Stability over Time and the Role of Attachment in Emerging Personality Disorder in Adolescence:

A Two Year Longitudinal Study.

Tessa Crombie

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Thesis declaration form

I confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

Signature:

Name:

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Overview

The overall focus of the thesis is on Personality Disorders (PD) and the factors that influence the development and maintenance of these. This thesis consists of three parts.

Part one presents a systematic literature review on the relationship between childhood emotional abuse and emotional neglect and Borderline Personality Disorder (BPD). Although existing research evidences the link between childhood trauma and BPD, no systematic review has considered the specific impact of emotional abuse and emotional neglect as opposed to sexual or physical abuse. Evidence of variables that may account for or contribute to the relationship is considered.

Part two is an empirical paper on the stability of PDs in adolescence over a two year period and the role that attachment plays in the maintenance of PD traits during this time. The results showed that PD traits in adolescence decline over time to an extent. Higher levels of overall quality of attachment and lower levels of alienation from peers, as measured at baseline, were predictive of improvement in the number of PD traits over time. The validity of the results is discussed in relation to problems of sample size and statistical power. The data collection for this study was conducted jointly with another trainee and in conjunction with a trial into Mentalization-Based Therapy (MBT) for adolescents with emerging PD.

Part three is a critical appraisal discussing reflections on issues that arose during the process of the research and commenting more generally on current debates within the field of PD research.

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Part 1: Literature Review

A Systematic Literature Review into the Relationship between Childhood Emotional Abuse and Emotional Neglect and Borderline Personality Disorder.

Abstract

Aims

This review aimed to evaluate literature concerning the relationship between childhood emotional abuse and emotional neglect and Borderline Personality Disorder (BPD) and to explore factors that account for or contribute to this relationship.

Method

A systematic literature search was conducted using the databases PsychINFO, MEDLINE, and EMBASE. Once exclusion criteria were applied, 39 studies were identified as appropriate for the review.

Results

Results showed higher levels of emotional abuse and neglect in BPD samples compared to healthy, Axis I and other Axis II controls. Studies highlighted changes in brain structure and psychological processes as potentially accounting for this relationship. Contributing factors were demographic variables, traits and other types of abuse. Evidence suggested that emotional abuse and neglect might be more significant in the development of BPD than other forms of abuse.

Conclusions

The current literature supports the link between emotional abuse and neglect and BPD. However, there is a lack of specificity to this relationship, with levels of abuse being higher across all clinical groups. Further research is needed to improve our understanding of the interaction between the relevant factors involved.

Introduction

Over recent years there has been a wealth of research considering the links between negative experiences in childhood and the development of Borderline Personality Disorder (BPD) in later life. This research has often considered childhood trauma in general or has focused specifically on the impact of sexual and/or physical abuse. Although a number of studies have looked more in depth at the impact of emotional abuse and emotional neglect on the development of BPD, as yet, there has been no systematic review in this area. Therefore, the current systematic review seeks to summarise the research to date on the specific impact of emotional abuse and/or emotional neglect on the development of BPD.

In the UK, the Department for Children, Schools and Families (DCSF) defined emotional abuse as "the persistent emotional maltreatment of a child such as to cause severe and persistent adverse effects on the child's emotional development" (DCSF, 2010, p. 38). This includes conveying to children that they are worthless or unloved, ridiculing them, not giving the child opportunities to express themselves, overprotecting them to the extent their learning is limited, or placing developmentally inappropriate expectations on them (DCSF, 2010). The DCSF (2010) states that emotional abuse is involved in all types of childhood maltreatment, but can also occur in isolation. Neglect is defined as "the persistent failure to meet a child's basic physical and/or psychological needs" (DCSF, 2010, p. 39). Emotional neglect includes failure to protect the child from emotional harm and unresponsiveness or neglect of the child's basic emotional needs (DCSF, 2010). Glaser (2002) further defined emotional abuse and neglect as being emotionally unavailable or unresponsive to the child, failing to recognise the child's boundaries and individuality, using the child for fulfilment of the parent's own psychological

needs and failing to promote social adaptation. An NSPCC survey conducted in 2000 found that, from a nationally representative sample of young people, 12% reported experiencing three or more forms of emotional abuse (Cawson, Wattam, Brooker & Kelly, 2000).

This, as well as other forms of childhood maltreatment, has often been linked with the development of BPD. BPD is a pervasive disorder characterised by emotional dysregulation, impulsivity and disrupted interpersonal functioning (American Psychiatric Association, 2001; Carlson, Egeland & Sroufe, 2009; Chanen & Kaess, 2012; Fonagy & Luyten, 2009). Common experiences of those with BPD are disorganised attachment patterns, an intense fear of abandonment, the inability to tolerate strong emotions and a disturbed sense of identity (Chanen & Kaess, 2012; Fonagy & Luyten, 2009). The prevalence of BPD in the general population is around 0.7% to 2.7%, rising to 20% prevalence amongst psychiatric outpatients and 40% with inpatients (Chanen & Kaess, 2012).

BPD and Childhood Trauma in General

Chanen and Kaess (2012) reviewed current research on the developmental pathways of BPD and stated that strong associations have been found between BPD and childhood trauma in both clinical and nationally representative samples. Research into the development of BPD generally takes one of two forms; either looking at the impact of a range of cumulative aversive events and how the volume of these contributes to the development of difficulties, or considering a specific type of adversity (e.g. child sexual abuse) and assessing the extent to which this triggers difficulties. Chanen and Kaess (2012) refer mainly to research conducted by the Children in the Community (CIC) study, which focuses on cumulative events. This is a large prospective study considering risk factors in the development of personality

disorders and other mental health diagnoses. This research has found increased levels of personality disorder traits in those with histories of childhood abuse and neglect. However, numerous other environmental risk factors, such as low socioeconomic status, parental illness and parental education, have also been identified, making it difficult to assess the unique impact of childhood trauma (Chanen & Kaess, 2012; Cohen, Crawford, Johnson & Kasen, 2005; Widom, Czaja & Paris, 2009). Many of the published findings from the CIC studies have been included in the systematic review below.

Ball and Links (2009) applied the Hill's Criteria of Causation (Hill, 1984)[1] to the current research into the relationship between BPD and child abuse. They concluded that there was sufficient evidence to satisfy most of the Hill's Criteria for Causation. However, they commented on the lack of evidence for specificity of the relationship, as not all those that have experienced trauma will go on to develop BPD and not all those with BPD will have experienced trauma. They raised the issue of distinguishing the individual impact of co-morbid factors and stressed the need for further research to develop a multifactorial model of the emergence of BPD, particularly focusing on gene-environment interactions and mediating variables (Balls & Links, 2009).

Given this lack of specificity, a developmental psychopathology perspective is appropriate here (Shiner, 2009). This considers development of normal and abnormal functioning across the life span and uses multiple levels of analysis (e.g. biological, psychological, social and the interaction between these) to establish risk and protective factors contributing to adaptation and maladaptation (Cicchetti, 2006).

[1]Hill's Criteria of Causation (Hill, 1984): relationship strength, temporality (causal variable occurs before outcome variable), dose-response (as causal variable increases, outcome variable increases), specificity (specific relationship between variables), consistency (found across studies), epidemiologic and biologic plausibility and analogy (the relationship is analogous to other causal relationships).

Key concepts in developmental psychopathology are equifinality and multifinality. Cicchetti (2006) defines equifinality as a diverse range of pathways leading to the same outcome. Therefore a number of different upbringings could result in the same disorder. Conversely, multifinality states that the impact of a component may be different depending on the system in which it operates, i.e. an adverse event will not necessarily lead to the same outcome in every individual, but can lead to a range of different disorders or the absence of disorder (Cicchetti, 2006). When considering protective factors, research has demonstrated that children can be very resilient to adversities in childhood and more exploration of this in relation to the development of BPD is needed (McGloin & Widom, 2001; Paris, 2003; 2007).

With this in mind, other researchers have contrasting views about the relative importance of childhood trauma in the aetiology of BPD. Paris (2007) stated that most people who experience abuse do not develop BPD or any other mental disorder and one third of BPD patients do not report experiencing abuse, while another third only report one-off, isolated incidents with perhaps minimal clinical relevance. He argued that the inconsistency is too great and therefore there is little evidence of any specific relationship between trauma and the symptoms of BPD (Paris 2003; 2007). Indeed, aversive childhood experiences have been implicated in a wide range of other Axis I and Axis II disorders (Cohen, Brown & Smailes, 2001). Recent systematic reviews and meta-analysis have demonstrated links between child abuse and psychosis (Read & Bentall, 2012; Skehan, Larkin & Read, 2012), bipolar disorder (Daruy-Filho, Brietzke, Lafer, & Grassi-Oliveira, 2011) and depression (Nanni, Uher & Danese, 2012). These associations with other disorders clearly highlight the lack of specificity and the multifinality of childhood trauma and mental health and the need for a richer understanding of how trauma contributes to the

development of mental health difficulties and whether certain types or aspects of the trauma are more predictive of particular disorders (Keyes et al., 2012).

The biological component of BPD also needs to be considered when exploring the relative influence of any childhood trauma in the development of BPD. Twin studies have shown that heritability can account for around half the variance seen in BPD and BPD symptoms of affective instability, self-harm and cognitive deficits have been shown to have genetic components (Paris, 2011; 2007; Posner et al., 2003). Paris (2007) hypothesised that the individual inherits a genetic pre-disposition in the form of a certain temperament or trait associated with BPD (e.g. emotional negativity, impulsivity) which then shapes how the individual responds to childhood traumas, contributing to the emergence or absence of BPD symptoms in later life. Temperament could shape the extent to which the individual experiences childhood adversity e.g. those who are more disinhibited may engage in more risk-taking behaviour in adolescence, thus exposing them to more potential adverse situations (Paris, 2007). Therefore, developing a greater understanding of this gene-environment interaction and the specific genes involved is key (Chanen & Kaess, 2012; Paris, 2011). When these aspects are considered the link between childhood trauma and BPD becomes a more complex one, with a multitude of interacting factors, for which continuing research is needed (Lenzenweger and Cicchetti, 2005).

BPD and Emotional Abuse and/or Emotional Neglect

The research and theories above have focused on all types of childhood trauma in combination rather than looking at the impact of emotional abuse or neglect in isolation, which is what the current review seeks to do. A number of theorists point to the key importance of neglect or emotional abuse in the development of BPD, therefore highlighting the need to understand further this

relationship in isolation from other forms of abuse. Fonagy and Luyten (2009) propose that it is early emotional neglect which might be crucial in predisposing an individual to developing BPD rather than physical or sexual abuse. Deficits in the ability to mentalize, that is "to perceive and interpret human behaviour in terms of intentional mental states" (Fonagy & Luyten, 2009, p. 1357), are now seen as a fundamental element in BPD. They hypothesise that growing up in an emotionally abusive or neglectful environment, in which the discussion and validation of mental states is absent, impacts on the development of the individual's ability to mentalize, thus predisposing them to BPD (Fonagy, 2000; Fonagy & Luyten, 2009).

This converges with Linehan's (1993) biosocial theory of the development of BPD, which postulates that growing up in an invalidating environment, where the child does not learn how to understand, regulate or tolerate their emotions, leads to emotional dysregulation, another hallmark of BPD. Crowell, Beauchaine and Linehan (2009) state that in an invalidating environment the child may often need more extreme displays of emotion to gain response from caregivers, potentially creating intermittent reinforcement of extreme emotional outbursts, such as those often witnessed in clients with BPD.

Both Fonagy and Luyten (2009) and Linehan (2009) stress the importance of secure attachment in allowing the child to develop the ability to mentalize and regulate their own emotions. In an environment that is invalidating and emotionally abusive, an insecure and disorganised attachment pattern is likely to develop (Fonagy, 2000; Fonagy & Luyten, 2009), with the child having no safe base from which to learn these skills, potentially leading to an increased likelihood of developing BPD later in life.

With these theories in mind, developing a greater understanding of the relationship between emotional abuse and emotional neglect and BPD is important to provide evidence support or refute these. One of the key difficulties is that emotional abuse and neglect often occur together with other forms of child abuse and more general adverse life circumstances, making it hard to identify the individual impact of this form of abuse (Chanen & Kaess, 2012). The extent to which the current literature attempts to make these distinctions will be considered.

Summary

In summary, over the past 20 years a body of evidence has emerged linking BPD and childhood trauma. However, there are still many discrepancies or unknowns within this relationship, with many who experience childhood trauma not developing BPD and not everyone with a diagnosis of BPD having a history of childhood adversity. There is also a lack of specificity with childhood trauma being associated with the development of a wide range of Axis I and II disorders. Therefore, more research is required in order to gain a fuller understanding of the nature of the relationship between childhood trauma and BPD and its potential causative impact (Lenzenweger and Cicchetti, 2005).

The previous reviews in this area have focused on looking at the experience of childhood trauma in general or specifically at physical or sexual abuse. Surprisingly, there is relatively limited data on the unique impact of emotional abuse and emotional neglect and BPD and factors that mediate or moderate this. No systematic literature review has been conducted in this area, despite leading theorists highlighting emotional abuse and emotional neglect as potential crucial factors leading to the development of key deficits witnessed in BPD (Crowell et al., 2009;

Fonagy & Luyten, 2009; Linehan, 1993). The current review hoped to rectify this gap by consolidating findings from research considering this relationship.

Aims

The current review aimed to systematically identify and appraise research which focuses on the impact of emotional abuse and emotional neglect on BPD. Further to this, it sought to draw together findings on the potential mediating and moderating variables within this relationship. The three research questions were:

1. Is there a relationship between BPD and childhood emotional abuse and neglect?
2. If so, what are the factors that might explain and/or mediate this relationship?
3. If so, what are the factors that contribute to and moderate this relationship?

Method

Systematic Literature Search

To conduct this systematic review the following procedure was employed. Firstly, appropriate electronic databases were identified as being PsychINFO, MEDLINE and EMBASE. PsychINFO focuses primarily of psychological literature and related disciplines. MEDLINE covers medicine, nursing, dentistry, the health care system and preclinical sciences. EMBASE consists of bio-medical literature and pharmacological literature. Combining these three databases ensured that approximately10,000 journals were included in the search. Search terms used were 'Borderline Personality dysfunct*' OR 'Borderline personality disorder*' AND 'neglect*' OR 'emotion* abuse' OR 'maltreatment'. In addition the subject headings of 'Borderline Personality Disorder' AND 'Child Neglect' OR 'Emotional Abuse' were used when possible. The results were restricted to English language, peer reviewed journals (only available on PsychINFO) and studies published from 2000

onwards. The initial search resulted in 469 studies, which was reduced to 298 once duplicates were removed. A breakdown of the stages is listed in Table 1.

Table 1
Breakdown of Search Strategy and Results

Database	Search Terms	Exclusion Criteria	Results
PsychINFO	Borderline Personality dysfunct* or Borderline personality disorder* AND neglect* or emotion* abuse or maltreatment and subject heading terms: Borderline Personality Disorder Child Neglect Emotional Abuse	Peer reviewed journal, English language, from 2000 *Duplicates removed*	109 *32*
MEDLINE	*As above*	English language, from 2000 *Duplicates removed*	209 *121*
EMBASE	*As above*	English language, from 2000 *Duplicates removed*	151 *145*
		Total *Duplicates removed*	469 *298*

The following initial exclusion criteria were used to screen the titles and abstracts:

1) Clearly irrelevant (i.e. study not on BPD or trauma)
2) No systematic measure of BPD specified
3) No systematic measure of childhood trauma specified
4) Review or theoretical papers (retained for introduction/discussion if relevant)
5) Single Case Studies

From this, 96 studies remained and their full texts were obtained. Additional hand searching was carried out in the Journal of Personality Disorders, American Journal of Psychiatry, Journal of American Academy of Child and Adolescent Psychiatry and Development and Psychopathology, as these were deemed prominent journals in the field. This produced three extra papers for inclusion. The Consort diagram in Figure 1 gives a summary of this procedure.

