pool at the University of Toronto Scarborough throughout the Fall 2011 semester. Students were compelled to complete a specified number of research hours as a requirement for this class. If they did not wish to participate in research, an alternative assignment was available. Participants self-selected where and when they chose to participate in research through an online system providing short descriptions of each study. Inclusion criteria included enrollment in the Introduction to Psychology course, ability to consent, and proficiency in the English language. There were no exclusion criteria. The Research Ethics Board of the University of Toronto approved this study.

Measures

Mood Assessment. For this particular study, six feeling states were assessed: Anxiety, sadness, joy, guilt, anger, and shame. Emotional intensity was assessed using a
nine point Likert scale, ranging from *none* to *extreme*. Participants were asked to report
the intensity to which they felt a particular emotion in that moment (i.e. during the study).

**Borderline Symptoms List-23** (BSL-23; Bohus et al., 2009). The BSL-23 is a
23-item self-report questionnaire that assesses symptoms of BPD. Two items related to
suicidal behaviour were removed as requested by the institutional review board.
Individuals were asked to recall how accurate a series of statements were in representing
problems they may have experienced in the past week. A Likert scale with five options
was provided, ranging from *not at all* to *very strong*. Internal consistency for the BSL-23
is high, with Cronbach’s α ranging from 0.94-0.97 (Bohus et al., 2009). For this study,
reliability was also high, with Cronbach’s α of 0.93. In addition, BSL-23 scores clearly
discriminated between borderline patients and a DSM-IV Axis I comparison group,
illustrating its relationship to an actual diagnosis of BPD (Bohus et al., 2009).

**Depression, Anxiety, and Stress Scale** (DASS; Lovibond & Lovibond, 1995).
The DASS is a 42-item self-report measure that is used to assess symptoms of
depression, anxiety, and life stress. One item related to suicidal behaviour was removed
as requested by the institutional review board. Only depression symptomology was
investigated in this study (14 items in total). A Likert scale with four options was used,
ranging from *did not apply to me* to *applied to me very much or most of the time*, to
ascertain the extent to which symptoms applied to individuals over the past week.
Internal consistency for the depression subscale is high, with a Cronbach’s α of 0.91
(Lovibond & Lovibond, 1995). For this study, reliability for the depression subscale was
also high, with a Cronbach’s α of 0.92. In addition, the DASS has been shown to
discriminate between depression and anxiety disordered groups well, and is related to DSM-IV diagnostic groups (Antony, Cox, Enns, Bieling, & Swinson, 1998).

**Procedure**

A series of questionnaires were given to students on a computer, using the program SurveyMonkey. SurveyMonkey is a website that is used to generate online surveys for participants. Data is generated from the surveys, at which point researchers can request to have this data emailed to them for further analyses. This was a mood induction task designed to assess emotional intensity at varying time points. Baseline emotions were examined and then a mood induction task was presented. A measure of a participant’s emotions was provided following this task, to establish how individuals reacted to mood induction. After this, a series of questionnaires were given as a filler task. Finally, emotions were reassessed after this filler. This was repeated again for a second time. Students were then thanked and a credit was awarded for their participation.

Individuals were given the mood assessment (MA) measure at the start of the study and asked to indicate how they were feeling in that particular moment. Participants were then asked to recall a time when they felt socially rejected. The instructions given were the following:

*Think of a time when you felt socially rejected. This could be a time when you were rejected by a romantic partner, friend, or family member. Examples may include a time when someone broke up with you or turned you down, you were ignored or forgotten, or you were teased or humiliated. This event may have caused you to feel embarrassed, anxious, sad, or angry.*
If a specific event did not come to mind, students were given the option to imagine an event in which social rejection or personal failure took place. A series of questions were provided to ascertain if individuals did in fact recall a memory, what the memory entailed, and the cognitive and emotional states that they experienced during that time. The MA was given again, with specific instructions to describe how they were feeling directly after the mood induction task. Following this, a series of questionnaires were completed to serve as a filler task after the first mood induction. Directly after the filler tasks were completed, participants were asked to report how they were feeling using the MA again. Then, students were asked to recall an instance of personal failure. The instructions given were the following:

*Think of a time when you failed at something that was important to you. This could be a time when you did a poor job in terms of academics, work, or concerning a talent that you possess (athletics, art, music). Examples may include a time when you received a low grade, were fired from a job, or lost an important competition. This event may have caused you to feel angry, sad, or disappointed.*

Specific questions about the particular event were asked to ensure participants recalled an event. If participants chose to imagine an event, questions about the imagined event were provided to ensure that participants did in fact visualize an event. Cognitive and emotional experiences surrounding the event were elicited. The MA was then given to indicate how they were feeling directly following this task. A series of questionnaires were provided as a filler task, and the MA was given following these tasks in order to ascertain how participants were feeling at the end of the study. The order of the tasks (i.e. social rejection or personal failure) was randomized to control for order effects.
While the procedure described above is novel, previous studies have utilized recalled negative memories in order to elicit particular emotions in mood-induction tasks. In Kuo and Linehan’s (2009) study, trained assessor’s asked participants to recall the most recent event in which they felt a particular emotion (e.g. sadness) and instructed participant’s to write out this event in vivid detail. Events were rated on intensity of emotional experiences and only those with high ratings of intensity (i.e. eight or higher on a 10-point scale) were rewritten to take two minutes when read out loud. On the day of the experiment, these memories were read out loud and participants were instructed to imagine themselves in that particular situation again. This task followed procedures that were described in a previous study (Pitman, Orr, Forgue, de Jong, & Clairborn, 1987).

