An Investigation of the Biosocial Model of Borderline Personality Disorder

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Abstract

Linehan's biosocial model of borderline personality disorder posits that the disorder is due to emotional dysregulation, which is in turn caused primarily by the interaction between an emotionally vulnerable child being raised in an invalidating environment. Despite the popularity of the therapy derived from this model, dialectical behaviour therapy, this model has not been empirically validated.

In the first study self-report measures of borderline traits, emotional dysregulation, childhood emotional vulnerability and invalidating parenting were administered to a community sample. Emotion dysregulation was found to strongly predict borderline traits; however the interaction between childhood emotional vulnerability and invalidating parenting was not found to be an important part of the model. Further, the effects of validating parenting upon borderline traits were not mediated by emotion dysregulation.

The second study extended the findings of the first, attempting to improve the measure of emotional dysregulation by including aspects of emotional dysregulation not currently assessed. Further, the applicability of the biosocial model to a comparison psychopathology, chronic worry, was assessed. It was found that childhood emotional vulnerability had a similar relationship to both forms of pathology, suggesting that this represents a general risk factor (akin to childhood neuroticism). Further, differing patterns of emotion regulation deficits were noted for each of the pathologies.

Overall these studies failed to support the biosocial model, raising questions as to the posited relationships between key constructs in the model and their specificity to borderline personality disorder. A potentially more accurate model is

posited, that highlights the role of a number of factors in the disorder and its evolving nature.

Declaration

I declare that this submission is my own work and that to the best of my knowledge and belief it contains no material previously published or written by another person, nor material which has been accepted for the awarding of another degree or diploma at a university or initiation of higher learning.

The data that was used for this submission was obtained via two research studies approved by the Macquarie University Human Research Ethics Committee (Reference Numbers: 5201200025 and 5201300082).

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Chapter I: Introduction

Section 1. Overview

Borderline personality disorder (BPD) is a chronic mental health condition, associated with significant impairments in interpersonal, behavioural and affective functioning. A number of theories have been posited to account for its development, one of the most significant being Linehan's biosocial model, the theory upon which her dialectical behaviour therapy (DBT) is based. It is theorised that BPD is primarily a disorder of emotion regulation, with this dysregulation being due to an emotionally vulnerable child having being raised in an emotionally invalidating environment. The aim of this thesis is to attempt to determine the validity of this model of BPD and, if this model is found to be lacking, posit a more accurate model of BPD.

The construct of emotion dysregulation and the empirical literature examining its aetiology are discussed. An attempt is made to reconcile the extant empirical literature with the biosocial model, including examining psychosocial risk factors associated with BPD, such as a history of child abuse and poor parenting. The clustering and complexity of these risk factors are highlighted, as is the resulting difficulty in ascertaining whether the factors support one model of BPD over another.

Various psychophysiological factors associated with the disorder are also discussed. It is noted that whilst such studies can offer some support for the model, they suffer from a range of methodological issues that make it difficult to draw strong conclusions from such research.

It is proposed that the biosocial model could derive support by either the treatment drawn from the model (i.e. DBT) demonstrating superiority in treating BPD over other therapies based upon less accurate models, or alternatively by directly testing the key aspects of the model via cross-sectional research. Unfortunately the treatment literature does not support the clear superiority of DBT over other forms of

therapy, with the limited studies available exhibiting a range of conclusions. Further, studies that directly test key aspects of the biosocial model have generally failed to support the central tenets of the model, in particular the importance of the interaction between childhood emotional vulnerability and invalidation. It is however, noted that several of these extant studies have methodological features that limit the extent to which conclusions about the model can be drawn.

Several a priori hypotheses are articulated based upon the biosocial model, in particular that the interaction between childhood emotional vulnerability and invalidating parenting would strongly predict adult emotional dysregulation, which in turn would mediate the relationship between these childhood factors and borderline personality traits. These hypotheses are then tested in two cross-sectional studies exploring the interactions between the various key constructs. The second study also examines whether the relationships between the constructs are similar for a comparison psychopathology (chronic worry) and the nature of the emotional dysregulation in each of the disorders.

Whilst it was found that emotional dysregulation strongly predicts borderline traits, and that this association appears stronger than in the case of chronic worry, several key hypotheses from the biosocial model were not supported. The interaction between childhood emotional vulnerability and invalidating parenting was not found to be critical in explaining adult emotional dysregulation. Emotion dysregulation only partly mediated the relationship between the childhood antecedents and borderline traits. Childhood emotional vulnerability had a similar role in the development of either chronic worry or borderline traits, suggesting that it may represent a more general risk factor for psychopathology.

Finally, a summary of the findings are presented, weaknesses in the study methodology are discussed, and future avenues for research are proposed, including the positing of a more appropriate model to account for the aetiology of BPD.

Section 2. Borderline Personality Disorder

2.1 Construct

Borderline personality disorder (BPD) has a long ontological history, indeed its name still bears the hallmarks of the time when such individuals were considered to be “borderline” schizophrenic (Gunderson, 2009). Later attempts, whilst still maintaining a strong psychoanalytic focus and bearing a limited relationship to our current understanding of the pathology, nevertheless began to view such clients’ difficulties as being due to personality problems (e.g. Kernberg, 1967). Efforts persisted to clarify the dysfunction (e.g. Grinker, Werble, & Drye, 1968) until it was formally incorporated in the Diagnostic and Statistical Manual of Mental Disorder (3rd ed.; DSM-III; American Psychiatric Association, 1980). With some minor modifications it has been retained in the subsequent editions of the DSM (i.e. American Psychiatric Association, 1994, 2013). As BPD is currently defined in DSM-5, as with all personality disorders, it is “an enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individual’s culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time and leads to distress or impairment” (American Psychiatric Association, 2013, p. 645), with 5 of the following criteria having to be met:

1. Frantic efforts to avoid real or imagined abandonment. (*Note:* Do not include suicidal or self-mutilating behaviour covered in Criterion 5.)

2. A pattern of unstable and intense interpersonal relationships characterised by alternating between extremes of idealisation and devaluation.

3. Identity disturbance: markedly and persistently unstable self-image or sense of self.

4. Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating). (*Note:* Do not include suicidal or self-mutilating behaviour covered in Criterion 5.)

5. Recurrent suicidal behaviour, gestures, or threats, or self-mutilating behaviour.

6. Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days).

7. Chronic feelings of emptiness.

8. Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights).

9. Transient, stress-related paranoid ideation or severe dissociative symptoms. (American Psychiatric Association, 2013, p. 663)

Whilst this definition has remained relatively constant over the recent incarnations of the DSM, it is not without its problems (Lewis & Grenyer, 2009), the shortcomings of the current system of personality disorder diagnosis being explicitly acknowledged in the DSM-5 (American Psychiatric Association, 2013).

Because 5 of 9 criteria are required for the diagnosis of BPD there are potentially 256 different combinations of symptoms which could result in an identical diagnosis. Further, two individuals with the same diagnosis may only share a single common feature. This is problematic as it makes it difficult to assert that the diagnosis is an

adept description of their pathology, except in the most general of terms. Despite this DSM-5 does not offer any means to distinguish between such clients.

In addition the diagnostic criteria do not address the impact of age upon presentation. It could be argued that a pattern of unstable interpersonal relationships, impulsivity and affective instability may be relatively common amongst most adolescents, whereas similar behaviours, even if demonstrated to a lesser degree, would be clearly indicative of personality dysfunction in older adults. Consequently the criteria as they currently stand probably do not allow for the accurate diagnosis of dysfunction across the lifespan.

Furthermore, there can be difficulties in distinguishing BPD from other mental health problems. A range of mental health issues, including anxiety, affective and other personality disorders are significantly more common amongst those diagnosed with BPD (Lenzenweger, Lane, Loranger, & Kessler, 2007). Further, some aspects of BPD overlap significantly with other disorders; in particular criteria 6 and 7 are likely to be also experienced by individuals suffering from affective and/or anxiety disorders. Consequently this creates difficulties in establishing diagnostic clarity and also raises questions of construct validity amongst the disorders.

Further, whilst factor analysis supports the view that BPD represents a unified construct, it has also identified that the diagnosis fits a 3 factor solution, spread unevenly across the 9 criteria (Sanislow et al., 2002). This is problematic, in that if an individual's dysfunction mainly occurs on an aspect against which most of the criteria load they are more likely to be diagnosed with BPD, relative to an individual whose dysfunction occurs in a factor which is only assessed by a few of the criteria. Thus the criteria as they currently stand have the potential to lead to both the under and over diagnosis of the disorder.

Another issue with the criteria is their dichotomous nature, where an individual is assessed as either possessing or not possessing an impediment of clinical significance, despite, with the possible exception of criteria 5, all of these traits existing upon a continuum. This leads to problems associated with determining appropriate cutoffs regarding what constitutes clinically significant symptoms. Further, it may result in the illogical situation where an individual who is severely affected by 4 of the 9 criteria would not be diagnosed with BPD, whilst another individual who met 5 criteria with symptoms of only marginal clinical significance would be diagnosed with the disorder.

A further issue with BPD is that there are concerns as to whether it fits the general description of personality disorders in the DSM, namely that it is "stable over time" (American Psychiatric Association, 2013, p. 645). It has been found that at least some aspects of BPD remit over time (Cohen et al., 2008; Paris & Zweig-Frank, 2001; Zanarini, Frankenburg, Hennen, Reich, & Silk, 2006; Zanarini, Frankenburg, Reich, & Fitzmaurice, 2010), with some features, such as suicidal and parasuicidal behaviour, tending to remit faster than others, such as feelings of chronic emptiness (Zanarini et al., 2007). These findings lead to two problems – first, if some clients remit whilst others do not, should both individuals be considered to have the same disorder in the first instance (or alternatively could their presentations have been similar but with different etiologies). Further, it suggests that the issues of meeting 5 out of 9 criteria are likely to be problematic in that at some point in the life trajectory of the disorder an individual will drop below the threshold in 5 of the 9 criteria, no longer meeting criteria for the disorder whilst still being mentally unwell and experiencing deficits associated with the disorder.

Perhaps the greatest concern regarding our current conceptualisation of BPD, is that whilst there is some level of consensus regarding the validity of the construct in the literature, a clear understanding as to the development of the disorder remains elusive, with multiple competing theories having been developed.

2.2 Theories of Development

A range of developmental models have been proposed to account for the development of BPD (Zanarini, 2000), with many of these focusing upon early childhood experiences.

Whilst genetics clearly play a role in the development of the disorder, with BPD possessing an estimated heritability of approximately 40% (Amad, Ramoz, Thomas, Jardri, & Gorwood, 2014), life experiences also have a significant role in the development of the disorder. Notably there exists a correlation between childhood abuse and borderline traits (Guzder, Paris, Zelkowitz, & Feldman, 1999), with this association being present in non-Western populations (Igarashi et al., 2010; Zhang, Chow, Wang, Dai, & Xiao, 2012) and with this relationship appearing to be stronger for BPD compared with other personality disorders (Zanarini, Gunderson, Marino, Schwartz, & Frankenburg, 1989). Further research has specifically focused upon parenting, noting that individuals with borderline personality disorder are at increased risk of having experienced poor parenting (Zweig-Frank & Paris, 1991), and further, relative to those with other personality disorders, those with BPD are similarly at increased risk of these failings having been present in both parents (Zanarini et al., 2000).

These findings suggest that developmental experiences may play a role in the aetiology of the disorder. What remains unclear is how these experiences lead to the development of the disorder, with a number of theories having been proposed.

Fonagy and other researchers (e.g. Fonagy & Bateman, 2007, 2008; Fonagy, Luyten, & Strathearn, 2011; Fonagy, Target, & Gergely, 2000; Sarkar & Adshead, 2006 etc.) have suggested that BPD is primarily due to a chronic disturbance in the attachment relationship between a child and their primary caregiver. It is posited that when a child experiences an emotional arousal, which is met by a manifestly inappropriate response, the child learns to respond to this by engaging a dissociative state. It is further suggested that as this interaction plays out repeatedly, that the child fails to develop organisation of self and the capacity for mentalisation, which in turn leaves them even more vulnerable to the impact of future trauma, with there being some evidence suggesting that the capacity to mentalise may be impaired in those with BPD (Fonagy et al., 1996).

Another set of theorists have suggested a somewhat different pathway, arguing that in individuals with BPD there is a basic disruption of the neurological basis that promotes the formation and maintenance of unconscious emotional memory (Baird, Veague, & Rabbitt, 2005). It is further posited that this interferes with the development of appropriate attachment relationships, and during the period of intense neurological change associated with adolescence, that this basic deficit manifests itself in the behavioural and emotional sequelae associated with BPD.

Several psychodynamic therapists have advanced models to account for the disorder (Goldstein, 1995), with some therapists asserting that borderline personality pathology is fundamentally a failure to develop a sense of self, resulting from a lack

of integration of object and self representations experienced under conflicting affective states (Kernberg & Michels, 2009).

From a cognitive therapy perspective, BPD (and indeed all personality disorders) are due to the adoption of maladaptive belief systems (Beck, Freeman, & Davis, 2004; Pretzer & Beck, 1996). Such beliefs may be due to an individual's developmental history, to their compensatory strategies or may involve maladaptive reactions to current events.

One notable form of cognitive therapy, schema therapy, has been found to be effective in treating BPD in a number of clinical trials (e.g. Giesen-Bloo et al., 2006; Nadort et al., 2009). Schema therapy practitioners have attempted to offer an account for the development of BPD (Kellogg & Young, 2006; Sempértegui, Karreman, Arntz, & Bekker, 2013; Young, 1990, 2002), with it being posited that BPD is due to a child's temperament/genetics, their childhood experiences and also interactions between the child's temperament and their parents' parenting style. It is posited that these factors lead to the development of early maladaptive schemas and also to the adoption of four key schema modes (and a deficit in the fifth, the healthy mode), which then lead to borderline symptomatology.

Judd (2005) developed a neuro-cognitive model to explain some of the cognitive aspects of the disorder. It is suggested that a child with inherited or acquired neuro-cognitive impairment, who then experiences poor parenting/maltreatment develops an insecure/disorganised attachment and dissociation, which then leads to impaired meta-cognition. In her view, this impaired meta-cognition then leads to a range of problems associated with BPD, including cognitive distortions and paranoia.

A cognitive analytic model of BPD has posited that in response to childhood abuse, individuals with BPD develop multiple, partly dissociated self-states, with the

alternating dominance of one state over the other being responsible for many of the features of the disorder (Ryle, 1997).

Evolutionary models of personality disorder have also been developed (e.g. Millon, 1990, 2011; Millon & Davis, 1996; Molina et al., 2009 etc.). From this perspective, it is argued that personality disorders, including BPD, represent adaptive evolutionary strategies, particularly in the context of a social environment that presents the individual with a unique set of challenges. From this perspective, BPD, whilst not allowing the individual to necessarily have a pleasant life, continues to exist because the strategies underpinning it allow for successful procreation under a specific set of environmental challenges.

Other theorists have emphasised the role of interpersonal problems in the genesis and maintenance of personality disorders (e.g. Benjamin, 1996; Hopwood, Wright, Ansell, & Pincus, 2013), seeing such disorders as being primarily an issue with how one relates to others (including the mental representations of self and others).

A neurodevelopmental model developed by Putnam and Silk (2005) focused upon emotion dysregulation as being key to the disorder. In their view, in addition to genetic factors, the key disturbance in childhood is that the childhood social environment fails to assist a child to learn how to manage emotionality, leading to emotion regulation difficulties in adulthood.

2.3 Biosocial Theory and Dialectical Behaviour Therapy

This neurodevelopmental model is similar to perhaps the most influential model of BPD, the biosocial model, the theory that underpins dialectical behaviour therapy (DBT). As outlined in Linehan's seminal text (Linehan, 1993), it is asserted that

borderline traits are, in essence, due to a disorder of the emotion regulation system. In her view this dysregulation is caused by two key components (see Figure 1.1).

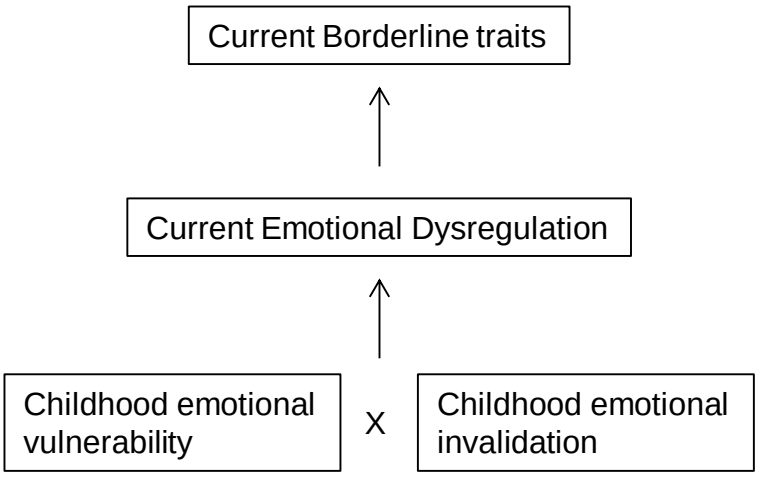

Figure 1.1 Simplified representation of the biosocial model. Interested readers are encouraged to refer to the significantly more detailed representation located at: Crowell, S. E., Beauchaine, T. P., & Linehan, M. M. (2009). A biosocial developmental model of borderline personality: Elaborating and extending Linehan's theory. *Psychological Bulletin, 135*(3), 495-510. doi: 10.1037/a001561

The first of these is emotional vulnerability. According to Linehan this constitutes abnormally high levels of emotion sensitivity and intensity, in addition to a protracted return to emotional equilibrium after experiencing an emotional event (Linehan, 1993). This emotional vulnerability is based upon biological predispositions which are subsequently influenced by life experiences, with these experiences determining whether borderline traits develop.

The other key component is an emotionally invalidating environment. In her view, an invalidating environment is one where the experiences of emotions are met with inappropriate, chaotic and excessive responses. This individual's experience of the emotion is disregarded, as well as the individual's views regarding the antecedents and consequences of the emotion.

In Linehan's view this experience firstly negates the invalidated individual's description and analysis of their lived reality. Second, it misattributes their lived

experiences as being due to socially undesirable attributes, such as being manipulative or overly sensitive (Linehan, 1993).

When this invalidation is chronic it leads to a number of undesirable outcomes (Linehan, 1993). First, the child does not learn to label and control emotional experiences. Second, the dismissal of emotional experience does not teach the child to learn to tolerate negative affective states. Third, the child learns that the exhibition of extreme emotional reactions is necessary to evoke recognition of emotional distress. Fourth, the child learns not to trust their own emotional experiences, but rather to invalidate their emotional states and look to the external environment for guidance regarding how to feel, think and behave.

Linehan asserts that when an emotionally vulnerable child is raised in an emotionally invalidating environment they will develop difficulties in managing emotions (emotional dysregulation) and therefore start to exhibit borderline traits. Whilst more recent incarnations of the biosocial model (e.g. Crowell, Beauchaine, & Linehan, 2009) have highlighted the role of other factors (e.g. childhood impulsivity), the focus of the model remains the interaction between emotional vulnerability and invalidation. Indeed Linehan even goes so far as to suggest that in the absence of this childhood emotional vulnerability, the outcome of an invalidating environment would result in a different personality disorder, that of a dependent personality (Linehan, 1993).

Other researchers (e.g. Fruzzetti, Shenk, & Hoffman, 2005) have highlighted the transactional nature of the model, arguing that dysregulated emotions invite invalidating responses from caregivers, in turn leading to increased emotional vulnerability (which in turn increases the likelihood of further dysregulation). Researchers have also explored the way in which gender may play a critical role in

the higher rates of BPD diagnosed amongst females (and the higher rates of antisocial personality disorder amongst males), positing a similar set of vulnerabilities by an altered developmental pathway leading to a differing diagnosis (e.g. Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009).

There are several potential problems associated with the biosocial model. First, as will be detailed later, “emotional dysregulation” is not a clearly defined term, with its meaning varying across researchers and with it being present in a range of disorders (thus raising questions regarding the specificity of the construct to BPD). Further, most models of emotional dysregulation argue that it is a multifaceted process, raising the question as to whether all (or specific) aspects of emotion regulation are impaired in BPD, and if so, whether this impairment is due to the model described.

Second, given the heterogeneity of the presentation of BPD, it is difficult for the model to account for the presence or absence of certain features in an individual, if all the features are due to the one dysfunction, namely a deficit in the capacity to regulate emotions.

Third, such a model has some difficulty in accounting for the differences in the remission rates of features of BPD throughout the lifespan. Some features of BPD, such as suicidal and parasuicidal behaviour, tend to remit more rapidly than others (Zanarini et al., 2007) – if all the features were due to the same underlying deficit (emotional dysregulation) then it would be anticipated that as emotional dysregulation improves so should each of the traits.

Finally, despite Linehan’s model being presented in her 1993 text, as will be detailed later, there has been very limited empirical investigation of its central tenet, namely that borderline traits are due to emotional dysregulation, caused in turn by an

emotionally vulnerable child having been raised in an emotionally invalidating environment.

Despite these failings, the model is scientific in the sense that it makes predictions that can be empirically tested and falsified (Popper, 1963). Further, the model has the benefit of being able to generate risky predictions (i.e. predications that, but for the theory, would be anticipated to fail) – particularly in relation: (a) to its identification of two key factors (emotional vulnerability and invalidation) and (b) its suggestion that that the interaction between these factors is of critical importance. Thus, if empirical observations were to confirm these predictions, it would provide strong support for the theory.

2.4 Genetics and the Biosocial Model

As previously mentioned, the heritability of BPD has been estimated at approximately 40% (Amad et al., 2014), with evidence supporting this rate being relatively consistent across countries (e.g. Distel et al., 2008) and with the heritability of the disorder tending to increase from adolescence to early adulthood (Bornovalova, Hicks, Iacono, & McGue, 2009). Even if the biosocial model is an accurate account of the disorder, it is unclear which components are genetically influenced, and to what extent. It is tempting to suggest that the innate emotional vulnerability posited by the model represents the majority of the genetic component. Indeed one study comparing individuals with and without BPD in terms of the role of trauma in childhood, found that the largest risk factor identified in the subsequent logistic regression was having a first degree relative with a neurotic spectrum disorder (Bandelow et al., 2005).

However there is also evidence suggesting that emotion dysregulation itself may at least be partly heritable (e.g. Jang, Dick, Wolf, Livesley, & Paris, 2005; Livesley, Jang, & Vernon, 1998), with a study focusing upon affective intensity (a construct similar to emotional vulnerability) and affective lability finding genetic factors accounted for approximately 40% and 25% of the variance respectively (Coccaro, Ong, Seroczynski, & Bergeman, 2012). Finally, it is unclear whether or not borderline traits may be directly heritable, or even that responding poorly to emotional invalidation may have a genetic component, with there being some evidence that maladaptive parenting is particularly pernicious when it is experienced by the offspring of individuals with personality disorders (Cheng, Huang, Liu, & Liu, 2011).

Indeed a range of polymorphisms have been implicated in the disorder, particularly with regard to the serotonergic system, including those related to serotonin synthesis and serotonin receptors and transporters (see Amad et al., 2014; Calati, Gressier, Balestri, & Serretti, 2013). Efforts in this area however have been hampered by the large number of polymorphisms, the limited number of studies completed and concerns regarding the quality of the extant studies (Carpenter, Tomko, Trull, & Boomsma, 2012). Further, the multifactorial nature of BPD (and the multifactorial nature of its aetiological pathway) may also hamper efforts to locate genes associated with the disorder (or with key constructs), as different aspects of the disorder may be due to differing neurobiological systems.

Determining the effects of environment and genes is complex and several models can be proposed: (a) an additive model, (b) a model that posits an interaction between environment and genes and (c) a model where the genetic contribution influences the individual's selection of, or exposure to, particular experiences and

environments (Kendler & Eaves, 1986). Indeed there is some evidence to suggest that the genes that lead to BPD also increase the likelihood of being exposed to adverse and traumatic life events including divorce, sexual and violent assault (Distel et al., 2011).

An extensive twin study explored the issue of familial aggregation and concluded that the resemblance amongst biological relatives could be entirely accounted for by genetic factors (Distel, Rebollo-Mesa, et al., 2009), perhaps suggesting that aspects of the home environment, including the propensity to experience parental invalidation, may be genetically based. Further, they found that the variation in borderline traits could be accounted for by a model that included both additive and dominant genetic factors. Only five gene-environment studies examining BPD were available for examination in a recent systematic review (Amad et al., 2014), with four these having the same lead author. All but one of these studies found a significant interaction between environmental and genetic factors.

Despite the aforementioned findings, care must be taken to not overemphasise the role of genetics in the development of the disorder. First, whilst a substantial proportion of the variation in the relevant constructs can be explained by genetic factors, a large proportion of the variance remains unexplained, suggesting that environmental factors play a significant role.

Second, care must be taken with conflating heritability with causality. Many genetic predispositions require an environmental trigger to make the gene active (see Caspi & Moffitt, 2006), and more importantly, many genetic influences on behavioural traits are indirect, for example the link between genes and suicidal behaviour may be due to endophenotypes such as trait aggression/impulsivity or neurocognitive function (see Mann et al., 2009). With this in mind, the proportion of the variation

explained by genetic factors must change depending upon the level of variation in environmental factors.

Third, the distinction between environment and genetic factors is, to a certain extent, a false dichotomy. Neither factor exists in isolation to the other, in that any adult human has, without exception, both a set of genes and also a set of life experiences. Further, the factors can interact in a variety of ways, not only are some genes likely to moderate the effects of exposure to certain environmental situations, but genetic components (e.g. being a child that is more difficult to soothe) may actually elicit particular environmental consequences (e.g. being subject to harsher parenting).

Fourth, given the strength of the association between some environmental risk factors (such as child abuse) and borderline pathology, it is challenging to assert that the disorder is primarily genetic in origin.

2.5 Conclusion

Despite the construct of BPD having a relatively long psychiatric history, there remain some issues with its current definition. Multiple theories have been formulated to account for the aetiology of this disorder, with most theories positing a developmental pathway which includes aspects of poor parenting and trauma. Several of the models are not mutually exclusive (e.g. the evolutionary model is compatible with most other models). The biosocial model is a prominent model which posits that BPD is predominately a disorder of emotion dysregulation, caused by an emotionally vulnerable child having been raised in an invalidating environment. Whilst genetics play a role in the development of the disorder, this does not ameliorate the importance of environmental factors in the disorder's development.

Section 3. Emotional Dysregulation

3.1 Construct

The centrality of emotion dysregulation to BPD is widely acknowledged by a range of researchers (e.g. Crowell et al., 2009; Glenn & Klonsky, 2009; Linehan, 1993; Putnam & Silk, 2005; Speranza, 2013 etc.). What remains unclear however, is: (a) how emotion dysregulation should be defined, with a range of views existing since at least the mid 1990's (e.g. Cole, Michel, & Teti, 1994; Thompson, 1994), and (b) whether emotion regulation difficulties are specific to BPD, given that they have also been linked to other disorders (e.g. generalised anxiety disorder; Mennin, Heimberg, Turk, & Fresco, 2005).

Gratz and Roemer define the construct as comprising of the following components:

1. Awareness and understanding of emotions.
2. Acceptance of emotions.
3. Ability to control impulsive behaviours and behave in accordance with desired goals when experiencing negative emotions.
4. Ability to use situationally appropriate emotion regulation strategies flexibly to modulate emotional responses as required in order to meet individual goals and situational demands. (Gratz & Roemer, 2004, p. 42)

This definition is not universally accepted, with a range of other perspectives existing. Some researchers, particularly those from an acceptance and commitment therapy (ACT) viewpoint, would de-emphasise the fourth component in Gratz and Roemer's definition. From the ACT viewpoint, the acknowledgement and acceptance of emotional experience, in conjunction with the capacity to pursue goals whilst

experiencing emotions, are considered the hallmarks of emotion regulation, conversely attempts to regulate emotions are considered to potentially contribute to psychopathology (Blackledge & Hayes, 2001). From this perspective it could be argued that the emotion dysregulation described by Gratz and Roemer actually conflates two (opposing) constructs. The first, comprising of emotional awareness, emotional acceptance and the capacity to pursue goal directed behaviour whilst experiencing emotions being associated with good mental health. The second, being the propensity to attempt to engage emotion regulation strategies being problematic and potentially contributing to psychopathology.