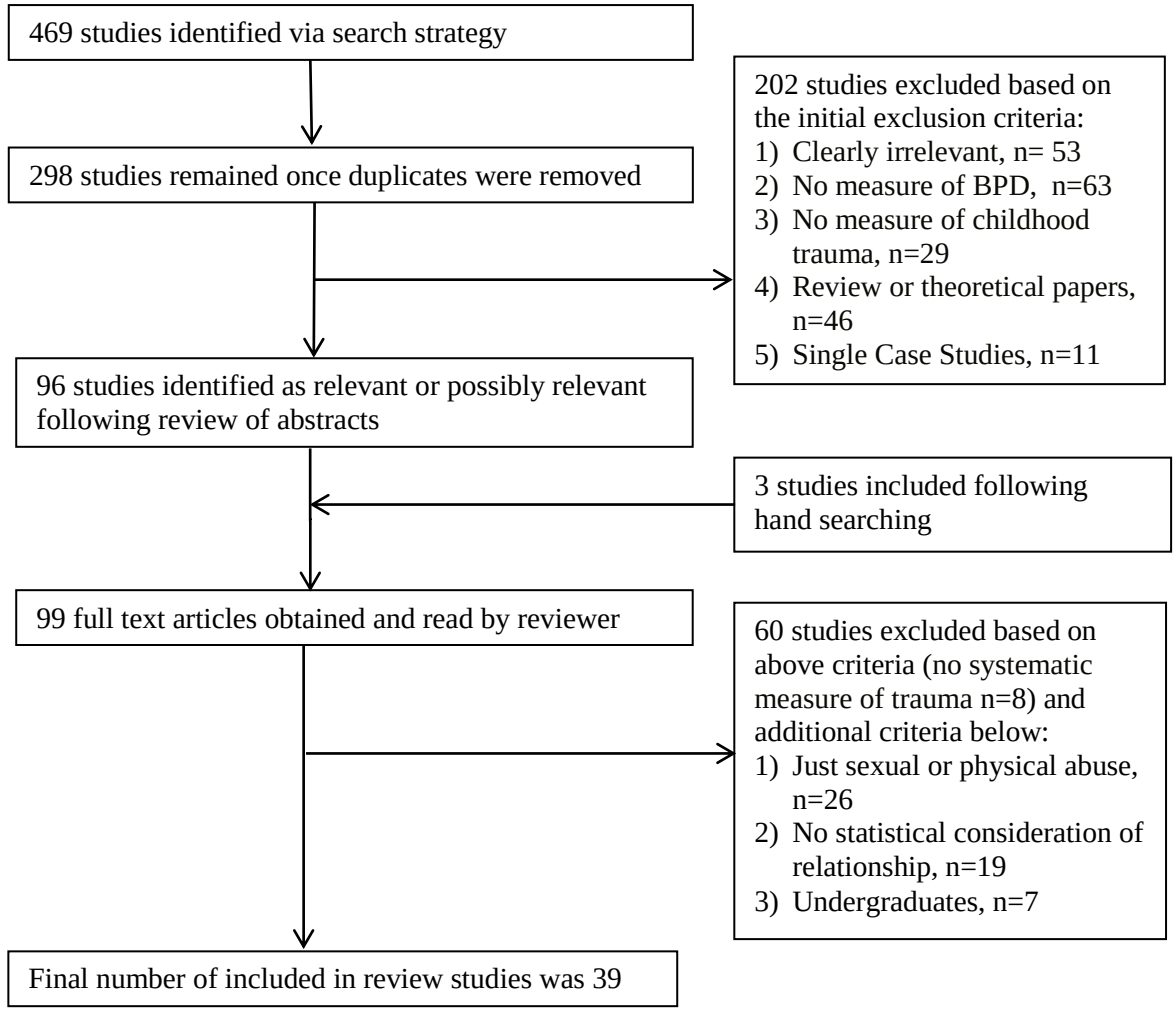

Figure 1. Consort diagram of search procedure

The 99 articles were then reviewed using both the criteria above and these additional exclusion criteria:

1) Studies focusing solely on childhood sexual abuse or physical abuse
2) Studies that did not statistically consider the relationship between BPD and emotional abuse and/or emotional neglect and/or a composite childhood trauma score
3) Studies on undergraduate populations (viewed as unrepresentative of the population in question)

This resulted in a further 60 articles being excluded, as detailed in Figure 1 above. The remaining 39 articles met all inclusion criteria for the study and were included in

the review. The reason for having this two stage exclusion process, with more stringent criteria at the second stage, was to ensure that no potentially suitable studies were excluded at the initial stages prior to full text review.

Data extraction and Study analysis

Following the systematic search, relevant data was extracted from the studies, including study design, the nature of participants, sample sizes, method of identifying BPD and childhood trauma, levels of trauma or BPD within the sample and relevant statistics pertaining to the relationship between trauma and BPD. The quality of the studies was assessed using the questions outlined by Young and Solomon (2009) for critically appraising research (see Appendix A) and the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement checklist for study reporting (Von Elm, Altman, Egger, Pocock, Gøtzsche & Vandenbroucke, 2008) (see Appendix B). The nature of the studies was considered too heterogeneous to permit a meta-analysis and therefore a more narrative approach was adopted. Reference to a significant finding should be interpreted as meaning statistically significant ($p<0.05$). Finally, studies that looked at factors that might account for the relationship (e.g. neurological research and mediation analysis) and those which might contribute to it (e.g. other trauma and moderating variables) were considered in more detail.

Results

Study Characteristics

Design, samples and participants. The Summary of Studies Table in Appendix C gives a detailed breakdown of the number of participants in each study, the nature of control groups used and the corresponding levels of emotional abuse and/or emotional neglect and BPD within each sample. It was difficult to make

comparisons between the levels of BPD and abuse within the samples as some studies gave percentage levels whereas others gave mean scores.

Regarding the types of samples used, three studies used adolescent or child samples, 28 used adult samples and eight were cohort studies. A breakdown of the nature of these samples and control groups is detailed in Table 2. In the adult samples, ages ranged from 18 to 60, with average ages being in the early thirties. In terms of gender, the majority of samples were between half and two thirds female, with seven studies using female only samples.

Table 2
Breakdown of samples used

Nature of Experimental group	n
BPD patients	19
BPD and comorbid depression patients	3
Adults who had experience childhood trauma	2
Substance abuse disorder patients	2
Incarcerated females	1
National sample	1
Child and adolescent	3
Cohort – Child to adulthood	6
Cohort – Follow-up of outcomes	2
Nature of control group	
Other Axis II disorder patients	4
Depression patients	3
Schizophrenia patients	1
Patients defined as 'other psychiatric diagnosis'	1
Healthy Controls	2
Sisters of those in BPD group	1
Non-abused matched control group	1
Multiple control groups (often healthy controls and other Axis II and/or Axis I groups)	9
No control group used	4

The same data set/participant group was shared across some studies, with results being analysed in different ways or additional data added. Three of the studies were from the large scale CIC sample (Crawford, Cohen, Chen, Anglin, & Ehrensaft, 2009; Johnson, Smailes, Cohen, Brown & Bernstein, 2000; Johnson, Cohen, Smailes, Skodol, Brown, & Oldham, 2001) and samples were also shared by Bornovalova, Levy, Gratz, and Lejuez (2010) and Gratz, Tull, Baruch, Bornovalova,

and Lejuez (2008), Grover et al. (2007) and Tyrka, Wyche, Kelly, Price & Carpenter (2009), Joyce et al. (2003 and 2006), and Zanarini et al. (2000) and Zanarini, Frankenburg, Hennen, Reich and Silk (2006). Therefore, only 33 separate samples were actually represented in the review rather than 39.

Measures. The measures used to assess BPD in each of the studies are detailed in Table 3. The majority of studies used the SCID-II (First et al., 1995) (either in questionnaire or interview form), which is seen as the gold standard measure for diagnosing PDs (Lobbestael, Leurgans & Arntz, 2011). It has been shown to have excellent inter-rater reliability for all twelve PD scales (Kappa ranged from 0.77 to 0.94) (Lobbestael et al., 2011). Ryder, Costa, and Bagby (2007) found the borderline scale to have good convergent and divergent validity, as well as a good relationship to the Five Factor Model of personality traits and to measures of functional impairment.

Table 3
Measures of BPD used in the studies

Measure of BPD	n
Structured Clinical Interview for DSM-IV – Axis II (SCID-II)	22
Borderline Personality Disorder Checklist (BPD Checklist)	1
Alcohol Use Disorder and associated disabilities diagnostic interview schedule (AUDADIS-IV)	1
Coolidge Personality and Neuropsychological index for Children – Borderline Personality subscale (CPNI-BP) *(completed by care-giver)*	1
Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV)	3
Shedler-Westen Assessment Procedure-200 (SWAP-200) *(rated by clinicians)*	1
Structured Interview for DSM-IV Personality (SIDP-IV)	1
Diagnostic Interview for Borderlines (DIB)	5
Personality Diagnostic Questionnaire (PDQ)	2
Borderline Syndrome Index (BSI)	1
Other[a]	1

[a] As their study was with adolescents, Rogosch and Cicchetti (2005) derived a BPD precursor composite score based on relevant items from other measures.

In terms of assessing childhood trauma nearly all studies used questionnaires completed retrospectively by participants (see Table 4). The difficulties with using these retrospective measures are discussed in the 'Quality of Studies' section below.

Table 4
Measures of Trauma within the studies

Measure of Trauma	n
Childhood Trauma Questionnaire (CTQ)	14
Childhood Experiences Questionnaire - Revised (CEQ-R)	5
Composite Score based on variety of assessments (records, self-report, maternal interviews) *(used in prospective cohort studies)*	4
Official Records (legal or medical)	3
Structured Trauma Interview (STI)	1
Childhood Experiences of Care and Abuse Questionnaire (CECA-Q)	1
Parental Bonding Instrument (PBI)	2
Traumatic Antecedents Questionnaire (TAQ)	2
Clinical Interview	2
Childhood Trauma Interview (CTI)	1
Early Trauma Inventory (ETI)	1
Structured Childhood Trauma Interview (VBG)	1
Interview for Traumatic Events in Childhood (ITEC)	1
Clinical Data Form *(rated by clinicians)*	1

The most commonly used questionnaire was the CTQ which screens for five types of maltreatment (emotional, physical, and sexual abuse, and emotional and physical neglect) and has been found to be reliable (test-retest reliability co-efficients of 0.80 to 0.83, internal consistency of Cronbach's alpha 0.79 to 0.94) and to have good construct validity (Bernstein, Fink & Handelsman, 1994; Fink, Bernstein, Handelsman, Foote & Lovejoy, 1995; Roy & Perry, 2004). In a review of instruments for assessing childhood trauma the CTQ was assessed to be an acceptable and in-depth measure for assessing trauma, as were the CECA (interview), CTI and ETI, which were also used by studies above (Roy & Perry, 2004). The four prospective cohort studies (Carlson et al., 2009; Crawford et al., 2009; Johnson et al., 2000; Johnson et al., 2001) derived composite scores based on a variety of measures used including legal records, maternal interviews and self-reports. Carlson et al. (2009) also used direct observations of early mother-infant bonding to assess attachment relationships and potential neglect.

A wide range of additional measures were used to consider other factors, including basic demographic data (age, gender, ethnicity and education level), psychopathology and family history (parental psychopathology and substance use).

Quality of Studies

Young and Solomon's criteria (2009) (Appendix A) for critically appraising research was used to assess the quality of methodology in each study with each one being analysed for the presence or absence of the given criteria based on the type of design (cohort n=8, cross-sectional n=18, or case-control n=13). Additionally, more qualitative comments about potential strengths and limitations of the studies were made. Appendix D gives results of this analysis including studies that raised concerns and why. Overall, the quality of the studies was high, with all being assessed as having a clear research question, an appropriate design and conducting the study according to the protocol outlined in the method. The necessary statistics were presented and these all showed evidence of a priori planning of statistical measures instead of conducting analysis in a posteriori fashion or 'data mining' (Field, 2009). There were some queries over the statistics stated by Lobbestael and Arntz (2010) as detailed in Appendix D. As described above, the measures used by the studies were mainly well known, validated and researched measures. However, this is probably partly reflective of the inclusion criteria adopted for this review. Methodological issues concerning sampling, design and statistics meant there was uncertainty over the validity of conclusions in five of the studies (Goodman et al., 2003; Gunderson et al., 2006; Heigeland & Torgersen, 2004; Lange, Kracht, Herholz, Sachsse & Irle, 2005; Lobbestael & Arntz, 2010), as detailed in Appendix D. In terms of reporting quality, from the items listed as being important in the STROBE Statement (Von Elm et al., 2008) (Appendix B), most studies were

presented to a high standard, giving the relevant methodological information, statistical results and with discussions outlining the key findings and limitations in an accessible manner.

More general limitations noted amongst the studies were small sample sizes (n=10) and sampling methods meaning results were not necessarily generalisable (n=13). Difficulties in generalising were due to exclusion criteria used (e.g. medication and/or substance misuse free, no Axis I disorders), samples being taken from a specific subset of the population (e.g. substance misusers, particular ethnic groups, pregnant women or specialist inpatient wards) and samples potentially being representative only of those with BPD who are higher functioning (as measured by educational and vocational achievements). All of these raise questions about how representative the samples, and therefore results, are of the wider BPD population. Another issue was limited numbers of those who had experienced childhood trauma and/or with BPD traits present in the sample (Goodman et al., 2003; Heigeland & Torgersen, 2004; Johnson et al., 2000; 2001).

In terms of study design, the majority of the studies were cross-sectional (n=18), with samples being assessed on levels of PD traits and split into groups accordingly (often comparing BPD with other types of PD or Axis I Disorders). Although ideal for exploring prevalence of disorders, cross-sectional studies cannot determine whether factors are causal or just associated as participants are only assessed at one point in time (Mann, 2003). This results in ambiguity in interpreting the nature of relationships found, particularly when considering complex developmental pathways as with BPD (Widom et al., 2009). When using this design it is hard to disentangle the effect of retrospectively measured trauma from other aversive experiences and the impact of subsequently symptomatology on self-

reports. Thirteen studies had case-control designs, with control groups consisting of individuals with other personality disorders, axis I disorders or healthy controls. The benefit of this design is that it ensures a high prevalence of the disorder in question within the sample as participants have been selected on this basis (Mann, 2003). However, there can be issues with sampling bias.

The main limitations of both groups of studies were the representativeness of samples and control groups (as discussed above) and the use of retrospective measures of trauma. All bar one of these studies used either retrospective questionnaires or interviews to assess childhood trauma and often gave this as a limitation of the study. The exception was Rogosch and Cicchetti's (2005) study which was with children and so used observational data taken at the time of study. The concern with the use of self-reports to assess childhood trauma centres on whether those with a diagnosis of BPD accurately report the abuse they have experienced. Zanarini et al. (2000) suggested that patients may have "misinterpreted, exaggerated, or even fabricated some of the reported caretaker behaviours" (p.270) or that they may minimise or withhold information. Similarly, Huang, Yang, Wu, Napolitano, Xi, and Cui (2012) stated there may be a memory bias towards traumatic events or a heightened sensitivity to parental failings in those with BPD. Battle et al. (2004) commented that over reporting abuse could result in eliciting sympathy or justifying symptoms and under reporting could be due to shame or inability to recall negative events. As one of the characteristics of BPD is a distorted perception of the self, world and others, so too might childhood memories of negative events be distorted (Machizawa-Summers, 2007). However, Laporte, Paris, Guttman and Russell (2011) and Laporte and Guttman (2001) gave evidence which supports the use of retrospective self-reports of BPD patients in measuring childhood trauma

histories. These studies obtained additional reports of abuse from other family members (sisters and parents respectively) and found strong corroboration between the results, indicating that patients accurately reported the abuse experienced. Further research like this is needed to explore the validity and reliability of self-reported accounts of trauma histories from BPD patients as frequently this is the easiest and potentially only method available to researchers.

The six prospective cohort studies following children into adulthood were well thought-out, using a comprehensive range of measures with stringent methodology. For example, the CIC studies involved multi-modal assessment during childhood and adolescence, including parental, teacher and medical reports as well as self-reports, thus adding to the validity and reliability of the data. This reflects the amount of time, planning and resources needed to make these studies work over such a long period of time. Interestingly, in all these cohort studies loss to follow-up appeared to be minimal. The advantage of prospective studies is that they can demonstrate cause and effect relationships better as potential causes are measured before the outcome occurs (i.e. trauma measured at the time of childhood and BPD traits measured as they emerge in later life) (Mann, 2003).