**Analyses**

In this particular study, an investigation of emotional reactivity will be focused on ratings after mood induction. Figure A1 (see Appendix) illustrates the MA means for the full sample for each of the six emotions. It also illustrates the different time points in which MA was obtained. Ratings of emotions after mood induction were provided the second and fourth time the MA was given (i.e. MA2 and MA4). Before reactivity scores were obtained, a series of t-tests were conducted to establish if MA2 and MA4 scores were significantly different in one particular emotion. For example, a paired samples t-test was computed for sadness MA2 scores and sadness MA4 scores. If the scores were not significantly different, sadness MA2 and MA4 scores were collapsed by averaging across the two scores. In the event that the scores were significantly different, they would not be collapsed and two sets of hierarchical regressions would be computed for one emotion.
A series of independent samples t-tests were conducted to ascertain if gender should be included in the model as a covariate. In the event that gender was associated with any of the measured variables, it was included as a covariate in the first step of the hierarchical regression models. Baseline emotion levels (i.e. MA1) were included as a covariate in the second step of the model, in order to account for preexisting emotion states. Then, the BSL-23 and the DASS depression subscale were inserted into the model simultaneously. Finally, an interaction term was added to the regression model in order to test for the effects of comorbidity. More specifically, a new variable was computed (BSL-23 x DASS depression subscale, labeled BPDxDep) to assess the multiplicative effects of comorbidity. Emotional reactions of anxiety, sadness, shame, guilt, anger, and joy from the mood assessment (i.e. MA2, MA4, or collapsed MA2 and MA4 scores) were regressed on gender, MA1 scores, BSL-23 and the DASS depression subscale, and BPDxDep. Each emotion was investigated separately. If the whole model was found to be significant for a particular emotion after adjusting for gender and MA1, this would suggest that emotion reactivity is related to variance shared by the BSL-23 and the DASS depression subscale. This suggests that variance accounted for by the two symptom clusters accounts for the significance in the model, after adjusting for gender and baseline emotions.

Including both BSL-23 and DASS depression subscale simultaneously into the model allowed for an investigation of the unique associations between symptom clusters and emotional experiences. The interaction term was added in the fourth step of the model to test for the multiplicative effect of BPDxDep symptomology. In other words, do individuals who endorse high levels of both borderline and depressive features
experience even greater reactivity than those who endorse high levels of only borderline or only depressive symptoms?

A second set of analyses was also conducted to identify group differences between borderline and depressive symptomology as they related to the six emotions mentioned previously. Top and bottom quartiles were used to differentiate groups and a series of analyses of covariance (ANCOVAs) were conducted to test group differences. As mentioned previously, baseline emotional experiences (i.e. MA1) were inserted as covariates in the model, to adjust for baseline emotional levels.

**Results**

**Participants**

One hundred and twenty-one university students were recruited for this study. Participants had a mean age of 18.97 (SD = 3.49, range 18-54) and were 83% female (n = 100). The sample was largely diverse in ethnoracial groups: Asian-American (n = 69; 57%); Caucasian (n = 17; 14%); Multi/Bi-racial (n = 10; 8.3%); Hispanic (n = 5; 4.1%); and African-American (n = 4; 3.3%). In addition, 13.2% (n = 16) of the sample consisted of international students. The mean score for the BSL-23 for the entire sample was 1.84 (SD = 0.59), and the mean score for the DASS was 1.99 (SD = 0.75). The two measures were highly correlated (r = 0.70). A series of independent samples t-tests were conducted to ascertain if gender should be included as a covariate in subsequent analyses. Results revealed that gender had a significant effect on DASS depression scores (t(93.17) = 3.64, p = 0.000, 95% CI [0.13, 0.44]) and on BSL-23 scores (t(39.74) = 2.49, p = 0.017, 95% CI [0.07, 0.64]). All other t-tests were non-significant (all ps > 0.16). As such, all subsequent analyses included gender as a covariate.
Manipulation Check

To ensure that the mood induction was successful, a series of paired samples t-tests were computed. The five negative affect emotions (anxiety, sadness, guilt, shame, and anger) were all summed together. Then, successive MA were compared. All MA were significantly different from the one another: MA1-MA2 ($t(116) = -3.21, p = 0.000, 95\% \text{ CI } [-4.29, -2.14]$), MA2-MA3 ($t(114) = 3.63, p = 0.000, 95\% \text{ CI } [2.04, 6.05]$), MA3-MA4 ($t(113) = -4.59, p = 0.000, 95\% \text{ CI } [-3.16, -2.16]$), and MA4-MA5 ($t(112) = 6.98, p = 0.000, 95\% \text{ CI } [2.48, 4.44]$). This manipulation check was not conducted for joy, as this emotion did not follow the expected pattern that the other emotions did.