In contrast, other researchers emphasise this fourth aspect as being the critical component of emotion dysregulation, with a tendency to ignore the first three factors (e.g. Garner & Spears, 2000).

Other researchers focusing specifically upon BPD have developed the emotional cascade model, which highlights the continuity between affective distress, inadequate emotion regulation strategies and the use of disordered behaviour (Selby & Joiner, 2009). In their view, emotional (dys)regulation strategies such as ruminating upon negative affect lead to increases in the intensity of the negative emotion. This in turn leads to further rumination, with this process continuing in a downward spiral. Engagement of dysregulated behaviour disrupts the sequence, thereby being effective in one sense (in that negative affect does not worsen) but being evidently maladaptive in another (given the negative consequences associated with dysregulated behaviour).

Yet other researchers examining the role of the display of emotions, suggest that these are also aspects of emotion regulation (e.g. Zeman & Garber, 1996), whilst others have emphasised the role of context with regard to the deployment of emotion

(dys)regulation strategies (e.g. Aldao & Nolen-Hoeksema, 2012), thus suggesting that an individual's regulatory capacity is not innate and static, but may vary depending upon contextual factors. Indeed, it could be argued that in some contexts, the expenditure of cognitive resources to attend to and be aware of emotional content may be maladaptive, despite this being generally viewed as a component of adaptive emotional regulation.

Another perspective is that emotional dysregulation, particularly as it pertains to BPD, should also incorporate cognitive processes associated with emotions. There is a substantial body of literature indicating a significant level of disturbance in this area amongst those with BPD, including the propensity to attend to negative stimuli, a disproportionally strong ability to recall negative events and the propensity to make negative interpretations of neutral stimuli (Baer, Peters, Eisenlohr-Moul, Geiger, & Sauer, 2012). Such disturbances are likely to lead to negative affectivity; consequently it could be argued that as such they should be considered as part of the construct of emotion dysregulation.

Other researchers suggest that emotion regulation involves components of affective intensity and sensitivity (e.g. Newhill, Mulvey, & Pilkonis, 2004). Indeed some authors have posited that the dysregulation involved in BPD comprises of four main deficits – emotion sensitivity, heightened and labile negative affect, an excess of maladaptive regulation strategies and a deficit of appropriate regulation strategies (Carpenter & Trull, 2012).

Similarly, in the most recent iteration of the biosocial model (Crowell et al., 2009), emotional dysregulation is identified as consisting of emotional sensitivity, intense responding to emotional stimuli and a slow return to emotional baseline. This definition is describing a fundamentally different construct to that identified by Gratz

and Roemer and other researchers in the field. In the model, they do however include another construct, labeled “reactions to emotional situations (transient emotional states)” which mediates the relationship between the developmental risk factors and adult borderline psychopathology. The issues they identify under this label fit well with the Gratz and Roemer definition, suggesting that the difference between Gratz and Roemer’s definition and that in the biosocial model may be due to labelling (as opposed to a genuine disagreement regarding the relevance of the construct identified by Gratz and Roemer to BPD).

3.2 Development of Emotional Dysregulation

Given the confusion that still exists regarding the definition of the construct of emotional dysregulation, it is unsurprising that there is a lack of clarity in the literature regarding the developmental trajectory that leads to these difficulties. Some models have been proposed to account for the development of emotional (dys)regulation (e.g. Calkins, 1994; Cole et al., 1994; Kopp, 1989), including of course, the biosocial model.

A number of environmental stressors, in conjunction with an individual’s temperament, have been posited to pose risks for developing difficulties in managing emotions (Thompson & Calkins, 1996). There is some evidence that a history of child abuse is a risk factor for elevated levels of emotion dysregulation (Gratz, Tull, Baruch, Bornovalova, & Lejuez, 2008; Robinson et al., 2009; Shields & Cicchetti, 1998; Soenke, Hahn, Tull, & Gratz, 2010), including indirect forms of abuse such as exposure to domestic violence (Zarling et al., 2013). Exposure to stressful life events correlates to subsequent declines in emotional regulation (Herts, McLaughlin, &

Hatzenbuehler, 2012), as has adult exposure to traumatic events (Bardeen, Kumpula, & Orcutt, 2013).

Exposure to a range of poor parenting practises, including harsh, coercive, psychologically controlling or ineffectual parenting, have been found to correlate with emotion regulation difficulties (e.g. Coplan, Reichel, & Rowan, 2009; Cui, Morris, Criss, Houltberg, & Silk, 2014; Denham, Mitchell-Copeland, Strandberg, Auerbach, & Blair, 1997; Hoffman, Crnic, & Baker, 2006; Raval, Raval, & Deo, 2014; Sarıtaş, Grusec, & Gençöz, 2013; Zarling et al., 2013), including when the poor parenting takes the form of emotion invalidation (Buckholdt, Parra, & Jobe-Shields, 2014). Unsurprisingly, it has also been found that attachment difficulties in childhood and adulthood correlate with emotion regulation difficulties (Bender, Esbjørn, Sømhovd, & Pons, 2012; Goodall, Trejnowska, & Darling, 2012).

Affective intensity also appears to be a risk factor for emotional dysregulation (Gratz et al., 2008) as does being of female gender (Bender, Reinholdt-Dunne, Esbjørn, & Pons, 2012). Maternal psychopathology has also been found to relate to child emotional dysregulation (Hoffman et al., 2006; Zarling et al., 2013) as have some maternal personality factors, such as neuroticism and disagreeableness (Coplan et al., 2009). Finally, emotional dysregulation appears to be at least partly heritable (Jang et al., 2005; Livesley et al., 1998).

When attempting to evaluate the developmental models of emotional dysregulation, two issues become clear. First, a large number of the risk factors are also risk factors for BPD (as will become apparent when examining the psychosocial factors associated with the disorder). Second, attempting to formulate a comprehensive model based upon the empirical literature and establishing the types of relationships that exist between the pertinent constructs is extremely difficult;

similar to the difficulties with reconciling the various models of BPD with the empirical literature (as will be detailed later).

3.3 Emotion Dysregulation, BPD and the Biosocial Model

Despite the ongoing debate regarding how the construct of emotion dysregulation should be defined, there have been a number of studies that have supported the view that emotional dysregulation is central to BPD (e.g. Bornovalova et al., 2008; Cheavens, Strunk, & Chriki, 2012; Reeves, James, Pizzarello, & Taylor, 2010 etc.).

One study using college samples found that emotional dysregulation strongly correlated with borderline traits, even after controlling for a range of measures of negative emotionality such as anxiety and depression (Glenn & Klonsky, 2009). Another study comparing those with and without BPD found that those with BPD exhibited a range of emotionally dysregulated behaviour, including being less willing to approach potentially distressing situations or to experience distress to pursue goal directed behaviour, but did not differ from the control group with regard to their performance on a task when distressed (Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2009).

Relative to healthy controls, individuals with BPD are more likely to suppress both positive and negative emotions (Beblo et al., 2013), with other research finding that maladaptive meta-cognitions about emotions correlate with borderline symptomatology (Manser, Cooper, & Trefusis, 2012). Another study found that a measure of affective control remained a significant predictor of BPD, even after accounting for variation associated with heightened level of affective intensity (Yen, Zlotnick, & Costello, 2002), whilst another found emotional avoidance appears to

mediate the relationship between borderline traits and the experiencing of positive emotions (Jacob, Ower, & Buchholz, 2013).

Significant interest has been expressed regarding the mediating role emotional dysregulation plays between many risk factors in adult psychopathology. One study found that emotional dysregulation differentiated between women with and without a history of deliberate self-harm (DSH), a behaviour frequently associated with BPD (Gratz & Roemer, 2008). Further, the study found that emotional dysregulation accounted for variation in DSH even after other risk factors were taken into account, and that some aspects of emotional dysregulation appeared to either partially (or in the case of emotional inexpressivity) fully mediate the relationship between these risk factors and DSH. In addition it was found that different aspects of emotional dysregulation predicted DSH – an examination of the 6 factor Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) used in this study found that "emotional non-acceptance" and "impulse control difficulties" were not related to a history of DSH, whereas the other factors were significantly correlated to such a history.

Some of the literature however raises doubts regarding the specificity of emotion dysregulation to BPD. Indeed emotion dysregulation is linked to a variety of problems, including post-traumatic stress disorder (N. H. Weiss et al., 2012), generalised anxiety disorder (McLaughlin, Mennin, & Farach, 2007; Mennin et al., 2005; Soenke et al., 2010), depressed affect (Mennin et al., 2005) and elements of psychopathy (Donahue, McClure, & Moon, 2014). Furthermore a recent meta-analysis found that emotion regulation difficulties correlate with a range of pathologies, including anxiety, substance abuse, eating and depressive disorders (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Consequently it would appear that,

whilst emotion dysregulation is an important aspect of BPD, the two constructs are not synonymous.

Further, other studies have found emotional dysregulation mediates the relationship between childhood abuse and generalised anxiety disorder (Soenke et al., 2010). This provides something of a challenge to the specificity of the biosocial model to BPD, in that, presuming child abuse is an invalidating experience, the specificity of the biosocial model rests upon the construct of emotional vulnerability being specific to BPD (a proposition, that as will be discussed later, is not defensible).

3.4 Operationalisation of Emotion Dysregulation

A number of researchers have operationalised emotion dysregulation, leading to such measures as the General Emotional Dysregulation Measure (GEDM; Newhill, Mulvey, & Pilkonis, 2007) and the Leahy Emotional Schema Scale (LESS; Leahy, 2002).

One of the most widely used of such measures is the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). In Gratz and Roemer's view, emotional dysregulation consists of the capacity to be aware of and understand emotions, to accept emotional experience, to engage in adaptive and appropriate behaviour whilst experiencing negative affect, and the capacity to utilise effective strategies to manage emotions. Subsequent exploratory factor analysis of the items proposed for the measure revealed a 6 factor solution (Gratz & Roemer, 2004). The DERS has been found to correlate significantly with a range of dysfunction, including deliberate self-harm (Gratz & Chapman, 2007; Gratz & Roemer, 2008) and BPD (Bornovalova et al., 2008).

There is however some concern that such self-report questionnaires for emotional dysregulation, whilst being accurate measures (in terms of the construct, as defined by their makers), only partly assess this domain. Indeed one study found that the DERS combined with behavioural measures of distress tolerance was significantly superior in predicting BPD than the DERS alone (Bornovalova et al., 2008), accurately classifying 84% of participants as judged against the gold standard of a structured clinical interview.

3.5 Conclusion

Emotion dysregulation appears to be an important construct, with it being a feature of many disorders, in particular BPD. Despite this, a consensus in the literature does not exist regarding the exact features of the construct, in particular whether it includes aspects of emotional control, emotional intensity and emotional responding.

Section 4. Psychosocial Factors associated with Borderline Personality Disorder

Environmental factors are considered to be an important component of the aetiology of BPD, with a large twin study finding that environmental factors accounted for 54.9% of the variation in borderline traits (Distel, Rebollo-Mesa, et al., 2009).

A number of psychosocial factors have been identified as increasing the risk of developing BPD (Keinänen, Johnson, Richards, & Courtney, 2012). In this section several such risk factors will be explored.

4.1 Childhood Trauma and Abuse

In absolute terms, the reported rates of abuse and neglect amongst individuals with BPD are extremely high with some studies reporting rates higher than 90% (e.g. Zanarini et al., 1997). This association is apparent even from a young age (Guzder et al., 1999) and is not unique to Western populations (Igarashi et al., 2010; Zhang et al., 2012). Further, these rates appear to be even higher than those reported by individuals with other personality disorders (Zanarini et al., 1989; Zanarini et al., 1997), with other trait based research reporting findings consistent with this conclusion (e.g. Miller et al., 2010).

One study comparing those with BPD to healthy controls found only 6.1% of those with BPD did not report severe trauma in childhood, relative to 61.5% of those in the control group (Bandelow et al., 2005). Those with BPD reported higher levels of many types of traumatic childhood experiences, including overt child abuse, separation from parents, marital discord in the parenting relationship and major childhood illnesses. Another study examining 6 to 12 year old children, found those with abuse histories were substantially more likely to perform more poorly on measures assessing BPD precursors than non-abused peers (Rogosch & Cicchetti, 2005), suggesting that the impact of the abuse occurs prior to the full development of the disorder.

Traumatic childhood events have been found to increase the likelihood of subsequently being diagnosed with BPD (Liotti, Pasquini, & Cirrincione, 2000). Similarly, other research has identified that the traumatic separation from a parent during childhood is a risk factor for BPD (Malone, Westen, & Levendosky, 2011), whilst another study has highlighted that early maternal separation can be a risk factor for BPD (Crawford, Cohen, Chen, Anglin, & Ehrensaft, 2009).

It could be argued that the parental separation in the aforementioned studies may have correlated with abuse and neglect (thus prompting the child's removal); one study avoids this issue by examining the outcomes of children who had been evacuated unaccompanied by their parents during the turmoil preceding and during World War II (Lahti et al., 2012), with these children being drawn from a variety of socio-economic backgrounds. Compared to healthy controls, men but not women, who had been separated from their parents were at a significantly greater risk of developing a dramatic personality disorder (of which BPD is one), particularly if the separation had occurred when they were younger than 5.

Not all studies have been supportive of the direct role of child abuse on the subsequent development of BPD, including a recent twin study examining the role of internalising and externalising disorders, BPD and child abuse (Bornovalova et al., 2013). Whilst there were correlations between the constructs, the study concluded that a causal relationship between abuse and BPD was unlikely; with (in their view) a more probable scenario being that the association between abuse and BPD was due to common genetic factors. This study however could be criticised on the grounds that child abuse was considered to be a dichotomous construct (either being present or not), rather than an attempt being made to grade how pervasive, severe or chronic the abuse was, when the abuse occurred or whether other environmental factors not considered to be abuse per se, such as poor parenting, were present. Consequently, the results need to be interpreted as being rather limited in scope, indicating (solely) that the genetic factors accounted for an increased likelihood of at least one form of abuse having occurred, at some point in the individual's developmental history.

It is also crucial to note that attempting to disentangle genetic and environmental factors is problematic, given the high correlations between the various

constructs, so that it may be relatively uncommon that a child with a parent with a high genetic load for BPD does not also experience maltreatment. Indeed one study examining children who had been removed by protective services to those in a control group found that maternal borderline status was a predictor of a child being in the child protection group; with this being a more direct predictor of group than even the child's reported maltreatment history (Perepletchikova, Ansell, & Axelrod, 2012).

4.2 Poor Parenting in Childhood

It should be noted that the boundary between child abuse and poor parenting is blurred and somewhat arbitrary. Despite this it would appear that some parenting practices that are not extremely abusive (to the extent that child protective services would become involved) still impact upon the development of BPD.

A study comparing those with BPD to healthy controls found those in the BPD group reported a much higher frequency of poor parenting behaviours, across a range of facets and from both parents compared to those in the control group (Bandelow et al., 2005). Similarly research has also found that BPD traits are significantly negatively correlated to recalled parental warmth and monitoring, whilst being significantly correlated to parental psychological intrusiveness (Miller et al., 2010).

A very large study with children at age 11 found that being raised in a household where conflict existed in the parenting relationship increased the likelihood of the child exhibiting borderline traits, particularly where this conflict was to the level where it could be considered domestic violence (Winsper, Zanarini, & Wolke, 2012). Further, it found that suboptimal parenting also was a significant risk factor.

Poor parenting is a risk factor for developing personality disorders in general, with an increasing number of problematic behaviours resulting in increased risk (J. G. Johnson, Cohen, Chen, Kasen, & Brook, 2006). Other research has highlighted that not only poor parenting, but poor parenting which is conflicted (e.g. where the parents display poor parenting but of differing kinds) more than doubles the odds ratio of developing a personality disorder, relative to parents who engage in poor parenting (but of the same kind) (Cheng et al., 2011). It does appear that poor parenting may be particularly germane to BPD, with a study completed in China comparing patients with BPD, those with another personality disorder, and those with a non-personality-disorder mental health problem with regard to their reported exposure to poor parenting (Huang et al., 2014). Of the groups examined, those with BPD experienced the highest levels of poor parenting, particularly with regard to high levels of maternal and paternal punishment and rejection. Similarly, individuals with BPD report poorer parenting than those with a diagnosis of bipolar II (Fletcher, Parker, Bayes, Paterson, & McClure, 2014).

Another study compared the reported childhood experiences of female inpatients with BPD, those with a differing personality disorder and those with non-personality disordered psychiatric diagnoses (Hernandez, Arntz, Gaviria, Labad, & Gutiérrez-Zotes, 2012). Whilst the limited sample sizes involved made it somewhat difficult for establishing significant differences between the groups, there was a trend for those with BPD to have received more abusive/inappropriate parenting, and less appropriate parenting than those with other personality disorders, who in turn fared somewhat worse than those with non-personality disordered diagnoses. Interestingly, significant correlations existed between borderline traits and both maternal overprotection and also a lack of maternal care. This is of note, given that

these constructs appear to be the opposite of one another. It is however possible the parental failure occurred in differing situations, with overprotection in situations where the child should have been encouraged to develop independence and alternately a lack of care when the child should have been protected.

Researchers have also noted that parenting behaviours and borderline behaviours may be reciprocal in nature, with poor parenting eliciting borderline behaviours, which in turn provoke further poor parenting. Support for this proposition has come from a large longitudinal study, which found that the developmental trajectories for borderline traits and poor parenting were moderately associated, suggesting that such a relationship between the factors existed (Stepp et al., 2014).

4.3 Insecure Attachment Relationships

One study examining those with BPD and normal controls found that the predominant attachment style in the BPD group was a fearful/anxious style, relative to a secure attachment being the most predominant amongst normal controls (Minzenberg, Poole, & Vinogradov, 2006). Further, it was found that the associations between a history of sexual abuse and measures of current clinical symptoms diminished once attachment-anxiety and attachment-avoidance were controlled for, suggesting attachment partially mediated these relationships.

It should also be noted that the attachment difficulties may be considered both a risk for BPD, and also a consequence of the other risk factors (such as child abuse and poor parenting). Indeed, as noted previously, several theorists have argued that attachment difficulties are the construct central to the disorder (e.g. Fonagy & Bateman, 2007, 2008; Fonagy et al., 2011; Fonagy et al., 2000; Sarkar & Adshead, 2006).

4.4 Invalidating Environment

In the view of the biosocial model, childhood emotional invalidation is viewed as critical to the genesis of the disorder. Studies have found that retrospectively reported levels of parental invalidation correlate with borderline traits (Robertson, Kimbrel, & Nelson-Gray, 2013; Sturrock & Mellor, 2014), although the relationship is not always found to be critical when more sophisticated modelling is completed with multiple constructs (e.g. Reeves et al., 2010).

One interesting study examining child sexual abuse, measured the severity and frequency of abuse amongst three groups, the total sample of abuse victims, those who had disclosed the abuse and those who were yet to disclose (P. Y. Hong, Ilardi, & Lishner, 2011). Measures were also taken of BPD symptomatology, the level of general parental invalidation and the specific level of invalidation that the participant received when disclosing the child sexual abuse (or thought that they would have received in the cases of those who had not disclosed). When the various measures were regressed upon BPD symptomatology, it was found that the level of general invalidation and also invalidation specifically in relation to the sexual abuse were strongly predictive of adult BPD traits, even after the frequency and severity of the abuse were taken into account.

Experimental studies, examining whether individuals with BPD respond differently to emotional invalidation, potentially suggesting a sensitivity to invalidation stemming from early developmental experiences, have been limited and had mixed results (e.g. Woodberry, Gallo, & Nock, 2008)

4.5 Emotional Vulnerability

The biosocial model also argues that emotional vulnerability is a key risk factor for BPD. Whilst studies do suggest individuals with BPD suffer some disturbance in an area akin to Linehan's construct of "emotional vulnerability", unfortunately aside from a few studies (e.g. Sauer & Baer, 2010), most of the research to date has focused upon adult (rather than childhood) emotional vulnerability. Consequently such research can only provide a modest amount of support for the construct being a childhood risk factor, in that it depends upon the assumption that the construct of emotional vulnerability remains stable throughout the lifespan.

Several studies have found that self-reported affective intensity is elevated amongst those with BPD (Cheavens et al., 2005; Yen et al., 2002). There is also some support for affective intensity playing a role in BPD independently of life experiences, with affective intensity having been found to predict borderline traits above and beyond that which could be accounted for by a history of childhood sexual assault (Rosenthal, Cheavens, Lejuez, & Lynch, 2005).

Another study found that in response to a negative evaluation, individuals with BPD reported experiencing shame to a higher degree and a longer duration than healthy controls (Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2010). Interestingly however, such differences were not found in response to a more general, non-shame inducing stressor.

One innovative study provided individuals with BPD and a healthy control group with handheld computers, which prompted them on an hourly basis to report on their current state of aversive tension. Those with BPD were found to report significantly more episodes of tension, a higher average level of tension, more rapid increases in aversive tension and longer lasting aversive states (Stiglmayr et al., 2005).

Other studies have found that the heightened affective intensity amongst those with BPD is dependent upon the type of emotion being experienced, with studies finding that those with BPD reportedly experience negative, but not positive emotions more intensely compared to healthy controls (Ebner-Priemer et al., 2007; Levine, Marziali, & Hood, 1997). Research examining the stability of measures associated with BPD has also found that negative affectivity directly relates to levels of BPD, in that a change in the levels of BPD traits (on some such measures) related to changes in levels of negative affectivity (Trull et al., 1998).

It has also been noted that, whilst in general individuals with BPD report high levels of negative affectivity, this is a characteristic of many forms of psychopathology (Rosenthal et al., 2008). This makes it difficult to interpret whether the negative affectivity that has been associated with BPD is specific to BPD or is characteristic of a number of different forms of psychopathology (of which BPD is just one).

One study comparing individuals with BPD to normal controls and those with other psychiatric diagnoses, including major depressive disorder noted that individuals with BPD typically had more variability to their mood than other groups included in the study (Cowdry, Gardner, O'Leary, Leibenluft, & Rubinow, 1991). Similarly, another study comparing individuals with BPD to healthy controls and those with anorexia nervosa noted that the BPD group typically reported greater short-term fluctuations in unpleasant affective states than healthy controls (Stein, 1996).

One study comparing those with BPD to either sufferers of bipolar II or other personality disorders, noted heightened levels of affective intensity amongst those with BPD (Henry et al., 2001). Another study found that affective intensity was not more prominent in those with BPD, relative to other personality disorders, however it

was noted that such individuals did experience greater emotional lability with regard to anger and anxiety
(Koenigsberg et al., 2002).

A small study examining affective intensity comparing subjects with BPD to those with bipolar II or cyclothymia found that those with the affective disorders reported significantly stronger positive emotions than those with BPD, but the groups did not significantly differ with regard to negative affectivity (Reich, Zanarini, & Fitzmaurice, 2011), thus suggesting that the nature of the emotion may also affect its relative intensity amongst those with BPD.

Finally some researchers have explored whether individuals with BPD are particularly emotionally sensitive, by determining whether they are better able to indentify emotions in others, relative to controls (e.g. Jovev et al., 2011). Such attempts have shown either null results or deficits in those with BPD (e.g. Guitart-Masip et al., 2009; Jovev et al., 2011; Merkl et al., 2010; Unoka, Fogd, Füzy, & Csukly, 2011). It would appear that this fundamentally mistakes what the "emotion sensitivity" in BPD is – it is not so much an external process in being overly proficient at identifying emotions in others, but rather an internal process, where the individual is prone to reacting more strongly to emotional stimuli that others may ignore. Indeed other research has found that those with BPD are hypervigilant to emotional cues, relative to those with Cluster C personality disorders, those with an axis I disorders and healthy controls (Sieswerda, Arntz, Mertens, & Vertommen, 2007).

Overall, it appears that a construct akin to Linehan's emotional vulnerability is present in those with BPD. Despite this, several key issues remain unclear, such as whether the type of the emotion is of importance, whether emotional vulnerability is

specific to BPD and whether this vulnerability also plays a role in the childhood development of the disorder (in addition to being present in adults with BPD).

4.6 Miscellaneous Childhood Risk Factors

Interestingly, aside from the impacts of trauma, abuse, poor parenting and the development of poor attachment relationships, there are a disparate range of other factors that have also been associated with the development of the disorder, highlighting that in any one individual with the disorder the aetiological pathway may be both complex and unique.

There is even some evidence suggesting that the developmental trajectory for developing BPD may begin prior to birth. One study utilising individuals with BPD and matched healthy controls found that a range of prenatal stressors, including maternal smoking and maternal traumatic stress were significantly related to a subsequent diagnosis of BPD (Schwarze et al., 2013). Further, such prenatal factors could be used to account for 25.7% of the variation in BPD. Finally a large longitudinal study found that the presence of family adversity during pregnancy increased the likelihood of borderline traits being present at age 11 (Winsper et al., 2012), with other studies finding that relative to healthy controls, those with BPD have higher rates of having being born prematurely (Bandelow et al., 2005).

Another study utilising a sample identified to be at risk for having been subjected to poor parenting examined a large number of variables, including perinatal difficulties, abuse history and childhood temperament and the subsequent development of borderline personality traits (Carlson, Egeland, & Sroufe, 2009). Significant correlations with adult borderline traits were found across a wide array of measures. The presence in infancy of factors such as infant emotionality (at 30

months), infant muscle tone/tension and a maternal history of medical problems all increased the risk of subsequent borderline traits. Early childhood factors including maltreatment, attachment disorganisation, life stress and maternal hostility were also significantly correlated with adult borderline traits. Significant risk factors in early childhood/adolescence included attention problems, disturbed self-representation and emotional and behavioural instability.

Other research has found significantly elevated rates of maternal loss events (e.g. losing a child, death of a spouse etc) within the first two years of life amongst individuals who have later developed BPD (Liotti et al., 2000).

Whilst most of the research has focused on parental behaviour, there are some indications that other social interactions increase the likelihood of the emergence of BPD symptoms. Bullying, particularly if it is chronic in nature is associated with the subsequent development of borderline traits (Wolke, Schreier, Zanarini, & Winsper, 2012).

4.7 Adult Risk Factors

It is unwise to limit the developmental factors associated with BPD to those occurring in childhood, particularly given that the life experiences of individuals with the disorder are markedly different from those of the general population.

Relative even to those with other personality disorders, those with BPD are significantly more likely to report being the adult victims of verbal, emotional, physical, and sexual abuse (McGowan, King, Frankenburg, Fitzmaurice, & Zanarini, 2012; Zanarini, Frankenburg, Reich, Hennen, & Silk, 2005; Zanarini et al., 1999), with each form of abuse (barring sexual abuse) being inversely related to the time taken for the disorder to remit (Zanarini et al., 2005).

The levels of BPD amongst incarcerated populations have repeatedly been found to be much higher than those in the general population (Sansone & Sansone, 2009), suggesting that this population is overexposed to the adversity associated with the criminal justice system. Individuals with BPD have significantly greater problems in their intimate relationships, both with regard to locating a non-personality disordered individual with whom to partner and also with regard to various measures of marital functioning (Bouchard, Sabourin, Lussier, & Villeneuve, 2009), similarly there is a significant correlation between spouse's levels of borderline traits (Distel, Rebollo-Mesa, et al., 2009), indicating an increased risk of the individual with borderline traits being exposed to the challenges inherent in being in a relationship with someone with borderline traits. Carers of those with BPD report elevated levels burden, even relative to carers of those caring for inpatients with other mental health conditions (Bailey & Grenyer, 2013), indicating both the current life difficulties facing those with BPD, and also suggesting a concomitant reduction in the capacity of their support network to offer further assistance to buffer against future adversities.