These cohort studies benefit from being free from potential retrospective bias in reporting (as discussed above) and allow for a wide range of potential covariates to be measured and for consideration of changes in the relationships between factors over time (Cohen et al., 2005). Lenzenweger and Cicchetti (2005) stressed the need for more prospective research that enables BPD to be studied before it emerges in order to gain a lifespan perspective of the disorder and consider prodromal presentations which may be important for prevention strategies. However, a drawback with these studies is that, by their nature, they can only be done on

populations that are presumed to be ‘at risk’, meaning that the actual prevalence of disorder within the sample may be low, therefore limiting statistical power and conclusions that can be drawn (Cohen et al., 2005). To overcome this, the CIC studies have focused more on PD Clusters using a dimensional instead of categorical approach to assess levels of PD (Cohen et al., 2005). Another issue is the potential impact of changes in diagnostic criteria since the studies began in the 1970s. The CIC team is aware of this and has reviewed the measurements used at each stage (Cohen et al., 2005). However, it may still mean that factors were missed in early data collection as they were not diagnostically relevant at the time. This could particularly be the case with neglect, where there have been cultural shifts in what parenting practices are considered neglectful since the 1970s, perhaps resulting in lower levels of neglect being recorded in the samples than would be considered today. Another difficulty is that reliance on legal records potentially only captures more severe cases of childhood maltreatment. Again, emotional abuse or neglect may be considered a more ‘hidden’ form of abuse and so be underreported (Cawson et al. 2000).

In summary, the studies in this review were generally of good quality and well reported. The main limitations centred on sampling, meaning results may not be generalisable, sample sizes being small and the use of retrospective self-reports to measure trauma. Prospective studies were rigorously conducted and provide important data in terms of establishing causality due to the temporal nature in which variables can be assessed.

Results of studies

The relationship between BPD and emotional abuse and neglect. This section considers the different evidence regarding the link between BPD and emotional abuse and neglect. Studies have been grouped in relation to the nature of comparison samples used.

BPD compared to healthy controls. Eleven studies involved comparisons of differences in levels of abuse between those with BPD and healthy controls. Table 5 summarises the nature of the trauma assessed and the findings for each of these studies.

Table 5
Studies comparing levels of emotional abuse and neglect in healthy controls and BPD

Reference	Trauma assessed	Results
Driessen et al. (2000)	Physical and Emotional abuse combined	HC < BPD
Giesen-Bloo & Arntz (2005)	Overall trauma	HC < BPD
Horesh et al. (2008)	Physical and Emotional abuse combined	HC < BPD
Lange et al. (2005)	Neglect	HC < BPD
Laporte & Guttman (2001)	Verbal abuse	HC < BPD
Laporte et al. (2011)	Emotional Abuse	HC < BPD
Lobbestael & Arntz (2010)	Overall trauma	HC < BPD
Lobbestael et al. (2005)	Emotional Abuse	HC < BPD
Sieswerda et al. (2006)	Emotional Abuse	HC < BPD
Weniger et al. (2009)	Neglect score	HC < BPD
Wingfield et al. (2011)	Emotional Abuse	HC < BPD

Note. HC = Healthy Control group, BPD = Borderline Personality Disorder group

All of these studies found significantly higher levels of abuse in the BPD group than in healthy controls. As can be seen in Table 5, five of the studies involved the more specific comparison of levels of emotional or verbal abuse and BPD traits and these echoed the result of studies comparing broader forms of childhood trauma. Interestingly, Laporte et al. (2011) used sisters of BPD participants as the control group and found that siblings with BPD reported higher

levels and more severe forms of emotional and sexual abuse, than their sisters. Most of the sisters were found to be psychopathology free, with only 3 of the 56 pairs being concordant for BPD. This evidence supports there being a link between emotional abuse and neglect and BPD, when compared with healthy controls.

BPD compared to Axis I diagnosis. Fourteen studies compared BPD patients with Axis I disorder patients as summarised in Table 6 below. Studies using 'non-PD groups' tended to include a range of Axis I disorders and all found significant differences between groups. Two focused specifically on emotional abuse and/or emotional neglect (Macizawa-Summers, 2007; Sieswerda, Arntz, Mertens & Vertommen, 2006). Huang et al. (2012) separately considered mother or father antipathy or neglect and, again, found significantly higher levels in the BPD group compared to non-PD patients on all scales.

Table 6 shows that of the six studies comparing BPD with controls with Major Depressive Disorder (MDD) (or comorbid samples), four of them found significantly higher levels of trauma in the BPD groups. However, Horesh, Ratner, Laor, and Toren (2008) and Wingenfeld et al. (2011) failed to find a significant difference between the groups. Four further studies compared BPD with other Axis I diagnoses and found significantly higher levels of verbal and/or emotional abuse and neglect in BPD groups when compared to anorexia (Laporte and Guttman, 2001), schizophrenia (but not comorbid schizophrenia and BPD) (Kingdon et al., 2010) and substance misuse (Bornovalova et al., 2010; Gratz et al., 2008) samples.

Table 6
Studies comparing levels of emotional abuse and neglect in Axis I and BPD groups

Reference	Trauma assessed	Comparison group	Results
Battle et al. (2004)	Emotional abuse	MDD	MDD < PD[a]
	Verbal abuse	MDD	MDD < PD[a]
	Neglect	MDD	MDD < PD[a]
Bellino et al. (2005)	Emotional and verbal abuse	MDD	MDD < BPD (with comorbid MDD)
Bornovalova et al. (2010)	Emotional abuse	Substance Users (split according to severity BPD traits)	More severe BPD reported more emotional abuse
Giesen-Bloo & Arntz (2005)	Overall trauma	Non-PD group	Non-PD < BPD
Gratz et al. (2008)	Emotional abuse and emotional neglect	Substance Users	More severe BPD reported more emotional abuse
Horesh et al. (2008)	Physical and emotional abuse combined	MDD	MDD = BPD
Huang et al. (2012)	Mother antipathy	Non-PD group	Non-PD < BPD
	Mother neglect	Non-PD group	Non-PD < BPD
	Father antipathy	Non-PD group	Non-PD < BPD
	Father neglect	Non-PD group	Non-PD < BPD
Joyce et al. (2003)	Overall trauma	-	Abuse predictive of BPD traits in a MDD sample
Joyce et al. (2006)	Overall trauma	-	Abuse predictive of BPD traits in MDD sample
Kingdon et al. (2010)	Emotional abuse and emotional neglect	Schizophrenia and Comorbid BPD and Schizophrenia	Schizophrenia < BPD BPD = Schizophrenia and comorbid BPD
Laporte & Guttman (2001)	Verbal abuse	Anorexia	Anorexia < BPD
Machizawa-Summers (2007)	Emotional abuse and emotional neglect	Non-PD group	Non-PD < BPD
Sieswerda et al. (2006)	Emotional abuse	Non-PD group	Non-PD < BPD
Wingfield et al. (2011)	Emotional abuse	MDD	MDD = BPD

Note. MDD = Major Depressive Disorder, BPD = Borderline Personality Disorder, PD = Personality Disorder.
[a] Chi-squared tests only conducted with a combined PD group (including schizotypal, avoidant and obsessive compulsive and BPD). Mean score of abuse in BPD group was higher than MDD.

From these comparisons it appears that generally those with BPD do experience more childhood emotional abuse and/or neglect compared to controls with Axis I disorders. However, not all researchers found this to be the case with

depression groups and so more research is needed in this area. It is important to note that in studies with healthy control groups, levels of abuse in the Axis I groups were still often significantly higher than in the healthy controls.

BPD compared to other PDs. Eleven studies compared levels of abuse and neglect between PD groups, as detailed in Table 7 below.

Table 7
Studies comparing levels of emotional abuse and neglect in BPD and other PD groups

Reference	Trauma assessed	Comparison group	Results
Battle et al. (2004)	Emotional Abuse Verbal Abuse Neglect	Other PD	BPD significantly more likely to report all three types of abuse
Giesen-Bloo & Arntz (2005)	Overall trauma	Cluster C and other Cluster B	Cluster C and other Cluster B < BPD
Goodman et al. (2003)	Emotional abuse	Other PD	Significant correlation between emotional abuse and affective liability and intensity in other PD group but not BPD
Huang et al. (2012)	Mother antipathy Mother neglect Father antipathy Father neglect	Other PD	Other PD < BPD Other PD < BPD Other PD < BPD Other PD < BPD
Joyce et al. (2003)	Overall trauma	Levels of BPD with MDD group	Trauma was risk factor for BPD and Avoidant PD
Joyce et al. (2006)	Overall trauma	Levels of BPD with MDD group	Trauma was risk factor for BPD and Avoidant PD
Lobbestael & Arntz (2010)	Overall trauma	Cluster C Other Cluster B	Cluster C < BPD Other cluster B < BPD, except ASPD = BPD
Lobbestael et al. (2005)	Emotional abuse	ASPD	ASPD = BPD
Sieswerda et al. (2006)	Emotional abuse	Cluster C and other Cluster B	Cluster C and other Cluster B < BPD
Zanarini et al. (2000)	Emotional abuse Verbal abuse	Other PD	Other PD < BPD
Zhang et al. (2012)	Emotional abuse and emotional neglect	Cluster A & C Other Cluster B	Cluster A & C < Cluster B Other Cluster B < BPD

Note. ASPD = Antisocial Personality Disorder, BPD = Borderline Personality Disorder, MDD = Major Depressive Disorder, PD = Personality Disorder

Table 7 shows the majority of studies found levels of emotional abuse, verbal abuse and/or emotional neglect to be higher in BPD participants than in participants

with other PDs. This includes studies looking more specifically at types of abuse/neglect from both parents (Huang et al., 2012; Zanarini et al., 2000). Zhang, Chow, Wang, Dai & Xiao (2012) found a significant difference between Cluster B PDs compared to Cluster A or C PDs, and with BPD compared to other Cluster B PDs. However, regression analysis showed emotional neglect to be a significant predictor for any PD diagnosis and emotional abuse to be a predictor of Cluster A and B diagnoses, not just BPD. Studies found levels of emotional abuse to be similar to BPD for antisocial PD (ASPD) (Battle et al., 2005; Lobbestael & Arntz, 2010; Lobbestael, Arntz & Sieswerda 2005) and avoidant PD (Joyce et al., 2003; 2006). Surprisingly, Goodman et al. (2003), who used measures of affective stability traits instead of BPD measures, found a significant correlation between this and emotional abuse in their other PD group but not the BPD group. It is difficult to compare this with other results due to the difference in variables measured.

In summary, a number of studies have shown that those with BPD tend to report experiencing more emotional abuse and emotional neglect than those with other PDs and that this abuse may be from both parents. However, once again, levels of abuse and neglect were still higher in groups of other PDs than in healthy controls or Axis I disorders, with some results suggesting that abuse and neglect could put individuals at risk for all PDs, not specifically BPD (Zhang et al., 2012).

Correlation studies. As detailed in Table 8 below, five studies explored correlations between abuse and BPD symptoms within their samples. Afifi et al. (2011) found that in a large population based study (over 30,000 people) the relationship between BPD and emotional abuse and neglect was still significant. However, in line with the PD studies above, this form of abuse was also found to be predictive of a number of PD diagnoses. Gratz, Latzman, Tull, Reynolds and Lejuez

(2011) demonstrated that the association between BPD and emotional abuse can be found in childhood. The other studies continued to support the link between BPD and emotional abuse, but not always with neglect.

Table 8
Correlational Studies

Reference	Trauma assessed	Comparison group	Results
Afifi et al. (2011)	Emotional abuse and neglect	Large Population based study	Emotional abuse and neglect was predictive of meeting criteria for BPD
Bierer et al. (2003)	Emotional abuse Emotional neglect	Correlations with PD diagnosis in mixed PD sample	BPD diagnosis was correlated with emotional abuse, but not neglect
Bradley et al. (2005)	Environmental stability	Levels of BPD symptoms in PD sample	Levels of BPD symptoms correlated with a number of family environmental variables (indicating potential emotional abuse and/or neglect)
Gratz et al. (2011)	Emotional abuse	Child/parent dyads	Emotional abuse was significantly correlated with BPD features in childhood
Specht et al. (2009)	Emotional abuse	Incarcerated females	Significant correlation between BPD traits and emotional abuse and with perceived lack of emotional support but not with neglect.

Note. BPD = Borderline Personality Disorder, PD = Personality Disorder

Levels of BPD in abused compared to non-abused samples. Instead of sampling based on the presence of BPD, three studies compared those who had experienced childhood abuse with those who had not in terms of levels of BPD traits. These are described in Table 9. Rogosch and Cicchetti (2005) found significantly higher levels of BPD precursors (e.g. negativity, conflicted relationships, self-harm and upsetting others) in maltreated compared to the non-maltreated children. This provides evidence that significant differences, resulting from childhood trauma, can be seen from an early age. Grover et al. (2007) showed that a community sample of adults who experienced any childhood abuse were more likely to have symptoms of PD across all the clusters than the non-abused, with significant individual disorders

being paranoid PD, narcisstic PD, BPD, ASPD, obsessive-compulsive PD, passive aggressive PD and depressive PD. The authors did not state whether BPD symptoms were more prevalent than symptoms of other PDs. Tyrka et al. (2009) extended these findings with the same sample but looking at different types of trauma and found higher levels of BPD in those with emotional abuse and neglect. These results suggest that those who experience childhood abuse, including emotional abuse and/or neglect, are significantly more likely to develop BPD traits and meet criteria for a BPD diagnosis in later life.

Table 9
Studies comparing BPD in abused and non-abused samples

Reference	Trauma assessed	Sample	Findings
Grover et al. (2007)	Overall trauma	Subclinical PD traits in a community sample	Non-abused < abused
Rogosch &Cicchetti (2005)	Overall trauma	BPD traits in child sample	Non-abused < abused
Tyrka et al. (2009)	Emotional abuse and neglect	Subclinical PD traits in a community sample	Non-abused < abused

Note. BPD = Borderline Personality Disorder

Cohort Studies. Results from the cohort studies mirror the majority of the findings shown above in terms of the impact of emotional abuse or neglect on the emergence of BPD symptoms in later life. Table 10 gives a summary of the results the cohort studies. Their longitudinal nature enables these studies to take a detailed look at various forms of abuse.

The results of the CIC studies highlight the impact of more specific aspects of emotional abuse and neglect, such as supervision neglect (Johnson et al., 2000), verbal abuse (Johnson et al., 2001) and the impact of early maternal separation (Crawford et al., 2009). Interestingly, Crawford et al. (2009) found that early separations, for reasons other than mother or child illness, had significantly higher BPD symptoms than others. Although separations of this type cannot be inferred to

equate to emotional abuse or neglect, it may be indicative of a lack of maternal investment in caregiving (Crawford et al., 2009). Early separations also led to a slower rate in the natural decline of BPD symptoms in adulthood. As these studies are all based on one sample of participants, convergence between results would be expected.

Carlson et al. (2009) considered a range factors linked with emotional abuse and/or neglect and many were found to correlate significantly with BPD symptoms at age 28 (as detailed in Table 10). This study highlights the potential damaging impact of negative interactions even at the earliest age. As discussed previously, the use of observation methods to look at parent-interactions adds weight to the findings of this study. These results were supported by Heigeland and Torgersen (2004) who found reports of emotional abuse, together with other factors relating to environmental instability and conflict, to be higher in those that developed BPD symptoms in later life than those that did not. Widom et al. (2009) found significantly higher numbers of adults who were abused before age 11 met criteria for BPD at follow-up (age 29) than non-abused matched controls.

Finally, the two outcome follow-up studies provide mixed results for the impact of emotional abuse and emotional neglect in terms of outcomes in BPD patients, with one finding no correlation between these (Gunderson et al., 2006) and one finding lower levels of abuse in childhood were predictive of better outcomes (Zanarini et al., 2006). The evidence from the cohort studies appears to support the notion of emotional abuse and neglect being important factors in the emergence of BPD. However, more research is needed on how this then impacts on outcomes in terms of symptom remission.

Table 10
Cohort studies

Reference	Focus of study	Results
Carlson et al. (2009)	Data on range of factors consistent with childhood trauma or maladaptation taken during childhood and adolescence	Early maltreatment, attachment disorganisation, maternal hostility, maternal boundary dissolution, life stress and parent-child relationship disturbance were all significant predictors of BPD symptoms in adulthood, as was a composite score of family disruption from 1 to 18 years.
Crawford et al. (2009) - CIC	Early maternal separation (under 5)	Participants with early separation had higher BPD symptoms and slower rates of symptom decline between the ages 22 and 30.
Gunderson et al. (2006)	Two year follow-up of outcomes	A history of abuse and/or neglect was not correlated with levels of BPD criteria met at two year follow-up.
Heigeland & Torgersen (2004)	Follow-up of adults who were inpatients at adolescence	Levels of environmental instability, abuse and overall trauma (including aspects such as rejection, conflict, loss and over-control) in adolescence were significantly higher in those that met criteria for BPD in adulthood.
Johnson et al. (2000) - CIC	Emotional, physical and supervision neglect	Rates of PD in adulthood were higher in those who experienced emotional, physical and supervision neglect in childhood. However, only supervision neglect was a significant predictor of BPD symptoms.
Johnson et al. (2001) - CIC	Verbal Abuse	Rates of BPD in adulthood were significantly higher in those who experienced verbal abuse when younger.
Widom et al. (2009)	Emotional neglect	Rates of BPD in adulthood were higher in children with legal records of emotional neglect when younger compared to non-abused matched controls
Zanarini et al. (2006)	Ten year follow-up of outcomes	Lower levels of emotional abuse and neglect were predictive of fewer BPD symptoms at ten year follow-up.