T-tests for Emotions

A series of paired samples t-tests were computed to ascertain if emotions assessed at MA2 and MA4 should be collapsed (i.e. they are not significantly different), or if they should remain separate (i.e. they are significantly different). T-tests for sadness, anxiety, shame, and guilt were all not significant (all $p$s > 0.36). The t-test comparing joy assessed at MA2 and joy assessed at MA4 was significant ($t(118) = 3.04, p = 0.003, 95\% \text{ CI } [0.14, 0.65]$), and the t-test comparing anger at MA2 and anger at MA4 was also significant ($t(117) = -2.47, p = 0.015, 95\% \text{ CI } [-0.75, -0.08]$). After a closer examination of the means, it was decided that the two assessment points for joy were to remain separate and that the two assessment points for anger should be collapsed. This decision was made based on the directionality of the effect. Anger followed the same pattern as all other emotions across the MA assessment time points, while joy did not (please see Figure A1). It seems that participants’ reaction patterns in ratings of joy were somehow unique when
compared to the reaction patterns of the other five emotions. As such, the two joy assessment points were kept separate.

**T-tests for Culture**

Due to a high number of Asian-American participants and evidence illustrating cultural differences in emotion reactivity between depressed Asian-Americans and European-Americans (Chentsova-Dutton et al., 2007), additional independent samples t-tests were computed to ascertain if culture should be included as a covariate in subsequent analyses. A dichotomous variable was created in order to compare Asian-Americans versus non-Asian participants on all the variables included in the analyses. T-tests for the BSL-23, DASS depression subscale, anxiety, sadness, guilt, shame, anger, joy1, and joy2 were all not significant (all $ps > 0.13$). As such, culture was not included as a covariate in any subsequent analyses.

**Hierarchical Regressions**

Seven hierarchical regressions were conducted to ascertain associations between specific emotional experiences and borderline and depressive symptomology. Seven hierarchical regressions were completed that included gender in step one, MA1 scores in step two, BSL-23 and DASS depression subscale scores in step three, and BPDxDep interaction term in step four. While only six emotions were investigated, mood assessments for joy were not collapsed. As such, associations with joy were assessed twice for MA2 and MA4 time points, labeled as joy1 and joy2 in all subsequent analyses.

Table A2 presents results for all seven hierarchical regressions. Gender was not significantly associated with any of the emotions. Baseline emotional levels were significantly associated with all six emotions. The common factor hypothesis of emotion
reactivity was supported, such that all of our models at step three were significant, with the exception of joy1 and joy2 models. Additionally, a unique association between borderline features and sadness emotion reactivity was found and a unique association between depressive features and anger emotion reactivity was found.

In step four, a unique negative association between the interaction term BPDxDep and guilt emotion reactivity was found. After this term was included into the model, the unique association between borderline features and guilt \((b = 0.61, p = 0.017)\) became significant, as did the unique association between depressive features and guilt \((b = 0.62, p = 0.004)\). While BPDxDep was not significantly associated with any other emotions, its inclusion in step four altered the results obtained in step three. After including BPDxDep into the model, unique associations between borderline features and sadness \((b = 0.63, p = 0.012)\) remained significant, while associations between depressive features and sadness \((b = 0.41, p = 0.041)\) became significant. A similar effect was observed for anger, such that unique associations between depressive features and anger remained significant \((b = 0.50, p = 0.015)\), while unique associations between borderline features and anger became significant \((b = 0.49, p = 0.046)\).

**Group Analyses**

High and low borderline and depression groups were created from quartile extremes (25\(^{th}\) and 75\(^{th}\) percentiles on borderline and depressive features). Group characteristics are presented in Table A3. A series of t-tests were conducted to ascertain if groups significantly differed on gender or age. High and low borderline groups did not differ significantly on gender \((t(48.45) = 1.74, p = 0.09, 95\% \text{ CI } [0.02, 0.35])\) or on age \((t(55) = 0.22, p = 0.83, 95\% \text{ CI } [-2.67, 3.30])\). Similarly, high and low depression groups
did not differ significantly on gender ($t(23) = 1.45, p = 0.16, 95\% \text{ CI} [0.04, 0.20]$) or on age ($t(51) = 0.82, p = 0.41, 95\% \text{ CI} [-2.00, 4.29]$). A series of two-way ANCOVAs were conducted to investigate group differences on the six emotional experiences outlined above. Baseline emotion (MA1) was included as a covariate. Results are displayed in Table A4. All models tested reached significance, including joy1 and joy2 models. Effects of baseline emotions were significant across all the models, except for guilt. No significant unique associations between the symptom lists and specific emotions were found (all $ps > 0.22$).