With regard to psychosocial development, one study found that those with BPD are much more likely to suffer impairments relative to healthy controls across a range of areas, including employment, home duties and global measures of functioning (Ansell, Sanislow, McGlashan, & Grilo, 2007), with their impairments in global measures of functioning being significantly greater than those suffering from anxiety or mood disorders. Another study found that the deficits in global functioning associated with borderline traits persisted, even after accounting for the variation associated with a range of axis I diagnoses (J. P. Hong et al., 2005)

When attempting to access appropriate psychotherapeutic assistance, those with BPD encounter stigma, being perceived to be challenging to treat and manage

(Bourke & Grenyer, 2013; Cleary, Siegfried, & Walter, 2002; James & Cowman, 2007), potentially impacting upon their capacity to access appropriate help as adults. Further, even after entering treatment, BPD (being a personality disorder) means such clients are at elevated risk of prematurely discontinuing therapy (relative to most other mental health conditions) (Swift & Greenberg, 2012).

Individuals with BPD are at a much higher risk of a raft of medical conditions, even after controlling for anxiety, mood and substance abuse disorders, including arthritis, hepatic disease and gastrointestinal diseases (El-Gabalawy, Katz, & Sareen, 2010). Rates of the disorder are considerably elevated amongst those with pain syndromes (Sansone, Whitecar, Meier, & Murry, 2001), with significant correlations being found to exist between pain conditions and borderline traits (McWilliams & Higgins, 2013). Those with BPD suffer significantly higher rates of problems across a range of facets of their sleep, including perceived sleep quality, frequency of nightmares and daytime sleepiness (Philipsen et al., 2005; Semiz, Basoglu, Ebrinc, & Cetin, 2008; Simor & Horváth, 2013). The comorbidity rates of BPD with other psychiatric illnesses are, predictably, very high (Barrachina et al., 2011; Zanarini et al., 1998). Further, research following individuals with the disorder over a 6 year period found that those who subsequently ceased to meet diagnostic criteria for the disorder still had high levels of axis I disorders, whilst the rates were even higher amongst those who had never experienced a remission of BPD (Zanarini, Frankenburg, Hennen, Reich, & Silk, 2004), suggesting that even achieving remission (at least as adjudged by DSM criteria) does not end the difficulties facing this population.

In reviewing the adult risk factors, it is somewhat striking to consider the breadth of difficulties and dysfunction associated with BPD, in that it appears that there are

few areas of an individual's life where BPD is not associated with poorer functioning and increased challenges. It is tempting to assume that such difficulties are a consequence of the disorder. However, while borderline traits are likely to lead to the aforementioned difficulties, it is premature to assume that the causality is unidirectional. Indeed it could also be argued that a number of the aforementioned factors may well maintain or exacerbate borderline symptoms.

Supporting this interpretation one prospective longitudinal study compared those with BPD who had recovered from the disorder during the study relative to those who never recovered (Keuroghlian, Frankenburg, & Zanarini, 2013). It was found that the never recovered group was significantly more likely to suffer from chronic medical conditions (e.g. obesity, urinary incontinence etc.). They reported significantly higher levels of poor lifestyle choices, including physical inactivity and levels of tobacco and alcohol use. They showed increased levels of healthcare utilisation, including both on measures that may directly relate to the disorder (e.g. emergency room visits) as well as those that would not appear to be related (e.g. frequency of imaging procedures such MRIs and CT scans).

4.8 Clustering and Complexity of Risk

When examining the risk factors associated with BPD, it is important to note that risk factors tend to cluster. Part of this is due to how constructs are defined, with some risk factors inherently being associated with others (for example, most forms of abuse will also contain elements of emotional invalidation).

This clustering is also due to some risk factors increasing the likelihood of other factors being present. Indeed the family aggregation of personality disorders has long been noted (Cheng, Huang, Liu, & Liu, 2010; B. A. Johnson et al., 1995). This

familial aggregation holds true specifically in relation to BPD (Gunderson et al., 2011; B. A. Johnson et al., 1995; Riso, Klein, Anderson, & Ouimette, 2000; M. Weiss et al., 1996), as well as to key traits underpinning the disorder (Gunderson et al., 2011).

There are several possible explanations for this. Research has generally supported the diagnosis of a personality disorder being associated with worse parenting (Laulik, Chou, Browne, & Allam, 2013) with the presence of a parental personality disorder strengthening the association between poor parenting and the child developing a personality disorder (Cheng et al., 2011). Further, the severity of the parental borderline personality symptoms is associated with increased numbers of problematic child rearing behaviours (J. G. Johnson, Cohen, Kasen, Ehrensaft, & Crawford, 2006). Consequently it is likely that children raised by their biological parents, where the parents have borderline traits, experience both genetic and environmental risk. Further, some risk factors, such as poor parenting, are likely to increase the exposure to additional psychosocial risk factors, including the development of poor attachment and the child displaying behaviours that may invite emotional invalidation.

Not only do the risk factors tend to cluster, but ascertaining how various combinations of factors interact is complex (and at this stage not well understood). It appears probable that risk factors may well be additive (i.e. the more risk factors present, the greater the likelihood of BPD), if not interactive (i.e. that several risk factors lead to an exponentially greater level of risk). Further, it could be anticipated particularly with regard to genetic factors that some protective factors may exist, rather than these merely being the converse of a given risk factor (i.e. absence of abuse in childhood). Indeed one study with individuals with BPD examining the role of a key component of BPD, impulsivity, and serious life events (e.g. child

maltreatment etc.) found that whether or not these events predicted rates of impulsivity was modulated by a specific genetic polymorphism (Wagner, Baskaya, Lieb, Dahmen, & Tadić, 2009).

4.9 Conclusion

Borderline personality disorder is associated with a range of psychosocial risk factors, in particular difficulties experienced in childhood. Other factors, such as experiences in adulthood and the interaction between various risk factors are also likely to be important. There is presently some limited support for the role of childhood emotional vulnerability and invalidation. Often the pertinent risk factors aggregate and a comprehensive model of the psychosocial risk factors remains elusive.

Section 5. Psychophysiological Factors associated with Borderline Personality Disorder

A concern that has been raised with the use of self-report measures to examine constructs germane to BPD, is that the disorder is associated with difficulties in emotional awareness and clarity (Salsman & Linehan, 2012). This raises the prospect that the accuracy of self-report measures focusing on the reporting of emotional content may diminish amongst individuals with greater levels of borderline traits. Further it has been argued that due to the dramatic features of the disorder, individuals with borderline traits may be inclined to overstate their experiences when completing self-report measures (Rosenthal et al., 2008). Consequently, concerns

can be raised regarding the accuracy of the self-report data used in the study of the psychosocial factors relevant to BPD.

The use of psychophysiological instruments can avoid these issues, with such studies potentially being able to lend support to the biosocial theory. For example, if chronic emotional invalidation were to be found to correlate with specific neurological changes which are also found in those with BPD it would support the importance of this aspect of parenting to the aetiology of the disorder. Similarly, if a study were to assess physiological aspects of arousal when emotional amongst children, and then find that heightened emotional reactivity and a slow return to baseline as a child correlated with adult borderline traits, this would support the contention that childhood emotional vulnerability is an important developmental risk factor. Consequently, various psychophysiological studies will be examined in the following sections, exploring what support they are able to proffer for the biosocial model.

5.1 Physiological Studies

A number of physiological studies have been conducted, primarily focusing upon emotional responding amongst those with BPD.

One study found scripts dealing with issues germane to the BPD diagnosis (abandonment and rejection) led to a potentiation of the startle response and increased autonomic arousal in individuals with BPD which was not observed in healthy controls (Limberg, Barnow, Freyberger, & Hamm, 2011). Similarly, heightened emotional reactivity has been found to correlate with BPD pathology in another study, which also noted a significant interaction between BPD pathology and post-traumatic stress disorder (PTSD) (Dixon-Gordon, Gratz, & Tull, 2013). Conversely, two studies comparing sufferers of BPD to healthy controls argued that

the main difference between the groups involved heightened levels of negative emotional intensity, rather than reactivity (Elices et al., 2012; Feliu-Soler et al., 2013).

Another study compared individuals with BPD to those with social anxiety disorder and healthy controls (Kuo & Linehan, 2009). Participants' responses on a number of physiological and self-report measures were noted, as were their responses when viewing either films designed to elicit emotion or engaging in personally relevant scripts designed to activate emotions in the given participant. In general, individuals with BPD had poorer vagal tone and higher levels of negative emotional activation at baseline across the measures, relative to healthy controls. In contrast, their levels of reactivity to the emotion inductions were generally not greater than the healthy controls.

Indeed a range of studies have resulted in largely null findings. An early study examined a range of physiological parameters such as heart rate, skin conductance, and startle response while females with BPD and healthy female controls were exposed to a range of slides designed to engender positive, neutral or negative affective states. No differences were found between the groups on any of the physiological measures assessed, however a number of differences were found on self-report measures (Herpertz, Kunert, Schwenger, & Sass, 1999). A follow up study comparing females with BPD to those with avoidant personality disorder found that neither self-report nor physiological measures indicated that the affective response was stronger amongst those with BPD (Herpertz et al., 2000).

Another study, noting that psychopathy and BPD share some aspects (e.g. impulsivity) but differ in emotional responding, explored a number of physiological responses to viewing unpleasant and pleasant images, including skin conductance, modulation of the startle reflex and electromyographic activity of the a facial muscle

(to detect emotional expression) (Herpertz, Werth, et al., 2001). Compared with healthy, noncriminal controls, inmates with psychopathy showed a lack of responsiveness across all measures, whereas the results of inmates with BPD were consistent with the healthy controls, except with regard to limited facial modulation when viewing either pleasant or unpleasant slides.

Another study explored physiological measures such as heart rate, skin conductance responses, and systolic/diastolic blood pressure with participants who had a history of childhood sexual and/or physical abuse. Self-report measures were also included. The subjects were placed into BPD, post-traumatic stress disorder or abused control groups and were exposed to neutral scripts and personalised scripts depicting traumatic events or abandonment (Schmahl et al., 2004). No significant differences were found for either heart rate or diastolic blood pressure across the groups, nor were there significant differences in the self-report measures. Significantly higher levels of blood pressure in response to the trauma script were experienced by the abused control and PTSD groups, relative to the BPD group.

A study exploring the role of dissociative experiences upon the startle response examined this response in unmedicated females with BPD and healthy female controls (Ebner-Priemer et al., 2005). Heart rate, skin conductance and orbicularis oculi electromyogram responses were monitored whilst the subjects were exposed to 15 startling tones. Only on the electromyograph (EMG) did those with BPD demonstrate an elevated startle response, with higher levels of dissociation being associated with a blunted response on this measure.

Overall these findings offer inconsistent support for the contention that individuals with BPD are particularly “emotionally vulnerable” (as this construct is conceptualised by Linehan).

5.2 Brain Structure

A neuro-imaging approach has also been adopted to explore whether individuals with BPD are characterised by structural differences, particularly in the areas of the brain associated with emotional processing. Early CT scans found very limited differences in neurological structure between BPD and non-BPD participants (Lucas, Gardner, Cowdry, & Pickar, 1989; Schulz et al., 1983). A more recent study using MRIs have found that individuals with BPD, relative to healthy controls, have smaller frontal lobes (Lyoo, Han, & Cho, 1998). Another study has found that individuals with BPD, relative to healthy controls, have significantly smaller amygdalae and hippocampuses (Driessen et al., 2000). Similarly another study comparing those with BPD to healthy controls noted that individuals with BPD had significantly smaller hippocampal and amygdala volumes, with the left orbitofrontal and right anterior cingulate cortices also being smaller (Tebartz van Elst et al., 2003). A voxel-based morphometric (VBM) study in females with BPD found that, compared to healthy controls, those with BPD had less gray matter volume in the left amygdala, but there were no other differences in volumes or density of gray matter or white matter elsewhere in the brain (Rüsch et al., 2003). In contrast a study conducted with males with BPD found that relative to healthy controls, males with BPD had smaller grey matter volumes in frontal, temporal and parietal cortices but similar amounts of white matter (Völlm et al., 2009).

Another MRI study comparing those with BPD to those without the disorder, found that those with the disorder exhibited higher levels of grey matter concentration in the amygdala, but lower levels in the left anterior cingulate cortex, consistent with

animal models of stress and depression (Minzenberg, Fan, New, Tang, & Siever, 2008).

A further study comparing individuals with BPD to healthy controls found no differences in amygdala volume. Amongst those with BPD, comorbid major depressive disorder correlated with greater amygdala volumes across both hemispheres, with a larger left amygdala in this group correlating with more depressive symptomatology (Zetzsche et al., 2006).

Whilst there has not been an abundance of studies examining whether structural differences exist when comparing BPD to other disorders, the studies that have been conducted have generally suggested that such differences do exist (e.g. Rossi et al., 2013) however these findings are far from universal (e.g. Brunner et al., 2010). Further, interpreting the implications of these structural studies for the biosocial model is challenging, beyond drawing a general conclusion that there appear to be structural differences in the brains of those with BPD, particularly in the areas associated with emotion processing, and possibly areas related to behavioural control.

5.3 Brain Function

A further series of studies have explored whether there are functional differences in individuals with BPD, and if so, where in the brain these differences occur.

As assessed by functional MRI (fMRI) individuals with BPD experience significantly more activation in the left amygdala than healthy controls (Donegan et al., 2003). Another study using proton magnetic resonance spectroscopy (MRS) with BPD and healthy subjects examining the amygdala found decreased N-acetylaspartate (NAA) and total creatine (Cr) concentrations in the left amygdala of

patients with BPD, particularly with those with comorbid PTSD, with it being concluded that this may indicate disturbed affect regulation and emotional information processing in this region for these individuals (Hoerst et al., 2010). Interestingly, these decreased concentrations did not correlate with a range of self-report measures of anxiety, depression and other psychopathology.

Another study utilising single voxel spectroscopy found significant absolute N-acetylaspartate concentrations (a lower concentration potentially demonstrating impending cell death) in the dorsolateral prefrontal cortex (a part of the brain related to behavioural control) in those with BPD, relative to control subjects (Tebartz van Elst et al., 2001).

Using positron emission topography (PET), one poorly designed study compared individuals with and without BPD (unfortunately the groups were poorly matched with regard to gender and almost the entire BPD group were experiencing substance withdrawal) (de la Fuente et al., 1997). Notwithstanding these limitations, when PET results were compared, individuals with BPD demonstrated hypometabolism of glucose at the premotor and prefrontal cortical areas, the anterior cingulated cortex and the thalamic, caudate and lenticular nuclei, concluding that there were substantial disturbances in metabolism in those with BPD. A better designed study, utilising only female participants found hypermetabolism in frontal and prefrontal lobes, and significant hypometabolism in the hippocampus and cuneus amongst BPD sufferers relative to controls (Juengling et al., 2003).

There has also been interest in the hypothalamic-pituitary-adrenal (HPA) axis in those with BPD. One study comparing those with BPD, those with major depressive disorder (MDD) and healthy controls in response to a dexamethasone suppression test, found that both those with BPD and MDD had higher levels of cortisol before

and after the administration of the compound (Carvalho Fernando et al., 2012). Curiously, subsequent regressions did not find that depressive or borderline symptoms significantly predicted pre or post measures of cortisol, but found that a childhood history of emotional abuse predicted elevated baseline levels of cortisol, whilst a history of physical abuse predicted *lower* levels of baseline cortisol. Further, a history of childhood emotional neglect predicted lower post-levels.

A 2010 review noted that there was substantially less research on the HPA axis with regard to BPD, relative to other psychiatric disorders, concluding that although there were heterogeneous results, that it appeared that amongst individuals with BPD there was an enhanced basal and stimulated cortisol release, suggesting an over activity of the HPA (Wingenfeld, Spitzer, Rullkötter, & Löwe, 2010).

Other researchers have explored whether those with BPD may respond abnormally to the release of cortisol. One experiment compared those with BPD and healthy controls when cortisol levels were increased artificially (by the injection of hydrocortisone), with it being found that the groups experienced similar effects upon their performance on a task measuring response inhibition for emotional face stimuli (Carvalho Fernando et al., 2013). It was however noted that amongst those with BPD, comorbid PTSD led to a decrease in reaction times (relative to those without PTSD).

Other researchers, drawing on the limited research available, have also suggested a potential role for neuropeptide dysfunction in the disorder (Stanley & Siever, 2010). It is posited that opioids, oxytocin, vasopressin, neuropeptide Y and neurokinin 1 disturbances may potentially play roles in the disorder.

Overall, whilst these brain function studies provide some evidence of functional disturbance amongst those with BPD, it is difficult to argue that these findings strongly support or contradict the biosocial model.

5.4 Experimental Research

A burgeoning area of research has begun to not only look at the functional differences in the brains of individuals with BPD, but has also begun to introduce experimental conditions to determine whether further differences are apparent when such individuals are faced with a variety of psychological challenges. This research offers perhaps the best methodology with which predictions from the biosocial model could be tested.

There have been a number of studies that could be interpreted to support the biosocial model's proposition that BPD involves impairments in a construct akin to Linehan's emotional vulnerability (e.g. Hazlett et al., 2012; Herpertz, Dietrich, et al., 2001; Koenigsberg et al., 2014; Koenigsberg et al., 2009; Minzenberg, Fan, New, Tang, & Siever, 2007; Niedtfeld et al., 2010). Further, a large meta-analysis noted an abnormal pattern of brain activation is present when individuals suffering from BPD are faced with processing negative emotions (Ruocco, Amirthavasagam, Choi-Kain, & McMain, 2013), suggesting that emotion processing difficulties are central to the disorder.

Other studies have provided some evidence to support the biosocial model's contention that individuals with BPD also suffer from impairments when attempting to regulate emotions (e.g. Ruocco, Medaglia, Ayaz, & Chute, 2010; Schulze et al., 2011). Interestingly, some other studies have suggested that such difficulties may be due to a trauma history, rather than BPD per se (e.g. Lang et al., 2012). Finally,

there is also some evidence that after individuals with BPD complete therapy to assist with emotion dysregulation, that this leads to relative improvements in the abnormal pattern of neurological activity in the amygdala associated with the disorder (Goodman et al., In Press).

The preceding studies generally support the biosocial model's contention regarding the importance of a disturbance in emotion processing to the disorder, both with regard to emotional dysregulation and emotional vulnerability. Unfortunately few of these experiments have been designed with regard to the biosocial model, making it difficult to interpret the findings as supporting (or contradicting) other key (and more contentious) aspects of the biosocial model. Indeed, the psychophysiological literature can be rather extensively criticised in this regard, as will be detailed below.

5.5 Issues with the Psychophysiological Literature

In reviewing the psychophysiological studies it could be argued that they are broadly consistent with the biosocial model, in that they appear to demonstrate difficulties in the amygdala, hippocampal and prefrontal regions (O'Neill & Frodl, 2012), suggesting problems with emotion processing, memory and executive function. Further, some studies have been supportive of the view that individuals with BPD have difficulties in areas consistent with the construct of emotional vulnerability. Finally many of the studies manage to avoid the potential bias introduced by the self-report measures that characterise much of the literature examining the psychosocial factors. There are however, several key limitations to the psychophysiological studies, particularly with regard to the evaluation of the biosocial model.

First, the extant studies have small sample sizes, are heterogeneous in design and have often not been replicated by other researchers, making it somewhat difficult to confidently draw conclusions from the research.

Second, findings which suggest that those with BPD have difficulties with emotions and executive function (particularly when feeling emotional) are hardly paradigm shifting insights into such individuals, but merely a reflection of general clinical experience. This being the case, whilst the results are consistent with the biosocial model, they are also consistent with most mainstream models of BPD (which would anticipate similar disturbances).

Third, the studies have an issue with causality in that the findings that BPD correlates with abnormal structure and function in the areas of the brain associated with emotion processing, could be due to: (a) the structural and functional abnormalities lead to BPD behaviours, (b) BPD behaviours lead to structural and functional abnormalities, or (c) that both issues are caused by a third construct (e.g. poor attachment etc.). Although some studies have begun to generate findings that may allow some inferences with regard to causality (e.g. Goodman et al.), such research is sparse. Further, it is unclear whether differences to healthy controls are due to the presence of BPD, or alternatively due to conditions that are frequently co-morbid with this condition (e.g. depression, anxiety, trauma history etc.). This issue is further compounded by the lack of studies comparing those with BPD to individuals with other forms of psychopathology, making it difficult to ascertain that a given difference is specific to BPD.

Fourth, research such as the Lang et al. (2012) study raises concerns regarding whether “healthy” controls can be considered healthy – in that exposure to trauma appeared to differentiate the groups, rather than a BPD diagnosis. Conversely, other

studies have found that co-morbid PTSD was responsible for at least some of the differences associated with BPD (e.g. Dixon-Gordon et al., 2013; Limberg et al., 2011). Consequently, without the other studies having screened for trauma (or other, hereunto unidentified critical experiences), it is uncertain whether the "healthy" controls, are in fact "healthy" or alternatively whether the results attributed to BPD status should be attributed to another diagnosis or characteristic.

Fifth, the studies all focus upon adults who already have received the diagnosis of BPD. The biosocial theory posits a developmental trajectory involving an innately emotionally vulnerable child being raised in an invalidating environment – a theory that is difficult to demonstrate without the studies having been conducted in childhood and then establishing which participants as adults develop the disorder.

Sixth, it could be argued that the biosocial theory actually posits that four distinct groups should emerge; those without any of the risk factors, those who were emotionally vulnerable children (but raised in an validating environment), those who were not emotionally vulnerable (but who were raised in an invalidating environment) and those who were emotionally vulnerable but experienced invalidation, with only the fourth profile correlating to symptoms of BPD. There does not appear to have been an attempt in the extant empirical literature to apply psychophysiological techniques to test this hypothesis.

Seventh, whilst some studies have reported null findings, care needs to be taken as to whether this represents a lack of difference, or alternatively a lack of precision in the measures being used. As was the case with the early CT scans which had difficulties in locating differences in structure (despite clear differences in behaviour, affect and cognition) in those with BPD, this would appear to be reflective of the crudity of the measure being used (rather than the absence of any differences being

present). Consequently, as the psychophysiological research tools become increasingly sophisticated, it may be that many “null” findings will be revised.

Finally, it has been noted there is a general dearth of studies examining the brain structure of individuals exposed to positive parenting experiences, despite there being evidence that such experiences are associated with altered brain structure (Whittle et al., 2014). Consequently it would appear that the literature, both in general, and specifically in relation to BPD only examines one side of the coin – it cannot even be asserted with confidence that the reported effects associated with poor parenting and trauma are directly the result of these experiences (rather, they may reflect the lack of appropriate parenting experiences).

5.6 Conclusion

A number of psychophysiological studies have found a range of differences associated with those with BPD. They support the view that those with BPD experience disturbances in emotion processing, however interpreting the significance of these studies, particularly with regard to the development of the disorder and the biosocial model is problematic.

Section 6. Synthesis of Bio-Psycho-Social Factors

Drawing together the results from the various developmental and neurophysiological studies into a coherent understanding of the disorder is a daunting task. Interpreting the causal relationships between risk factors over such a long developmental timeframe is complicated. The relationship between two given factors may represent:

1. A direct causal relationship (in either direction or alternatively being transactional).
2. An indirect causal relationship mediated (or partly mediated) by another factor.
3. A correlational relationship.

Further complicating the situation, a number of dynamics may have implications for the nature of these relationships:

1. When the risk factor occurs (e.g. separation from one's parents at birth may well have a very different effect, compared with separation after attachment relationships have been established).
2. The presence of other risk factors.
3. The order in which the risk factors are introduced.
4. The presence of protective factors (e.g. certain genes may determine whether trauma significantly affects an individual).
5. The interaction of the particular combination of risk factors present.
6. The presence of factors that may moderate given outcomes (e.g. gender may moderate the response to poor parenting, leading to externalising behaviour in males but internalising behaviour in females).

Finally, when considering developmental risk factors associated with BPD, an argument can be raised with regard to how to define the developmental timeframe that a model must account for, with cogent arguments being able to be mounted that the timeframe:

1. Ends once sufficient aetiological risk factors are present.

2. Ends once a vicious cycle commences between the risk factors (which will eventually lead to BPD).

3. Ends once the individual begins to display borderline traits.

4. Ends once the individual begins to display borderline traits that are “clinically significant”.

5. Ends once the individual meets criteria for BPD.

6. Ends after the individual meets criteria for BPD, but only once the disorder stabilises.

7. Ends after the individual meets criteria for BPD, but only just prior to the disorder going into remission.

8. Never ends, with the individual’s condition constantly evolving throughout the lifespan.

An argument could be mounted for each of these “endpoints”, with the appropriateness of any developmental model being contingent on the endpoint’s (somewhat) arbitrary placement.

Consequently, attempting to determine whether the various bio-psycho-social studies are congruent with the biosocial model (or other models of BPD) is problematic, with the aforementioned issues creating something of a Gordian knot. Whilst there are some findings, such as the heightened emotional intensity in those with BPD that fit well with the model, other findings, such as having differing forms of poor parenting increases the likelihood of developing a personality disorder (e.g. Cheng et al., 2011) fit less well given the emphasis the biosocial model places upon one type of parenting (i.e. invalidating parenting).

A way to resolve this issue would be to directly test hypotheses derived from the biosocial model. Two main hypotheses can be advanced and therefore tested. If the biosocial model is the most accurate account of the development of BPD, then it would be anticipated that:

1. Therapy for BPD based upon this model (i.e. DBT) would be clearly more effective than therapy based upon inaccurate models.
2. The relationships between self-report measures of the relevant constructs would conform to the model. Namely, borderline traits would be associated with high levels of emotional dysregulation, which in turn would mediate a strong interaction effect between childhood emotional vulnerability and an invalidating environment.

Unfortunately, as will be noted, currently there is not sufficient evidence to support either of these two hypotheses.

Section 7. Test of Hypothesis 1 - Treatment Studies

A measure against which the biosocial theory could be tested is the effectiveness of DBT, the therapy based upon the biosocial model. All things being equal, it could be hypothesised that should DBT prove superior to other therapeutic modalities in the treatment of BPD, then this would support the biosocial model (relative to the other therapeutic models).

A number of randomised controlled trials (RCT) comparing DBT to treatment as usual (TAU) have found that DBT is generally a more effective treatment (e.g. Koons et al., 2001; Pasieczny & Connor, 2011; Verheul et al., 2003), including when other comorbidities are present (e.g. Linehan et al., 1999). RCT's comparing DBT to

treatment by experts across a range of measures have found that DBT is either equivalent to, or superior to, treatment by experts (Linehan et al., 2006; Neacsiu, Lungu, Harned, Rizvi, & Linehan, 2014).

Findings when DBT is compared to other comprehensive modalities which would be anticipated to be effective in the treatment of BPD have been mixed. One well designed RCT compared DBT to supportive therapy (ST) and transference focused psychotherapy (TFP) (Clarkin, Levy, Lenzenweger, & Kernberg, 2007). All three therapies led to improvements; however there were some differences across the therapies, with TFP and ST significantly improving aggression, whilst DBT and TFP improved suicidality. When examining 12 key outcome variables, TFP improved 10, ST improved 6 and DBT improved 5.

A RCT compared DBT to general psychiatric management (McMain et al., 2009), finding that after 1 year of treatment both groups had improved across a range of measures including suicidal and para-suicidal behaviours, however there were no significant differences between the groups. Differences between the groups also did not emerge at 2 year follow up (McMain, Guimond, Streiner, Cardish, & Links, 2012).