Note. BPD = Borderline Personality Disorder, CIC = Children In the Community Study, PD = Personality Disorder

What factors explain and/or mediate this relationship? Having looked at evidence to support the link between emotional abuse and neglect and BPD, factors that could potentially explain why the relationship between BPD and childhood trauma exists were then considered. The primary focus was on studies of biological mechanisms and studies that explicitly used mediation analysis. A mediating variable can be defined as a third variable which is influenced or generated by the independent variable (e.g. childhood trauma) which then influences the dependent variable (e.g. BPD), thus mediating the relationship (Baron & Kenny, 1986).

Neuroimaging evidence. A number of studies used neuroimaging to consider the impact of childhood trauma on brain development and how this may result in BPD symptoms. These showed BPD participants had significantly smaller hippocampal and amygdaloid volumes than healthy controls (Driessen et al., 2000; Weniger, Lange, Sachsse & Irle, 2009). However, there was no significant difference in volumes between those with BPD that had been abused in childhood and those that had not. Lange et al. (2005) found reduced glucose metabolism in right-sided ventromedial temporal and left-sided medial parietal/posterior cingulate cortices in BPD patients, all of whom had reported childhood abuse and experienced dissociative symptoms, compared to healthy controls. The authors proposed that childhood trauma may lead to stress-related neural degeneration in temporo-parietal areas which may produce BPD symptoms (Lange et al., 2005). The lack of non-abused BPD comparisons means that it cannot be stated whether these changes are specifically linked to trauma or whether they would be present in all BPD patients.

Mediation analysis. Disappointingly, few of the studies included in this review performed additional analysis to extend their understanding of significant findings. Only four of the 39 studies looked at potential mediating factors in the

relationship between emotional abuse and BPD using the Baron and Kenny (1986) criteria. Results showed emotional dysregulation (Gratz et al., 2008), schema modes (particularly disconnection/rejection and impaired limits) (Specht, Chapman & Cellucci, 2009) and self-representation in middle childhood (Carlson et al., 2009) to mediate the relationship between BPD and childhood abuse. However, all studies used a composite score of maltreatment, rather than looking more specifically at emotional abuse and/or neglect. Rogosch and Cicchetti (2005) found attentional networks and processes did not mediate the relationship in abused children. These studies point to the importance of key psychological processes, such as the formation of schemas and representation of the self, in mediating the impact that the experience of trauma in childhood has on the development of BPD symptoms in later life. This is based on only a handful of results and further research is needed in the area.

What factors contribute to or moderate the relationship? This final section considers additional factors identified by studies that lead to an increased risk of BPD and, when applicable, how these impact on the relationship between emotional abuse and emotional neglect and BPD.

Demographic variables. A number of studies found significantly higher rates of BPD symptoms in females compared to males (Afifi et al., 2011; Bornovalova et al., 2010; Gratz et al., 2008; Huang et al., 2012; Kingdon et al., 2010; Zanarini et al., 2000). Both Bierer et al. (2003) and Goodman et al. (2003) found emotional abuse to be significantly correlated with BPD symptoms in males and not females. Higher rates of symptoms were found in younger participants (Afifi et al., 2011; Joyce et al., 2003, 2006; Zanarini et al., 2006). Other higher risk demographic factors were being African-American (Afifi et al., 2001; Gratz et al., 2008), marital status (Afifi et al. 2011; Widom et al., 2009; Zanarini et al., 2006) and being unemployed or having a

lower household income (Bornovalova et al., 2010; Widom et al., 2009; Zanarini et al., 2006). A number of other more family related factors were significant predictors of BPD, e.g. parental psychopathology (Afifi et al., 2011; Helgeland & Torgersen, 2004; Widom et al., 2009; Zanarini et al., 2006), witnessing domestic violence in childhood (Afifi et al., 2011, Zanarini et al., 2006) and parental imprisonment and suicide attempts (Afifi et al., 2011).

Significantly, Widom et al. (2009) found that the impact of child abuse and neglect (all types) became non-significant in a regression model when other family and lifestyle characteristics were included (e.g. parental substance use, employment, education level and history of Axis I disorders), concluding that maltreatment may represent a marker for family dysfunction which is actually more significant in leading to a greater risk of BPD. This sheds doubt on there being a causal relationship between trauma and BPD. Helgeland and Torgersen (2004) was the only study to consider protective factors. They found in their cohort study that environmental instability (including emotional abuse and neglect) and the absence of protective factors (e.g. artistic talents and superior school performance) were both independent predictors of BPD in a regression analysis.

Comorbidity. Mood disorders and substance misuse were highly prevalent within BPD samples (Afifi et al., 2011; Bornovalova et al., 2010; Huang et al., 2012; Gratz et al., 2008; Joyce et al., 2003; Widom et al., 2009; Zanarini et. al, 2006) and abused samples (Tyka et al., 2009), as were other PDs diagnoses (Afifi et al., 2011; Zanarini et al., 2006). In their regression analysis, Afifi et al. (2011) found comorbidity with Axis I and Axis II disorders had the highest odds ratios in being predictive of a Cluster B diagnosis (higher than all types of childhood abuse) and Zanarini et al. (2006) found comorbidity led to significantly worse outcomes at a ten

year follow-up. As discussed, the majority of studies found significantly higher levels of all abuse present in those with Axis I and other Axis II disorders compared to healthy controls, not just in BPD participants (Zhang et al., 2012).

Traits. A number of studies looked in more detail at underlying traits associated with BPD. Studies showed traits such as impulsivity (Bornovalova et al., 2010; Laporte et al., 2011), novelty seeking (Joyce et al., 2003), stress reactivity (Bornovalova et al., 2010), affective liability (Laporte et al., 2011) and hypervigilance for emotional cues (Sieswerda et al., 2006) were significantly higher in BPD samples compared to others. The difficulty with interpreting these results from retrospective studies in relation to trauma is that causality cannot be inferred, as the presence of these traits may be because of experiencing childhood trauma or may have been premorbid. Many of these traits map directly onto the criteria required for a BPD diagnosis and are inherently higher within this population. In their cohort study, Crawford et al. (2009) found angry temperament, crying or demanding behaviour and anxious or avoidant attachment styles in infancy were predictive of BPD symptoms in later life, perhaps providing more substantial support for the role of these traits in developing BPD due to the prospective nature of the study. Lange et al. (2005) and Weniger et al. (2009) also found significant differences in intelligence and memory in BPD groups compared to healthy controls.

Gratz et al. (2011) was the only study to conduct a formal moderation analysis, using the Aiken and West (1991) criteria for looking at interactions. The relationship between emotional abuse and BPD features in their child community sample increased in magnitude as the level of affective dysfunction moved from low to high therefore moderating the relationship between the two. The authors stated that this implies that emotional abuse only leads to BPD pathology when an

underlying trait vulnerability of affective dysfunction is present. Impulsivity was not found to be a significant moderator.

Childhood sexual abuse and physical abuse. Thirteen studies found significantly higher rates of sexual and physical abuse in BPD samples compared to controls (Afifi et al., 2011; Bradley, Jenei, & Westen, 2005; Carlson et al., 2009; Crawford et al., 2009; Driessen et al., 2000; Giesen-Bloo & Arntz, 2005; Huang et al., 2012; Kingdon et al., 2010; Lange et al., 2005; Laporte & Guttman, 2001; Sieswerda et al., 2006; Weniger et al., 2009; Zanarini et al., 2006). However, five studies failed to find a significant difference in levels of sexual abuse (Bellino et al., 2005; Bierer et al., 2003; Laporte et al., 2011; Widom et al. 2009; Zanarini et al., 2000) and two found no significant difference in levels of physical abuse (Battle et al., 2004; Wingenfeld et al., 2011) compared to their respective control groups.

Grover et al. (2007) and Tyrka et al. (2009), using the same sample, found no significant differences in levels of both Axis I and Axis II disorders between those who reported sexual and physical abuse and those who reported only emotional abuse, concluding that this form of abuse is as significant as others. Furthermore, Gratz et al. (2008), Machizawa-Summers (2007) and Zhang et al. (2012) found emotional abuse and emotional neglect to be significant predictors of BPD symptoms above other types of abuse. Similarly, Specht et al. (2009) showed lack of emotional support to be the only independent predictor of BPD in regression analyses.

Taking a slightly different perspective, Bradley et al. (2005) used the Baron and Kenny (1986) criteria to look at whether sexual abuse and physical abuse were mediated by family environment (measured by quality of parental relationship, stability, warmth and separations) and found both to have a direct effect on BPD symptoms and to be partially mediated by family environment. This perhaps

highlights the interplay between emotional abuse/neglect (as represented by unstable family environments) and other forms of childhood abuse.

Nature of studies. Finally, whether results differed significantly depending on type of methodology or sample used was considered. It can be seen that many of the studies were conducted to high standards with strong degree of convergence between the findings of the studies and no major anomalies arising. Therefore, methodological issues are not considered to be a confounding factor with the results. There was no difference observed between those studies using retrospective measures of trauma and those using prospective, supporting the validity of using retrospective measures. There was agreement of findings between those samples selected on the basis of experience of abuse compared to those selected on the basis of BPD symptoms, and likewise with child and adolescent studies compared to adult studies. All of this supports the reliability and validity of the findings of this literature review and the studies within it.

Discussion

The aim of this literature review was to establish the current evidence base for a link between the experience of emotional abuse and emotional neglect in childhood and the development of BPD symptoms in later life and then to consider which factors may account for this relationship and which may contribute to it. From reviewing 39 studies, there appears to be a considerable amount of evidence to support the relationship between emotional abuse and neglect and BPD. This evidence comes from studies comparing levels of abuse between those with BPD and healthy controls, Axis I disorders and other Axis II disorders, as well as cohort studies and abused compared to non-abused samples.

This main finding supports the theories put forward in the introduction by Linehan (1993) and Fonagy and Luyten (2009). Both theories postulate that emotional abuse and neglect results in invalidating environments, which potentially lead to the child failing to develop key processes required for later life. In Linehan's (1993) theory, this is the ability to regulate their emotions and soothe themselves. As the child's emotional needs are not met due to the environment (e.g. not comforted or even ridiculed when upset) they develop more extreme emotional reactions (e.g. outbursts or self-harming) in order to gain a response from caregivers. Beliefs may form about their emotions being bad or dangerous, developing into negative core beliefs, as seen in BPD (Linehan, 1993). This can be viewed in conjunction with Fonagy's (2000) mentalization theory, in which the invalidating environment results in the child failing to learn about their own and others mental states, leading to difficulties understanding the intentions of others' which impacts on interpersonal functioning, again as seen in BPD.

However, although the evidence supports this relationship, there is still a lack of specificity, with research suggesting that experience of emotional abuse and neglect is still significantly higher within Axis I, particularly depression, and Axis II populations compared to non-clinical groups, albeit higher still within BPD groups. Added to this, a significant number of people experience abuse and do not go on to develop BPD or any other disorder. For example, Widom et al. (2009) found that only 14.9% of those abused met criteria for BPD, which, although significantly higher than controls, still means that only a minority of those abused developed BPD. Likewise not all those with BPD reported childhood abuse, with prevalence rates ranging between 28.6% (Afifi et al., 2011) and 92% (Kingdon et al., 2010) for

emotional abuse and 21% (Afifi et al., 2011) and 77.1% (Zanarini et al., 2001) for emotional neglect.

This finding could perhaps be accounted for with reference to current evolutionary-neurodevelopmental theory on differential susceptibility to the environment (Ellis, Boyce, Belsky, Bakermans-Kranenburg & Van Ijzendoorn, 2011). This states that some individuals have a neurobiological susceptibility to both negative and positive environments leading to necessary adaptations to ensure evolutionary survival. Therefore, susceptible children in high-stress (i.e. abusive) environments will adapt in ways which allow them to respond best to the threat at the time by reducing pain, even if from the outside these appear risky and self-destructive, whereas susceptible children in positive environments will flourish. In contrast, low-susceptible children will fair similarly regardless of environmental context (Ellis et al., 2011). Although more research is needed, particularly into how to determine which individuals are susceptible at the neurobiological level (which is in its infancy), this could account for why a subset of individuals develop pathology following child abuse and others do not. Similarly, those with BPD who do not report childhood abuse may be highly susceptible individuals responding to 'lesser' adversity within the environment not detected by child abuse measures (e.g. harsh parenting but not to a neglectful extent) (Ellis et al., 2001).

This neurobiological or genetic susceptibility is further supported by Belsky et al. (2012). They considered the prevalence of borderline personality related characteristics (BPRS) at age 12 in a large study of same sex twins in relation to etiological factors of inheritability (as measured by a family history of psychiatric disorders) and maltreatment (physical/sexual abuse and maternal negative expressed emotion). They found those who experienced maltreatment developed more BPRS

and that this was specific to the child's unique personal experience of maltreatment, as it was not attributable to features of family environment shared with twins (either monozygotic or dizygotic). Further to this, children were more likely to develop BPRS following maltreatment when there was a family history of psychiatric disorder. This demonstrates that inherited liability and maltreatment both contribute to borderline traits and the effect of these is more potent when combined. Belsky et al. (2012) stated that a family history of psychiatric disorder may be an indicator of vulnerability for the individual to have a more intense emotional response to maltreatment, increasing the likelihood of developing borderline traits. However, more research is needed to delineate the biological nature of this relationship and establish the links between this and the cognitive, emotional and behavioural symptomatology seen in BPD.

When reviewing factors that might account for the relationship between BPD and abuse, neuroimaging studies highlighted reduced hippocampal and amygdaloid volume (Driessen et al., 2000; Weniger et al., 2009) and reduced glucose metabolism (Lange et al., 2005) in BPD patients. However, based on the current evidence, it cannot be determined whether these changes are due to experiencing trauma or are universal across those with BPD due to the nature of samples used. Psychological processes of emotional dysregulation (Gratz et al., 2008), schema modes (Specht et al., 2009) and self-representation in late childhood (Carlson et al., 2009) were found to mediate the relationship between overall childhood trauma and BPD. These perhaps indicate the key psychological processes that dictate the lasting impact of trauma on personality development and centre around the concept of self and identity, something often impoverished in BPD (Carlson et al., 2009).

In terms of contributing factors, research looking at how underlying traits impact on the relationship between BPD and trauma was fairly limited. As discussed in the introduction, Paris (2007) stressed the need to consider the interplay between genetically predisposed temperaments or traits, trauma and the development of BPD, hypothesising that trauma only leads to the development of BPD in the presence of certain trait vulnerabilities predisposing the individual to react to trauma in a way that increases the likelihood of BPD. Gratz et al. (2011) found that affective dysfunction (encompassing traits such as affective lability, reactivity and emotional intensity) moderated the impact of emotional abuse on BPD features in children, with the strength of the relationship increasing when affective dysfunction was high, thus supporting Paris' (2007) theory. Other studies found levels of impulsivity, novelty seeking and angry temperament to be significantly higher in BPD groups, but did not conduct more formal moderation or mediation analysis.

Additional factors putting individuals at further risk of developing BPD were gender, comorbid mood, anxiety or substance abuse disorders, parental psychopathology, and other factors indicative of unstable environments while growing (e.g. criminality, conflict/hostility, loss and few protective factors). These factors echo those found in previous reviews (Chanen & Kaess, 2012; Cohen et al., 2005). It is of note that when researchers included other lifestyle and family characteristics in covariance analysis the effect of trauma became insignificant potentially supporting the notion that instead of a causal relationship directly with trauma, it could be other co-occurring factors present in dysfunctional family environments that lead to BPD rather than the trauma itself (Widom et al., 2009). Of course it becomes quite hard even conceptually to distinguish between neglect and dysfunctional family environments. However, from the perspective of prevention it

is evidently important that researchers arrive at a more refined understanding of the nature of environmental toxicity in the causation of personality disorder.