**Chapter 3**
**Discussion: Interpretations and Implications**

In the present study we aimed to answer three main questions: does a common factor of emotion reactivity exist between individuals who endorse BPD features and those who endorse depressive features? How do these individuals differ on emotion reactivity in terms of specific emotional experiences? And finally, do individuals who endorse both BPD and depressive features exhibit heightened emotion reactivity? Overall, results largely supported our common factor hypothesis, such that the regression models investigating anxiety, sadness, shame, guilt, and anger were all significant. Associations between low joy and BPD and depressive features were not found in our regression analyses. However, these associations were found in our group analyses. Our hypotheses concerning associations between BPD and depressive features and specific emotions were partially supported. Unique associations between BPD features and sadness, anger, and guilt were found. Further, unique associations between depressive features and the same three emotions were also found. Finally, our initial hypothesis that
a multiplicative effect would be found, such that individuals who endorse both BPD and depressive features would exhibit a greater magnitude of emotion reactivity above and beyond what is seen in the unique associations described above, was not supported. Specifically, the BPDxDep interaction term was only significantly related to guilt, in the opposite direction that was hypothesized.

For ease of interpretation, group analyses were also conducted. Groups were created by differentiating individuals in quartile extremes on BPD and depressive features. All models reached significance, including joy1 and joy2 models. No unique associations between BPD and depressive features and any of the six emotions were obtained.

**Common Factor**

As was previously mentioned, researchers investigating common factors between BPD and depression usually find that both share internalizing symptoms. In this study, a more descriptive account of this common factor was established – emotion reactivity to negative affect. It is important to note that this establishes that both BPD and depressive features are associated with emotion reactivity. As was mentioned above, the relationship between BPD and emotion reactivity was less than clear. This study corroborates findings that individuals endorsing BPD features do experience heightened reactivity, specifically to negative emotions. Similarly, individuals experiencing mild to moderate depressive features seem to also exhibit increases in emotional reactivity to negative emotions – reaffirming the importance of this symptom in specific depression populations. More specifically, such individuals experience heightened reactivity in the five emotions investigated: Anxiety, sadness, guilt, shame, and anger.
The finding that joy was not associated with BPD or depressive features in the regression analyses was surprising – as it was initially proposed that low joy would be related to both symptom clusters. This hypothesis converges with evidence that BPD and depression groups are both related to decreases in positive affect (Staebler et al., 2009). While the results did not support this view, it is possible that this was due to a methodological error. Specifically, the task that was employed prompted participants to recall and outline an event in which they experienced a personal failure or interpersonal rejection. Specific questions that targeted joy were not investigated, and this could explain the above findings. In addition to this, Figure A1 (see Appendix) illustrates the directionality of the mean scores at each time point that the mood assessment was administered. Evidently, the directionality of emotion reactivity specifically in terms of joy did not follow the pattern of the other emotions – it seems that participants did not recover after administration of the filler tasks and joy steadily declined throughout the duration of the experiment. This could indicate that the task utilized in this study exerted different effects on joy. Furthermore, in a study validating a new emotion sensitivity measure, the questionnaire was subdivided into negative egocentric sensitivity (NES), a factor that was largely correlated with neuroticism, and positive interpersonal sensitivity (PIPS), a factor that was largely correlated with empathy (Guarino, Roger, & Olason, 2007). It was found that individuals with high PIPS scores were able to identify emotions in others more accurately – a finding that has not been consistently found in either BPD or depression. As such, it is possible that joy is an “other focused” emotion that is related to the PIPS dimension of emotion sensitivity. Considering the nature of the task utilized, associations with joy may have been found if participants were prompted to analyze
emotions of others. Future research should investigate emotion sensitivity in BPD and depression in emotions of others, using new paradigms that do not rely solely on facial recognition tasks.

While the regression models tested for joy were not significant, an effect was found in our group analyses, such that both joy1 and joy2 models were significant. These results do not converge with the previous analyses. Nevertheless, it is possible that an effect was obtained at the extreme ends of the continuum (i.e. the quartile extremes). This could indicate that associations with joy and BPD and depression are present in more severe forms of psychopathology. Future research should investigate this relationship in clinical populations.

**Unique Associations**

Unique associations between BPD and depressive features did not completely agree with the proposed hypotheses. BPD features were uniquely associated with anger, while depressive features were uniquely associated with sadness and guilt. Unexpectedly, BPD features were also uniquely associated with sadness and guilt and depressive features were also associated with anger. Contrary to our hypotheses, unique associations between features of psychopathology and the other emotions (shame, anxiety, and joy) were not found.