Similarly, a study with adolescents with borderline traits found that the addition of emotion regulation training (an aspect of DBT) to TAU failed to lead to significant improvements in outcomes (Schuppert et al., 2012).

A small RCT was completed with individuals with BPD and comorbid opiate dependence who received either DBT or comprehensive validation and a 12 step program for 12 months (Linehan et al., 2002). Both treatments were effective at reducing opiate use (although there were some differences in the patterns of reduction between groups) and led to improvements in levels of psychopathology,

with it being noted that the DBT group suffered significantly more participants exiting treatment.

Another small RCT comparing DBT and TAU + waitlist (WL) with women with BPD found that both groups improved with regard to rates of hospitalisation and engaging in DSH (Carter, Willcox, Lewin, Conrad, & Bendit, 2010), with limited differences between the interventions. The DBT intervention did however appear to be superior with regard to improving indices of quality of life and level of disability (as measured by bedridden days in the previous month).

A recent review of the evidence for DBT, noted that whilst there was evidence supporting its effectiveness with BPD, there was insufficient evidence to conclude that it was especially effective relative to other psychotherapies (Bendit, 2014). Finally, a recent Cochrane review found that when the results from the available studies were pooled that DBT demonstrated a moderate to large effect size relative to TAU interventions for improving anger, parasuicidality and mental health, but no advantage with regard to patient retention (Stoffers et al., 2012). Further, the review highlighted the very sparse nature of the investigations of other comprehensive therapies, either comparing such therapies against each other or TAU.

7.1 Issues with the Treatment Studies

There are several issues with the extant outcome research with regard to BPD treatment. First, given at least some aspects of BPD tend to improve over time (Cohen et al., 2008; Paris & Zweig-Frank, 2001; Zanarini et al., 2006; Zanarini et al., 2010) any research without the use of a control group runs the risk of misattributing gains that are made throughout the treatment period to the effectiveness of the treatment, rather than this being due to the natural progression of the disorder.

Equally, depending upon the criteria used, some studies may underestimate the treatment's relative effectiveness if the primary measures assess the aspects of BPD that are slow to remit.

Second, as found in the Cochrane review (2012), there are a rather limited number of studies comparing DBT to another specified therapy, waitlist or treatment as usual. Consequently there is not an extensive body of empirical literature from which to draw conclusions.

Third, completing long term therapy is, by its nature, a time demanding and financially expensive exercise. As a consequence the studies that do exist often do not have overly large sample sizes, reducing their power.

Fourth, although all things being equal, a RCT comparing therapies should be able to determine whether one therapy is superior to the other, all things are rarely equal. There are a number of factors such as therapist experience, therapist skill and modality adherence (both ensuring that a therapist assigned to a modality does not use any techniques beyond those proscribed by the therapy, in addition to ensuring a comprehensive use of the techniques specific to the given therapy) that may confound the results. Further, given the inherently risky nature of BPD, it is questionable as to whether a therapist or health service is able to maintain therapeutic purity, in that should a therapist determine that a technique or intervention is likely to benefit a client, withholding the intervention to preserve therapeutic purity is unlikely to (or should not) occur. Consequently it is difficult to ensure that the differences (or lack of differences) found in the extant studies are not due to methodological issues.

Fifth, the studies that are available do not clearly demonstrate the superiority of DBT to other therapeutic modalities. Whilst there is some evidence of superiority

relative to TAU, the results are inconsistent. Further, when examining the limited studies directly comparing DBT to other forms of therapy tailored to the treatment of BPD, the results are confused, with a mix of findings suggesting variously that limited differences exist, that DBT may be superior in some areas and inferior in others.

Sixth, much of the research that supports a given treatment, including many of the RCT's supporting DBT, have been performed by clinicians with a strong therapeutic allegiance to the given therapeutic orientation. This is problematic as other research has concluded that such allegiances have the possibility of distorting the outcomes of such comparative studies (Luborsky et al., 1999), thus raising questions as to the reliability of the current studies.

Seventh, even if DBT were to be found to be consistently superior to other forms of therapy, this would only provide indirect support for the biosocial theory. Determining why a therapy is effective is difficult – whilst the biosocial model suggests that the improvements are likely due to improved emotion dysregulation, it could also be possible that the validation emphasised heavily in DBT leads to a superior therapeutic relationship, with this relationship helping to resolve attachment issues (considered critical to the attachment models of BPD), and therefore leading to remission. Indeed, it is interesting to note that the phone coaching component of DBT (where the therapist is available to the client when they are in a distressed state) could be highly compatible with an attachment focused therapy, with regard to the client being provided the readily available secure base that was denied to them in childhood.

Equally, it is possible that the mixed results may not be due to a failing on the behalf of the biosocial model, but rather it demonstrating a failing of DBT. It may be that DBT is not reliably superior at addressing the issues of emotional dysregulation

(that the biosocial model posits are central to BPD), and so the results are a comment on the efficacy of DBT, rather than the model. There is however some evidence that at least partially contradicts this hypothesis, suggesting that DBT skill use mediates improvement in some aspects of the disorder (e.g. Neacsiu, Rizvi, & Linehan, 2010).

7.2 Conclusion

Whilst it would appear that there is some evidence that DBT is an effective therapy for BPD, there is insufficient evidence to conclude that it is exceptionally so. Consequently the existing treatment studies cannot provide strong support either for or against the biosocial model of BPD.

Section 8. Test of Hypothesis 2 - Direct Tests of the Biosocial Model

Given the length of time since the model's presentation in Linehan's text (Linehan, 1993), it is surprising that the central tenets of the model have not been extensively tested. This is despite the explosion in the use of DBT (Swenson, 2000), the therapy derived from the model. Indeed given the aforementioned difficulties in inferring support or otherwise for the model from the general literature or outcome studies, it would appear that a direct test of the model's predictions with regard to the interactions of the relevant constructs would be the most appropriate means of testing the model. It can be speculated as to why this line of inquiry has not been more extensively examined. One reason may be that the insight that is most

clinically useful (that BPD is caused by extensive emotional dysregulation), remains valid even if the underlying assumptions about what caused the dysregulation remain untested or unsupported (or even entirely inaccurate).

There are some studies that have attempted to test the model to varying degrees, with perhaps two studies (Reeves, 2007; Sauer & Baer, 2010), having come closest to testing the central assumptions of the biosocial model, namely that an emotionally vulnerable child raised in an invalidating environment will develop emotion regulation difficulties that will persist into adulthood manifesting themselves as borderline traits/BPD.

Neither of these studies supported the posited interaction between childhood emotional vulnerability and invaliding parenting, with the interaction failing to predict emotion dysregulation in the first study (Reeves, 2007) and borderline traits in the second (Sauer & Baer, 2010). There are, however, plausible explanations to account for these empirical studies not demonstrating this interaction effect.

The Reeves (2007) study used the General Emotional Dysregulation Measure (GEDM; Newhill et al., 2007) as the measure of emotional vulnerability. Whilst this may appear to be appropriate given that the measure was designed to assess emotional vulnerability as conceptualised by Linehan, there are two issues associated with the study's use of this measure. First, the biosocial theory posits that the emotional vulnerability occurs at an early age however the GEDM requests respondents to indicate how they view themselves (without specifying a timeframe) and so is likely to be measuring current vulnerability rather than childhood vulnerability. Second, this study sought to assess emotional dysregulation using the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). As the GEDM and the DERS contain several items that are almost identical it could be

argued that they are measuring very similar constructs. Consequently the research may have failed to appropriately delineate between the constructs of emotional vulnerability and emotional dysregulation, making the results of subsequent statistical analyses difficult to interpret.

The Sauer and Baer (2010) study was designed to validate recently developed self-report measures of constructs relevant to the biosocial model, including the Emotional Vulnerability–Child Scale (EV-Child; Sauer & Baer, 2010). This measure is based upon the Affect Intensity Measure (AIM; Larsen, Diener, & Emmons, 1986) but rewritten for greater applicability to the retrospective examination of emotional vulnerability as conceptualised by Linehan (making it an excellent tool for the measurement of the construct, and avoiding issues found with the use of the GEDM). Whilst this study was not primarily designed as a direct test of the biosocial model, the authors did complete some analysis of the relevant constructs, finding that an interaction effect between emotional vulnerability and invalidating parenting was not a significant predictor of borderline traits. It should be noted that the study did not involve the validation of an emotional dysregulation measure. For this reason, key parts of the biosocial model could not be tested, most notably whether emotional dysregulation mediates the relationship between the childhood antecedents and borderline traits. Further, proponents of the biosocial model could argue that the interaction effect in the biosocial model is supposed to be an important predictor of emotional dysregulation (rather than borderline traits), and thus the study was not a fair test of the model (although it should be noted that such an argument nevertheless would require modification of the biosocial model, given that as the model stands the interaction effect is likely to be important to both constructs).

Beyond these two main studies, several others have been completed that are relevant, but which for a variety of reasons, should be treated with some caution.

Arens et al. (2011) completed a study with several methodological strengths, but also some methodological issues. It compared clinical (with BPD), clinical (with a depressive disorder) and nonclinical groups on a range of measures taken 5 years previously, and then used logistic regressions with these measures to attempt to predict into which group the participants would be allocated. Accurate predictions regarding the allocation to the BPD group could be made based upon historical antecedents, with a significant temperament by parenting interaction being noted in the logistic regression. The authors concluded that the results confirmed the biosocial model (Arens et al., 2011). However a number of factors undermine its support for the biosocial model. The aspect of parenting that they included in the interaction effect was “overprotective” parenting – a construct far removed from parental invalidation. Furthermore, the temperamental aspect that they included as the other part of the interaction effect was “harm avoidance” – another construct not particularly consistent with the biosocial model’s concept of emotional vulnerability. Further, in the logistic regression they only included the interaction effect, not testing for the main effects of either of these constructs, making it very difficult to interpret what the interaction effect actually represented. Consequently it is difficult to concur with the author’s conclusions that this study confirmed *the* biosocial model advocated in the DBT literature (e.g. Crowell et al., 2009; Linehan, 1993), despite it potentially supporting *a* biosocial model.

Another study utilising a sample of BPD sufferers explored the role of personality traits, childhood emotional abuse and also BPD symptomatology (Martín-Blanco et al., 2014). A significant interaction effect was found between a “neuroticism-anxiety”

personality trait and the experience of emotional abuse, when used to predict BPD severity. There were some flaws with the study, foremost that the measure used to establish the level of “neuroticism-anxiety” was based upon current functioning. Therefore in terms of using this measure to predict BPD severity one must assume that adult levels of “neuroticism-anxiety” are consistent with childhood levels of this trait. Further, with regard to the biosocial model, the constructs examined differed from the constructs in the model (i.e. invalidating environment and emotional vulnerability) and a measure of emotion dysregulation was not included, thus limiting the study’s capacity to examine other key predictions of the model.

Another study found thought suppression mediated the relationship between constructs consistent with BPD and affective intensity, and partially mediated the relationship between the BPD constructs and perceived parental criticism (Cheavens et al., 2005). This study is somewhat hard to reconcile with the biosocial model on a number of grounds. First, thought suppression plays a significant mediational role despite it not typically being considered to be a form of emotion dysregulation. Second, assuming some stability in the trait of affective intensity from childhood, it would appear that a component of emotional vulnerability’s effect upon BPD was being mediated by a construct other than emotional dysregulation. Third, parental criticism, whilst it may involve elements in common with invalidation is a different construct, and so its influence on BPD would not be anticipated by the biosocial model.

Yet another study has supported the role of emotional dysregulation in BPD, finding that distress tolerance (an aspect of emotional dysregulation) moderates the relationship between affective intensity and borderline traits (Bornovalova, Matusiewicz, & Rojas, 2011). It should however be noted that key features of the

model, including the affective intensity being present in childhood, the role of childhood invalidation and the interaction between these constructs was not examined.

A study that examines some issues germane to the biosocial model was completed with inner city substance users (Gratz et al., 2008). Participants completed self-report measures of negative affective intensity/reactivity, childhood trauma, emotional dysregulation and were assessed via clinical interview for BPD against the DSM-IV (American Psychiatric Association, 1994) criteria. Child maltreatment and negative affective intensity/reactivity significantly predicted the number of BPD criteria met, however the interaction effect between these two independent variables was not significant. These variables also significantly predicted levels of emotional dysregulation. However once the levels of emotion dysregulation were included in the regression both of these factors ceased to predict the number of BPD criteria met, suggesting that emotional dysregulation mediates the relationship. These findings only partly support the biosocial model, highlighting the central role of emotional dysregulation, but failing to find a strong interaction effect between the putative antecedents. It should however be noted that the antecedents varied somewhat from those proposed in the model – adult affective intensity/reactivity, whilst potentially being indicative of childhood emotional vulnerability, is not the same construct. Further, child maltreatment is not synonymous with an invalidating environment.

An additional study compared the responses of individuals with BPD, those without BPD (but similarly high levels of negative affect and impulsivity) and a third group without BPD (and not matched to the other two groups with regard to levels of negative affect and impulsivity) when exposed to a social stress task (Scott, Levy, &

Granger, 2013). On measures of subjective negative affect and levels of salivary cortisol and alpha-amylase there was little difference between the two non-BPD groups, whilst those with BPD displayed a distinct pattern of results consistent with higher baseline arousal and greater affective intensity in response to the stressor. It could be argued that these results partially support the biosocial model, particularly with regard to individuals with BPD being emotionally vulnerable. It should however be noted that there are some issues with applying this study to the biosocial model. First, the measures were all based upon current functioning – the biosocial model suggests that the emotional vulnerability exists from an early age (and so one would have to assume the stability of this construct over time when using this study's results to infer levels of childhood emotional vulnerability). Further, it is difficult to interpret the findings with regard to the group with high levels of negative affect but without BPD represented. They could be considered to be individuals who had significant levels of emotional vulnerability (but weren't invalidated, and therefore never developed BPD) – or alternatively individuals who developed some measure of psychopathology in adulthood (as evidenced by their negative affect) but through an aetiological pathway that did not include childhood emotional vulnerability. Thus, it makes it difficult to draw conclusions from this study, at least in regard to the validity of the biosocial model.

A further study that tested some features of the model attempted to explain current BPD traits, examining the roles of invalidation (in the forms of past parental invalidation, current parental invalidation and invalidation by a current partner), mediated via poor distress tolerance and emotion dysregulation (Sturrock & Mellor, 2014). Whilst this study broadly supported the importance of invalidation with regard to current presentation of borderline traits, in some respects this study only partly

supports the model. In particular, it did not include any measures of childhood emotional vulnerability in the model, and as a consequence could not test the critical interaction between invalidation and vulnerability proposed by the biosocial model. As a consequence only a small proportion of the variation in emotional dysregulation was able to be explained. It is therefore unknown whether this was due to the failure to include the putatively critical elements or whether these would not have made a substantial contribution.

A recent study tested the role of parental attachment to the use of positive and negative emotion regulation strategies in the context of BPD symptomatology in a clinical sample (who did not necessarily possess significant borderline traits) (Kim, Sharp, & Carbone, 2014). It found that attachment security's effect on borderline symptoms was mediated by the use of positive emotion regulation strategies, but not by the use of negative emotion regulation strategies. Further, the associations between the relevant constructs were generally stronger for paternal (rather than maternal attachment). These findings are somewhat inconsistent with the biosocial model, which highlights the importance of invalidation (rather than attachment) in emotion (dys)regulation and does not draw a distinction between the gender of the person providing the (in)validation.

Another study explored maternal and dyadic affective behaviours and their relationship to the severity of borderline traits over 3 consecutive years amongst female adolescents, with the sample being recruited to have an overrepresentation of adolescents with high affective instability (Whalen et al., 2014). Over time, displays of maternal positive (but not negative) affective behaviours were associated with a reduction in the severity of borderline traits. Similarly positive dyadic behaviours were associated with a remission of BPD symptoms. These results are somewhat

difficult to reconcile with the biosocial model, in particular that the enhancement of positive behaviours and interactions over time appeared to be more important than negative behaviours and interactions, being somewhat inconsistent with the view that invalidation (being a negative behaviour) is of critical importance. It should however, be noted that the construct of invalidation was not directly assessed, and that the study was conducted in late adolescence (whereas the biosocial model may be applicable to the processes occurring in childhood).

A large longitudinal study found that the relationship between suboptimal parenting and BPD status at age 11 was partially mediated by IQ and axis I DSM-IV diagnoses at ages 7-8 (Winsper et al., 2012). This finding is somewhat contrary to the biosocial model, in that given the primacy and specificity given to the role of emotional invalidation, if the suboptimal parenting is considered to include invalidating parenting then the model would not predict that either IQ/axis I diagnoses should play a mediating role – if suboptimal parenting does not include parental invalidation then it could be questioned as how the study could construct a model of BPD without accounting for this factor.

One study examined some constructs relevant to the biosocial model, comparing the aetiology of BPD to bipolar II (Fletcher et al., 2014). It found that having experienced poor parenting was significantly more common in the BPD group with the BPD group also generally demonstrating higher levels of emotion dysregulation (both in terms of the presence of unhelpful responses and the absence of helpful responses). This supports the biosocial model, in that it links poor parenting to emotion dysregulation, however it also provides some contradictory evidence given the forms of poor parenting in question did not directly involve emotional invalidation.

Yet another study explored the differences between models of BPD by examining which core constructs from the various models were the best predictors of BPD in a community sample (Cheavens et al., 2012). Measures included those assessing emotional dysregulation, interpersonal difficulties and a sense of self. Whilst all three areas were found to be predictive of borderline symptoms, only emotional dysregulation was uniquely predictive of borderline traits amongst individuals with higher levels of these traits, thus supporting the biosocial theory's contention regarding emotional dysregulation's central role in the pathology.

In contrast, there is other evidence suggesting that another set of constructs, those of maladaptive schemas, may be central to BPD pathology. Such schemas predict personality pathology, with the resolution of such schemas being strongly associated to symptomatic remission in pathology (Nordahl, Holthe, & Haugum, 2005). Further, schema based therapy, targeting maladaptive schemas (rather than focusing upon emotion dysregulation directly) has been associated with very large effect sizes in the treatment of BPD (Sempértegui et al., 2013). Consequently to account for such findings, further theorising may be necessary for the biosocial model to be supported (i.e. the construct of maladaptive schemas have to be placed somewhere within the model). It could, however, be suggested that such schemas are a consequence of emotion dysregulation, rather than directly causing BPD – without having established to what extend borderline traits are accounted for by the biosocial model, it is difficult to ascertain the role of constructs not considered crucial by the model.

8.1 Conclusion

Various studies have been conducted that relate to the biosocial model. None, however, fully or adequately test the biosocial model, primarily through not measuring all of the key constructs identified in the model. Two studies that most closely tested the model (Reeves, 2007; Sauer & Baer, 2010) failed to find that the purportedly critical interaction between emotional vulnerability and invalidation was important in accounting for symptoms of emotion dysregulation/BPD.

Section 9. Current Studies

Given the difficulties associated with drawing inferences from the general literature and the treatment outcome research, and that the two studies best positioned to directly test the biosocial model possess methodological limitations, a decision was made to complete two studies to test predictions derived from the model. Several specific a priori hypotheses were derived from the biosocial model with regard to the key constructs of borderline traits, emotional dysregulation, childhood emotional vulnerability and having experienced emotionally (in)validating parenting in childhood. If these hypotheses were supported by empirical findings it would provide strong support for the biosocial model of BPD. Conversely if these hypotheses were not supported, or an alternate model better fitted the data this would undermine the validity of the biosocial model.

9.1 Hypotheses

1. The interaction between childhood emotional vulnerability and having experienced emotionally invalidating parenting in childhood would strongly predict adult emotional dysregulation.
2. Adult emotional dysregulation would be a strong predictor of adult borderline traits.
3. The relationship between adult borderline traits, and childhood emotional (in)validation and emotional vulnerability would be fully mediated by adult emotional dysregulation.
4. Emotional dysregulation would be more strongly associated with borderline traits than a comparison psychopathology.
5. (In)validating parenting would feature more prominently and/or operate differently in the account of borderline traits than a comparison psychopathology.
6. Childhood emotional vulnerability would feature more prominently and/or operate differently in the account of borderline traits than a comparison psychopathology.
7. These findings would be supported with individuals drawn from a broad age range.

The first study sought to test the central tenet of the biosocial model, namely that the constructs of having been an emotionally vulnerable child and having experienced an invalidating environment, would lead to emotion dysregulation as an adult, with this emotional dysregulation mediating the relationship between the aforementioned constructs and levels of borderline traits.

Chapter II: Study 1 - An Investigation of the Biosocial Model of Borderline Personality Disorder [1]

[1] This study was published as Gill, D. J., & Warburton, W. A. (2014). An investigation of the biosocial model of borderline personality disorder. *Journal of Clinical Psychology, 70*(9), 866-873. doi: 10.1002/jclp.22074. Some changes in formatting have been applied.

Abstract

Objectives: We sought to test the biosocial theory of borderline personality disorder (BPD) which posits that borderline traits are due to emotional dysregulation, caused by a combination of an emotionally vulnerable child being raised in an emotionally invalidating environment.

Methods: 250 adults (76% female, median age = 32.06 years) from a non-clinical population completed self-report measures assessing current levels of borderline traits and emotional dysregulation. They also completed retrospective measures of childhood emotional vulnerability and parental invalidation.

Results: Invalidating parenting and emotional vulnerability independently predicted emotion dysregulation, but an interaction effect was not found. Having experienced validating parenting was found to be a protective factor for developing borderline traits but was not significantly related to emotional dysregulation.

Conclusions: Data in this sample did not support the underlying genesis of BPD proposed by the biosocial theory and a model that more parsimoniously explains the development of BPD is proposed.

Introduction

Dialectical behaviour therapy is a common treatment for borderline personality disorder (BPD). It is based upon the biosocial theory which posits that borderline traits are due to extensive emotion dysregulation, caused in turn by an interaction between an individual's innate emotional vulnerability and the experience of being raised in an emotionally invalidating environment (Linehan, 1993). Whilst recent revisions of the model have hypothesised that childhood impulsivity may also play a role in the development of borderline traits (Crowell et al., 2009), the main emphasis of the biosocial theory rests upon a strong interaction between childhood emotional vulnerability and invalidating parenting.

The biosocial model has received some empirical support (Arens et al., 2011), but the posited interaction between childhood emotional vulnerability and invaliding parenting has failed to predict emotion dysregulation in at least one study (Reeves, 2007) and borderline traits in another (Sauer & Baer, 2010). Unfortunately both of these studies had methodologies that make it hard to make firm conclusions about the impact of the proposed interaction. In the Reeves (2007) study the measure used for childhood emotional vulnerability (GEDM; Newhill et al., 2007) does not focus upon the respondent's childhood and contains some items that are almost identical to the those used in the measure of current emotional dysregulation (DERS; Gratz & Roemer, 2004), thus confusing construct validity. The Sauer and Baer (2010) study did not incorporate a measure of emotion dysreuglation and so it was not possible to determine whether this contruct mediated the effect of the childhood measures on current borderline traits.

The current study sought to test whether the best fitting model of precursors to borderline traits would include a significant interaction effect between childhood

emotional vulnerability and parental invalidation, leading to current emotional dysregulation, resulting in borderline traits.

Anecdotally, this prediction is contrary to the authors' clinical experience, where clients may possess borderline traits whilst only reporting either a history of emotional vulnerability or invalidating parenting. These anecdotal reports from clinicians, considered in conjunction with the findings of Reeves (2007) and Sauer and Baer (2010), lead to the hypothesis that an interaction effect would not play an important role in predicting either emotional dysregulation or borderline traits.

An argument could be mounted that the interaction that the biosocial theory posits is not a statistical interaction, but rather is a functional one, whereby an emotionally vulnerable child is likely to elicit invalidating responses from their environment, leading to further emotionality. The most recent incarnation of the biosocial theory suggests that this functional interaction occurs (Crowell et al., 2009). Consequently, if this interpretation of the biosocial theory is correct it would be expected that the measures of emotional vulnerability and parental invalidation would be highly correlated and share substantial co-variance (as a causal cycle is posited to exist between the two factors). Further, it is possible that both functional and statistical interactions may be present. The current study sought to test both forms of interaction, but based on emerging evidence, hypothesised that neither would be present.

Method

Participants

The study was conducted online, with 150 subjects recruited from the general population. The sample was further supplemented by 100 first year psychology students completing the study for course credit.

The final sample consisted of 250 participants (60 male, 190 female) with a mean age of 32.06 years (S.D. = 15.80) and a mean number of years of education of 16.14 (S.D. = 3.25). 60% of participants identified with an Australian background and the sample was predominately middle class (24% with a household income of $37,000 or less; 24% $37,001-80,000 and 52% over $80,000).

Measures

Emotion dysregulation

The issue of what constitutes emotional dysregulation was considered prior to the commencement of the study, given that the definition varies across theorists. For the purposes of this study, emotional dysregulation was conceptualised as encompassing a maladaptive reaction that an individual may make to an emotion rather than a high level of intensity for the emotion itself, a distinction made by previous researchers examining emotional dysregulation (e.g. Gratz & Roemer, 2008; Mennin et al., 2005). With regard to the biosocial model, whilst this distinction differs from the definition utilised in the most recent description of the model (Crowell et al., 2009), a similar construct is identified further down the model, prior to the emergence of borderline traits (albeit with the label of “Reactions to emotional situations”).

Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). was selected with these considerations in mind, with its items focusing upon the second order responses to emotions (rather than the intensity of emotions themselves). It was developed with consideration of Linehan's theory and comprises of 6 facets of emotion dysregulation. It has demonstrated good internal consistency (α = .93 - .94), test-retest reliability (ρ_I = .88, p < .01) and predictive power in relation to the anticipated behavioural outcomes of emotional dysregulation (Gratz & Roemer, 2004, 2008). Further, it is a significant predictor of borderline personality disorder (Gratz et al., 2008).

Borderline Traits

Borderline Personality Questionnaire (BPQ; Poreh et al., 2006). is a self-report measure that has been previously used with nonclinical populations as a dimensional measure of borderline traits (Fonseca-Pedrero et al., 2011). It contains 9 subscales, one for each of the facets of BPD as per the DSM-IV-TR (American Psychiatric Association, 2000). The subscales demonstrate adequate internal consistency (α = .78 - .93) (Fonseca-Pedrero et al., 2011), and the measure has performed favourably when compared to other measures of BPD in the screening of outpatient youth for the disorder (Chanen et al., 2008).

Childhood Invalidation

Recalled Childhood Socialization of Emotion Scale (RCSES; Krause, Mendelson, & Lynch, 2003). is a self-report measure retrospectively rating the levels of validation and invalidation experienced as a child, through assessing the

manner in which the respondent's parent would respond to a series of common scenarios experienced during childhood (e.g. a child becoming upset at losing a prized possession). It has previously been used in a number of studies to assess perceived levels of parental invalidation (e.g. Sauer & Baer, 2010; Thomas, DiLillo, Walsh, & Polusny, 2011), having significantly predicted borderline symptoms and shown a modest relationship to parent's self-report of their parenting (Sauer & Baer, 2010).

In this study the measure used did not include all the questions, but instead removed the scenarios identified as being redundant by Sauer & Baer (2010), with such refinements having previously resulted in highly internally consistent measures of parental validation/invalidation (α = .88 - .95). In this study the RCSES scales were reversed (cascading from left to right) in order to keep the meaning of a high score consistent with the other measures used. Both invalidation and validation scales were used in this study to determine (a) whether they differ as risk and protective factors and (b) whether one construct is merely the inverse of the other.

Childhood Emotional Vulnerability

Emotional Vulnerability–Child scale (EV-Child; Sauer & Baer, 2010). is a self-report measure retrospectively rating emotional vulnerability, based upon the Affective Intensity Measure (AIM; Bryant, Yarnold, & Grimm, 1996) but also incorporating some items relating to Linehan's conceptualisation of emotional vulnerability involving a slow return to baseline from emotional arousal. It has high levels of internal consistency (α = .91) and enjoys a modest convergence with retrospective parental ratings of childhood vulnerability (Sauer & Baer, 2010).