Perhaps one of the more significant findings to emerge from this review is that studies have shown the impact of emotional abuse and neglect being equal to that of sexual and physical abuse, which has perhaps traditionally been viewed as more severe (Tyrka et al., 2009). A number of cases actually showed this form of abuse to have more predictive power for the emergence of BPD symptoms than sexual or physical abuse in regression analyses (Gratz et al., 2008; Machizawa-Summers, 2007; Zhang et al., 2012). Bradley et al.'s (2005) finding that family environment (warmth and stability within family and nature of relationship with parents) partially mediates the relationship between both sexual and physical abuse and BPD is interesting. Further replication and exploration of this is needed. When linking this to the theories of invalidating environments discussed above, it could be understood that within a more stable family environment the impact of sexual and physical abuse is partially negated by the protective factors, such as positive attachments and more adaptive ways of coping. Whereas, this negation is absent in more chaotic and emotionally neglectful families, resulting in a more severe and lasting impact of abuse leading to an increased risk of BPD. As sexual and physical abuse rarely occur in the absence of emotional abuse or neglect it is difficult to separate out the unique impact of this form of abuse (Gratz et al., 2008) and discussion of the extent to which this is possible (either by study design or use of statistical measures) is largely absent in the literature, which often combines all forms of abuse. The evidence above strongly suggests that there may be a unique impact of emotional abuse and further research on disentangling this is needed.

Limitations of Current Evidence

Despite there being a large body of literature included in this review that on the whole supports a link between emotional abuse and neglect and BPD, there are still a considerable number of limitations with the current evidence base. Of the studies discussed, very few went beyond initial analysis to enable a richer understanding of mediating and moderating factors. This is particularly important given the lack of specificity within the relationship and the findings of some covariance analysis (Widom et al., 2009). Studies where these processes were explored tended to focus only on overall trauma experience.

As discussed in the analysis of quality of studies, there continue to be questions about the validity and reliability of using retrospective self-report measures with this participant group, where the nature of BPD pathology could result in a bias towards reporting emotionally abusive and neglectful memories (Huang et al., 2012; Zanarini et al., 2000). However, the convergence of results between studies using retrospective and prospective measures provides support for their use, as does Laporte et al. (2011) and Laporte and Guttman (2001) who corroborated patients' reports of trauma with that of siblings and parents respectively.

There is a lack of representative sampling, with the majority of studies drawing participants from inpatient or outpatient populations, with limited data on those with BPD who do not enter into services or do so only at crisis points. Added to this, negative experiences of treatment, both in the past and on-going, which increase the chance of an individual being in the 'system' and therefore available for research, may also confound participants' self-reports of trauma, particularly when currently in settings potentially experienced by individuals as abusive, theoretically heightening previous memories of similar experiences.

Although increasing in number, there is a need for more prospective longitudinal studies with adequate systematic assessment of on-going traumatic and aversive experiences throughout childhood and adolescence. However, with the current body of evidence on emerging personality disorder growing, there is the potential for BPD traits emerging at an early age to impact on the parenting received, thus confounding experiences and the potential direction of causality (i.e. BPD traits emerging in childhood lead to more emotionally abusive and neglectful parenting rather than the other way round). With this is mind, there is an urgent need for rigorous genetically informed studies attempting to delineate gene environment interactions (Paris, 2007). Ellis et al. (2011) stressed the importance of method of assessment of environment in determining the outcome of results, with previous studies failing to replicate findings of genetic and environmental interaction studies when using self-report measures of environment as opposed to interview-based measures. They stressed the need to be able to measure environmental factors as accurately as genetic ones in future research.

Limitations of the Current Literature Review

There are limitations to the current review. A number of studies only conducted analysis using overall trauma scores, meaning the results did not reflect the unique impact of emotional abuse/neglect as was the aim. However, cutting out studies that used composite scores would have resulted in a significant loss of data, which was not felt appropriate. A number of studies looked at family environment/instability and it was felt that there was a potential overlap between this and emotional abuse and, especially, emotional neglect, again making it harder to look at the unique impact. The disparity in ways of reporting between the studies,

particularly with levels of abuse and/or symptoms between participant groups, made comparisons between these factors difficult.

The fairly rigorous exclusion criteria for the review may have resulted in some interesting research findings being missed. However, this was felt necessary to ensure that only good quality studies were included, which was evident in the analysis. However, surprisingly, there was still a certain amount of heterogeneity with the measures used to establish levels of BPD. This was potentially due to the varied nature of samples used by the studies and measures needing to be appropriate for the population.

Despite these limitations, the advantages of the current review are that it has been conducted in a systematic way, with a detailed analysis of quality methods used by the studies included prior to exploration of the results.

Future Research and Implications

There are a number of areas highlighted above that warrant future research in order to address the current limitations in the evidence. These centre on the need to account for the apparent multifinality of emotional abuse and neglect. Namely, to establish what factors are predictive of abuse resulting in BPD as opposed to other mental health diagnoses or no diagnosis. The theories and evidence so far suggest that developing a greater understanding of the interaction between genetic pre-dispositions or traits, trauma and BPD is critical to this (Ellis et al., 2011; Paris, 2011). This should involve replicating and extending existing research looking at the mediating and moderating impact of schema/self-representation, affective dysfunction and protective factors. Also, continuing neuroimaging research on changes in brain functioning is necessary. Paris (2011) called for collaborative research considering both biological and environmental factors and the need to

develop research on endophenotypes, or biological markers, within personality disorder, an area in its relative infancy.

Further exploration of findings related to the interaction between different types of trauma and the potential mediating role of emotional neglect/family environment in this is needed. In addition to this, consideration of the nature of trauma would be beneficial, with a potential hypothesis being that it is perhaps the severity of abuse experienced (e.g. number of abuse incidents, frequency, level of physical harm caused) that dictates the impact of abuse, with more severe abuse leading to an increased likelihood of developing BPD over and above other disorders. This research is nearly absent to date, perhaps due to limited sample sizes making it difficult to consider reliably these more detailed aspects in adequate numbers for quantitative research.

As stated by previous authors (Lenzenweger & Cicchetti, 2005; Paris, 2011), there is a need for more prospective research, particularly with at risk groups, in order to gain a lifespan perspective of the development of BPD, which would be the ideal way of further researching these factors. Obviously, the time and economic investments often involved in prospective research present barriers to this. In line with current theoretical developments on differential susceptibility (Ellis et al., 2011), it would be appropriate to give stronger consideration to positive environments experienced, protective factors and the subsequent impact these have on functioning and the development of pathology.

The findings of this review have clinical implications for the psychological assessment and treatment of BPD. It highlights the need for clinicians to take a history of emotional abuse and neglect in their assessments as well as of physical and sexual abuse. The impact of this should then be fed into formulation and treatment

models accordingly. It also has implications in terms of preventative interventions, by giving clinicians, teachers, policy makers etc. the understanding that emotional abuse and neglect can have as serious an impact as other forms of child abuse on development and psychological functioning in later life. Raising awareness of this means the relevant systems can ensure necessary intervention and support is given to families where emotional abuse and neglect is suspected. Educational campaigns for children, adolescents and/or parents on what constitutes emotional abuse and neglect and how to seek help could be important in ensuring early intervention.

Conclusions

The current systematic literature review provides substantial support for a link between the experience of emotional abuse and emotional neglect in childhood and the development of BPD symptoms in later life. However, there is still a lack of specificity to this relationship and research on the factors that account for and contribute to it is in its infancy. The potential importance of changes in brain structure/functioning and certain temperamental/trait and psychological factors have been identified as having a possible impact. There is also some evidence that emotional abuse and neglect may lead to a greater risk of developing BPD than sexual or physical abuse and that it may mediate the impact of these. Further research is needed within this area to develop a more comprehensive understanding of the nuances of this relationship. This could have important clinical implications in the treatment of those who have experienced this form of abuse and in developing preventative initiatives.

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Part 2: Empirical Paper

Stability over Time and the Role of Attachment in Emerging Personality Disorder in Adolescence: A Two Year Longitudinal Study.

Abstract

Aims

Longitudinal studies have found that Personality Disorders (PD) are not as stable as originally conceived (Morey & Hopwood, 2013; Skodol, 2012). The aims for this study were, firstly, to test the hypothesis that PD traits in adolescents decline over time, with symptoms enduring for some, as found in previous research. The second aim was to explore whether parent and peer attachment relationships, as measured at baseline, were predictive of improvement in levels of PD of traits at follow-up.

Method

Assessments were completed by adolescents at baseline, on admission to an inpatient unit, and at a two year follow-up. These entailed self-report questionnaires on personality traits, depression and attachment. The final sample consisted of 31 participants (23 female, 8 male) with a mean age of 15.72 years old at baseline and 17.97 years old at follow-up.

Results

A repeated measures t-test showed a significant decline in the number of PD traits over time. Logistical regression analysis indicated that overall quality of attachment and, more specifically, levels of peer alienation were predictive of improvement at follow-up (meeting criteria for fewer PD traits) when baseline numbers of PD traits were controlled for.

Conclusions

The results support previous literature showing a gradual decline in PD traits over time, as well as highlighting the potential importance of attachment factors in delaying this decline. However, due to lack of statistical power, the findings here cannot be taken as valid and further replication is needed.

Introduction

The Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV) defines a Personality Disorder as "an enduring pattern of inner experience and behavior 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, 2000, p.287). To meet criteria for diagnosis the individual must have impairment in at least two of the following areas: cognition, affectivity, interpersonal functioning and impulse control. Although its onset in adolescence is part of the diagnostic criteria, until recent years, clinicians avoided applying the label of Personality Disorder (PD) to adolescents. However there is now a growing body of research and reviews supporting the presence of PD traits in childhood and adolescence (Cohen, Crawford, Johnson & Kasen, 2005; Fonagy and Luyten, 2009; Shiner, 2009; Tackett, Balsis, Oltmanns & Krueger, 2009). PD traits in adolescence can impact on functioning both in the present (e.g. heightened risk taking behaviours and impairment in relationships) and in future adult life (Shiner, 2009; Skodol, Johnson, Cohen, Sneed & Crawford, 2007), highlighting the need for further understanding and targeted intervention at critical periods (Tackett et al., 2009). The impact of PD traits during this period is compounded by the presence of Axis I disorders, which appear to be highly comorbid (Johnson, Cohen, Skodol, Oldham, Kasen & Brook, 1999; Shiner, 2009).

Stability of PDs in adults

Until recently, stability over time was considered one of the key features of PDs. However, increasing evidence from adult studies suggests that PDs are not as stable as originally thought (Morey & Hopwood, 2013; Skodol, 2012). Currently,

there are four major longitudinal studies of PD from which the bulk of stability research arises: the Longitudinal Study of Personality Disorders (LSPD), the Children in the Community (CIC), the Collaborative Longitudinal Personality Disorders Study (CLPS) and the McLean Study of Adult Development (MSAD) (see Morey & Hopwood, 2013, for review).

The LSPD (n=258) assessed PD traits in an undergraduate population over a 16 year period (Lenzenweger, 2006). The results on stability to date have demonstrated that dimensionally measured PD traits were relatively stable over time (based on both clinical interview and self-report). However, individual growth curve analysis showed significant individual variation both in elevation and rate of change in PD symptoms, concluding that change over time is by no means uniform and contradicting the DSM model of stability (Lenzenweger, 2006). The CIC Study (Cohen et al., 2005), begun in 1975, followed a random sample of individuals (n≈800) living in the New York area from infancy until present day. Assessments took place at under 10 and mean ages of 14, 16, 22 and 33 and have involved measures of Axis I and Axis II disorders, as well as demographic and lifestyle characteristics. Cohen et al.'s (2005) review of CIC findings to date suggested relative stability over time, but also a natural decline. The results of these studies will be discussed in more detail below.

The CLPS (n=668) is based on a clinical population and compares a PD group with a Major Depressive Disorder (MDD) group on rates of Axis I and Axis II disorders (Gunderson et al., 2006; Gunderson et al., 2011). Strikingly, at 10 year follow-up the CLPS study found the remission rate for Borderline PD (BPD) was 85% (defined as meeting 2 or fewer BPD criteria over a 12 month period), with only an 11% relapse rate for those in remission (Gunderson et al., 2011). However, this

group continued to have higher levels of functional impairment compared with those with MDD (Gunderson et al., 2011). These results mirror the findings of the MSAD, which focuses on a group of hospitalised BPD patients (n=290) followed up every 2 years (currently at 16 years) (Zanarini, Frankenburg, Hennen, Reich & Silk, 2006). They found 88% remission rates by the 10 year follow-up, with 61.6% of these remitting in the first 4 years.

These results, particularly from the CLPS and MSAD studies, refute the notion that PDs in adulthood are stable over time. Johnson, Cohen, Kasen, Skodol, Hamagami and Brook (2000) suggested that perhaps PDs should be characterised as having a more variable course with asymptomatic periods, comparing them to physical disorders such as multiple sclerosis and arthritis (Johnson et al., 2000). However, Morey & Hopwood (2013) highlighted methodological concerns when reviewing research on stability over time, including the definition of PD used, the nature of how stability is being measured and the approach used to assess PD (quality and focus of measures used). They stated the need for further research addressing these issues and exploring the development of PDs both in early and later life (Morey & Hopwood, 2013). One current area of debate is whether PD is diagnosed in a categorical (presence or absence of PD) or a dimensional way (symptom counts). Morey and Hopwood (2013) found that longitudinal studies using dimensional measures demonstrated higher estimates of stability than those using categorical criteria, thus evidencing the importance of considering this. This reflects a wider on-going debate or shift in the conceptualisation of PDs as to whether they are on a continuum with 'normal' personality, and therefore dimensional, or are distinctly different and 'abnormal', and therefore categorical (Morey & Hopwood, 2013; Tackett et al., 2009).

Stability of PD during adolescence and early adulthood

Historically, the DSM has advised against diagnosing PDs in adolescence as personality has been considered to be fluid during this period and there are concerns around the impact of stigma attached to this diagnosis at an early age (Chanen, Jackson, McGorry, Allot, Clarkson & Yuen, 2004; Rossouw, 2012). However, recent research has explored the stability of PD traits from adolescence to adulthood and estimated that the prevalence of PDs in adolescence appears similar to adulthood with around one in ten adolescents being likely to meet the criteria for a PD (Shiner, 2009). Rossouw (2012) conducted a systematic literature review into the stability of PD traits from adolescence to adulthood considering both the stability of symptoms and of diagnoses over time. This resulted in 18 prospective studies being reviewed, twelve of which came from the CIC longitudinal study (described above). The findings of the review showed moderate evidence of rank order stability (order of individuals' scores remain the same) over time and that PD traits were relatively stable over time, with a peak in adolescence and a gradual decline in early adulthood (Rossouw, 2012). Findings persistently showed that meeting criteria for a PD in adolescence was predictive of poorer outcomes in adulthood, particularly when comorbid with Axis I disorders, even if the criteria for PD were no longer met.

The CIC studies have demonstrated that PD symptoms present in adulthood have their origins in childhood, with elevated symptoms in early adolescence having negative prognostic implications over the next ten or twenty years (Cohen et al, 2005). Traits were found to be moderately stable during adolescent years and similar to stability witnessed in adult community samples. Importantly, PD traits in adolescence were predictive of PD traits in adulthood. As in Rossouw's review (2012), Cohen et al. (2005) concluded, based on evidence from the CIC studies, that,

although symptoms are stable to a degree, there is a decline in symptoms over time (age 9 to 27), with adolescence being their highest point. For example, Johnson et al. (2000) found a 28% decline in overall levels of PD traits from adolescence to early adulthood within this community sample. This is thought to be partly due to already established maturational processes, such as developmental declines in impulsivity, attention seeking and dependency, and increases in social competence and goal related self-control (Cohen et al., 2005; Johnson et al., 2000). The decline seems to stop at 28, with no age differences found between 28 and 33 years old. The level of stability was comparable across the different PD clusters and developmental stages. Individuals maintained similar symptom rankings relative to same age peers, even as symptom levels declined over time (Cohen et al., 2005). However, there was a sub-group of individuals with the highest symptom scores in adolescence that increasingly differed from a normative symptom group at assessments. The authors highlighted the need for further research to explore which factors were delaying or preventing symptom decline for this sub-group (Cohen et al., 2005; Skodol et al., 2007). Skodol et al. (2007), with the CIC sample, focused on functional impairments and found that those with stable traits from adolescence to adulthood had significant levels of functional impairment, when controlling for Axis I disorders, whereas those whose symptoms remitted in adulthood showed only mild impairment.