Unique associations between depressive features and sadness reactivity were not surprising. These findings corroborate diagnostic criteria and literature cited above concerning associations between sadness and depression (APA, 2013; Beck, 1976). In addition, unique associations between depressive features and guilt were also not surprising. This is consistent with DSM-V defined criteria, as well as the aforementioned
The relationship between anger and depressive features was not expected. Nevertheless, relationships between anger and depression have been established in both normal (Kashdan & Roberts, 2007) and patient populations (Besharat, Abdolmanafi, Farahani, & Khodaii, 2011). In a recent study investigating the relationship between depression and anger, Besharat, Nia, and Farahani (2013) found that this association was mediated by poor emotion regulation strategies and anger rumination. More specifically, this model was found significant in three of the five dimensions of anger – anger arousal (closely linked to anger reactivity), anger-eliciting situations, and hostile outlook. It seems that individuals with depression may experience heightened anger reactions because of hostile outlooks and anger-eliciting situations.

These findings are especially interesting taking into consideration the events that participants were asked to recall – those in which they felt rejected or experienced a personal failure. Hostile outlooks on such events and heightened anger arousal could explain the associations seen between depressive features and anger. It would be interesting to test out the meditational effects of hostile outlooks and heightened arousal in the elicitation of anger. Future research should investigate factors that may mediate the relationships between depressive features and anger.

Unique associations between BPD features and anger comply with previous research as well as diagnostic criteria (APA, 2013; Koenigsberg et al., 2002). However, associations between BPD features and sadness were not expected. It was anticipated that the common variance between BPD and depressive features would account for a relationship with sadness, and that only unique features of depression would be related to sadness. It is possible that symptoms of affective lability in BPD as described by the
BSL-23 (e.g. “My mood rapidly cycled in terms of anxiety, anger, and depression”) were uniquely related to sadness reactivity. It is also possible that other symptoms of BPD, such as feeling worthless or being disgusted with oneself, were uniquely related to sadness. Furthermore, associations between BPD features and guilt were also unexpected. A large majority of the research on BPD and specific emotions focuses on shame (e.g. Rusch et al., 2007). Associations between guilt and BPD have yet to be truly investigated. Shame has been defined as an emotion that is associated with self-blame, in comparison with guilt, which is defined as an emotion that is associated with blaming the particular behaviour, but not the self (Lewis, 1971; Niedenthal, Tangney, & Gavanski, 1994). It is possible that individuals with BPD tend to blame external factors, such as actions, versus blaming internal factors, such as intelligence, when confronted with memories of interpersonal rejection or personal failure. This may be linked to features of BPD that are related to the externalizing dimension. As defined previously, the externalizing dimension is characterized by turning distress outwards (e.g. substance abuse, hostility, etc.). However, it is important to note that guilt has also been associated with adaptive, reparative, and empathic behaviour (Thompson & Hoffman, 1980). It is possible that in BPD, blame may be placed on behaviours or on individuals, resulting in maladaptive guilt reactions. Future research should investigate associations between BPD, guilt, and subsequent reactions to feelings of guilt.

No unique associations between either symptom clusters and anxiety and shame were surprising. However, this implies that the shared variance was significantly related to both emotions. Our initial hypothesis suggests that individuals who endorse BPD symptoms and those who endorse symptoms of depression share emotion reactivity to
negative affect, and that this would be reflected by significance of regression models as a whole. While unique features of BPD and depression were not related to shame or to anxiety reactivity, both were still related to these emotions via shared symptoms between BPD and depressive features. On the other hand, sadness, guilt, and anger may be related to commonalities between BPD and depressive features, but they were also associated with unique features of each symptom cluster. The question now becomes, what is unique to BPD features that is related to these three emotions? Similarly, what is unique to depressive features that is related to these three emotions? While both were related to sadness, it is possible that this relationship is due to low mood in depression, while it is due to affective lability in BPD. In addition, guilt may be related to excessive rumination concerning past failures in depression, while it may be related to externalization and blaming the other in BPD. Finally, anger may be related to anger towards the self that is internalized in depression, while it may be related to hostility and aggression in BPD. It is important to note that these relationships are purely speculative, and that future research should attempt to elucidate what factors makes these relationships unique to each symptom cluster.

It should be noted that the group analyses did not reveal the same results as the regression analyses. In the group analyses, no unique associations were found between BPD features, depressive features, and any of the six emotions. This should not take away from the obtained results, however. It is likely that unique associations were not established in the group analyses due to low power. Taking this into consideration, it is imperative that future research examine these associations in clinical samples, in order to
explore differences in emotion reactivity across BPD and depressive groups more explicitly.