Results

Calculating Scores

The standard manner of establishing the total score on the BPQ was considered inappropriate for this study as it involves summing all the scores despite each subscale having a variable number of items that may be endorsed at differing rates. This creates the potential for some of the subscales to have a greater influence on the overall score, whereas the DSM-IV-TR does not give primacy to any of the traits (American Psychiatric Association, 2000). Consequently the scoring of the BPQ was completed by transforming the mean level of endorsement of each of the subscores into a z-score prior to the creation of an overall mean score, thus not favouring one facet of BPD over the others. A similar issue existed for the DERS and so the same solution was applied (i.e. the mean of each subscale was transformed into a z-score before an average score was determined).

RCSES-Invalidation and RCSES-Validation were calculated with regard to the refinements suggested in previous research (Sauer & Baer, 2010) in order to deliver a two factor solution.

Data Transformation and Internal Consistency

Data normality was found to be an issue for RCSES-Invalidation scores so a logarithmic transformation was used to resolve this issue (the transformed variable was used for all subsequent analysis).

Internal consistency for each of the measures was found to be adequate to excellent for all the scales, other than the quasi-psychotic states subscale of the BPQ which was marginal (see Table 2.1).

Table 2.1
Descriptive statistics and internal reliability

	Mean	S.D.	Crombach's alpha
EV-Child	3.11	0.83	.94
RCSES-Validation	3.99	1.39	.95
RCSES-Invalidation (prior to transformation)	2.79	1.31	.93
DERS (Mean of z-scores)	0	.71	.81
DERS_NA	2.28	0.96	.91
DERS_GDB	2.96	0.98	.88
DERS_ICD	1.95	0.82	.88
DERS_LEA	2.40	0.76	.81
DERS_LS	2.15	0.90	.92
DERS_LEC	2.18	0.74	.84
BPQ (Mean of z-scores)	0	.72	.89
BPQ_Impulsivity	.18	.20	.68
BPQ_Affective_Instability	.35	.31	.87
BPQ_Abandonment	.18	.20	.74
BPQ_Relationships	.32	.31	.82
BPQ_Self_Image	.30	.29	.82
BPQ_Suicide/Self-Mutilation	.16	.23	.78
BPQ_Emptiness	.26	.28	.84
BPQ_Intense_Anger	.26	.28	.86
BPQ_Quasi-Psychotic States	.19	.21	.62

Note: NA Nonacceptance of Emotional Responses, GDB Difficulties in Goal Directed Behaviour, ICD Impulse Control Difficulties, LEA Lack of Emotional of Emotional Awareness, LS Limited Access to Emotion Regulation Strategies, LEC Lack of Emotional Clarity.

Model Comparisons

The childhood emotional vulnerability (EV-Child), RCSES-Invalidation and RCSES-Validation scores were converted to z-scores to allow for the calculation of interaction effects (obtained by multiplying the standardised scores together and generating the variables EV*Invalid and EV*Valid). The demographics variables were recoded into dichotomous or ordinal variables for analysis. The model used composite rather than latent variables due to the large number of parameters.

A model was designed using AMOS 7.0, whereby all of the demographic variables, childhood emotional vulnerability (EV-Child), RCSES-Invalidation, RCSES-Validation, EV*Invalid and EV*Valid were entered as independent variables with direct effects upon emotional dysregulation (DERS; the mediating variable). All possible co-variances between the independent variables were estimated. borderline traits (BPQ total score) was then entered as the dependent variable, with all variables (including DERS) identified as having a direct effect upon this measure.

Because the model had many paths and co-variances, a large number of which were not significantly different from zero, the model was simplified by removing non-significant paths and co-variances. The initial approach was based on chi-squared difference tests. However, because of the large sample size, the removal of non-significant paths and co-variances often led to significant changes in the value of chi-squared, even though, by other fit indices less affected by sample size, the goodness of fit of the model had not been reduced (and in some cases even improved). Consequently it was determined to prune the model on the basis of the significance of the co-variances and direct effects. The model was pruned of all weak co-variances, rerun and further co-variances were pruned. This process was repeated until only significant ($p < .05$) co-variances remained. The same pruning technique was then utilised for the direct effects.

The resulting model was a very good fit to the data (GFI = .960, CFI = .982, RMR = .044, SRMR = .0663, NFI = .930). The model was also parsimonious (RMSEA = .035, PCLOSE = .873) and had an acceptable chi-squared statistic $\chi^2(58, N = 250) = 76$, $p = .056$ ($\chi^2/df = 1.31$).

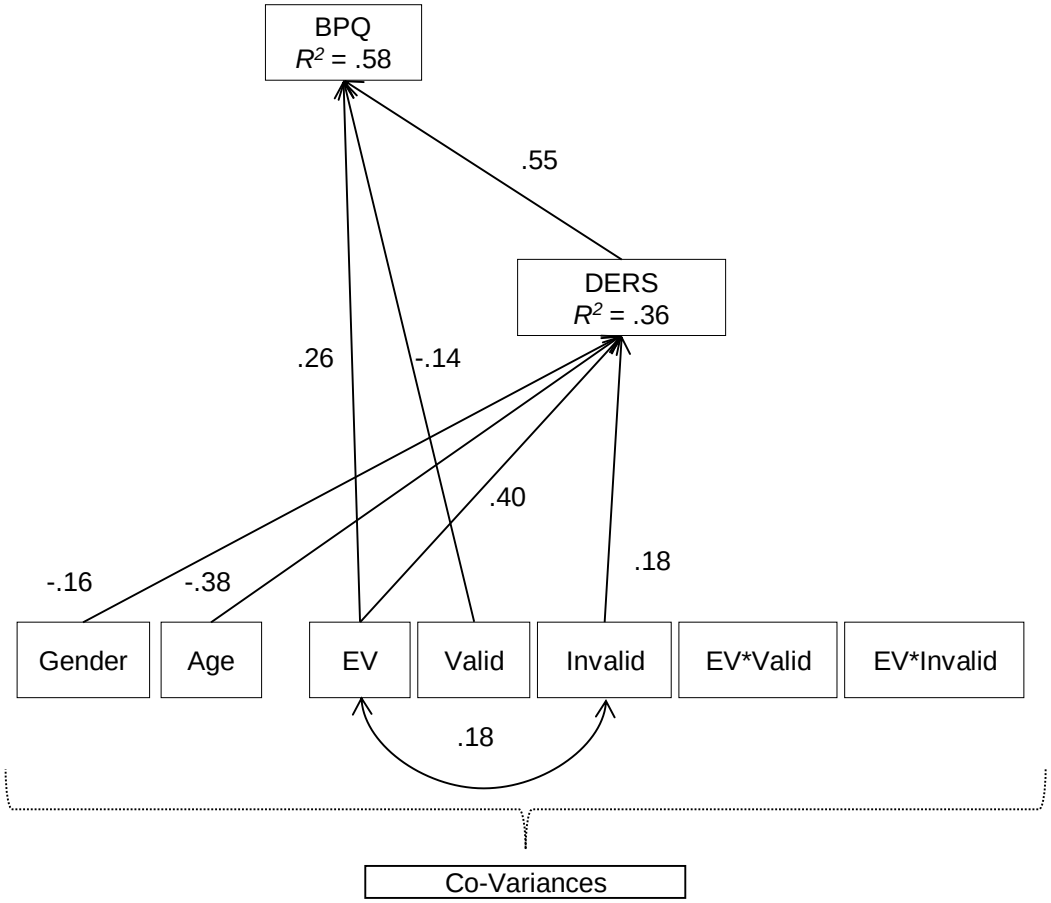

Figure 2.1 Model with all non-significant direct effects removed ($p < .05$). The integers represent standardized estimates for each direct path or co-variance, with each of these being significant ($p < .05$). For simplicity of presentation not all the demographics measures are displayed nor are all the significant co-variances.

The final model was compared to the model where all the direct paths between the variables were retained, yielding a non-significant difference $\chi^2(17, N = 250) = 15.3$, $p = .57$.

Correlational Data

Table 2.2
Correlation between measures of emotional vulnerability and parenting environment

	EV-Child	RCSES-Validation	RCSES-Invalidation
EV-Child	1.00		
RCSES-Validation	-.15*	1.00	
RCSES-Invalidation	.26**	-.53**	1.00

*Note: *p < .05, **p < .01*

Discussion

In line with the biosocial theory, and as expected, emotional dysregulation significantly predicted borderline personality traits. However, contrary to the biosocial theory (Crowell et al., 2009), the hypothesis that emotional vulnerability and invalidating parenting would individually predict emotional dysregulation but have a limited interaction with each other was supported. In addition, both the correlation and co-variance between invalidating parenting and emotional vulnerability, although significant, was not strong, a finding at odds with the contention that the interaction between these two constructs is of a functional (rather than statistical) nature. These findings suggested that both emotional vulnerability and invalidating parenting independently exert their effects upon borderline traits. This has clinical implications, particularly in the case of patients with emerging borderline traits, where both aspects should be assessed and may be targets for intervention.

It is noteworthy that there was a strong relationship between borderline traits and emotional dysregulation, which was operationalised in this study as an individual's reaction to the experience of emotion, rather than the strength of the emotion itself. Despite this strong relationship the items contained in the emotion dysregulation measure do not appear to encompass the same constructs as the measure of borderline traits which are more behaviourally based. Together these findings support the notion that an individual's reaction to emotions, rather than the intensity of the emotions they feel, is more fundamental to the development of borderline traits.

An unanticipated finding was that perceived parental validation correlated significantly and negatively with borderline traits but not with emotional dysregulation, suggesting that it may exert a protective effect against the development of borderline

traits. This is a significant finding, as it suggests that emotion validation is not merely the opposite of emotion invalidation, given the differing roles they occupy in the model. Rather, based upon the items in the measure, it involves being taught how to respond to emotional challenges in a constructive and helpful manner. This finding is also of note as it highlights that not all borderline aetiology is mediated via emotion dysregulation.

Further Research

It should be noted that this study used retrospective self-report measures, cross-sectional rather than longitudinal data to explore the dynamics between the various factors, and a nonclinical sample to examine a clinical construct. Future studies would benefit from replicating this study in a clinical sample. Longitudinal studies are also indicated, however to follow a sufficiently large sample from early childhood to the development of BPD would be costly.

It would also be of interest to establish whether the narrow focus on invalidating parenting may be misguided. It is plausible that invalidating parenting per se may not be of particular importance but rather the measure of invalidating parenting may be tapping into a broader factor of generally harmful parenting (which may include a wide range of behaviours). It is possible that other types of poor parenting beyond emotionally invalidating parenting may also influence the development of borderline traits. Consequently including a measure assessing various types of poor parenting would assist to clarify this situation.

Similarly it could be argued that the construct of an emotionally vulnerable child may be indistinguishable from the broader construct of neuroticism, a construct that correlates with borderline traits (Distel, Trull, et al., 2009), but also to a range of

other physical and mental health conditions (Claridge & Davis, 2001). Consequently if the measure of emotional vulnerability substantially measures neuroticism, its inclusion in a model for BPD, whilst accounting for variation in the associated measures, may not possess the capacity to explain why an individual develops BPD (as opposed to another disorder).

Finally, it would also be of interest to ascertain whether poor parenting exerts an effect in a non-linear fashion. The classification of BPD as a form of complex post-traumatic stress disorder has previously been postulated (Driessen et al., 2002). This would suggest that negative childhood events may have to reach a threshold of stress in order to induce a trauma response, with poor parenting (up until a point) not exerting a significant effect upon longer term behaviour.

Chapter III: Overview and Review of Study 1

The first study's (Gill & Warburton, 2014a) findings were not consistent with hypotheses generated by the biosocial model of BPD. An interaction effect between childhood emotional vulnerability and childhood invalidation was not crucial in explaining current levels of emotional dysregulation. The effects of childhood experiences and vulnerability were not all mediated by emotion dysregulation, with both emotional vulnerability and parental validation having a direct effect on borderline traits. Despite the inclusion of the two factors putatively key to the development of current levels of emotional dysregulation (in addition to demographic factors), the majority of variation in emotion dysregulation remained unexplained.

Several other findings of interest were also made, including that a model with a rather limited number of constructs was able to account for the majority of the variation in borderline traits. In addition, the very strong association between the DERS and borderline traits suggested that the aspects of emotional dysregulation assessed in this measure were germane to such traits. Finally, the key measures of these constructs performed well, demonstrating good internal consistency, suggesting they would be of use in further research.

The research raised a number of further questions. First, the success in predicting borderline traits was largely due to the very strong relationship between borderline traits and emotional dysregulation; the two key childhood constructs exhibited some difficulties in accounting for large amounts of variation in the DERS score. Indeed both age and gender were highly significant predictors of DERS (even with emotional vulnerability and childhood invalidation present in the model).

Second, subsequent analysis of the data (not included in the published report, but included in Appendix B) found that there was an inconsistent relationship between the borderline traits and the DERS factors. Most of the factors of the DERS

correlated highly with each of the borderline traits, with the notable exception of the construct “Lack of emotional awareness”, which had a weak and inconsistent relationship with all of the borderline traits. This raises two main possibilities: (a) that, as some other studies have found (e.g. Bardeen, Fergus, & Orcutt, 2012), lack of emotional awareness should perhaps not be considered as an aspect of emotion dysregulation (or the DERS), or (b) that only certain aspects of emotion dysregulation are central to BPD. This second explanation could lead one to conclude that emotion dysregulation may consist of far more than the six factors identified in the DERS, with specific pathologies exhibiting a combination of deficits unique to the disorder. Indeed, it could be anticipated that other individuals (e.g. perhaps those suffering from spectrum disorders) may suffer a deficit in the area of emotion awareness (but not other deficits particular to BPD). This could also account for the somewhat confusing situation where emotion dysregulation is considered key to BPD, but elevated levels of emotional dysregulation are associated with a range of disorders (Aldao et al., 2010; McLaughlin et al., 2007; Mennin et al., 2005; Soenke et al., 2010; N. H. Weiss et al., 2012).

With regard to aspects of emotion dysregulation missing from the DERS, it could be suggested that the strategies associated with the regulation of emotion should also be included. As previously noted, a number of theorists consider these to be components of emotion dysregulation. Further, it appears that one of the constructs “Limited access to emotion regulation strategies” may be indirectly assessing this construct, with a high score on this factor potentially indicating both a genuine, as well as perceived, lack of strategies to regulate emotion.

Third, whilst the items in the EV-Child measure appear to be a faithful operationalisation of Linehan’s construct of “emotional vulnerability”, close

examination suggests that they may be measuring the construct of neuroticism, a general non-specific risk factor linked to a range of adult psychopathology (Fryers & Brugha, 2013). This would appear to be due to the constructs of “neuroticism” and “emotionally vulnerability” being very similar, rather than any inadequacy on behalf of the EV-Child measure to operationalise “emotional vulnerability”. It could therefore be argued that whilst “childhood emotional vulnerability” was an important part of the model in predicting emotion dysregulation and BPD traits, it could be questioned as to whether the same construct would play a role in a number of pathologies, rending the construct as a non BPD-specific risk factor. Indeed, it could be argued that the key constructs identified in the first study, such as emotion dysregulation and childhood parental invalidation and emotional vulnerability may be predictive of a number of disorders, raising questions as to the specificity of this aetiological model to BPD.

In order to address some of these issues, a second study was developed, utilising a new sample of undergraduate students participating in the study for course credit. The aims of the study were to (a) determine whether childhood emotional vulnerability was a specific risk factor for BPD, (b) whether the biosocial model would be equally predictive of another form of psychopathology, (c) to determine whether emotional dysregulation, as presently assessed by the DERS was equally predictive of another form of psychopathology, and (d) whether the DERS could be improved by including aspects of maladaptive responses to emotions (which are not currently assessed in the DERS).

Chapter IV: Study 2 - Specificity of Emotion Dysregulation and Childhood Emotional Vulnerability to Borderline Traits[2]

[2] This study has been submitted for publication and is presently referenced as Gill, D. J., & Warburton, W. A. (2014). *Specificity of emotion dysregulation and childhood emotional vulnerability to borderline traits.* Manuscript submitted for publication.

Abstract

The biosocial model of borderline personality disorder considers emotional dysregulation to be central to the disorder. Whilst the Difficulties in Emotion Regulation Scale (DERS) correlates strongly with the disorder, elevated scores on this measure have also been associated with other disorders. Similarly, another key construct in the biosocial model, childhood emotional vulnerability, may be a more general risk factor for adult psychopathology and thus conceptually indistinct from childhood neuroticism. We sought to clarify these issues by examining the relationship of these constructs to borderline traits and another psychopathology (chronic worry), in a nonclinical sample (N = 271). Concerns were also held that the DERS fails to assess key aspects of emotional dysregulation and so an attempt was made to expand this measure. Results suggest that the DERS is an incomplete measure of emotional dysregulation and that childhood emotional vulnerability may be conceptualised as a general risk factor for adult psychopathology.

Introduction

Emotion regulation is a problem present in many clinical disorders, with emotional dysregulation being viewed as a key deficit in individuals with borderline personality disorder (BPD) (Glenn & Klonsky, 2009). Dialectical behaviour therapy, based upon the biosocial model of BPD, posits that this emotion dysregulation is caused by an interaction between an emotionally vulnerable child being raised in an emotionally invalidating environment (Linehan, 1993). A study by Gill and Warburton (2014a), using structural equation modelling, confirmed the importance of emotion dysregulation in BPD but failed to support the existence of an interaction between emotional vulnerability and an invalidating parenting environment. Instead, they found these factors independently predicted emotion dysregulation but that an interaction between factors was not important.

Several aspects of Gill and Warburton's revised model of BPD require further examination. First, it is uncertain whether an "emotionally vulnerable" child is distinct from a "neurotic" child, or "invalidating parenting" is distinct from generally poor parenting. Consequently it could be argued that the best fit model in their study shows that a neurotic child exposed to poor parenting is more likely to develop borderline traits; an aetiological formulation so generic that may fit any number of mental disorders.

Second, more recent incarnations of the biosocial theory have suggested childhood impulsivity may also play a role in the development of borderline traits (Crowell et al., 2009), but its interaction with other aetiological components remains unclear.

Finally, it is uncertain whether the measure used to assess emotion dysregulation in previous studies, the Difficulties in Emotion Regulation Scale

(DERS; Gratz & Roemer, 2004) is too narrow in scope, not allowing for a comprehensive assessment of the emotion regulation difficulties encountered by those with BPD nor distinguishing between the regulation difficulties associated with BPD and other psychological disorders.

Difficulties in Emotion Regulation Scale

Several concerns can be raised in relation to the DERS, a major weakness being that the original DERS was purposely formulated without including measures of maladaptive responses to emotions, on the basis that whether a response is adaptive or not is context dependant. This position may, however, ignore several important factors. First, some responses to emotions, such as becoming fearful of the emotion or becoming cognitively inflexible are likely to be maladaptive in almost all circumstances. Second, even a potentially adaptive response, such as becoming angry, may be maladaptive if it is repeatedly utilised without regard to context. Third, a potentially adaptive response, such as becoming self-reflective may be maladaptive if utilised to an extreme degree (e.g. where it may develop into rumination). Fourth, from a clinical perspective it would appear that such maladaptive responses account for a significant amount of distress and impairment of function, and so the DERS may currently fail to assess critical components of emotion dysregulation. Finally, elevated scores on the DERS have been noted with a number of different conditions (Ehring & Quack, 2010; Mennin, McLaughlin, & Flanagan, 2009; Svaldi, Griepenstroh, Tuschen-Caffier, & Ehring, 2012). Consequently it could be hypothesised that the use of different maladaptive emotion regulation strategies may distinguish between the disorders. This is particularly problematic when examining BPD, as models utilising the DERS would struggle to

explain what distinguishes the emotion regulation difficulties in BPD from any of the other disorders that are associated with elevated scores on this measure.

Another major concern with the DERS involves its psychometric properties. Most notably, there is a lack of clarity about what the latent constructs measure. Several of the items appear to lack criterion validity with regard to the construct they putatively assess. For example one item "When I'm upset, I start to feel very bad about myself" would appear to relate to engaging in self-recrimination for feeling emotional – despite this, the DERS assigns this to the subscale of "Limited access to emotion regulation strategies". As each subscale has good internal consistency (Gill & Warburton, 2014a; Gratz & Roemer, 2004), such anomalies suggest that the latent constructs may measure a somewhat different construct to that labelled.

Present Study

In order to clarify these issues related to emotion dysregulation and BPD, a modified version of Gill and Warburton's BPD model was constructed and tested. In addition, further factors related to emotion dysregulation, such as maladaptive strategies or beliefs about emotions, were added to the DERS for analysis. Further, it was anticipated that if such additional aspects are germane to emotion dysregulation, that they would be able to account for variation in measures of psychopathology, over and above that predicted by the present DERS factors.

Second, in order to determine whether being an emotionally vulnerable child was a general risk factor for (any) adult psychopathology, this study assessed the relationship between emotional vulnerability, BPD and a comparison psychopathology – generalised anxiety. It was expected that if the construct was a

general risk factor it would share a similar relationship with the two measures of psychopathology.

Finally, a measure of childhood impulsivity was included in the model to determine its role in the development of BPD.

Method

The current study was conducted online by first year psychology students completing the study for course credit, with a sufficient sample being sought to complete the proposed structural equation modelling and factor analysis. Incomplete questionnaires were removed from the sample, as were questionnaires where random responding was a concern (as indicated by the survey being completed within 5 minutes of commencement). Data on household income was also removed due to the number of incomplete responses. These were the sole data exclusions and all other data manipulations used in the study are reported below.

The final sample consisted of 271 participants (48 male, 223 female) with a mean age of 20.51 years (S.D. = 5.56). 49.1% of participants identified with an Australian background. 18.1% of participants had engaged in therapy (being therapy that occurred more frequently than once a month), with 85.7% of those identifying that therapy had made them somewhat or much better.

Measures

Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). focuses upon an individual's response to emotions rather than the frequency or intensity of the emotions themselves. It comprises of 6 facets of emotion dysregulation has good internal consistency (α = .93 - .94), test-retest reliability (ρ_I = .88, p < .01) and predictive validity in relation to self-harm, a common behavioural consequence of emotional dysregulation (Gratz & Roemer, 2004, 2008).

Additional items for this measure were constructed by a group of five psychologists experienced in working with emotionally dysregulated clients. These psychologists examined the original DERS and were asked to consider aspects of

emotion dysregulation that they had observed in their clients that were not currently assessed by the DERS. Consistent with the definition used by the original authors of the DERS, the psychologists were asked to conceptualise emotional dysregulation as a secondary response to the experience of an emotion (rather than the intensity of the underlying emotion). In contrast to the definition used by the authors of the original DERS, the psychologists were encourage to consider maladaptive responses to emotions as being a component of emotion dysregulation. During the process of identifying the new aspects the psychologists were allowed access to the list of new areas that the other psychologists had identified, in order to minimise areas of conceptual overlap between the newly identified aspects. Seventeen aspects of emotion dysregulation not currently captured by the DERS were identified, with these falling into the following five groupings:

1. Maladaptive reactions to emotions
    a. Blaming others/becoming angry
    b. Comparing emotional experience to others
    c. Intellectualising/over-rationalising
    d. Sustaining emotion on purpose/making no effort to change
    e. Avoidance/fear
    f. Self-blame/deprecation
    g. Suppressing the emotion
    h. Rumination
    i. Questioning of self
    j. Aberrant responses to emotions
2. Maladaptive attitudes to emotions
    a. Dismissive

   b. Overconfident regarding ability to manage emotions
   c. Locus of control for emotional experience located in others
3. Problematic emotional expression
   a. Exaggerated expression of emotion
   b. Inhibited expression of emotion
4. Cognitive inflexibility when experiencing emotions
   a. Cognitive inflexibility when experiencing emotions
5. Experiencing emotion contagion
   a. Experiencing emotion contagion

For each of the 17 missing aspects, 4-5 items were developed. When combined with the standard DERS items, this resulted in 105 items for this measure.

Recalled Childhood Socialization of Emotion Scale (RCSES; Krause et al., 2003). presents participants with a series of common childhood scenarios which would evoke an emotional response from a child (e.g. feelings upset after watching a scary TV show) and asks the participant to rate how likely their parent would have engaged in a particular response (e.g. tell me that I was over-reacting). These responses then yield two scores, assessing the parenting on the level of validation and invalidation. It has previously been used for this purpose in a number of other studies (e.g. Sauer & Baer, 2010; Thomas et al., 2011). Based upon the data from our previous study (Gill & Warburton, 2014a), it had been determined that only 3 of the given scenarios would be required to accurately assess the constructs of validating and invalidating parenting and so consequently only these scenarios were used in the present study.

Emotional Vulnerability–Child scale (EV-Child; Sauer & Baer, 2010). is a self-report measure retrospectively rating emotional vulnerability, incorporating aspects of the Affective Intensity Measure (AIM; Bryant et al., 1996) and some items relating to Linehan's conceptualisation of emotional vulnerability involving a slow return to baseline once emotionally activated. It contains items such as "In scary situations, I got more scared than most other children" and "It took me a long time to calm down after getting upset about something". It demonstrates high levels of internal consistency (α = .91 - .94), is strongly related to borderline symptoms, emotion dysregulation, thought suppression and being fearful of emotions (Gill & Warburton, 2014a; Sauer & Baer, 2010).

Barrett Impulsivity Scale-11 (BIS-11; Patton, Stanford, & Barratt, 1995). the BIS-11 is a 30 item scale which is arguably the most commonly used measure of impulsivity for clinical and research purposes (Stanford et al., 2009). A validated retrospective self-report measure of childhood impulsivity was unable to be found. We therefore examined the factor structure of the scale when it had been utilised with a preteen sample (Cosi, Vigil-Colet, Canals, & Lorenzo-Seva, 2008), with three factors having been identified (motor impulsivity, not planning impulsivity and cognitive impulsivity). Eleven items spread across these factors were selected and modified to assess childhood impulsivity, with the modification primarily involving a re-wording of the item to allow for retrospective rating. There was a concern that the cognitive impulsivity domain would not be appropriately assessed and so an item was added to assess this domain ("I was a quick thinker"). Finally, one key item to assess childhood impulsivity was also developed ("I was impulsive"). Participants

were then asked to rate themselves on these items, as they applied to them when they were a child.

Given that this study was the first time that these items had been used in this way, care was taken when examining the results in order to ensure the measure's validity. An analysis of inter-item correlations of the items revealed weak and inconsistent correlations between the item "I was a quick thinker" and the other items in the measure, raising questions as to whether this item was assessing childhood impulsivity (or perhaps perceived cognitive ability). As a consequence this item was removed from the measure. The remaining items were found to be highly reliable ($\alpha = .87$), suggesting a homogenous construct was being measured. Further, these items correlated with adult levels of impulsivity (as assessed by one of the BPQ subscales) with $r(269) = .33$, $p < .01$, suggesting that each scale was assessing a related construct but that the ratings of childhood impulsivity were not merely reflective of individuals rating their current levels of impulsivity.

Borderline Personality Questionnaire (BPQ; Poreh et al., 2006). is a self-report measure assessing the nine borderline domains, as identified by the DSM-5 (American Psychiatric Association, 2013) by asking dichotomous items such as "I often feel empty inside" and "The people I love often leave me". It has previously been used a dimensional measure of borderline traits, with the subscales demonstrating adequate internal consistency ($\alpha = .78$ - $.93$) (Fonseca-Pedrero et al., 2011). Further, when used for the screening of outpatient youth with the disorder, it has compared favourably to comparable measures (Chanen et al., 2008).

Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). is a measure of chronic worry. It has been found to have a test-retest reliability of .93 and an internal consistency of .95 (Meyer et al., 1990), with evidence existing for its convergent and divergent validity (Brown, Antony, & Barlow, 1992). Further, there is also some support for its use as a screening measure of generalised anxiety disorder (GAD) (Behar, Alcaine, Zuellig, & Borkovec, 2003).

Given the number of items, all questionnaires after the initial demographics measures were randomised to ensure that participant fatigue would not systematically influence the results. For the same reason the emotion dysregulation items were also randomised within this measure.

Results

Factor Analysis of Emotion Dysregulation Measures

A mean score was calculated for each of the original DERS factors (DERS-O) and the seventeen additional aspects of emotional dysregulation identified by experts (DERS-N). These scores were then correlated against each other and also against the measures of psychopathology (the BPQ and the PSWQ). Problems were identified with the "Overconfident regarding ability to manage emotions" score, with this having a negative correlation with the other emotional dysregulation scores and exhibiting a linear negative relationship with the two measures of psychopathology. It was concluded that this measure was actually assessing a lack of confidence in managing emotions and consequently the items contributing to this measure were reverse coded and the subscale was renamed ("Lack of confidence regarding ability to manage emotions").

An analysis of all of the new emotion dysregulation items showed they had high internal consistency ($\alpha = .93$) and there was a strong significant correlation between the total mean score for the new items and the total mean score for the original DERS ($r = .81$, $p < .01$).

An exploratory factor analysis was completed upon all original and additional emotion dysregulation items, following a similar procedure to that used by Gratz and Roemer (2004); a principal axis analysis with promax rotation. This solution was unsatisfactory with 23 factors having eigenvalues greater than 1. Whilst the associated scree plot indicated 5 factors, these 5 factors only accounted for a minority (43.60%) of the variance of all the items. Various attempts were made to resolve these difficulties by utilising such strategies as removing items that correlated

poorly with the overall sample, to computing subscales and completing a factor analysis on the scales (rather than items). These attempts were unsuccessful.

An attempt to create a viable 23 factor measure was unsuccessful due to the large number of items loading onto the first factor, with insufficient items with discreet factor loadings to account for more than 4 factors in total.

Calculation of Measures for Analysis

The standard manner of establishing the total score on the BPQ was considered inappropriate for this study as it involves summing all the scores despite each subscale having a variable number of items that may be endorsed at differing rates. This creates the potential for some of the subscales to have a greater influence on the overall score, whereas the DSM-5 does not give primacy to any of the traits (American Psychiatric Association, 2013). Consequently the scoring of the BPQ was completed by transforming the mean level of endorsement of each of the subscores into a z score prior to the creation of an overall mean score, thus not favouring one facet of BPD over the others.

A decision was made to only use the original DERS items to assess emotion dysregulation, in order to allow for comparisons with other published studies utilising this measure. However, similar to the BPQ, concerns exist regarding the capacity of one domain of emotional dysregulation to unduly influence the overall score due to differing number of items in the scale or rates of endorsement. Consequently the total score (“DERS”) was calculated as the mean standardised score for each of the six domains in the original DERS.

The PSWQ does not have this difficulty and so was calculated in the standard fashion.

As previously noted, the item “I was a quick thinker” appeared to be measuring a construct other than that of childhood impulsivity. Consequently this item was removed from the impulsivity measure, with an overall measure of childhood impulsivity (“IMPULSE”) being calculated as the mean of the remaining items.

As had previously been found (Gill & Warburton, 2014a), normality was an issue for RCSES-Invalidation which was resolved via a logarithmic transformation. The childhood emotional vulnerability (“EV-CHILD”), RCSES-Invalidation (“INVALID PARENT”) and RCSES-Validation (“VALID PARENT”) scores were converted to z-scores to allow for the calculation of interaction effects (obtained by multiplying the standardised scores together and generating the variables “EV*INVALID” and “EV*VALID”).

Some of the independent demographic variables were categorical or had problems with normality, necessitating their transformation into dichotomous variables. Correlations were calculated between the main variables (see Table 4.1).

Table 4.1
Correlation between dependent and independent variables

	BPQ	PSWQ	DERS	EV-CHILD	VALID PARENT	INVALID PARENT	IMPULSE
BPQ	1.00						
PSWQ	0.56**	1.00					
DERS	0.69**	0.53**	1.00				
EV-CHILD	0.49**	0.49**	0.43**	1.00			
VALID PARENT	-0.32**	-0.07	-0.19**	-0.20**	1.00		
INVALID PARENT	0.28**	0.09	0.23**	0.27**	-0.67**	1.00	
IMPULSE	0.13*	0.00	0.21**	0.12*	-0.06	0.12*	1.00

*Note: *p < .05, **p < .01*

Regressions

Eleven of the subscales derived from the 17 aspects of emotion dysregulation not currently assessed by the DERS had acceptable reliability (i.e. $\alpha \geq .70$). The other six aspects had poor levels of reliability, with these issues unable to be adequately resolved due to one of two reasons: multiple weak inter-item correlations suggesting that they were not identifying a single construct, or, alternatively, there being only a small number of items allocated to construct, with the result that if only one or two items were not accurately assessing the given construct, their removal would leave too few remaining items to reliably assess the construct.

Three of the remaining scales had a significant number of respondents endorsing the lowest score for each item on the scale, thus impairing normality; these scales were transformed into dichotomous variables, based upon the respondent endorsing the items on average more than “sometimes”.

The regression against each measure of psychopathology was completed stepwise, with the first block containing all the subscales from the DERS and the second block containing the new subscales (see Table 4.2).

Table 4.2
Regression of emotion dysregulation subscales against measures of psychopathology

		b	se	β	t
BPQ	DERS-O: Limited Access to Emotion Regulation Strategies	.32	.06	.42	5.56**
	DERS-O: Lack of Emotional Clarity	.15	.04	.18	3.64**
	DERS-O: Impulse Control Difficulties	.18	.05	.22	3.55**
	DERS-N: Reaction to emotion: Avoidance/Fear	-.14	.05	-.18	-3.11**
	DERS-N: Attitude towards emotion: Other directed	-.11	.04	-.13	-2.71**
	DERS-N: Reaction to emotion: Comparing emotional experience	.10	.04	.15	2.56*
	DERS-N: Reaction to emotion: Self-blame/deprecation	.10	.05	.13	2.05*
	DERS-N: Reaction to emotion: Sustaining emotion on purpose (dichotomous variable)	.15	.07	.09	2.03*
PSWQ	DERS-O: Nonacceptance of Emotional Responses	.11	.05	.13	2.11[a]
	DERS-O: Limited Access to Emotion Regulation Strategies	.13	.08	.14	1.65*
	DERS-N: Attitude towards emotion: lack confidence	.32	.05	.33	5.95**
	DERS-N: Reaction to emotion: Rumination	.21	.06	.25	3.42**

Note. DERS-O: Original DERS subscales, DERS-N: New aspects of emotion dysregulation not previously assessed by the DERS, BPQ R^2 = .60, PSWQ R^2 = .45.
*[a] p < .10, * p < .05, ** p < .01*

Model Development

The model used composite rather than latent variables due to the large number of parameters relative to the size of the sample.

Variables were fitted into a three level model using AMOS software. The lowest level comprised of the independent variables, the middle level consisted of the

DERS, and the upper levels consisting of the PSWQ and the BPQ. All independent variables were posited to have a direct and indirect (via the DERS) relationship with the PSWQ and the BPQ. Demographic variables were also entered at the lowest level. All independent variables were posited to co-vary, as were the error terms for the BPQ and the PSWQ.

As Gill and Warburton (2014a) found, the removal of non-significant paths and co-variances led to significant changes in the chi-squared value, despite other fit indices (less affected by sample size) remaining stable. Consequently the same solution was applied, namely to prune the model on the basis of the significance of the co-variances and direct effects. The model was pruned of all weak co-variances, rerun and further co-variances were pruned. This process was repeated until only significant ($p < .05$) co-variances remained. The same pruning technique was then utilised for the direct effects.

This resulting model (see Figure 4.1) was a very good fit to the data (goodness of fit index = .98, comparative fit index = 1.00, root mean square residual = .03, standardised root mean square residual = .05, normed fit index = .96). The model was also parsimonious (root mean square error of approximation = .00, probability of close fit = .99) and had an acceptable chi-squared statistic $\chi^2(45, N = 271) = 43.22$, $p = .548$ ($\chi^2/df = .96$).

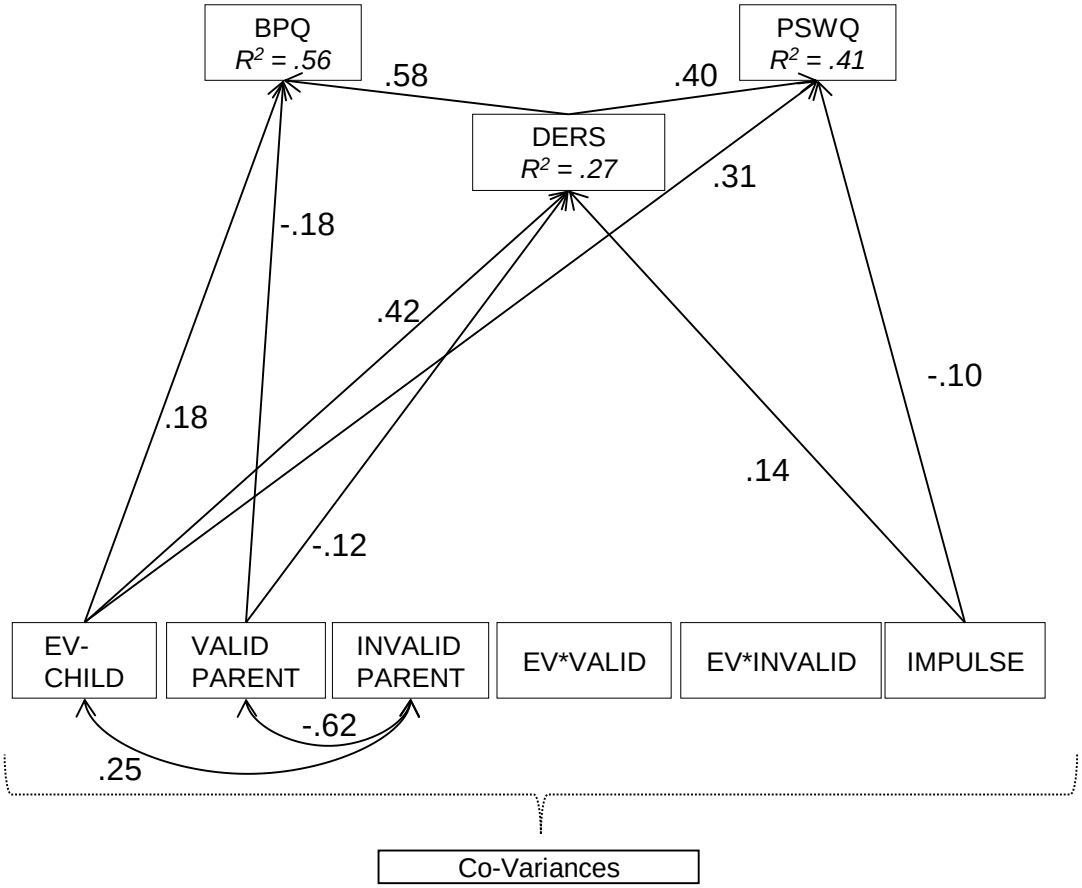

Figure 4.1. Model examining the biosocial model in relation to the BPQ and the PSWQ with all non-significant direct effects removed ($p < .05$). The integers represent standardized estimates for each path. For simplicity of presentation not all significant co-variances are noted, nor are all independent variables.

Discussion

As with our previous study, emotional dysregulation, as measured by the DERS, significantly predicted borderline personality traits. Similarly, and in contrast to the biosocial theory, the hypothesis that emotional vulnerability and invalidating parenting would correlate with emotional dysregulation but have a limited interaction with each other was supported. The prediction that childhood impulsivity would contribute to current emotional dysregulation was confirmed. It was found that the overall model was a better predictor of borderline traits than chronic worry; however this would appear to be primarily due to the strength of the relationship between borderline traits and the DERS. Further it appears childhood emotional vulnerability, but not (in)validating parenting, has a similar relationship to chronic worry as to borderline traits. Finally, it was of interest that invalidating parenting, a precursor to BPD in the biosocial model, did not predict the DERS, BPQ or PSWQ whilst validating parenting was a negative predictor for the DERS and BPQ, perhaps suggesting it may be a protective factor.

It is noteworthy that in comparing chronic worry to borderline traits that the relationship to childhood emotional vulnerability operated in a similar fashion across the psychopathologies, both directly and indirectly predicting the pathologies. This, in conjunction with the almost identical correlation between being an emotionally vulnerable child and each measure of psychopathology, is consistent with the hypothesis that this construct is more parsimoniously explained as childhood neuroticism (rather than it being uniquely associated with borderline traits).

Results suggested, as hypothesised, that emotion dysregulation is multi-faceted. The items measuring facets not assessed by the DERS demonstrated a high level of internal consistency, their mean correlated strongly with the DERS and

several of the subscales enhanced the capacity of the DERS to predict two different forms of psychopathology (indeed they were significant predictors of psychopathology whilst some of the original subscales were not). This supports the contention that maladaptive responses to emotions can and should be considered in the formulation of a measure of emotion dysregulation.

In the regressions of the original and new subscales upon measures of psychopathology it was noted that (a) the importance of a given subscale was dependent upon the form of psychopathology being examined and (b) some subscales, despite exhibiting positive correlations with the measure of psychopathology, exhibited significant negative relationships to the same psychopathology once other factors were taken into account.

This has important implications, first suggesting that certain aspects of emotion dysregulation are more or less relevant, depending upon the disorder in question.

Second, the stronger relationship between the DERS and BPQ in the model may be due to the particular scales used in the DERS being more relevant to borderline traits – if a different subset of scales of emotional dysregulation were used then the PSWQ may have exhibited a stronger relationship to emotional dysregulation than the BPQ. As a consequence it is problematic to suggest that emotional dysregulation *per se* is central to borderline pathology – rather it is a particular pattern of dysregulation that is critical and that distinguishes borderline traits from those of chronic worry/GAD.

Third, it suggests that whether an emotion regulation response is adaptive or not is contingent upon the other forms of emotion dysfunction present. This may be of clinical significance. If in treating an individual with borderline traits a clinician

were to try to decrease the client's reliance on others for emotion regulation or encourage them to be less avoidant of emotional experiences, prior to addressing other factors such as increasing their emotional clarity or impulsivity in relation to emotions, a worsening of symptoms would be anticipated.

Finally, the analysis raises questions as to whether the current iteration of the DERS contains aspects that are less relevant to the emotion dysregulation construct than others. It includes scales which appear to be redundant in the case of BPD and GAD, whilst failing to assess other relevant aspects of emotion dysregulation.

Implications and Further Research

It seems that a significant revision of the DERS is warranted, in particular an expansion of the measure, the removal of redundant subscales and a clarification of its factor structure.

Further, several studies have now cast doubt upon the validity of the biosocial model's hypothesis regarding the aetiology of emotional dysregulation, particularly with respect to borderline traits (Gill & Warburton, 2014a; Reeves, 2007; Sauer & Baer, 2010). It would be useful to conduct a similar study with a clinical population to replicate these findings. In addition, both the Gill and Warburton (2014a) study and the current study have been unable to account for a significant proportion of the variation in emotional dysregulation with the putative antecedents. Developing a more comprehensive model to explain the development of such dysfunction may be beneficial.

Chapter V: Conclusion

Section 1. Review of Study 2

The second study supported some hypotheses derived from the biosocial model. In particular (in)validating parenting appeared to have a relationship to borderline traits that did not exist with regard to chronic worry. Further, the relationship between emotional dysregulation, as assessed by the DERS, was more strongly related to borderline traits than to chronic worry.

The study however, failed to support other hypotheses derived from the biosocial model. Critically, the construct of childhood emotional vulnerability did not appear to have a special relationship to borderline traits, sharing a similar relationship to chronic worry, suggesting that this construct may represent a general risk factor (akin to childhood neuroticism) for psychopathology. Further, evidence was provided suggesting that borderline traits and chronic worry were associated with particular patterns of emotion dysregulation, rather than emotional dysregulation being a construct specific to BPD. Finally, the results of the first study were replicated, with regard to the interaction between childhood emotional vulnerability and invalidating parenting not being critical in accounting for the development of adult emotion dysregulation and borderline traits.

Section 2. Summary of Findings

These two studies provide only limited support for the biosocial model of BPD, and suggest that an alternative model could be developed to better account for the development of borderline traits. The key findings were as follows:

1. Current emotional dysregulation was found to be a highly significant predictor of borderline traits in both studies
2. A strong interaction effect between childhood emotional vulnerability and childhood invalidation was not supported, indeed in both studies this interaction effect was dropped from the model due to lack of significance.
3. The effects of (in)validating parenting on BPD were not solely due its impact upon emotion dysregulation.
4. Childhood emotional vulnerability does not appear to be a unique risk factor associated with BPD; rather the second study suggested that this construct probably represents something akin to childhood neuroticism.
5. In a general sense, emotional dysregulation does not appear to enjoy a special relationship to BPD (relative to other psychopathologies). Rather a particular pattern of emotional dysregulation is critical, with some aspects of emotional dysregulation actually appearing to be protective in the context of other forms of emotional dysfunction.
6. Despite the biosocial model's emphasis upon two key constructs; childhood emotional vulnerability and an invalidating environment, a majority of the variation in emotion dysregulation was unaccounted for.

2.1 Hypotheses Revisited

Several a priori hypotheses were made based upon the biosocial model, listed below:

1. The interaction between childhood emotional vulnerability and having experienced emotionally invalidating parenting in childhood would strongly predict adult emotional dysregulation.

2. Adult emotional dysregulation would be a strong predictor of adult borderline traits.

3. The relationship between adult borderline traits, and childhood emotional (in)validation and emotional vulnerability would be fully mediated by adult emotional dysregulation.

4. Emotional dysregulation would be more strongly associated with borderline traits than a comparison psychopathology.

5. (In)validating parenting would feature more prominently and/or operate differently in the account of borderline traits than a comparison psychopathology.

6. Childhood emotional vulnerability would feature more prominently and/or operate differently in the account of borderline traits than a comparison psychopathology.

7. These findings would be supported with individuals drawn from a broad age range.

In reviewing the two studies, hypotheses 1, 3 and 6 were not supported by the results whilst 2, 4, and 5 were. It should be noted that in the case of hypothesis 4 evidence was presented suggesting that it is a particular pattern of emotional dysregulation which is central to BPD (rather than all aspects of emotional dysregulation bearing a particularly strong relationship to BPD). Support for hypothesis 7 was mixed, given the mixed support for the other hypotheses.

Section 3. Critique of the Studies

Several aspects of the studies deserve critical comment, foremost the use of a nonclinical sample. However it should be noted that BPD is not uncommon – indeed it would be anticipated that amongst a large nonclinical sample that several individuals would have clinically significant borderline traits. Further it is recognised that most personality traits exist upon a continuum, consequently if the results were to differ with a clinical sample it would suggest that the biosocial theory requires further modification to account for why a process (i.e. the interaction) is critical in clinical populations but is not important in nonclinical populations.

An issue exists with the BPQ, in that some items assess whether a particular behaviour has “ever” occurred. This is problematic in that such items do not allow for a change in presentation over time. An attempt was made in the second study to adjust the items where this occurred by re-administering such items but specifying that the behaviour should have occurred in the preceding two years (see Appendix C). A decision was made not to include the analysis of these items in Study 2 given that the sample was relatively young (and the issue of change over time is likely to be of greater importance with older age groups) and also to ensure consistency in the BPQ across the studies. It would however be anticipated that making such adjustments may improve the apparent strength of the relationship between current emotional dysregulation and current borderline traits (although possibly leading to a decline in the apparent strength of the relationship between the childhood factors and current borderline traits).

Another issue with the study involves criterion validity. Although the measures used for invalidating parenting and childhood emotional vulnerability both have been validated to a certain extent, the validation has utilised retrospective recall on behalf

of other parties and comparing these to self-report data. Consequently it is possible that both the target individual in question (and other observers) may be heavily influenced by the target individual's current presentation (e.g. whether they appear to be currently emotionally vulnerable) and their current relationship with their parent (e.g. the level of invalidation currently experienced in the relationship). It is therefore possible that the retrospective reports of childhood experiences and functioning may reflect current functioning and experiences, rather than being an unadulterated retrospective account, and for this reason retrospective data needs to be interpreted cautiously.

Even assuming criterion validity, causality also remains an issue. Whilst a model was able to be derived that gave a reasonable account of BPD, caution should be taken assuming that this demonstrates causality (or the direction of this causality). A cogent argument could be presented that the model best describing the actual developmental trajectory could be an inversion of the current model. A child may present with nascent borderline traits (which subsequently continue through to adulthood, thus leading to a high BPQ score), and these borderline behaviours make managing emotions difficult (thus leading to a high DERS score). As the child experiences difficulties in managing their emotions this leads to more extreme levels of emotionality (thus leading to a high EV-Child score) and are more difficult for parents to manage, thus being more likely to provoke an invalidating response from the child's parents (thus leading to a high RCSES-Invalid score). Such a model would be equally likely to fit the data, despite all the causal relationships being inverted.

Further, caution must be drawn with regard to assuming the relationship between emotional dysregulation and BPD is causal or direct. It could well be that both

constructs are linked by a common relationship to a third construct, for example maladaptive schemas or a poorly integrated sense of self.

A concern could also be raised regarding the measure of invalidation not measuring as broad a construct as denoted in the biosocial model. An argument could be presented that the biosocial model focuses on an invalidating environment, with this environment including factors beyond the relationship between a child and their parent. This is a valid argument, and it could be anticipated that if one were somehow able to accurately measure such a broad construct that invalidation may have been able to account for a greater proportion of the variation in emotional dysregulation/borderline traits. However, whilst some attempts have been made to purportedly assess an invalidating childhood environment, they have typically focused on parenting (e.g. Mountford, Corstorphine, Tomlinson, & Waller, 2007), acknowledging the parents' central role. Further, even if a broader measure was developed, it would be anticipated that it would increase the main effect of invalidation on emotional dysregulation – it is unclear why the broadening of the measure of the construct would potentially lead to the posited interaction effect with childhood emotional vulnerability.

Another issue with invalidation is that questions can be raised as to whether it is a causal developmental antecedent leading to BPD. Previous studies have found that inappropriate parenting practices tend to cluster (Sheffield, Waller, Emanuelli, Murray, & Meyer, 2005; Warburton, 2007). Further, whilst abusive situations may include invalidation, they also involve a range of other damaging characteristics. Consequently it is difficult to assert that the invalidation aspect is the critical component in a specific abusive situation, or more broadly, that invalidation's

association with BPQ is not due to the propensity of an invalidating parent to engage in other forms of poor parenting.

Finally, concerns could be raised with regard to various aspects of the statistical methods used to analyse the data in both studies, including whether it could have been more appropriate to split the sample into clinical and nonclinical components, whether the data transformation of invalidating parenting was appropriate, whether the use of non-standard calculation of the DERS/BPQ was appropriate and whether different forms of analysis (such as logarithmic regressions) would have been more apt.

Whilst such concerns may have some validity, it is questionable as to whether these present a fundamental challenge to the central conclusions drawn by the studies. Rather, it is more likely that changing the form of the analysis would have altered the relative strength of the associations between the various constructs. It is somewhat implausible that altering one aspect of the analysis (such as the method of calculating the DERS/BPQ) would lead to a dramatic revision in the conclusions drawn. In particular it appears unlikely that this would allow the interaction effect between an emotionally vulnerable child and invalidating parenting (that was found to be redundant) to become of crucial significance in accounting for the DERS; with the DERS in turn mediating all the relationships between borderline traits and the relevant childhood antecedents.

Section 4. Further Clarification of the Biosocial Model

These studies support several avenues of further research. First, as noted above, it is possible that these findings may be due to the nonclinical nature of the samples. Consequently replicating the studies with a clinical sample would be valuable.

Second, the construct of childhood emotional vulnerability warrants further attention. If this construct is actually childhood neuroticism then it would be anticipated that it would be an antecedent of a variety of forms of psychopathology, beyond the development of BPD or chronic worry, a hypothesis which could be tested by comparing its relationship to other forms of adult dysfunction. Indeed, if this was found to be the case it would suggest that the EV-Child measure may be a useful clinical tool, given the absence of other tools retrospectively measuring childhood neuroticism, with those tools currently available often overtly (and unhelpfully) including aspects of child psychopathology.

Third, it would be of benefit to attempt to determine whether invalidation is the critical component of the poor parenting or whether other forms of maladaptive parenting better account for emotional dysregulation and borderline traits. Similarly, other research has suggested that childhood invalidation may be a factor in other forms of psychopathology, such as eating disorders (Ford, Waller, & Mountford, 2011; Haslam, Mountford, Meyer, & Waller, 2008). Whilst the second study did not find that this form of parenting contributed substantially to chronic worry, this may reflect the pathogenesis of chronic worry (rather than suggesting invalidating parenting has a unique relationship to BPD).

Section 5. Comprehensive Framework Model

Whilst the two studies failed to support several key hypotheses derived from the biosocial model, they do provide some guidance towards the development of a more appropriate aetiological model. Clearly, emotional dysregulation is an important component of BPD, and should be retained in a future model. Childhood neuroticism (or a construct akin to this) appears to also be of import. Whilst parental invalidation was found to be significant, clinical experience, in addition to the plethora of studies linking childhood abuse and trauma to BPD, suggest that the environmental factors could be further elaborated, with childhood abuse and trauma being appropriate constructs to examine.

Whilst there may be some benefit in attempting to further test the biosocial model, it could also be argued that its central prediction, regarding the purportedly critical interaction between childhood emotional vulnerability and invalidating parenting has failed to be supported by four separate studies (Gill & Warburton, 2014a, 2014b; Reeves, 2007; Sauer & Baer, 2010). Whilst efforts could continued to be made to alter the study design, either by utilising different measures or samples in order to locate the elusive interaction effect, at some point it may be appropriate to radically revise the model. Based both on the findings of the two studies presented, and the extant empirical literature, it could be argued that given the breadth of risk factors previously identified, that a more comprehensive model needs to be developed, one that does not rely upon a few key constructs. Consequently, based upon both the aforementioned research and the current studies, the following comprehensive framework model is proposed (see Figure 5.1).