All these findings are based on one community sample. Studies on clinical populations are limited and have found mixed results. Chanen et al. (2004) demonstrated the stability of PD diagnosis in adolescent outpatients, with 74% of participants assessed at 16 still meeting criteria for a PD at 18 as measured by the Structured Clinical Interview for DSM Axis II Disorders (SCID–II), with stability shown to be higher in females. However, this still represents a 26% decline in

symptoms. When using inpatient adolescent samples, Mattanah, Becker, Levy, Edell and McGlashan (1995) failed to demonstrate stability over a two year follow-up with a categorical approach and stability was found to be low to moderate in a subsequent study with the same sample using a dimensional/continuum approach to diagnosis (Grilo, Becker, Edell & McGlashan, 2001). Levy et al. (1999) extended these findings by demonstrating that a PD diagnosis at baseline was associated with increased drug use and inpatient admissions at follow-up, but not a measure of global functioning.

From the research reviewed here, it is apparent that further exploration into the stability of personality disorders between adolescence and early adulthood is needed, particularly with clinical samples (Chanen & Kaess, 2012; Morey & Hopwood, 2013; Shiner, 2009; Tackett et al., 2009). It appears that there is evidence of stability to a point but also of a natural decline (Cohen et al., 2005). As yet, researchers do not seem to have explored data further to look at what factors influence this decline and therefore may predict whether personality traits are likely to persist into adulthood for an individual. The current study seeks to extend this research by looking at the stability of PD traits over a two year period in an adolescent inpatient sample and then by considering the role of attachment in the maintenance of PD traits during this time.

Attachment and PD

Attachment has long been linked to the development of PD traits, in particular BPD (Fonagy, 2000; Levy, 2005), with poor attachment relationships with a primary caregiver thought to be mirrored in maladaptive relationships in later life (Tackett et al., 2009). Theories of attachment stem from the work of Bowlby (1980) who postulated that early attachment between a child and caregiver has implications

for the child's emerging concept of the self and the development of their view of the social world and relationships. Bowlby (1980) stated that attachment related behaviour in infancy was part of an evolutionary based biological system, which increases the likelihood of protection from danger and comfort during times of stress, thus enhancing chances of survival (Bowlby, 1980; Levy, 2005). Attachment can be categorised as secure or insecure, consisting of anxious-ambivalent, avoidant and disorganised/disorientated[2] styles (Ainsworth, Blehar, Waters & Wall, 1978; Main & Solomon, 1986). Research suggests attachment styles are largely due to parental factors as opposed to genetic influences and are independent of temperament (Levy, 2005). Neurotransmitters involved in the brain's reward systems, dopamine, opiates, and the hormone oxytocin, have been linked to the experience of secure attachment evidencing its biological basis (Baird, Veague & Rabbitt, 2005; Fonagy & Luyten, 2009)

In a secure attachment relationship the infant has their subjective experiences adequately understood and mirrored back in a containing way by a trusted other. Through this process the infant begins to develop an internalised sense of themselves as an intentional being; *"She thinks of me as thinking and therefore I exist as a thinker"* (Fonagy, 2000, p.1132). This allows the infant to develop the capacity to regulate their own affect and the capacity to mentalize, i.e. to think about their own and other's mental states and intentions (Fonagy, 2000; Fonagy & Luyten, 2009; Fonagy & Bateman, 2008). This psychological containment is necessary for developing a coherent sense of self. A caregiver who can be reflective and mentalize

[2] Attachment styles (as measured by the Strange Situation experiment; Ainsworth et al., 1978; Main & Solomon, 1986): *Secure* - the child has a secure base in the caregiver from which to explore and learn about the world and themselves. *Anxious-ambivalent* - the child is distressed by mother's departure but unable to be soothed by her on return. *Avoidant* - distant from mother and avoids her on return. *Disorganised/disorientated* - showing a lack of coherent response e.g. approach mother but then collapse or freeze (often witnessed in those who have experienced maltreatment; Fonagy & Luyten, 2009).

themselves increases the likelihood of a secure attachment (Fonagy, 2000). Fonagy (2000) stated that insecure attachment from unresponsive parenting may lead to a less integrated self-representation and fragmented and incoherent internal working models. In cases of maltreatment or neglect, the infant may internalise a sense of self as unlovable and unworthy or even as dangerous and learn to see others as abandoning or rejecting (Fonagy, 2000). It is these maladaptive internal working models that are thought to lead to many of the difficulties witnessed in BPD in later life e.g. being fearful of attachment/abandonment, intolerance of being alone, disturbed interpersonal relationships and impairments in the ability to mentalize (Fonagy & Luyten, 2009; Levy, 2005).

Fonagy, Gergely and Target (2007) have recently advanced theories of attachment with the notion of epistemic trust, emanating from a pedagogic stance of intergenerational transmission of cultural learning. This states that not only is secure attachment essential in allowing the infant to develop the ability to mentalize and construct a coherent sense of self, but it also engenders *"basic epistemic trust"* (Fonagy et al., 2007, p.313). This epistemic trust means that the infant more readily learns from an adult with whom they have a secure attachment bond on the assumption that information given will be reliable and relevant. This is a heuristic strategy, allowing the infant to learn faster without having to scrutinise the validity of the source or learn by trial and error, which is a lengthier process and potentially riskier depending on the situation (Fonagy et al., 2007; Allen & Fonagy, in press). This type of efficient learning from others is necessary for the infant to learn how to negotiate the social world in which we live. It is thought that this epistemic trust continues throughout life, meaning that learning from others in adulthood takes place more readily when it is felt that the other is able to mentalize about our minds and

respond to us sensitively (Allen & Fonagy, in press). In those with an insecure attachment relationship, it may be that this epistemic trust was never formed or was destroyed by the caregiver's actions towards the infant, thus potentially inhibiting this form of learning from others, both during infancy and in later life. This may lead to rigid and inflexible social rules based on mistrust, affecting future relationships and the formation of further attachment bonds, as seen in PDs (Allen & Fonagy, in press).

Research has shown low levels of secure attachment within PD groups when using both interview and self-report measures (Levy, 2005). In terms of adolescent samples, Nakash-Eisikovits, Dutra and Westen (2002) found secure attachment to be negatively correlated with all PDs in an adolescent patient sample (n=294), based on a range of clinician rated measures. Disorganised attachment was positively correlated with all PDs, except for antisocial and histrionic (Nakash-Eisikovits et al., 2002). Crawford, Cohen, Chen, Anglin and Ehrensaft (2009) found participants in the CIC study with early maternal separations (under age 5 for over a month) had higher levels of BPD symptoms in adulthood and a slower rate of symptom decline, particularly when separations were for reasons other than mother or child illness. The authors suggested that these early separations may signify a possible lack of maternal investment in caring, leading to the child developing internal working models that their mother did not care about their needs and impacting on attachment (Crawford et al., 2009).

The CIC studies have also found that self-reported attachment anxiety (abandonment fears) and attachment avoidance (uncomfortable with intimacy) in peer and adult relationships during mid to late adolescence (as opposed to parent-child relationships) were associated with higher BPD symptoms in adulthood,

independent of early separation (Crawford et al., 2009). An earlier study showed Cluster B and C PDs in adulthood to be associated with anxious attachment styles in adolescence, whereas Cluster A was associated with avoidant attachment (Crawford et al., 2006). They concluded that not only could the development of PDs be linked to insecure attachment and early separations, but also that attachment insecurity may play a key role in maintaining symptoms from adolescence into adulthood when they would otherwise decline (Crawford et al., 2006; 2009). They encouraged further research to test this hypothesis.

The Current Study

To summarise, recent longitudinal studies have demonstrated that PDs in adulthood are not as stable as originally thought (Gunderson et al., 2011; Zanarini et al., 2006). Research on the stability of PD traits from adolescence into early adulthood has mirrored these findings, concluding that although stable to a degree, there is a natural decline in PD traits over time (Cohen et al., 2005; Rossouw, 2012). However, there appears to be a subgroup of individuals with more enduring traits and higher levels of functional impairment (Cohen et al., 2005; Skodol et al., 2007). To date, few studies have considered what factors may be predictive of change over time in PD traits during adolescence. As discussed above, insecure attachment has long been seen as fundamental in the development of PD traits (Fonagy & Luyten, 2009; Levy, 2005) and researchers have hypothesised that poor attachment could be indicative of the maintenance of symptoms during this developmental period (Crawford et al., 2006; 2009). The current longitudinal study seeks, firstly, to provide further evidence of the nature of change in PD symptoms during adolescence over a two year period and, secondly, to explore whether any changes seen are linked to attachment security.

The study uses the Inventory of Parent and Peer Attachment (IPPA) (Armsden & Greenberg, 1987) as a measure of attachment. As with Crawford et al. (2009), this measure focuses more on current relationships with parents and peers, rather than infant attachment, reflecting a shift in research to consider the importance of attachment across the lifespan (Gullone & Robinson, 2005). However, as stated above, attachment is thought to be relatively stable across time, thus the adolescent-parent attachment relationship will be somewhat reflective of the quality of infant-parent attachment relationship (Armsden & Greenberg, 1987; Bowlby, 1980; Laible, Carlo & Raffaelli, 2000). Both positive parent and peer attachment have been found to be linked to improved self-esteem and life satisfaction, as well as facilitating a move towards independence and identity formation (Armsden & Greenburg, 1987; Laible et al., 2000).

The CIC studies have also evidenced the high comorbidity rates across PD types and between PDs and Axis I disorders, in particular depression, anxiety and disruptive disorders. The presence of an Axis I disorder in adolescence is highly predictive of the emergence or continuation of PD traits in adulthood and vice versa (Cohen et al, 2005; Shiner, 2009). With this in mind, depression has been considered as a covariate in the current study, along with demographic variables of gender and age at admission. Johnson et al. (2000) found age to be inversely associated with levels of PD traits during adolescence. However, no gender differences were found.

Hypothesis

Given the theories and research outlined above, this study aims to test the following two hypotheses:

Hypothesis 1: There will be a natural decline in PD symptoms over time, although symptoms will endure for some.

Hypothesis 2: The quality of self-reported parent and peer attachment relationships, as measured at baseline, will be predictive of the improvement in the number of PD traits at follow-up when covariates of age, gender, baseline PD traits and depression at baseline are controlled for.

Method

Design and Setting

This study was a two year follow-up of outcomes of adolescents admitted to a tertiary adolescent mental health inpatient unit. The primary binary outcome variable was change in PD traits from admission to follow-up (improved versus deteriorated), with the main independent variable being attachment. Other key variables of age, gender, number of PD traits at baseline and depression at baseline were used as covariates. Recruitment was conducted retrospectively at point of follow-up. The study was conducted as part of a larger trial on the effectiveness of Mentalization Based Therapy (MBT) for adolescents with comorbid emerging BPD and depression taking place at the adolescent inpatient service (Rossouw, Fonagy & Eparu, unpublished). The majority of participants' results were included in the MBT trial as part of the Treatment As Usual (TAU) group. Recruitment was carried out with another doctoral trainee researching predictors of continuing self-harm (Given-Wilson, 2013) (see Appendix E for further details on joint working).

In order to establish the sample size needed for sufficient statistical power for the intended logistical regression analysis, the "G*Power 3.1.3" software program (Faul, Erdfelder, Lang & Buchner, 2007) was used. Due to a lack of research in the area and no previous studies using a logistical regression, it was difficult to ascertain an appropriate predicted odds ratio in order to calculate power. After reviewing the literature on power calculations (Kraemer & Kupfer, 2006; Kraemer et al., 2003) and consulting with an expert in the field, an odds ratio of 3 was decided on as this was considered representative of a clinically relevant change. Alpha was set at 0.05 and the desired power set at 0.8, as is conventional. This calculation indicated a required sample size of 50 for the study to be sufficiently powered.

Participants

Sampling began by retrospectively screening the initial assessments and clinical notes of adolescents who completed assessment packs on admission between January 2009 and July 2011 to assess their suitability for inclusion in the study. Of the 141 notes screened, 74 were excluded due to a diagnosis of a developmental disorder (i.e. Autistic Spectrum Disorder or a Learning Disability) (n=37) or a Psychotic illness/Bipolar disorder (n=37). A further six were excluded as English was not their first language or because their admission was very brief (less than a week). This resulted in 61 adolescents being deemed eligible for inclusion in the study, of which 31 consented to take part and completed the follow-up assessment. The consort diagram in Figure 1 gives a breakdown of the recruitment process. Therefore, 51% of those eligible for participation completed the study, which is a lower recruitment rate than in other studies with similar populations (e.g. Levy et al. (1999) had a 61% completion rate). This could partly be due to the retrospective model of recruitment as well as limited or out of date contact details for participants.

In the final sample, 22 participants had comorbid BPD traits and depression and so were simultaneously recruited to take part in the larger MBT trial (Rossouw, Fonagy & Eparu, unpublished) as part of the TAU group. A further nine participants without this comorbidity were recruited solely for the purpose of this study in order. Within the MBT trial participants were not randomised into the TAU or MBT groups, instead recruitment was conducted longitudinally with the TAU group taken from admissions which preceded the introduction of the MBT model at the service. At this time TAU within the unit consisted of a mixture of individual, family and group therapy sessions, as well as regular monitoring by the nursing and psychiatry teams and crisis management when needed. However, there was no overarching model in place and so treatment could be fairly idiosyncratic, with not all clients receiving family therapy, and partly dependent on length of admission.

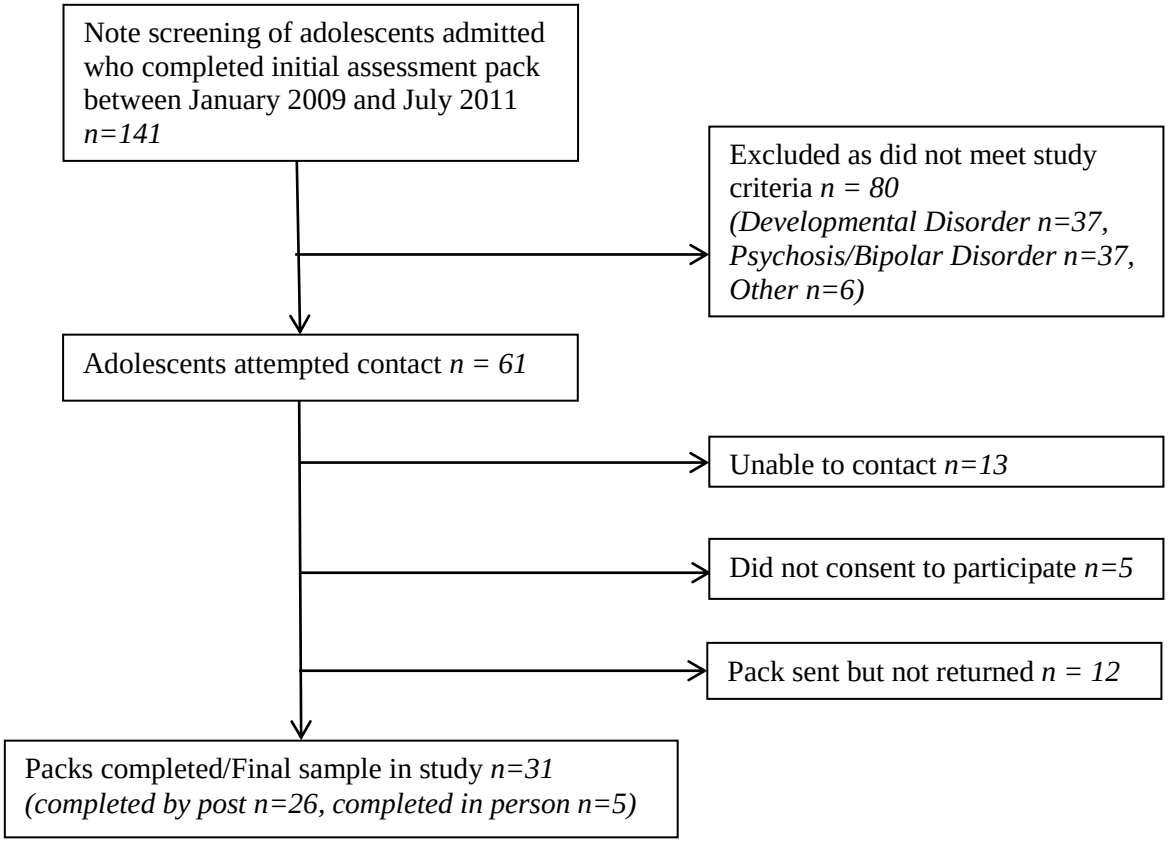

Figure 1. Consort diagram of recruitment stages

To consider whether the final group of participants were representative of the sample as a whole, t-tests and chi-squared tests were used. There was no significant difference in age at admission between those who participated and those who did not (participated – mean = 15.72 years, s.d. = 1.33, n=31; did not participate – mean = 15.41, s.d. = 1.56, n = 30; t (59) = -0.81, p = .42, n.s.) or in gender (participated - female n = 23, male n = 8; did not participate – female n = 24, male n = 6; χ^2 (1) = .291, p = .76, n.s.). Therefore, it can be concluded that our final sample of participants was similar to the larger participant pool in terms of age and gender. Further analysis of differences between the two groups was not possible due to unavailable data.