**Interaction effects**

In the current investigation, an interaction term between BPD and depression symptom clusters was computed to test the multiplicative effect hypothesis. It was initially expected that individuals who endorse both symptom clusters would exhibit heightened reactivity to all emotions, and would exhibit greater reactivity than individuals who just endorsed BPD or depression symptoms. The multiplicative effect hypothesis in this study was not supported in either regression or group analyses. This was surprising considering evidence suggesting that individuals with comorbid diagnoses usually exhibit poor outcomes in comparison with individuals with only one diagnosis (Bunce & Coccaro, 1999; Soloff et al., 2000). More surprisingly, we found that individuals who endorse both BPD and depressive symptoms were negatively associated with guilt reactivity, which suggests that these individuals experienced less guilt when confronted with aversive memories. There are several possible explanations for this unexpected finding. Such findings could be attributed to the current sample. In this study, an undergraduate sample was recruited to examine associations between symptom clusters and emotion reactivity. Levels of pathology were considerably lower than would be expected in clinical populations. As such, the relationships between feelings of guilt and clinical comorbidity may corroborate the initial hypothesis. This does not explain, however, the negative relationships between guilt and endorsement of both BPD and depressive features. Another possibility is that symptoms of depression dampen the relationship between BPD symptoms and guilt, or vice versa. For instance, if individuals
who endorse BPD symptomology tend to feel guilt due to associations with externalizing behaviours, depression may dampen this reactivity because of its strong associations with internalization of guilt. In a sense, these associations may cancel themselves out in individuals who endorse both BPD and depressive symptomology. Yet another possibility is that individuals who endorsed both depressive and BPD features suppressed feelings of guilt, therefore underreporting reactions of guilt. Suppression is a maladaptive emotion regulation tool that is associated with decreases in the behavioural expression of an emotion but no decreases in the actual emotional experience. Suppression is also associated with impairments in memory, which the tasks outlined above heavily relied on (Gross, 2002). To test this hypothesis, one could apply behavioural and physiological measures of emotion reactivity in a guilt mood induction task in order to ascertain discrepancies between self-reported guilt and physiological levels of arousal. Future research should examine differences between comorbid and pure conditions in order to establish differential relationships in guilt reactivity across a variety of measures (e.g. behavioural, physiological, etc.).

Limitations and Implications

While the findings in the current investigation are interesting, there are some important limitations that must be outlined. This study utilized an undergraduate sample and tested associations between emotion reactivity and symptom clusters. As such, conclusions concerning relationships between diagnostic groups and emotion reactivity cannot be established. Due to low rates of psychopathology, it is also possible that specific associations between BPD, depression, and emotional experiences were not observed because of the nature of the sample. In addition, the use of analogue samples in
assessing psychopathology is controversial. Generalizability of the results obtained above to individuals in clinical populations is questionable, for student samples are a specific subset of the population. Moreover, it has been posited that students and individuals diagnosed with depression exhibit distinct forms of the disorder (Gotlib, 1984). While others refute this theory (e.g. Vredenburg, Flett, & Krames, 1993), it is still important to be cognizant of these limitations when interpreting the above findings. As such, it is imperative that future research examines these relationships in patient populations, to more clearly ascertain similarities and differences between BPD, depression, comorbid BPD and depression, and emotion reactivity.

The ethnoracial labels that were used in this study were derived from the National Institute of Health. While these labels are commonly used in research in America, the current sample was a Canadian sample. This is problematic because the above labels are not congruent with the commonly used labels in Canadian research. For instance, the use of the word American instead of Canadian presents as an issue for correct identification. While it may be assumed that individuals were able to self select the most appropriate label, this is not necessarily the case. In addition, the ethnoracial labels are too broad; both South and East Asians can identify as Asian-American. This is problematic for it creates heterogeneity in ethnoracial groups, therefore specific cultural effects cannot be clearly delineated. It is imperative that in future research appropriate ethnoracial groups are used and that more specific labels be used in order to decrease errors in identification and heterogeneity.

The current study also investigated emotion reactivity aggregated across two tasks – interpersonal rejection and personal failure. While this allows for a general examination
of emotion reactivity, it is likely that individuals react differentially across these two tasks. For example, it is possible that an individual feels angry when recalling instances of interpersonal rejection, but feels shameful when recalling instances of personal failure. In a recent study, Tomko and colleagues (2012) found that individuals with depression were less likely to spend time with others when angry, while this was not observed in individuals with BPD. Such findings illustrate the importance of the interplay between symptom clusters, emotions, and environmental events. Establishing differences in emotion reactivity across a variety of situations is necessary, for it is more reflective of every day life events. Future studies should investigate these differences in order to ascertain relationships based not solely on symptom clusters, but also on environmental conditions. Such research will also allow for a more holistic investigation of similarities and differences between BPD and depression.

Furthermore, the current study focused solely on emotion reactivity and its relationship to BPD and depressive features. The three tenets of the emotion sensitivity model as proposed by Linehan (1993) were not completely investigated. It is important that all tenets of this model be examined, in order to elucidate what factors of emotion dysregulation are related to both disorders, and which are uniquely related to BPD or depressive symptoms. As such, future research should investigate all three tenets of the emotion sensitivity model and associations between specific emotions and BPD and depression.