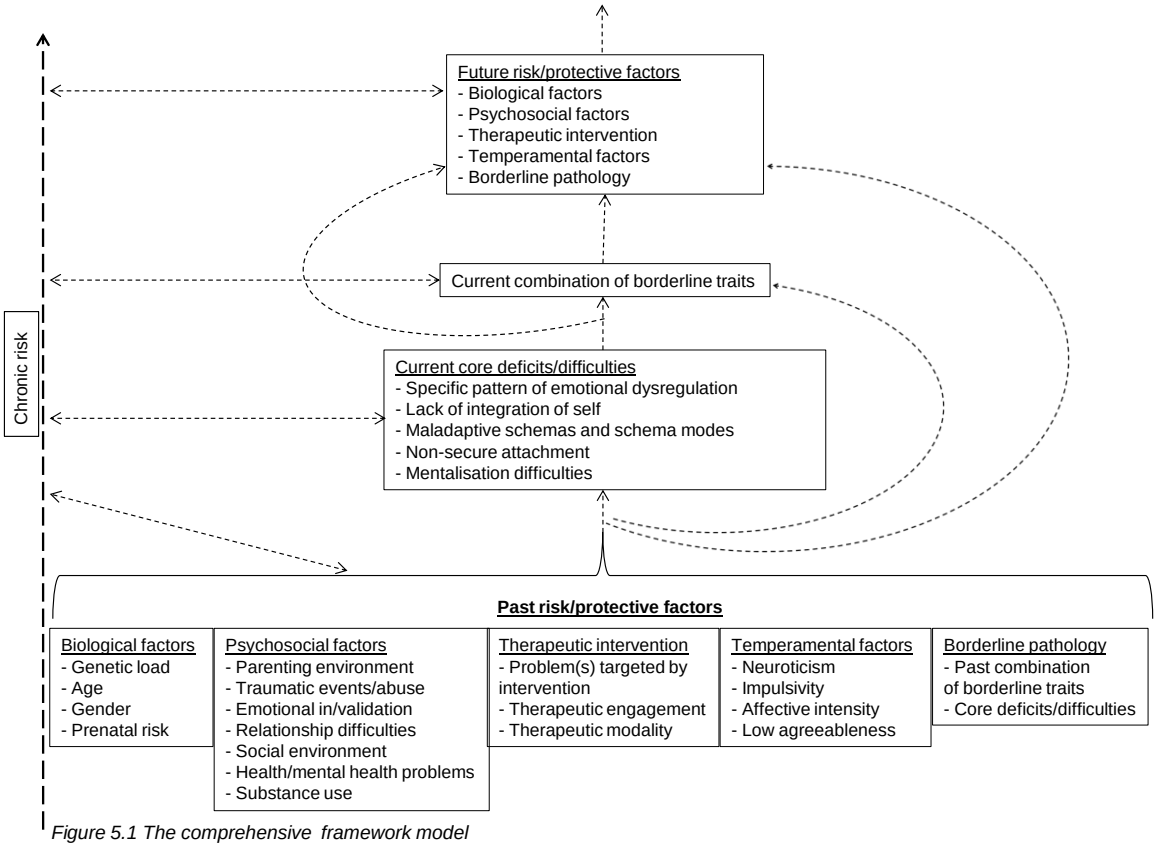

Figure 5.1 The comprehensive framework model

There are several features of the model that require exposition.

Past risk factors. A range of biopsychosocial risk factors are associated with BPD. Whilst some, such as a history of child abuse and a family history of the disorder are well recognised, the relationship between the risk factors remains unclear. It is considered probable the relationship between the factors is complex, with the relationships potentially being either correlational or causal. Furthermore, some risk factors, such as genetic load and experiencing poor parenting are expected to enjoy substantial overlap. It is likely that some of the relationships between factors include elements of transaction, interaction, mediation or moderation. In addition, it is posited that the nature of these relationships may change depending upon a given combination of factors – of particular note an individual's age is likely to have a significant influence upon the other relationships

present (particularly with regard to responses to psychosocial risk factors such as poor parenting). Given the research regarding the early emergence of borderline traits, it is likely that the pre-existence of borderline traits is in and of itself a risk factor for later borderline symptomatology (of either greater or lesser severity, depending upon the other factors present, in particular an individual's age). Indeed, as with most learning, the more an individual "practices" borderline traits and responds to situations in emotionally dysregulated ways, the more neurologically ingrained such ways of responding and being are likely to become. Whilst it is acknowledged that borderline traits correlate with difficulties across almost every area of life, only factors that could be reasonably presumed to play a significant role in the ongoing development of the disorder are noted.

With regard to parenting, it is considered likely that the overall parenting environment, rather than a unique feature of parenting by one parent is likely to be of importance. This recognises that many types of unhelpful parenting often cluster in the same home, thus impacting the overall environment. To this end it is posited that a set of parenting failures will distinguish BPD-genic parenting from parenting not leading to BPD (as distinct from the biosocial model, which emphasises the importance of one feature, emotional invalidation, being of primary importance). Second, the parenting should be considered as an environment, as opposed to a parent being regarded as a discrete entity. Indeed, as has previously been found with the development of personality disorders (Cheng et al., 2011), the combination of parenting received by an individual may also be of importance, with conflicted styles within the parenting team leading to elevated risk. Similarly, other research has found that parental failure associated with BPD often involves failures by both parents (Zanarini et al., 2000). Finally, with regard to modern child raising practices,

where some children spend extended periods of their early childhood in the care of those other than their biological parents, the parenting environment may be considered to extend beyond those who are a child's parents.

With regard to therapeutic intervention, this aspect is not typically included in models attempting to account for the naturalistic development of a psychopathology. However, its inclusion here is considered important, given that the problems inherent to BPD often lead to individuals having a fairly extensive engagement with psychiatric services, with this appearing to be more extensive than both healthy controls and individuals with mood and/or affective disorders (Ansell et al., 2007). Further, given the high levels of psychiatric comorbidity with other disorders (McGlashan et al., 2000), such interventions may be targeting psychopathology other than BPD, with the effects of the intervention on the borderline traits being incidental.

Current core deficits/difficulties. Several key deficits and difficulties are noted in the literature to underpin BPD, these are likely to mediate the relationship between historical risk factors and current borderline traits. It is also probable, as was found in the two studies, that such mediation may only be partial. Hence it is posited that even if these constructs are taken into account, that further symptomatology could be accounted for by the inclusion of the historical risk factors.

Future risk factors. Clinically it is observed that individuals with BPD often make very poor life choices, choosing to become involved in dysfunctional relationships, making decisions that place them in jeopardy of further trauma and evoke invalidating and punitive responses from peers. Borderline behaviour, particularly parasuicidal behaviour can often lead to further life difficulties. It is

therefore posited that future risk is a combination of current under-functioning and historical risk factors.

Chronic risk. It is also suggested that some distal historic risk factors may well continue to directly exert and influence behaviour in the distant future, not completely mediated by their capacity to increase the likelihood of immediate future risk factors. In particular, it is suggested having experienced poor parenting in early childhood (and the subsequent attachment difficulties) is likely to influence adult behaviour, even if one subsequently develops a more healthy attachment style.

Unceasing development. Finally, it is noted that this model does not consider BPD to have a clearly defined beginning or endpoint. As has previously been noted, drawing a distinction with regard to the end of the development of the disorder remains somewhat arbitrary. Some risk factors appear to predate birth (Schwarze et al., 2013; Winsper et al., 2012), whilst in a substantial proportion of individuals the disorder reoccurs even after it is judged to have gone into remission (Zanarini, Frankenburg, Reich, & Fitzmaurice, 2012). Consequently the model does not designate an endpoint, viewing the current disorder as a manifestation of antecedents and providing the basis of future difficulties, with future risk factors becoming in turn historic risk factors.

5.1 Differences Between the Comprehensive Framework and Biosocial Models

Relative to the biosocial model, this comprehensive framework model differs on several key predictions, which could be empirically tested. If the comprehensive

framework model is a more accurate aetiological account of borderline traits, some of the empirical findings that would be anticipated would include:

1. After taking into account childhood emotional vulnerability and an invalidating environment (notwithstanding the broad nature of this construct), other risk factors such as childhood trauma and relationship problems will predict substantial amounts of variation in the aspects of emotional dysregulation germane to BPD and also borderline traits.
2. After taking into account the aspects of emotional dysregulation germane to BPD, the other identified deficits and difficulties will be able to predict additional variation in borderline traits.
3. After taking into account aspects of emotional dysregulation germane to BPD and the other identified deficits and difficulties, historical risk factors (in particular past levels of borderline traits) will predict further variation in current levels of borderline traits.
4. The impact of psychosocial risk factors upon the key deficits and borderline functioning will vary according the age at which the psychosocial risk factor was introduced (rather than a broad age range of “childhood” being considered a critical period).
5. Parenting that leads to BPD (and related deficits) will be more complex than merely being invalidating parenting. Hence if a comprehensive measure of parenting associated with BPD is developed, this measure will be a superior predictor of borderline traits than a pure measure of invalidating parenting. Further, the best measure of a parenting environment leading to BPD will consider the interaction of various parenting styles (rather than the mean level of invalidation across parents being of primary importance).

6. Borderline traits and the associated deficits may be found in individuals who have not been exposed to significant levels of invalidation as a child (but who have experienced other risk factors as identified in the comprehensive framework model).

7. Current emotional vulnerability/neuroticism is likely to offer additional predictive power with regard to current borderline traits, even after childhood emotional vulnerability/neuroticism are taken into account.

8. Clinically, even if aspects of emotional dysregulation germane to BPD are normalised, borderline traits will continue to persist provided that the other key deficits remain present (i.e. emotional dysregulation is not viewed as being the primary deficit via which all risk is mediated).

Two primary criticisms can be made of the model that is presented. First, this model, whilst potentially accounting for a significant amount of the variation borderline traits, does not explain the process by which these traits develop. This is a purposeful feature of the model – attempting to explain the exact processes via which the dysfunction occurs is premature. As per the case of the biosocial model, an extensive explanation has been made to account for a process - despite the existence of this process not being supported by the empirical literature. Consequently it would be preferable to develop a comprehensive model of the disorder, based upon clinical findings, prior to attempting to develop accounts for the mechanisms by which these interactions occur.

The second criticism of the model is that certain factors of the model may be more or less important than others. This is likely to be the case. Indeed, the purpose of the model being proposed is to stimulate research into this area, in order to clarify

the important aetiological features of the disorder. In particular it is likely that some features of the disorder may depend on the individual's age; it is unlikely that some of the core deficits as identified in the model, in particular the capacity to mentalise and to have an integrated sense of self, would be important in younger children, despite these factors being potentially of critical importance to adults. Further, given that there are 256 possible combinations of symptoms that can lead to a diagnosis of BPD, it is considered likely that there may be numerous pathways to the disorder, depending upon the combination of symptoms present. Indeed, it is possible that some factors may be critical to the development of one of the 9 criteria, but of no import to the development of the other 8.

Section 6. Summation

The biosocial model is an important model of BPD, given that it underpins one of the main therapies for the treatment of the condition. However there is limited empirical literature to support its key assertions.

The two studies presented in this thesis failed to support several key aspects of the model, in particular that adult emotional dysregulation is due to an emotionally vulnerable child being exposed to an emotionally invalidating environment. A potentially more accurate model, the comprehensive framework model is presented, with it being argued that this offers a substantially more complete aetiology of the disorder.

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Appendices

Appendix A: Measures Used in Study 1

Demographics
1. What is your gender?
O Male
O Female

2. Which of the following best describes your hand preference?
O I am right handed
O I am left handed
O I am ambidextrous

3. How old are you?

4. Which of the following best describes your cultural background?
O Australian/New Zealander
O African
O Asian
O European
O Indian
O Middle Eastern
O North American
O Pacific Islander
O South American

5. How many years of education have you had in total? (e.g. primary school, high school, TAFE, university)

6. Which best describes your current marital status?
O Never married
O Married/De facto
O Separated
O Widowed
O Divorced

7. What best describes your current work status?
O Full time work
O Part time work
O Retired/Pensioner
O Home-maker
O Student
O Unemployed

8. What is your household income per year?
O $0 – $6,000
O $6,001 – $37,000
O $37,001 – $80,000
O $80,001 – $180,000
O $180,001 and over

Emotional Vulnerability–Child scale (EV-Child)
Below are some statements about your emotional style when you were a child. Please read each statement and rate how much it applied to you, when you were a child, using the following scale. Mark the appropriate response.

	Never	Almost never	Occasionally	Usually	Almost always	Always
1. My emotions tended to be more intense than those of most children.	O	O	O	O	O	O
2. When I got angry it was a very intense anger.	O	O	O	O	O	O
3. People who knew me would have said I was emotional.	O	O	O	O	O	O
4. Sad stories, TV shows, or movies deeply affected me.	O	O	O	O	O	O
5. When I felt sad, this emotion was very strong.	O	O	O	O	O	O
6. When I felt anxiety, it was a very strong feeling.	O	O	O	O	O	O
7. The sight of someone who was hurt affected me strongly.	O	O	O	O	O	O
8. People who knew me would have said that I got upset very easily.	O	O	O	O	O	O
9. If things didn't go my way, I got quite distressed.	O	O	O	O	O	O
10. People who knew me would have said that I was a tense or high-strung child.	O	O	O	O	O	O
11. Seeing something violent or scary in a book, TV show, or movie made me very upset.	O	O	O	O	O	O
12. Things that seemed minor to others caused strong negative emotions in me.	O	O	O	O	O	O
13. In scary situations, I got more scared than most other children.	O	O	O	O	O	O
14. When I felt guilty, this emotion was quite strong.	O	O	O	O	O	O
15. I was easily bothered by things that others just brushed off or ignored.	O	O	O	O	O	O
16. When I did something wrong, I had strong feelings of shame or guilt.	O	O	O	O	O	O
17. When I got upset, I stayed upset for quite a while.	O	O	O	O	O	O
18. When I felt nervous I got shaky all over.	O	O	O	O	O	O

	Never	Almost never	Occasionally	Usually	Almost always	Always
19. My negative emotions were long-lasting.	O	O	O	O	O	O
20. When I tried something new for the first time, I got shaky all over.	O	O	O	O	O	O
21. It took me a long time to calm down after getting upset about something.	O	O	O	O	O	O

Recalled Childhood Socialization of Emotion Scale (RCSES)

INSTRUCTIONS: In the following items, please indicate on a scale from 1 (very unlikely) to 7 (very likely) the likelihood that your primary caregiver (be they female or male; biological or step-parent or foster-parent) would have responded in the ways listed for each item. Please read each item carefully and respond as honestly and sincerely as you can. If the item never happened to you, try your best to recall a similar event and how your primary caregiver would have responded to the best of your recollection. For each response, please mark a number from 1-7. My primary caregiver as a child was (choose one)

O Biological Mother
O Biological Father
O Step-mother
O Step-father
O Foster-mother
O Foster-father
O Other

Scenario 3. If I lost some prized possession and reacted with tears, my caregiver would:

	1 Very unlikely	2	3	4 Medium	5	6	7 Very likely
a) get upset with me for being so careless and crying	O	O	O	O	O	O	O
b) tell me that I was over-reacting	O	O	O	O	O	O	O
c) help me to think of places I hadn't looked yet	O	O	O	O	O	O	O
d) distracted me by talking about happy things	O	O	O	O	O	O	O
e) tell me it's ok to cry when you feel unhappy	O	O	O	O	O	O	O
f) tell me that's what happens when you're not careful	O	O	O	O	O	O	O

Scenario 5. If I was going over to spend the afternoon at a friend's house and became nervous and upset because my caregiver couldn't stay there with me, my caregiver would:

	1 Very unlikely	2	3	4 Medium	5	6	7 Very likely
a) distract me by talking about all the fun I was going to have with my friend	O	O	O	O	O	O	O
b) help me think of things that I could do so that being at the friend's house without him or her wasn't scary (e.g. take a favorite book or toy with me)	O	O	O	O	O	O	O

	1 Very unlikely	2	3	4 Medium	5	6	7 Very likely
c) tell me to quit over-reacting and being a baby	O	O	O	O	O	O	O
d) tell me that if I didn't stop, that I wouldn't be allowed to go out anymore	O	O	O	O	O	O	O
e) feel upset and uncomfortable because of my reactions	O	O	O	O	O	O	O
f) encourage me to talk about my nervous feelings	O	O	O	O	O	O	O

Scenario 7. If I was about to appear in a recital or sports activity, and became visibly nervous about people watching me, my caregiver would:

	1 Very unlikely	2	3	4 Medium	5	6	7 Very likely
a) help me think of things that I could do to get ready for my turn (e.g. to do some warm-ups and not to look at the audience)	O	O	O	O	O	O	O
b) suggest that I think about something relaxing so that my nervousness would go away	O	O	O	O	O	O	O
c) remain calm and not get nervous herself / himself	O	O	O	O	O	O	O
d) tell me that I was being a baby about it	O	O	O	O	O	O	O
e) tell me that if I didn't calm down, we'd have to leave and go home right away	O	O	O	O	O	O	O
f) encourage me to talk about my nervous feelings	O	O	O	O	O	O	O

Scenario 9. If I was panicky and couldn't go to sleep after watching a scary TV show, my caregiver would:

	1 Very unlikely	2	3	4 Medium	5	6	7 Very likely
a) encourage me to talk about what scared me	O	O	O	O	O	O	O
b) get upset with me for being silly	O	O	O	O	O	O	O
c) tell me that I was over-reacting	O	O	O	O	O	O	O
d) help me think of something to do so that I could get to sleep (e.g. take a toy to bed, leave the lights on)	O	O	O	O	O	O	O
e) tell me to go to bed or I wouldn't be allowed to watch any more TV	O	O	O	O	O	O	O
f) do something fun with me to help me forget about what scared me	O	O	O	O	O	O	O

Scenario 10. If I was at a park and appeared on the verge of tears because the other children were being mean to me and wouldn't let me play with them, my caregiver would:

	1 Very unlikely	2	3	4 Medium	5	6	7 Very likely
a) NOT get upset herself / himself	O	O	O	O	O	O	O
b) tell me that if I started crying then we'd have to go home right away	O	O	O	O	O	O	O
c) tell me it's ok to cry when I feel bad	O	O	O	O	O	O	O
d) comfort me and try to get me to think about something happy	O	O	O	O	O	O	O
e) help me to think of something else to do	O	O	O	O	O	O	O
f) tell me that I would feel better soon	O	O	O	O	O	O	O

Scenario 12. If I was shy and scared around strangers and consistently became teary and wanted to stay in my bedroom whenever family friends came to visit, my caregiver would:

	1 Very unlikely	2	3	4 Medium	5	6	7 Very likely
a) help me think of things to do that would make meeting her / his friends less scary (e.g. to take a favorite toy with me when meeting the friends)	O	O	O	O	O	O	O
b) tell me that it is ok to feel nervous	O	O	O	O	O	O	O
c) try to make me happy by talking about the fun things we can do with the friends	O	O	O	O	O	O	O
d) feel upset and uncomfortable because of my reactions	O	O	O	O	O	O	O
e) tell me that I must stay in the living room and visit with the friends	O	O	O	O	O	O	O
f) tell me that I was being a baby	O	O	O	O	O	O	O

Difficulties in Emotion Regulation Scale (DERS)

Please indicate how often the following statements apply to you by writing the appropriate number from the scale below on the line beside each item:

	1 Almost never	2 Sometimes	3 About half the time	4 Most of the time	5 Almost always
1. I am clear about my feelings	O	O	O	O	O
2. I pay attention to how I feel	O	O	O	O	O
3. I experience my emotions as overwhelming and out of control	O	O	O	O	O
4. I have no idea how I am feeling	O	O	O	O	O
5. I have difficulty making sense out of my feelings	O	O	O	O	O
6. I am attentive to my feelings	O	O	O	O	O
7. I know exactly how I am feeling	O	O	O	O	O

	1 Almost never	2 Sometimes	3 About half the time	4 Most of the time	5 Almost always
8. I care about what I am feeling	O	O	O	O	O
9. I am confused about how I feel	O	O	O	O	O
10. When I'm upset I acknowledge my emotion	O	O	O	O	O
11. When I'm upset, I become angry with myself for feeling that way	O	O	O	O	O
12. When I'm upset, I become embarrassed for feeling that way	O	O	O	O	O
13. When I'm upset, I have difficulty getting work done	O	O	O	O	O
14. When I'm upset, I become out of control	O	O	O	O	O
15. When I'm upset I believe that I will remain that way for a long time	O	O	O	O	O
16. When I'm upset, I believe that I'll end up feeling very depressed	O	O	O	O	O
17. When I'm upset, I believe that my feelings are valid and important	O	O	O	O	O
18. When I'm upset, I have difficulty focusing on other things	O	O	O	O	O
19. When I'm upset, I feel out of control	O	O	O	O	O
20. When I'm upset, I can still get things done	O	O	O	O	O
21. When I'm upset, I feel ashamed with myself for feeling that way	O	O	O	O	O
22. When I'm upset, I know that I can find a way to eventually feel better	O	O	O	O	O
23. When I'm upset, I feel like I am weak	O	O	O	O	O
24. When I'm upset, I feel like I can remain in control of my behaviours	O	O	O	O	O
25. When I'm upset, I feel guilty for feeling that way	O	O	O	O	O
26. When I'm upset, I have difficulty concentrating	O	O	O	O	O
27. When I'm upset, I have difficulty controlling my behaviours	O	O	O	O	O
28. When I'm upset, I believe that there is nothing I can do to make myself feel better	O	O	O	O	O
29. When I'm upset, I become irritated with myself for feeling that way	O	O	O	O	O
30. When I'm upset, I start to feel very bad about myself	O	O	O	O	O

	1 Almost never	2 Sometimes	3 About half the time	4 Most of the time	5 Almost always
31. When I'm upset, I believe that wallowing in it is all I can do	O	O	O	O	O
32. When I'm upset, I lose control over my behaviours	O	O	O	O	O
33. When I'm upset, I have difficulty thinking about anything else	O	O	O	O	O
34. When I'm upset, I take time to figure out what I'm really feeling	O	O	O	O	O
35. When I'm upset, it takes me a long time to feel better	O	O	O	O	O
36. When I'm upset, my emotions feel overwhelming	O	O	O	O	O

Borderline Personality Questionnaire (BPQ)
Please mark the response that you feel best DESCRIBES YOUR USUAL SELF (for the past two years or longer) in relation to each statement. Mark True if you think the statement is true. Mark False if you think the statement is false. There are no right or wrong answers and there are no trick questions. Please respond as honestly as you can, but don't ponder too long over each item. Please answer every question, even though sometimes you may find it hard to decide.

	True	False
1. I often do things without thinking them through.	O	O
2. I often become depressed or anxious 'out of the blue'.	O	O
3. People often leave me.	O	O
4. I am rarely disappointed by my friends.	O	O
5. I feel inferior to other people.	O	O
6. I have threatened to hurt myself in the past.	O	O
7. I do not believe that I have the skills to do anything with my life.	O	O
8. I rarely get angry at other people.	O	O
9. Sometimes I feel like I am not real.	O	O
10. I will not have sex with someone unless I have known them for quite some time.	O	O
11. I sometimes feel anxious or irritable and become sad a few hours later.	O	O
12. When people close to me die or leave me, I feel abandoned.	O	O
13. I often exaggerate the potential of friendships only to find out later that they will not work out.	O	O
14. If I were more like other people I would feel better about myself.	O	O
15. I have deliberately tried to hurt myself without trying to kill myself.	O	O
16. In general, my life is pretty boring.	O	O
17. I frequently get into physical fights.	O	O
18. People are sometimes out to get me.	O	O
19. My friends have told me that my mood changes very quickly.	O	O
20. I am afraid to spend time alone.	O	O

	True	False
21. People who seem trustworthy often disappoint me.	O	O
22. I have made a suicide attempt in the past.	O	O
23. I often feel like I have nothing to offer others.	O	O
24. I have trouble controlling my temper.	O	O
25. I can read other people's minds.	O	O
26. I have tried 'hard' street drugs (e.g. cocaine, heroin).	O	O
27. My mood frequently alternates throughout the day between happiness, anger, anxiety and depression.	O	O
28. When my friends leave, I am confident I will see them again.	O	O
29. My friends often disappoint me.	O	O
30. I have cut myself on purpose.	O	O
31. I often feel lonely and deserted.	O	O
32. I have no difficulty controlling my temper.	O	O
33. I sometimes see or hear things that others cannot see or hear	O	O
34. It is not unusual for me to have sex on the first date.	O	O
35. I sometimes feel very sad but this feeling can change quickly.	O	O
36. People often let me down.	O	O
37. I wish I could be more like some of my friends.	O	O
38. I used to try to hurt myself to get attention.	O	O
39. I am often different with different people in different situations so that sometimes I am not sure who I am.	O	O
40. I easily become irritated by others.	O	O
41. Sometimes I can actually hear what other people are thinking.	O	O
42. I get high on drugs whenever I feel like it.	O	O
43. I rarely feel sad or anxious.	O	O
44. No one loves me.	O	O
45. When I trust people, they rarely disappoint me.	O	O
46. I feel that people would not like me if they really knew me well.	O	O
47. I get angry easily.	O	O
48. It is impossible to read others' minds.	O	O
49. I sometimes feel very happy but this feeling can change quickly.	O	O
50. I find it difficult to depend on others because they will not be there when I need them.	O	O
51. The relationships with people I care about have lots of ups and downs.	O	O
52. I feel comfortable acting like myself.	O	O
53. I have never made an attempt to hurt myself.	O	O
54. I rarely feel lonely.	O	O
55. I often find that the littlest things make me angry.	O	O
56. Sometimes I can't tell between what is real and what I have imagined.	O	O
57. When I drink, I drink too much.	O	O

	True	False
58. I consider myself to be a moody person.	O	O
59. I have difficulty developing close relationships because people often abandon me.	O	O
60. My friends are always there when I need them.	O	O
61. I wish I were someone else.	O	O
62. I feel like my life is not interesting.	O	O
63. When I am angry, I sometimes hit objects and break them.	O	O
64. I often receive speeding tickets.	O	O
65. I often feel like I am on an emotional 'roller coaster'.	O	O
66. I feel like my family has deserted me.	O	O
67. I am very comfortable with who I am.	O	O
68. I often do things impulsively.	O	O
69. My life is without purpose.	O	O
70. I am not sure what I want to do in the future.	O	O
71. At times I eat so much that I am in pain or have to force myself to throw up.	O	O
72. People tell me that I am a moody person.	O	O
73. The people I love often leave me.	O	O
74. In social situations, I often feel that others will see through me and realise that I don’t have much to offer.	O	O
75. I have been in the hospital for trying to harm myself.	O	O
76. I often feel empty inside.	O	O
77. Others often make me angry.	O	O
78. I often become frantic when I think that someone I care about will leave me.	O	O
79. I am confused about my long-term goals.	O	O
80. Others say I'm quick tempered.	O	O

Appendix B: Correlations between the DERS/BPQ subscales in Study 1

Appendix B
Correlation between BPQ and DERS subscales

	DERS_Non-Acceptance of Emotional Responses	DERS_Difficulties Engaging in Goal-Directed Behaviour	DERS_Impulse Control Difficulties	DERS_Lack of Emotional Awareness	DERS_Limited Access to Emotion Regulation Strategies	DERS_Lack of Emotional Clarity
BPQ_Impulsivity	.26**	.28**	.40**	.06	.31**	.20**
BPQ_Affective Instability	.50**	.58**	.67**	.01	.72**	.41**
BPQ_Abandonment	.43**	.40**	.60**	.01	.61**	.34**
BPQ_Relationships	.39**	.45**	.55**	-.07	.59**	.25**
BPQ_Self-Image	.52**	.51**	.54**	.14*	.63**	.46**
BPQ_Suicide/Self-Mutilation	.27**	.34**	.42**	.08	.40**	.26**
BPQ_Emptiness	.47**	.49**	.55**	.13*	.64**	.43**
BPQ_Intense Anger	.38**	.41**	.56**	.06	.54**	.29**
BPQ_Quasi-Psychotic States	.22**	.28**	.37**	-.07	.32**	.24**

*Note: *p < .05, **p < .01*

Appendix C: Measures Used in Study 2

Demographics

1. Is English your primary language?
O Yes
O No

2. What is your gender?
O Male
O Female

3. Which of the following best describes your hand preference?
O I am right handed
O I am left handed
O I am ambidextrous

4. How old are you?

5. Which of the following best describes your cultural background?
O African
O Asian
O Australian/New Zealander
O European
O Indian
O Middle Eastern
O North American
O Pacific Islander
O South American

6. How many years of education have you had in total? (e.g. primary school, high school, TAFE, community college, university)

7. Which best describes your current marital status?
O Never married
O Married/De facto
O Separated
O Widowed
O Divorced

8. What best describes your current work status?
O Full time work
O Part time work
O Retired/Pensioner
O Home-maker
O Student
O Unemployed

9. What is your household income per year? (in Australian dollars)
O $0 – $6,000
O $6,001 – $37,000
O $37,001 – $80,000
O $80,001 – $180,000
O $180,001 and over

10. Have you engaged in regular therapy (being therapy that occurred more frequently than once a month)?
O Yes
O No

10a. (only displayed to participants endorsing the preceding item) Thinking about the therapy that you received, please rate how helpful you found the therapy. If you have seen more than one therapist, please rate the therapy/therapist that you felt had the greatest impact upon you (this impact could be positive or negative). I felt that the therapy:
O Made me much worse
O Made me somewhat worse
O Neither made me better or worse
O Made me somewhat better
O Made me much better

Emotional Vulnerability–Child scale (EV-Child)
Below are some statements about your emotional style when you were a child. Please read each statement and rate how much it applied to you, when you were a child, using the following scale. Mark the appropriate response.