As stated above, the final sample of 31 was 74% female (n=23) and 26% male (n=8). Mean age at admission was 15.72 years old (s.d. = 1.33, range = 12.73 – 17.80) and mean age at follow-up was 17.97 years old (s.d. = 1.33, range = 14.50 – 20.74). The medium length of admission was 104 days (range 19 – 671 days). The mean time to follow-up from admission date was 2.25 years (s.d. = 0.57). In terms of ethnicity, 64.5% were White British, 12.9% were Black or Black British and 22.6% were other ethnicities or did not state their ethnicity. This ethnic breakdown is consistent with the area's demographic make-up, as reported in the NHS Trust's National Ethnicity Census for 2008 (NELFT, 2008). A clinical records review showed that the majority of participants (n=21) had had further contact with mental health services during the follow-up period.

Ethics

Ethical approval was obtained previously from the local NHS research committee for the MBT trial under which the current study was conducted (REC Reference Number: 10/H0701/123; Appendix F). An amendment was sought to add

additional researchers to the trial and also to gain approval to pay participants, which was deemed necessary to increase response rates (Appendix F). Participants were made aware that all results would remain anonymous and that they had the right to withdraw at any time.

Measures[3]

Millon Adolescent Clinical Inventory (MACI) (Millon, 1993; Millon, Millon, Davis & Grossman, 2006). The MACI was administered at baseline and follow-up to assess levels of PD traits within the sample. The MACI is a widely used measure for assessing personality traits in adolescents, consisting of 160 items with responses of true or false. Results give scores on 31 scales. There are 12 scales relating to personality patterns which parallel diagnoses in the DSM-IV: Introversive (Schizoid), Inhibited (Avoidant), Doleful (Depressive), Submissive (Dependent), Dramatizing (Histrionic), Egotistic (Narcissistic), Unruly (Antisocial), Forceful (Sadistic), Conforming (Compulsive), Oppositional (Passive-Aggressive), Self-Demeaning (Masochistic), Borderline Tendency (Borderline). Other scales include expressed concerns (e.g. social insensitivity, childhood abuse), clinical syndromes (e.g. eating dysfunction, depressive affect) and modifying indices (e.g. level of disclosure or wanting to appear desirable). Scores of over 75 on a scale indicate the presence of a trait and over 85 indicate the prominence of a trait. Norms are gender and age (12-15 or 16-19) specific and based on expected prevalence rates for each scale within the adolescent population (Millon, 1993). Only the scores for the personality pattern scales have been used in this study.

The MACI has been shown to have test-retest reliability ranging across subscales from .71 – .90 (Murrie & Cornell, 2010) and internal consistency ranging

[3] A number of other self-report measures not reported here were also administered at baseline and follow-up as part of data collection for the wider MBT trial and the other trainee's study. The MACI and BDI-Y have not been included in the appendices due to copyright protection.

from .71 - .93 (Millon, 1993; Pinto & Grilo, 2004). Criterion and concurrent validity have been established among general populations and clinical samples (Hiatt & Cornell, 1999; Pinto & Grilo, 2004) and it has been shown to have adequate performance in predicting classes of diagnoses (as rated by clinicians) (Pinto & Grilo, 2004). Baum, Archer, Forbey and Handel (2009) conducted a systematic literature review on studies using the MACI and found a substantial and growing research base for this measure and its clinical utility. The adult version of the MACI, the Millon Clinical Multiaxial Inventory (MCMI), was previously used in a study on adolescent inpatients exploring the prevalence of certain personality traits with adolescents grouped according to attachment style (Rosenstein & Horowitz, 1996), thus supporting its use with the variables in question in the current study.

The Inventory of Parent and Peer Attachment (Short Version) (IPPA) (Armsden & Greenberg, 1987; Laible et al., 2000*). (Appendix G)* The IPPA was administered at baseline to assess attachment within the sample. The IPPA (short version) is a 24 Item self-report questionnaire for adolescents measuring parent attachment (12 items) and peer attachment (12 items) (Laible et al., 2000). Each item is rated on a 5-point scale ('almost always or always true' to 'almost never or never true') with some items being reverse scored. Participants respond according to the relationship with the parent or peer they feel has influenced them most. Items load onto three factors, creating three subscales relating to the degree of mutual trust, quality of communication and the extent of anger and alienation. Armsden and Greenburg (1987) found good test–retest reliability for a sample of 18–20-year-olds over a three-week period (r=0.86 for peer attachment and r= 0.93 for parent attachment) and good internal consistency with Cronbach's alpha coefficients ranging between 0.72 and 0.91 for the sub-scales across both the parent and peer

scales. Laible et al. (2000), using the same shortened version of the IPPA as in the current study, found a Cronbach's alpha of .85 for the parent scales and .84 for the peer scales. Its convergent validity has been supported on the basis of moderate correlations between the IPPA and related measures, including the Family Self-Concept subscale of the Tennessee Self-Concept Scale (r = 0.78 with parent attachment; r = 0.28 with peer attachment) and the Social Self-Concept subscale (r = 0.46 with Parent attachment; r = 0.57 with Peer attachment) (Armsden & Greenberg, 1987; Gullone & Robinson, 2005).

Beck Depression Inventory – Youth (BDI-Y) (Beck, Beck & Jolly, 2001). The BDI-Y was administered at baseline to assess levels of depression with the sample for use as a covariate. The BDI-Y is a subscale of the Beck Youth Inventory, which assesses emotional and social impairment in children and adolescents. The BDI-Y focuses on feelings of sadness, negative thoughts about one's self and future, and associated bodily symptoms. It consists of 20 items rated on a four-point Likert scale (0=Never – 3=Always). Raw scores range from 0 to 60 and are converted to t-scores based on age and gender norms. A t-score of above 56 reflects elevation to a clinical level. The BDI-Y has good reliability and validity among both clinical and general adolescent populations, with coefficients for internal consistency ranging from 0.91 in females and 0.92 in males (Beck et al., 2001; Beck, Steer & Garbin, 1988; Bose-Deakins & Floyd, 2004; Gross & Hersen, 2008).

Procedure

Baseline assessments were completed within one week of the participant's admission to the unit, in accordance with the unit's routine practice. At follow-up, participants were contacted by telephone approximately eighteen months after discharge and given details about the nature of the study to ascertain if they were

willing to participate. Contact details were obtained from the inpatient service's electronic clinical records system, which was linked with other local mental health services. Messages were left on answer phones asking individuals to contact researchers up to five times over a period of three months before they were considered non-contactable. If interested in the study, participants were sent an assessment pack by post (including a pre-paid return envelope) with the questionnaires (Appendix G), information sheets for young people and parents/carers, a consent form (Appendix H) and all relevant instructions. If the participant was under 16 years old, their parents/carers were also contacted and asked for consent. All participants' GPs were informed of participation. Participants were given the option of coming into the unit or completing the questionnaires over the telephone if they preferred. However, only a minority chose this alternative (n=5). Once the assessments were returned, participants were posted a cheque for £10.00 for their time. If a pack had not been returned within a month of being sent then a maximum of three further follow-up calls were made to encourage completion.

Analysis

Analysis was conducted using SPSS Version 21. Preliminary analysis involved using Missing Value Analysis (MVA) to deal with missing data within the sample due to some incomplete baseline questionnaires. Following this, new variables were computed based on overall levels of PD traits (rather than separate types). The sample was then split into two groups of improved versus deteriorated/unchanged (see results section below) and variables were checked to ensure that parametric assumptions of normal distribution were met.

The main analysis consisted of a repeated measures t-test to assess whether there were significant differences in the number of PD traits present at baseline and

follow-up (hypothesis one). Next, exploration of associations within the data and identification of covariates for analysis took place using a correlation matrix. Factor analysis was used to reduce the attachment subscales to an underlying factor, creating a new 'overall quality of attachment' variable. Hypothesis two, concerning the relationship between change in PD traits over time and attachment, was evaluated with independent t-tests assessing differences in attachment scales at baseline between those who improved and those who deteriorated or showed no change at follow-up. Finally, a series of logistical regression analyses were used to consider the extent to which overall quality of attachment and, in a separate model, the individual attachment subscales were predictive of change over time in number of PD traits, when controlling for identified covariates. These models were refined with the removal of non-significant covariates. Each of these steps is discussed in more detail in the results section below.

Results

Preliminary Analysis

Missing value analysis. Due to a number of missing values within the sample, MVA was used to handle missing data. Little's Missing Completely At Random (MCAR) test was non-significant across all variables with missing values, meaning the data could be considered missing at random. Therefore, the Expectation and Maximisation (EM) algorithm function in SPSS was used to compute values for missing data. This is a single imputation iterative approach that first estimates a value for the missing data using a regression equation based on the values of actual observed data. It then determines the Maximum Likelihood (ML) parameters for this estimate and iteratively calculates the best prediction for a value (Dempster, Laird & Rubin, 1977; Graham, 2012). This is thought to be far superior to alternative single

imputation procedures for handling missing data, such as case and pairwise deletion or mean substitution (Graham, 2012).

Computation of variables. Due to the limited sample size, there was insufficient power to consider changes in individual PD types or PD Clusters. Therefore, the MACI PD scores were aggregated into new PD variables giving overall levels of PD traits within the sample. A sum of the number of PD subscales on the MACI that reached cut-off criteria (75 or over) was calculated for both baseline and follow-up for each participant. The number of PD subscales at follow-up was subtracted from the number at baseline in order to create an overall change score (lower numbers indicating more PD subscales reached criteria at follow-up than baseline and therefore representing an increase in PD symptoms over time). A median split (median = 1) was used to split the sample into two groups of those who improved (scoring above 1) (n=17) (labelled 'Improved') and those who deteriorated or remained the same (scoring below 1) (n=14: deteriorated n=9, unchanged n=5) (labelled 'Deteriorated'). This new variable was labelled 'Improvement'. Although this method of dichotomising data is controversial, it is often widely used within psychological research (DeCoster, Iselin & Gallucci, 2009). Farrington and Loeber (2000) support dichotomisation to enable the use of logistical regression therefore obtaining odds ratios, which are seen as giving a meaningful, interpretable and realistic measure of strength of association and highly applicable to considering risk factors in relation to diagnoses. This approach fits with an underlying conceptualisation of PD as being categorical, as adopted here with use of the cut-off scores on the MACI.

Normality Checks. Normality checks were performed to assess whether the data was normally distributed and therefore suitable for the use of parametric tests or

whether any transformations were required. The variables were all found to be within acceptable limits of normal distribution based on Skewness and Kurtosis scores and Kolmogorov-Smirnov tests of normality and inspection of histograms (Field, 2009).

Primary Analysis

Hypothesis 1: Change in PD traits over time. At baseline, 90.3% (n=28) met the criteria for at least one PD as measured by the MACI. By follow-up, this number had reduced to 80.6% (n=25). Table 1 gives a breakdown of the number of participants reaching cut-off for each of the MACI PD subscales at both time points. The most common PD traits in the sample at baseline were introversive, inhibited, doleful, self-demeaning and borderline tendency. At follow-up, although decreased, these were broadly similar. However, there was an increase in submissive traits and a decrease in borderline tendency traits. There may have been variation in which traits were met by a participant at each point which is not reflected in this data.

Table 1
Breakdown of number of participants meeting cut-off individual PD subscales

MACI PD Subscale	Number at baseline	Number at follow-up
Introversive (Schizoid),	16	8
Inhibited (Avoidant),	16	10
Doleful (Depressive),	17	12
Submissive (Dependent),	4	9
Dramatizing (Histrionic),	2	3
Egotistic (Narcisstic),	0	1
Unruly (Antisocial),	3	4
Forceful (Sadistic),	3	2
Conforming (Compulsive),	1	5
Oppositional (Passive-Aggressive),	12	5
Self-Demeaning (Masochistic),	15	9
Borderline Tendency (Borderline).	15	5
Any PD subscale	*28*	*25*

To assess the first hypothesis that there would be a natural decline in PD symptoms over time (although symptoms will endure for some), a repeated measures t-test was used to look at the difference between the total number of PD subscales meeting criteria for a PD at baseline and at follow-up (score of 75 or over on each of

the 12 PD subscales on the MACI). The descriptive statistics for these, including the PD change score, are shown in Table 2. A significant difference was found between the number of criteria met at the two time points ($t(30) = 2.44$, $p = .021$*), meaning there was a reduction in PD symptoms over time, as predicted.

Table 2
Descriptive statistics for number of PD traits on MACI at both time points (≥75)

	Mean (n=31)	SD	Range
No. of PD Traits Baseline	3.35	2.07	0 - 7
No. of PD Traits Follow-up	2.35	1.91	0 - 6
Change in PD traits [a]	1.00	2.28	-3 - 6

[a] Number of PD traits at baseline minus number of PD traits at follow-up

As outlined above, 17 participants showed a reduction in the number of PD traits over the two year period, nine showed an increase and five remained the same. In order to consider the clinical relevance of this, individual participants' change scores within the sample were examined. This showed that only three participants that met criteria for PD traits at baseline did not meet criteria for any PD traits at follow-up, demonstrating a clear clinically significant improvement. One participant had no PD traits at both time points, whereas two who had no traits at baseline had developed traits during the follow-up period. The rest showed varying changes in the number of PD traits over time, with no clear pattern emerging from the data. This demonstrates that, despite the overall statistically significant reduction in the number of traits, there were still a large number of participants meeting criteria for at least one, and often multiple, PD traits within the sample. Not only does this support the current evidence base that PD traits do endure for some during adolescents, it highlights that perhaps in terms of clinical relevance the improvement within the sample was somewhat minimal.

Exploration of correlations within data. Pearson's correlations were used to explore associations between PD traits (using four different variables: improvement, change in number of PD traits and baseline and follow-up numbers of

PD traits) and baseline measures used in the study. The correlation matrix in Table 3 shows high correlations within the different PD subscales and within the different attachment subscales as would be expected. No association was found between gender and PD variables. Gender was correlated with levels of depression, with depression being higher in females. It was also associated with attachment variables, with males showing higher levels of attachment with parents and less alienation from peers. No associations were found between age and the other variables. Depression at baseline was correlated with PD traits at baseline and variables reflecting change in PD over time, but it was not correlated with the number of PD traits at follow-up. Depression was also correlated with levels of parental attachment at baseline. In terms of attachment variables, increased alienation from parents was correlated with PD traits at baseline and poor communication with peers was correlated with PD traits at follow-up. From exploring these associations it was concluded that baseline depression and gender were important covariates to include in the regression model. Further consideration of the relationship between the different attachment subscales was warranted due to the high correlations between them.

Table 3
Correlation Matrix (Pearson's r, n=31)

	PD Variables				Demographics		Clinical Measures	Attachment scales (IPPA)					
	Improvement	Change	PD T1	PD T2	Gender	Age	BDI-Y	PC	PT	PA	PeC	PeT	PeA
PD Variables													
Improvement	––												
Change	.81**	––											
PD T1	.54**	.62**	––										
PD T2	-.38*	-.52**	.35*	––									
Demographics													
Gender	-.06	-.03	-.07	-.03	––								
Age	-.00	.10	.07	-.04	-.18	––							
Clinical Measures													
BDI-Y	.37*	.36*	.66**	.29	-.40*	-.13	––						
Attachment scales (IPPA)													
PC	.01	-.09	-.31	-.23	.47**	-.24	-.42*	––					
PT	-.03	-.14	-.30	-.17	.35*	-.08	-.38*	.83*	––				
PA	-.04	.13	.39*	.27	-.51**	.05	.64**	-.78**	-.69**	––			
PeC	.30	.22	-.11	-.39*	.07	-.23	.08	.36*	.39*	-.33	––		
PeT	.34	.03	-.15	-.20	.15	-.19	.02	.49**	.66**	-.42*	.68**	––	
PeA	-.33	-.10	.14	.27	-.40*	.19	.32	-.58**	-.35*	.71*	-.48**	-.47**	––
OA[a]	.19	-.02	-.31	-.31	.42*	-.20	-.37*	.87**	.85**	-.84**	.65**	.77**	-.75**

Note. Improvement = Improved (1)/Deteriorated (0), Change = no. PD T1- no. PD T2, PD T1 = number of personality disorders at baseline, PD T2 = number of personality disorders at follow-up, Gender = Male (1)/Female (0), Age = age at admission, BDI-Y = Beck Depression Inventory-Youth, PC = IPPA Parent Communication Scale, PT = IPPA Parent Trust Scale, PA = IPPA Parent Alienation Scale, PeC = IPPA Peer Communication Scale, PeT = IPPA Peer Trust Scale, PeA = IPPA Peer Alienation Scale, OA = Overall quality of Attachment.