In the current study, participants were asked to recall two negative events. An examination of positive events and emotion reactivity (e.g. a time when they were complimented; a time when they were successful) was not undertaken. This may explain
the lack of association between BPD and depression symptom clusters and emotional experiences of joy. An assessment of heightened positive and negative emotion reactivity is important in determining associations between psychopathology and emotions across a full array of experiences. Future studies should aim to investigate emotion reactivity across both positive and negative life experiences.

Taking into consideration the above limitations, the current results still possess far-reaching implications. The broad aim of the current investigation was to ascertain common and unique factors of BPD and depressive features. In doing so, it was established that features of BPD and depression are related to emotion reactivity, but that associations with specific emotions are different. This more clearly explains the high comorbidity rates between BPD and depression, along with the differences in presentation between individuals with either BPD or depression. Future research should aim to investigate the mediating role of cognitive distortions and perceptions in these populations, to explain associations between BPD, depression, and specific emotional experiences.

Furthermore, the above findings have clear implications for the implementation of therapy. In therapy, recollections of negative events often take place. Taking into consideration high emotion reactivity associated with such recollections, it is encouraged that therapist not only focus on the event, but also pay due attention to the emotional experiences of the patient. This may be especially important with specific emotional experiences and how they differentially relate to BPD or depression. For instance, if a therapist is confronted with guilt reactivity in a patient with depression, intervening in the rumination process may be most beneficial. On the other hand if confronted with guilt
reactivity in a patient with BPD, cognitive distortions related to externalized blame may be targeted in therapy. Further, if confronted with lack of guilt in a patient with both BPD and depression, cognitive reappraisal as a tool to regulate emotions can replace maladaptive suppression. Along these lines, it has been found that individuals with depression who experience anger tend to suppress these emotions (Allan & Gilbert, 2002; Gilbert, Gilbert, & Irons, 2004). Considering associations between depressive features and anger reactivity, it is encouraged that therapists identify these emotions and discourage suppression. By doing so, they can provide patients with the appropriate tools to regulate particular emotional experiences more effectively.

**Conclusion**

In the current investigation, a common factor of emotion reactivity was observed between BPD and depression symptom clusters. This finding reaffirms previous notions that high comorbidity rates between BPD and depression can be explained by a shared factor – emotion reactivity to negative affect. In addition to this, specific emotional experiences were uniquely associated with BPD and depressive features, illustrating differences in emotion reactivity profiles. In particular, BPD features were related to sadness, guilt and anger, and depressive features were also related to these three emotions. While no associations between joy and the above symptom clusters were found, this may be an artifact of the current methodology. In addition, the multiplicative effect hypothesis was not supported. This is likely due to the nature of the current sample (i.e. nonclinical sample). Surprisingly, it was found that individuals who endorse both symptom clusters experience significantly less guilt reactivity. This relationship suggests that individuals who endorse both BPD and depressive features may experience a
dampening in reactivity, or that they may utilize suppression to regulate emotions, resulting in underreporting of reactivity. Implications for the current findings span theoretical and practical fields of psychology by attempting to clearly identify similarities and differences between these two highly comorbid conditions, and by identifying specific emotional experiences that should be of interest in therapy.
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Appendix 1: Mood Assessment Means for the Entire Sample

Figure 1. MA1 = Initial mood assessment. MA2 = Mood assessment after first mood induction. MA3 = Mood assessment after completion of filler tasks. MA4 = Mood assessment after second mood induction. MA5 = Final mood assessment after completion of filler tasks.
## Appendix 2: Hierarchical Regression for Six Emotions and Borderline and Depressive Features