	Never	Almost Never	Occasionally	Usually	Almost always	Always
1. My emotions tended to be more intense than those of most children.	O	O	O	O	O	O
2. When I got angry it was a very intense anger.	O	O	O	O	O	O
3. People who knew me would have said I was emotional.	O	O	O	O	O	O
4. Sad stories, TV shows, or movies deeply affected me.	O	O	O	O	O	O
5. When I felt sad, this emotion was very strong.	O	O	O	O	O	O
6. When I felt anxiety, it was a very strong feeling.	O	O	O	O	O	O
7. The sight of someone who was hurt affected me strongly.	O	O	O	O	O	O
8. People who knew me would have said that I got upset very easily.	O	O	O	O	O	O
9. If things didn't go my way, I got quite distressed.	O	O	O	O	O	O
10. People who knew me would have said that I was a tense or high-strung child.	O	O	O	O	O	O
11. Seeing something violent or scary in a book, TV show, or movie made me very upset.	O	O	O	O	O	O
12. Things that seemed minor to others caused strong negative emotions in me.	O	O	O	O	O	O

	Never	Almost Never	Occasionally	Usually	Almost always	Always
13. In scary situations, I got more scared than most other children.	O	O	O	O	O	O
14. When I felt guilty, this emotion was quite strong.	O	O	O	O	O	O
15. I was easily bothered by things that others just brushed off or ignored.	O	O	O	O	O	O
16. When I did something wrong, I had strong feelings of shame or guilt.	O	O	O	O	O	O
17. When I got upset, I stayed upset for quite a while.	O	O	O	O	O	O
18. When I felt nervous I got shaky all over.	O	O	O	O	O	O
19. My negative emotions were long-lasting.	O	O	O	O	O	O
20. When I tried something new for the first time, I got shaky all over.	O	O	O	O	O	O
21. It took me a long time to calm down after getting upset about something.	O	O	O	O	O	O

Childhood Impulsivity

Below are some statements about how impulsive you were when you were a child. Please read each statement and rate how much it applied to you, when you were a child, using the following scale. Mark the appropriate response.

	Never	Almost Never	Occasionally	Usually	Almost always	Always
1. I did things without thinking	O	O	O	O	O	O
2. I acted on impulse	O	O	O	O	O	O
3. I acted on the spur of the moment	O	O	O	O	O	O
4. I did not pay attention	O	O	O	O	O	O
5. I got easily bored when solving thought problems	O	O	O	O	O	O
6. I liked to think carefully about things	O	O	O	O	O	O
7. I made up my mind quickly	O	O	O	O	O	O
8. I planned for my future	O	O	O	O	O	O
9. I planned my spare time	O	O	O	O	O	O
10. I planned what I had to do in advance	O	O	O	O	O	O
11. I was a quick thinker	O	O	O	O	O	O
12. I was impulsive	O	O	O	O	O	O

	Never	Almost Never	Occasionally	Usually	Almost always	Always
13. My thoughts raced too fast	O	O	O	O	O	O

Recalled Childhood Socialization of Emotion Scale (RCSES)
Please select from the list below your primary caregiver as a child. If you had a number of different primary carers throughout childhood select the one that you feel had the greatest impact upon you (this impact could be positive or negative). The questions following this one will be focusing upon this caregiver.
O Biological Mother
O Biological Father
O Step-mother
O Step-father
O Foster-mother
O Foster-father
O Other relative (not listed above)
O Other non-relative (not listed above)

INSTRUCTIONS: In the following items, please indicate on a scale from 1 (very unlikely) to 7 (very likely) the likelihood that your primary caregiver (be they female or male; biological or step-parent or foster-parent) would have responded in the ways listed for each item. Please read each item carefully and respond as honestly and sincerely as you can. If the item never happened to you, try your best to recall a similar event and how your primary caregiver would have responded to the best of your recollection. For each response, please mark a number from 1-7.

Scenario 3. If I lost some prized possession and reacted with tears, my caregiver would:

	1 Very unlikely	2	3	4 Medium	5	6	7 Very likely
a) get upset with me for being so careless and crying	O	O	O	O	O	O	O
b) tell me that I was over-reacting	O	O	O	O	O	O	O
c) help me to think of places I hadn't looked yet	O	O	O	O	O	O	O
d) distracted me by talking about happy things	O	O	O	O	O	O	O
e) tell me it's ok to cry when you feel unhappy	O	O	O	O	O	O	O
f) tell me that's what happens when you're not careful	O	O	O	O	O	O	O

Scenario 5. If I was going over to spend the afternoon at a friend's house and became nervous and upset because my caregiver couldn't stay there with me, my caregiver would:

	1 Very unlikely	2	3	4 Medium	5	6	7 Very likely
a) distract me by talking about all the fun I was going to have with my friend	O	O	O	O	O	O	O
b) help me think of things that I could do so that being at the friend's house without him or her wasn't scary (e.g. take a favorite book or toy with me)	O	O	O	O	O	O	O
c) tell me to quit over-reacting and being a baby	O	O	O	O	O	O	O

	1 Very unlikely	2	3	4 Medium	5	6	7 Very likely
d) tell me that if I didn't stop, that I wouldn't be allowed to go out anymore	O	O	O	O	O	O	O
e) feel upset and uncomfortable because of my reactions	O	O	O	O	O	O	O
f) encourage me to talk about my nervous feelings	O	O	O	O	O	O	O

Scenario 9. If I was panicky and couldn't go to sleep after watching a scary TV show, my caregiver would:

	1 Very unlikely	2	3	4 Medium	5	6	7 Very likely
a) encourage me to talk about what scared me	O	O	O	O	O	O	O
b) get upset with me for being silly	O	O	O	O	O	O	O
c) tell me that I was over-reacting	O	O	O	O	O	O	O
d) help me think of something to do so that I could get to sleep (e.g. take a toy to bed, leave the lights on)	O	O	O	O	O	O	O
e) tell me to go to bed or I wouldn't be allowed to watch any more TV	O	O	O	O	O	O	O
f) do something fun with me to help me forget about what scared me	O	O	O	O	O	O	O

Difficulties in Emotion Regulation Scale (DERS) with additional items

Please indicate how often the following statements apply to you by marking the appropriate number for each item:

	1 Almost never	2 Sometimes	3 About half the time	4 Most of the time	5 Almost always
1. When I'm upset, I look for someone to blame.	O	O	O	O	O
2. When I'm upset, someone is to blame.	O	O	O	O	O
3. When I'm upset, someone else is at fault.	O	O	O	O	O
4. When I'm upset, I end up feeling angry.	O	O	O	O	O
5. When I'm upset, I compare myself to others.	O	O	O	O	O
6. When I'm upset, I always notice how easy everyone else has it.	O	O	O	O	O
7. When I'm upset, I wish I could be more like other people.	O	O	O	O	O

	1 Almost never	2 Sometimes	3 About half the time	4 Most of the time	5 Almost always
8. When I'm upset, I think that others manage themselves better than I do.	O	O	O	O	O
9. When I'm upset, I over-intellectualise about the experience.	O	O	O	O	O
10. I use my intellect to avoid experiencing emotions.	O	O	O	O	O
11. When I'm upset, I tell myself that "there is no need to be emotional; you just need to be rational".	O	O	O	O	O
12. I avoid experiencing emotions by being overly rational.	O	O	O	O	O
13. When I'm upset, I don't want to change how I feel.	O	O	O	O	O
14. When I'm upset, I try to maintain the emotion I am feeling.	O	O	O	O	O
15. When I'm upset, there is no point to calming down.	O	O	O	O	O
16. When I'm upset, I try to stay that way.	O	O	O	O	O
17. When I'm upset, I am afraid of my emotions getting out of control.	O	O	O	O	O
18. When I'm upset, I will do almost anything to avoid the feeling.	O	O	O	O	O
19. I get scared of feeling upset.	O	O	O	O	O
20. Feeling a strong emotion frightens me.	O	O	O	O	O
21. When I'm upset, I am the only one to blame.	O	O	O	O	O
22. Being upset makes me feel worthless.	O	O	O	O	O
23. When I'm upset, I feel that I am not worth much for feeling that way.	O	O	O	O	O
24. When I'm upset, I think that only weak people feel the way I do.	O	O	O	O	O
25. When I'm upset, I try to shut down the feelings.	O	O	O	O	O
26. When I'm upset, I focus on pushing the feelings away.	O	O	O	O	O
27. When I'm upset, I try to block the feelings out.	O	O	O	O	O
28. When I'm upset, I try to force myself to feel differently.	O	O	O	O	O
29. When I'm upset, I spend a lot of time thinking about what upset me.	O	O	O	O	O

	1 Almost never	2 Sometimes	3 About half the time	4 Most of the time	5 Almost always
30. When I'm upset, I spend time thinking about things that have upset me in the past.	O	O	O	O	O
31. When I'm upset, I can't think of anything else but how upset I am.	O	O	O	O	O
32. When I'm upset, I spend a lot of time worrying about how I will cope in the future.	O	O	O	O	O
33. When I'm upset, I feel that I don't know who I am anymore.	O	O	O	O	O
34. When I'm upset, I start to question what this means about me.	O	O	O	O	O
35. When I'm upset, I feel uncertain about myself.	O	O	O	O	O
36. When I'm upset, I start to question who I am.	O	O	O	O	O
37. Being happy feels strange to me.	O	O	O	O	O
38. Feelings of intimacy make me feel uncomfortable.	O	O	O	O	O
39. I enjoy being angry.	O	O	O	O	O
40. I find that I enjoy being sad.	O	O	O	O	O
41. Being upset agrees with me in a funny sort of way.	O	O	O	O	O
42. I disregard my feelings.	O	O	O	O	O
43. When I'm upset, I tell myself that I should just get over it.	O	O	O	O	O
44. I find it hard to respect people who focus upon their feelings.	O	O	O	O	O
45. Feelings are unimportant to me.	O	O	O	O	O
46. Dealing with my emotions is simple.	O	O	O	O	O
47. I have complete control over my emotions.	O	O	O	O	O
48. I can cope with strong emotions with little effort.	O	O	O	O	O
49. I'm surprised when people can't manage their emotions.	O	O	O	O	O
50. When I'm upset, I need others to help me feel better.	O	O	O	O	O
51. I think that other people can influence my feelings more than I can.	O	O	O	O	O
52. When I'm upset, I have to rely on others to help me calm down.	O	O	O	O	O

	1 Almost never	2 Sometimes	3 About half the time	4 Most of the time	5 Almost always
53. I tend to stay upset unless people around me stop doing things that upset me.	O	O	O	O	O
54. I find it hard to tolerate being around others who are upset.	O	O	O	O	O
55. If someone near me is upset I find myself feeling like they do.	O	O	O	O	O
56. Being around people who are upset disrupts me from what I am doing.	O	O	O	O	O
57. If someone near me is upset I have trouble staying calm.	O	O	O	O	O
58. It is hard to see things from another's perspective if I am feeling emotional.	O	O	O	O	O
59. I am stubborn about my ideas when I am upset.	O	O	O	O	O
60. When I'm upset, I become even less likely to change my mind about something.	O	O	O	O	O
61. When I'm upset, I become even more certain of my beliefs.	O	O	O	O	O
62. It is important for me to make sure others can't tell how I am feeling inside.	O	O	O	O	O
63. When I'm upset, I hide my emotions from others.	O	O	O	O	O
64. I try to make sure that others can't tell when I am upset.	O	O	O	O	O
65. I feel uncomfortable expressing any emotion in front of others.	O	O	O	O	O
66. When I'm upset, I exaggerate how upset I feel.	O	O	O	O	O
67. I over-exaggerate to others how upset I am.	O	O	O	O	O
68. I overstate to others how upset I feel.	O	O	O	O	O
69. When I'm upset, I tend to become melodramatic.	O	O	O	O	O
70. When I'm upset, I feel guilty for feeling that way.	O	O	O	O	O
71. When I'm upset, I feel ashamed with myself for feeling that way.	O	O	O	O	O
72. When I'm upset, I become embarrassed for feeling that way.	O	O	O	O	O
73. When I'm upset, I become angry with myself for feeling that way.	O	O	O	O	O

	1 Almost never	2 Sometimes	3 About half the time	4 Most of the time	5 Almost always
74. When I'm upset, I become irritated with myself for feeling that way.	O	O	O	O	O
75. When I'm upset, I feel like I am weak.	O	O	O	O	O
76. When I'm upset, I have difficulty concentrating.	O	O	O	O	O
77. When I'm upset, I have difficulty focusing on other things.	O	O	O	O	O
78. When I'm upset, I have difficulty getting work done.	O	O	O	O	O
79. When I'm upset, I have difficulty thinking about anything else.	O	O	O	O	O
80. When I'm upset, I can still get things done.	O	O	O	O	O
81. When I'm upset, I lose control over my behaviors.	O	O	O	O	O
82. When I'm upset, I have difficulty controlling my behaviors.	O	O	O	O	O
83. When I'm upset, I become out of control.	O	O	O	O	O
84. When I'm upset, I feel out of control.	O	O	O	O	O
85. I experience my emotions as overwhelming and out of control.	O	O	O	O	O
86. When I'm upset, I feel like I can remain in control of my behaviors.	O	O	O	O	O
87. I am attentive to my feelings.	O	O	O	O	O
88. I pay attention to how I feel.	O	O	O	O	O
89. When I'm upset, I acknowledge my emotions.	O	O	O	O	O
90. When I'm upset, I believe that my feelings are valid and important.	O	O	O	O	O
91. I care about what I am feeling.	O	O	O	O	O
92. When I'm upset, I take time to figure out what I'm really feeling.	O	O	O	O	O
93. When I'm upset, I believe that I'll end up feeling very depressed.	O	O	O	O	O
94. When I'm upset, I believe that I will remain that way for a long time.	O	O	O	O	O
95. When I'm upset, I believe that wallowing in it is all I can do.	O	O	O	O	O
96. When I'm upset, it takes me a long time to feel better.	O	O	O	O	O

	1 Almost never	2 Sometimes	3 About half the time	4 Most of the time	5 Almost always
97. When I'm upset, I believe that there is nothing I can do to make myself feel better.	O	O	O	O	O
98. When I'm upset, I know that I can find a way to eventually feel better.	O	O	O	O	O
99. When I'm upset, my emotions feel overwhelming.	O	O	O	O	O
100. When I'm upset, I start to feel very bad about myself.	O	O	O	O	O
101. I have difficulty making sense out of my feelings.	O	O	O	O	O
102. I have no idea how I am feeling.	O	O	O	O	O
103. I am confused about how I feel.	O	O	O	O	O
104. I know exactly how I am feeling.	O	O	O	O	O
105. I am clear about my feelings.	O	O	O	O	O

Penn State Worry Questionnaire (PSWQ)

Instructions: Rate each of the following statements on a scale of 1 (“not at all typical of me”) to 5 (“very typical of me”).

	1 Not at all typical of me	2	3	4	5 Very typical of me
1. If I do not have enough time to do everything, I do not worry about it.	O	O	O	O	O
2. My worries overwhelm me.	O	O	O	O	O
3. I do not tend to worry about things.	O	O	O	O	O
4. Many situations make me worry.	O	O	O	O	O
5. I know I should not worry about things, but I just cannot help it.	O	O	O	O	O
6. When I am under pressure I worry a lot.	O	O	O	O	O
7. I am always worrying about something.	O	O	O	O	O
8. I find it easy to dismiss worrisome thoughts.	O	O	O	O	O
9. As soon as I finish one task, I start to worry about everything else I have to do.	O	O	O	O	O
10. I never worry about anything.	O	O	O	O	O
11. When there is nothing more I can do about a concern, I do not worry about it any more.	O	O	O	O	O
12. I have been a worrier all my life.	O	O	O	O	O
13. I notice that I have been worrying about things.	O	O	O	O	O
14. Once I start worrying, I cannot stop.	O	O	O	O	O
15. I worry all the time.	O	O	O	O	O
16. I worry about projects until they are all done.	O	O	O	O	O

Borderline Personality Questionnaire (BPQ)

Please mark the response that you feel best DESCRIBES YOUR USUAL SELF (for the past two years or longer) in relation to each statement. Mark True if you think the statement is true. Mark False if you think the statement is false. There are no right or wrong answers and there are no trick questions. Please respond as honestly as you can, but don't ponder too long over each item. Please answer every question, even though sometimes you may find it hard to decide.

	True	False
1. I often do things without thinking them through.	O	O
2. I often become depressed or anxious 'out of the blue'.	O	O
3. People often leave me.	O	O
4. I am rarely disappointed by my friends.	O	O
5. I feel inferior to other people.	O	O
6. I have threatened to hurt myself in the past.	O	O
7. I do not believe that I have the skills to do anything with my life.	O	O
8. I rarely get angry at other people.	O	O
9. Sometimes I feel like I am not real.	O	O
10. I will not have sex with someone unless I have known them for quite some time.	O	O
11. I sometimes feel anxious or irritable and become sad a few hours later.	O	O
12. When people close to me die or leave me, I feel abandoned.	O	O
13. I often exaggerate the potential of friendships only to find out later that they will not work out.	O	O
14. If I were more like other people I would feel better about myself.	O	O
15. I have deliberately tried to hurt myself without trying to kill myself.	O	O
16. In general, my life is pretty boring.	O	O
17. I frequently get into physical fights.	O	O
18. People are sometimes out to get me.	O	O
19. My friends have told me that my mood changes very quickly.	O	O
20. I am afraid to spend time alone.	O	O
21. People who seem trustworthy often disappoint me.	O	O
22. I have made a suicide attempt in the past.	O	O
23. I often feel like I have nothing to offer others.	O	O
24. I have trouble controlling my temper.	O	O
25. I can read other people's minds.	O	O
26. I have tried 'hard' street drugs (e.g. cocaine, heroin).	O	O
27. My mood frequently alternates throughout the day between happiness, anger, anxiety and depression.	O	O
28. When my friends leave, I am confident I will see them again.	O	O
29. My friends often disappoint me.	O	O
30. I have cut myself on purpose.	O	O
31. I often feel lonely and deserted.	O	O
32. I have no difficulty controlling my temper.	O	O
33. I sometimes see or hear things that others cannot see or hear	O	O
34. It is not unusual for me to have sex on the first date.	O	O

	True	False
35. I sometimes feel very sad but this feeling can change quickly.	O	O
36. People often let me down.	O	O
37. I wish I could be more like some of my friends.	O	O
38. I used to try to hurt myself to get attention.	O	O
39. I am often different with different people in different situations so that sometimes I am not sure who I am.	O	O
40. I easily become irritated by others.	O	O
41. Sometimes I can actually hear what other people are thinking.	O	O
42. I get high on drugs whenever I feel like it.	O	O
43. I rarely feel sad or anxious.	O	O
44. No one loves me.	O	O
45. When I trust people, they rarely disappoint me.	O	O
46. I feel that people would not like me if they really knew me well.	O	O
47. I get angry easily.	O	O
48. It is impossible to read others' minds.	O	O
49. I sometimes feel very happy but this feeling can change quickly.	O	O
50. I find it difficult to depend on others because they will not be there when I need them.	O	O
51. The relationships with people I care about have lots of ups and downs.	O	O
52. I feel comfortable acting like myself.	O	O
53. I have never made an attempt to hurt myself.	O	O
54. I rarely feel lonely.	O	O
55. I often find that the littlest things make me angry.	O	O
56. Sometimes I can't tell between what is real and what I have imagined.	O	O
57. When I drink, I drink too much.	O	O
58. I consider myself to be a moody person.	O	O
59. I have difficulty developing close relationships because people often abandon me.	O	O
60. My friends are always there when I need them.	O	O
61. I wish I were someone else.	O	O
62. I feel like my life is not interesting.	O	O
63. When I am angry, I sometimes hit objects and break them.	O	O
64. I often receive speeding tickets.	O	O
65. I often feel like I am on an emotional 'roller coaster'.	O	O
66. I feel like my family has deserted me.	O	O
67. I am very comfortable with who I am.	O	O
68. I often do things impulsively.	O	O
69. My life is without purpose.	O	O
70. I am not sure what I want to do in the future.	O	O
71. At times I eat so much that I am in pain or have to force myself to throw up.	O	O

	True	False
72. People tell me that I am a moody person.	O	O
73. The people I love often leave me.	O	O
74. In social situations, I often feel that others will see through me and realise that I don't have much to offer.	O	O
75. I have been in the hospital for trying to harm myself.	O	O
76. I often feel empty inside.	O	O
77. Others often make me angry.	O	O
78. I often become frantic when I think that someone I care about will leave me.	O	O
79. I am confused about my long-term goals.	O	O
80. Others say I'm quick tempered.	O	O

Borderline Personality Questionnaire (BPQ) – current focus

Please mark the response that you feel best describes you (just in the past two years). Mark True if you think the statement is true. Mark False if you think the statement is false. There are no right or wrong answers and there are no trick questions. Please respond as honestly as you can, but don't ponder too long over each item. Please answer every question, even though sometimes you may find it hard to decide.

	True	False
6a. In the past two years I have threatened to hurt myself.	O	O
15a. In the past two years I have deliberately tried to hurt myself without trying to kill myself.	O	O
22a. In the past two years I have made a suicide attempt.	O	O
26a. In the past two years I have tried 'hard' street drugs (e.g. cocaine, heroin).	O	O
30a. In the past two years I have cut myself on purpose.	O	O
38a. In the past two years I have tried to hurt myself to get attention.	O	O
75a. In the past two years I have been in the hospital for trying to harm myself.	O	O
53a. In the past two years I have NOT made an attempt to hurt myself.	O	O

Appendix D: Final Ethics Approval Letters

MACQUARIE UNIVERSITY

DUNCAN GILL <duncan.gill@students.mq.edu.au>

Approved- Ethics application -Warburton (Ref: 5201200025)

1 message

Ethics Secretariat <ethics.secretariat@mq.edu.au> 9 March 2012 at 15:00
To: Dr Wayne Warburton <wayne.warburton@mq.edu.au>
Cc: Duncan James Gill <Duncan.Gill@students.mq.edu.au>

Dear Dr Warburton

Re: "An investigation into the biosocial theory and the development of borderline personality traits" (Ethics Ref: 5201200025)

Thank you for your recent correspondence. Your response has addressed the issues raised by the Human Research Ethics Committee and you may now commence your research.

The following personnel are authorised to conduct this research:

Chief Investigator- Dr Wayne Warburton

Co-Investigator- Mr Duncan James Gill

NB. STUDENTS: IT IS YOUR RESPONSIBILITY TO KEEP A COPY OF THIS APPROVAL EMAIL TO SUBMIT WITH YOUR THESIS.

Please note the following standard requirements of approval:

1. The approval of this project is conditional upon your continuing compliance with the National Statement on Ethical Conduct in Human Research (2007).

2. Approval will be for a period of five (5) years subject to the provision of annual reports. Your first progress report is due on 09 March 2013.

If you complete the work earlier than you had planned you must submit a Final Report as soon as the work is completed. If the project has been discontinued or not commenced for any reason, you are also required to submit a Final Report for the project.

Progress reports and Final Reports are available at the following website:

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/forms

3. If the project has run for more than five (5) years you cannot renew approval for the project. You will need to complete and submit a Final Report and submit a new application for the project. (The five year limit on renewal of approvals allows the Committee to fully re-review research in an environment where legislation, guidelines and requirements are continually changing, for example, new child protection and privacy laws).

4. All amendments to the project must be reviewed and approved by the Committee before implementation. Please complete and submit a Request for Amendment Form available at the following website:

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/forms

5. Please notify the Committee immediately in the event of any adverse effects on participants or of any unforeseen events that affect the continued ethical acceptability of the project.

6. At all times you are responsible for the ethical conduct of your research in accordance with the guidelines established by the University. This information is available at the following websites:

http://www.mq.edu.au/policy/

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/policy

If you will be applying for or have applied for internal or external funding for the above project it is your responsibility to provide the Macquarie University's Research Grants Management Assistant with a copy of this email as soon as possible. Internal and External funding agencies will not be informed that you have final approval for your project and funds will not be released until the Research Grants Management Assistant has received a copy of this email.

If you need to provide a hard copy letter of Final Approval to an external organisation as evidence that you have Final Approval, please do not hesitate to contact the Ethics Secretariat at the address below.

Please retain a copy of this email as this is your official notification of final ethics approval.

Yours sincerely
Dr Karolyn White
Director of Research Ethics
Chair, Human Research Ethics Committee

MACQUARIE UNIVERSITY

DUNCAN GILL <duncan.gill@students.mq.edu.au>

Approved- Ethics application- Warburton (Ref No: 5201300082)

1 message

Ethics Secretariat <ethics.secretariat@mq.edu.au> 11 April 2013 at 09:29
To: Dr Wayne Warburton <wayne.warburton@mq.edu.au>
Cc: Mr Duncan James Gill <duncan.gill@students.mq.edu.au>

Dear Dr Warburton

Re: "Investigating the specificity of the biosocial model of borderline traits" (Ethics Ref: 5201300082)

Thank you for your recent correspondence. Your response has addressed the issues raised by the Human Research Ethics Committee and you may now commence your research.

This research meets the requirements of the National Statement on Ethical Conduct in Human Research (2007). The National Statement is available at the following web site:

http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/e72.pdf.

The following personnel are authorised to conduct this research:

Dr Wayne Warburton
Mr Duncan James Gill

NB. STUDENTS: IT IS YOUR RESPONSIBILITY TO KEEP A COPY OF THIS APPROVAL EMAIL TO SUBMIT WITH YOUR THESIS.

Please note the following standard requirements of approval:

1. The approval of this project is conditional upon your continuing compliance with the National Statement on Ethical Conduct in Human Research (2007).

2. Approval will be for a period of five (5) years subject to the provision of annual reports.

Progress Report 1 Due: 11 April 2014
Progress Report 2 Due: 11 April 2015
Progress Report 3 Due: 11 April 2016
Progress Report 4 Due: 11 April 2017
Final Report Due: 11 April 2018

NB. If you complete the work earlier than you had planned you must submit a Final Report as soon as the work is completed. If the project has been discontinued or not commenced for any reason, you are also required to submit a Final Report for the project.

Progress reports and Final Reports are available at the following website:

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/forms

3. If the project has run for more than five (5) years you cannot renew approval for the project. You will need to complete and submit a Final

Report and submit a new application for the project. (The five year limit on renewal of approvals allows the Committee to fully re-review research in an environment where legislation, guidelines and requirements are continually changing, for example, new child protection and privacy laws).

4. All amendments to the project must be reviewed and approved by the Committee before implementation. Please complete and submit a Request for Amendment Form available at the following website:

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/forms

5. Please notify the Committee immediately in the event of any adverse effects on participants or of any unforeseen events that affect the continued ethical acceptability of the project.

6. At all times you are responsible for the ethical conduct of your research in accordance with the guidelines established by the University. This information is available at the following websites:

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If you will be applying for or have applied for internal or external funding for the above project it is your responsibility to provide the Macquarie University's Research Grants Management Assistant with a copy of this email as soon as possible. Internal and External funding agencies will not be informed that you have final approval for your project and funds will not be released until the Research Grants Management Assistant has received a copy of this email.

Please retain a copy of this email as this is your official notification of final ethics approval.

Yours sincerely
Dr Karolyn White
Director of Research Ethics
Chair, Human Research Ethics Committee