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Anxiety</th>
<th>Sadness</th>
<th>Guilt</th>
<th>Shame</th>
<th>Anger</th>
<th>Joy1</th>
<th>Joy2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\Delta R^2$</td>
<td>$\beta$</td>
<td>$\Delta R^2$</td>
<td>$\beta$</td>
<td>$\Delta R^2$</td>
<td>$\beta$</td>
<td>$\Delta R^2$</td>
</tr>
<tr>
<td>Step 1</td>
<td>.00</td>
<td>.00</td>
<td>.01</td>
<td>.01</td>
<td>.03</td>
<td>.00</td>
<td>.00</td>
</tr>
<tr>
<td>Gender</td>
<td>.01</td>
<td>.01</td>
<td>.11</td>
<td>.17</td>
<td>.03</td>
<td>.02</td>
<td></td>
</tr>
<tr>
<td>Step 2</td>
<td>.59**</td>
<td>.31**</td>
<td>.27**</td>
<td>.40**</td>
<td>.27**</td>
<td>.38**</td>
<td>.58**</td>
</tr>
<tr>
<td>Baseline$^a$</td>
<td>.77**</td>
<td>.56**</td>
<td>.52**</td>
<td>.64**</td>
<td>.52**</td>
<td>.62**</td>
<td>.76**</td>
</tr>
<tr>
<td>Step 3</td>
<td>.03*</td>
<td>.08**</td>
<td>.07*</td>
<td>.04*</td>
<td>.08**</td>
<td>.01</td>
<td>.00</td>
</tr>
<tr>
<td>BPD</td>
<td>.09</td>
<td>.24*</td>
<td>.07</td>
<td>.13</td>
<td>.13</td>
<td>-.08</td>
<td>-.07</td>
</tr>
<tr>
<td>Dep</td>
<td>.12</td>
<td>.11</td>
<td>.20</td>
<td>.10</td>
<td>.21*</td>
<td>.00</td>
<td>.07</td>
</tr>
<tr>
<td>Step 4</td>
<td>.01</td>
<td>.02</td>
<td>.03*</td>
<td>.01</td>
<td>.02</td>
<td>.00</td>
<td>.00</td>
</tr>
<tr>
<td>BPD x Dep</td>
<td>-.40</td>
<td>-.66</td>
<td>-.90*</td>
<td>-.36</td>
<td>-.62</td>
<td>.25</td>
<td>-.32</td>
</tr>
<tr>
<td>Total R$^2$</td>
<td>.62**</td>
<td>.41**</td>
<td>.37**</td>
<td>.46**</td>
<td>.38**</td>
<td>.39**</td>
<td>.58**</td>
</tr>
</tbody>
</table>

$^a$Specific baseline emotions were inserted into the model depending on which emotion was being investigated. For example, for anxiety, baseline anxiety levels were inserted into the model, while baseline sadness levels were inserted into the model for sadness. *Note. Baseline = Mood assessment 1 baseline emotion (anxiety, sadness, guilt, shame, anger, joy). BPD = borderline personality disorder features. Dep = Depressive features. BPDxDep = Borderline and depression features interaction term. *$p < .05$. **$p < .01$. 
Appendix 3: Group Characteristics of Upper and Bottom Quartiles of BPD and Depressive Features

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Age</th>
<th>Female</th>
<th>Pathology Mean Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>High BPD Features</td>
<td>28</td>
<td>19.23 (1.18)</td>
<td>26</td>
<td>2.29</td>
</tr>
<tr>
<td>Low BPD Features</td>
<td>30</td>
<td>19.55 (7.64)</td>
<td>23</td>
<td>1.48</td>
</tr>
<tr>
<td>High Dep Features</td>
<td>30</td>
<td>20.31 (7.39)</td>
<td>30</td>
<td>2.19</td>
</tr>
<tr>
<td>Low Dep Features</td>
<td>24</td>
<td>19.17 (1.16)</td>
<td>22</td>
<td>1.38</td>
</tr>
</tbody>
</table>

*Note.* Pathology mean scores were computed from the BSL-23 for BPD features and from the DASS depression subscale for depression features. BPD = Borderline personality disorder. Dep = Depression.
## Appendix 4: ANCOVAs for High/Low Borderline and Depressive Features Groups and Emotion Reactivity

<table>
<thead>
<tr>
<th>Factor</th>
<th>Anxiety</th>
<th>Sadness</th>
<th>Guilt</th>
<th>Shame</th>
<th>Anger</th>
<th>Joy1</th>
<th>Joy2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$F$</td>
<td>$p$</td>
<td>$F$</td>
<td>$p$</td>
<td>$F$</td>
<td>$p$</td>
<td>$F$</td>
</tr>
<tr>
<td>Baseline$^a$</td>
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<td>.000</td>
<td>6.32</td>
<td>.017</td>
<td>2.52</td>
<td>.123</td>
<td>30.78</td>
</tr>
<tr>
<td>BPD</td>
<td>.90</td>
<td>.351</td>
<td>1.57</td>
<td>.220</td>
<td>.60</td>
<td>.444</td>
<td>.33</td>
</tr>
<tr>
<td>Dep</td>
<td>.06</td>
<td>.815</td>
<td>.00</td>
<td>.950</td>
<td>.09</td>
<td>.763</td>
<td>.00</td>
</tr>
<tr>
<td>Total</td>
<td>29.85</td>
<td>.000</td>
<td>13.52</td>
<td>.000</td>
<td>6.25</td>
<td>.002</td>
<td>17.22</td>
</tr>
</tbody>
</table>

$^a$Specific baseline emotions were inserted into the model depending on which emotion was being investigated. For example, for anxiety, baseline anxiety levels were inserted into the model, while baseline sadness levels were inserted into the model for sadness. 

*Note.* Baseline = Mood assessment 1 baseline emotion (anxiety, sadness, guilt, shame, anger, joy). BPD = borderline personality disorder features. Dep = Depressive features.