[a]Overall quality of Attachment was derived following factor analysis of attachment subscales as detailed below.

*$p < .05$. ** $p < .01$.

Factor analysis for attachment variables. As the attachment variables were found to be highly correlated (Table 3), a Principal Components Analysis (PCA), using oblimin rotation, was used to see if the six attachment variables could be reduced to fewer underlying factors. The aim of this was to increase the reliability of the attachment measure and to reduce the number of variables to input into the logistical regression analyses, thereby also increasing the power (Field, 2009; Floyd & Widaman, 1995). Prior to conducting the PCA, the following checks were conducted to ensure this was appropriate for the data set as outlined by Field (2009). The Kaiser-Meyer-Olkin Measure of Sampling Adequacy (KMO) was sufficiently high (KMO = .65) and the Bartlett's Test of Sphericity was significant (χ^2 (15) = 129.92, $p < .001$). The communalities were all above .4 thus confirming that each item shared some common variance with other items. Given these overall indicators, PCA was deemed to be suitable (Field, 2009). Only one factor emerged from the data which explained 62.8% of the variance (Eigenvalue = 3.77). As there was only one factor, no rotations were performed. Table 4 details the factor loadings and communalities for each of the attachment variables. All of the factor loadings were high, indicating a strong pattern within the data. A substantial amount of variance for each variable was accounted for by the factor produced; the highest being 76.4% for the parent communication scale and the lowest being 42.3% for the peer communication scale.

Table 4
Factor loadings and Communalities for PCA of Attachment variables

	Factor Loadings	Communalities
Parent Communication	.874	.764
Parent Trust	.845	.714
Parent Alienation	-.846	.716
Peer Communication	.650	.423
Peer Trust	.770	.594
Peer Alienation	-.748	.560

In summary, the analysis indicated one distinct factor underlying the attachment variables, which appeared to explain a sufficient amount of the data. Exploration of this factor showed it met the criteria for normal distribution (as outlined previously). This factor was named 'overall quality of attachment' and was used in subsequent analysis. The correlations between this factor and other variables can be seen at the bottom of Table 3 above. It was found to correlate highly with the other attachment variables, as expected, and also with gender and depression.

Attachment and improvement over time. Subsequent analysis focused on the improvement score (improved = 1, deteriorated or unchanged = 0) as the primary outcome variable, as outlined above. The second hypothesis in this study was that the quality of self-reported parent and peer attachment relationships, as measured at baseline, would be predictive of the number of PD traits at follow-up when covariates of age, gender, number of PD traits at baseline and level of depression at baseline were controlled for. To assess this initially, independent samples t-tests were used to see if there was a significant difference between the two groups on baseline levels of attachment, prior to considering covariates. Table 5 shows that no significant differences were found for the overall quality of attachment scale or any of the individual subscales. However, baseline levels of Peer Trust were higher and levels of Peer Alienation were lower in the improved group, although the difference missed statistical significance.

Table 5
Independent T-tests for IPPA scales based on Improvement

Scale	Improvement (improved -n=17, deteriorated - n=14)	Mean	s.d.	t	df	Sig.
Overall Quality of Attachment	Improved	0.16	1.04	-1.04	29	.31
	Deteriorated	-0.21	0.90			
Parent Communication	Improved	11.71	4.88	-.04	29	.97
	Deteriorated	11.64	4.63			
Parent Trust	Improved	13.47	4.62	.15	29	.88
	Deteriorated	13.74	5.39			
Parent Alienation	Improved	12.06	4.01	.21	29	.84
	Deteriorated	12.36	3.90			
Peer Communication	Improved	12.53	3.24	-1.68	29	.10
	Deteriorated	10.46	3.61			
Peer Trust	Improved	14.71	4.01	-1.92	29	.06
	Deteriorated	12.02	3.70			
Peer Alienation	Improved	10.94	4.60	1.91	29	.07
	Deteriorated	13.62	2.79			

Hypothesis 2: Prediction of improvement over time. To test the hypothesis that attachment scores predicted the stability of PD once other identified covariates were controlled for, a logistical regression analysis was performed to see if overall quality of attachment was a predictor of improvement over time when controlling for age, gender, depression and the number of PD traits at baseline. As can be seen in Table 6, the initial model with just the demographic measures, which previous work identified as potentially predictive of stability, was not significant (χ^2 (2) = .11, R^2 = .00, p = .95, n.s). The second model, with depression and number of PD traits at baseline added, was significant (χ^2 (4) = 10.17, R^2 = .28, p = .04) and explained 28% of the variance. The third model, with overall quality of attachment, added was again significant (χ^2 (5) = 18.12, R^2 = .44, p<0.01) and the variance explained by the model had increased to 44%. This represented a significant increase in the variance accounted for from the previous model (χ^2 (1) = 7.95, p<0.01). Both number of PDs at baseline and overall quality of attachment were found to be significant

independent predictors of recovery (see Table 6). Age, gender and depression were not significant predictors.

Following this, a second logistical regression analysis was performed with the individual attachment scales entered in a stepwise method to see whether any of them predicted improvement independently (see Table 7). The initial two models were the same as above. In the third model, the peer alienation scale was the only measure to be retained in the model with the stepwise approach. This model was significant (χ^2 (5) = 21.40, R^2 = .50, p<0.01), explaining 50% of the variance, which accounted for significantly more variance than the previous model (χ^2 (1) = 11.22, p<0.01). Peer alienation was found to be a unique predictor of improvement over time as was the number of PD traits at baseline.

Table 6

Logistical regression exploring the role of overall attachment in improvement over time when controlling for covariates

	Model 1				Model 2				Model 3			
	B	Wald test	OR	95% CI	*B*	Wald test	OR	95% CI	*B*	Wald test	OR	95% CI
Gender	0.28	0.11	1.32	0.26 – 6.80	-0.02	0.00	0.99	0.10 – 11.68	0.73	0.21	2.07	0.10 - 46.27
Age	-0.02	0.1	0.98	0.56 – 1.69	-0.11	0.10	0.89	0.44 – 1.79	0.02	0.00	1.02	0.47 - 2.24
PD T1					0.69	3.99*	1.98	1.01 – 3.88	1.06	4.09*	2.90	1.03 - 8.11
BDI-Y					0.00	0.00	1.00	0.91 – 1.10	0.02	0.12	1.02	0.90 - 1.17
OA									1.73	5.56*	5.62	1.34 - 23.59
Chi-Square		0.11				10.17*				18.12**		

Note. OR = odds ratio, CI = confidence interval, PD T1 = number of PD traits at baseline, BDI-Y = Beck Depression Inventory-Youth, OA = Overall quality of Attachment
*$p < .05$. ** $p < .01$.

Table 7

Logistical regression exploring the role of attachment scales in improvement over time using a stepwise model when controlling covariates

	Model 1				Model 2				Model 3			
	B	Wald test	OR	95% CI	*B*	Wald test	OR	95% CI	*B*	Wald test	OR	95% CI
Gender	0.28	0.11	1.32	0.26 - 6.80	-0.02	0.00	0.99	0.08 - 11.68	1.12	0.30	3.06	0.06 - 169.08
Age	-0.02	0.01	0.98	0.56 - 1.69	-0.12	0.10	0.89	0.44 - 1.79	0.28	0.35	1.32	0.53 - 3.28
PD T1					0.69	3.99*	1.98	1.01 - 3.89	1.10	4.58*	3.01	1.10 - 8.23
BDI-Y					0.00	0.00	1.00	0.91 - 1.10	0.04	0.38	1.04	0.92 - 1.17
PeA									-0.57	5.92*	0.57	0.36 - 0.90
Chi-Square		0.11				10.17*				21.40**		

Note. OR = odds ratio, CI = confidence interval, PD T1 = number of PD traits at baseline, BDI-Y = Beck Depression Inventory-Youth, PeA = Peer Alienation scale. Attachment variables excluded from analysis through stepwise method: Parent Communication, Parent Trust, Parent Alienation, Peer Communication and Peer Trust.
*$p < .05$. ** $p < .01$.

Two further models were performed to see if the fit of the model was improved when only the significant predictors were used (Table 8). The first contained baseline number of PDs and overall quality of attachment and was significant (χ^2 (2) = 17.33, R^2 = .43, *p<0.001*) explaining 43% of the variance. The second contained baseline number of PDs and the peer alienation scale. Again, this was significant (χ^2 (2) = 19.57, R^2 = .47, *p<0.001*), accounting for 47% of variance. This shows that the other variables in previous models were accounting for a very small proportion of the variance.

Table 8
Logistical regressions with significant predictors of baseline PD traits and overall attachment/peer alienation

	B	Wald test	OR	95% CI
PD T1	1.26	5.97*	3.51	1.28 - 9.63
OA	1.59	4.72*	4.93	1.17 - 20.75
Chi-Square		17.33***		
PD T1	1.23	7.06**	3.41	1.38 - 8.44
PeA	-0.45	5.63*	0.64	0.44 - 0.93
Chi-Square		19.57***		

Note. OR = odds ratio, CI = confidence interval, PD T1 = number of PD traits at baseline, OA = Overall quality of Attachment, PeA = Peer Alienation scale.
*$p < .05$. ** $p < .01$. ***$p<.001$.

These final logistical regression models suggest that an individual with a higher overall quality of attachment was between 1 and 20 times more likely to show improvement in number of PD traits at two year follow-up. Likewise, an individual with higher levels of alienation from their peers was 0.44 and 0.93 times less likely to show improvement at two year follow-up.

Discussion

The results indicated a significant reduction in the number of PD traits present at two year follow-up. This echoes previous findings that there is a natural decline in PD traits during this period (Cohen et al., 2005; Grilo et al., 2001; Johnson et al., 2000; Rossouw, 2012). As concluded by Johnson et al. (2000), this natural decline is probably accounted for by the developmental maturational processes of

improved socialisation and social competence and a reduction in problematic patterns of interpersonal behaviours (e.g. impulsivity, attention seeking).

However, nine participants showed deterioration over the two year period, five showed no change in the number of PD traits, and 25 out of 31 were still meeting criteria for at least one PD trait at follow-up. This supports the notion that, despite an overall reduction, traits do still endure into late adolescence/early adulthood for some (Chanen et al., 2004; Cohen et al., 2005; Johnson et al., 2000; Rossouw, 2012). It is likely that participants with more enduring traits will have increased functional impairments into adulthood (Skodol et al., 2007).

These results, when combined with findings from adult longitudinal studies, reinforce the need to question increasingly whether PDs are as stable as historically thought (Gunderson et al., 2011; Skodol, 2012; Zanarini et al., 2006). Although still chronic and disabling conditions, the course of PDs seems to be far more variable and symptomatic than stable and life-long, as currently described in the DSM-IV criteria (Johnson et al., 2000; Lenzenweger, 2006; New, Triebwasser, & Charney, 2008). The pattern here perhaps reflects the findings of the LSPD study, that there is considerable individual variation in rates of change of PD symptoms over time, highlighting an increasing need for research into what factors influence these changes (Lenzenweger, 2006).

The current study sought to research further into the stability of PD traits in adolescence by considering whether quality of attachment was predictive of change over time. Attachment levels were not found to differ significantly between those who improved or deteriorated. Peer Trust was higher in the improved group and Peer Alienation lower but not to a statistically significant level. However, once traditional covariates were included in the model, logistical regression suggested that

the number of PD traits at baseline and overall quality of attachment/peer alienation were in combination predictive of improvement and together these variables accounted for a significant proportion of variance in outcome. Other covariates of gender, age, and depression at baseline were not found to be significant predictors. Contrary to what might be expected, the more severe cases (greater number of PD traits) at baseline were predictive of improvement over time. This is likely to be due to regression to the mean, with those with higher scores at baseline having more potential to show improvement/reduction in scores, than those that met criteria for fewer PD traits to begin with (Chanen et al., 2004). Therefore, once regression to the mean was controlled for, quality of attachment became a significant predictor.

Higher overall quality of attachment was predictive of improvement over time in the number of PD traits met. This supports current theories regarding the potential role of attachment in the development and maintenance of PDs during adolescence (Crawford et al., 2006; 2009; Fonagy, 2000; Levy, 2005). Theories predict that those with better attachment relationships with parents and peers (a proxy of the quality of attachment in infancy; Armsden & Greenburg, 1987; Bowlby, 1980; Laible et al., 2000), have more positive internal working models of the self and relationships and are better able to mentalize, thus reducing the risk of developing PDs (Fonagy, 2000; Levy, 2005). Extending this, conceivably those with more positive attachments in adolescence are more likely to show improvement over time in levels of PDs and are able to overcome psychological adversity as they mature into adulthood. This perhaps links with the notion of epistemic trust, with those with more positive attachment experiences being more able to learn implicitly about the social world from parents and trusted peers, enabling the individual to change and develop more flexible and adaptive social rules from others and, therefore, better

equipping them going forward into adult life than those who are unable to learn in this way (Fonagy et al., 2007, Allen & Fonagy, in press).

Interestingly, Peer Alienation was the only individual attachment subscale found to be predictive of change, with higher levels of alienation from peers predicting deterioration over time. This implies that perhaps during adolescence, peer relationships play a more significant role in development than parental ones. Laible et al. (2000) looked at attachment (using the IPPA) in relation to adjustment in adolescents (measured as levels of depression, aggression and sympathy) and found that those best adjusted had good attachment relationships with parents and peers. However, when there was a discrepancy between the two, it was those with stronger peer attachments who showed better adjustment than those with stronger parent attachment. This demonstrates that during adolescence peer attachment may actually have greater significance in terms of adjustment than parent attachment (Laible et al., 2000), findings which have been echoed in the current study in relation to improvement in PD traits.

Although interesting results, the small sample gathered in this study resulted in substantial lack of statistical power and therefore the findings reported above may be suggestive but cannot be considered as statistically valid. Although the models developed seem to fit observed data well, the odds ratios are potentially unrealistically high as the sample size was too limited relative to the number of predictors used. The use of factor analysis with the attachment subscales attempted to reduce this to an extent. The limited sample led to reduced variability within the data set and so the results may not be representative of the wider pool of participants eligible for recruitment and the wider population as a whole. This is evidenced by the failure to find a predictive link between depression and change over time, when

the link between PD traits and Axis I disorders has been fairly robustly established in previous research (Cohen et al., 2005, Rossouw, 2012).

Also, as discussed in part one of this thesis, there are numerous other factors which have been linked to the development of PDs and so may be important in the maintenance of PD traits, which have not been considered within this study but may well account for the observed associations. These include the role of trauma, genetic differences, other Axis I disorders, temperament and potential mediating variables such as mentalization and self-concept (Carlson, Egeland, & Sroufe, 2009; Chanen & Kaess, 2012; Cohen et al., 2005). In addition, screening of clinical records in this study showed that two thirds of the sample had subsequent contact with mental health services during the follow-up period and obviously any treatment received acts as a further confounding variable to the results and has not been considered here.

Limitations

As above, the main limitation within the study was lack of statistical power. However, there are also a number of other limitations which should be considered. The decision to dichotomise the data based on those who improved or deteriorated, although a commonplace method, could be criticised for adding further loss of power (due to reducing variability within data that would have been maintained with a more continuous approach) (DeCoster et al., 2009). However, given the categorical approach to diagnosis adopted here (cut-off score on MACI leading to a trait being classed as present or absent) and the focus on identifying risk factors predictive of future deterioration, it appeared to be an appropriate method to use within the data, as discussed by Farrington and Loeber (2000). In terms of the use of a categorical approach measure of PD, this was thought to be the best way to combine data to look at overall PD traits as the sample size was insufficient to allow for exploration of

separate subscales. However, previous research has shown that this may lead to an underestimation of stability compared with using a dimensional approach (Morey & Hopwood, 2013).

The recruitment rate of only 51% of the eligible original pool of eligible participants was disappointing. This can be attributed partly to the practicalities of following-up participants after a two year interval, in that a number of the contact details were inaccurate or outdated. Added to this, as recruitment was retrospective (i.e. participants were asked to consent at follow-up rather than at baseline) individuals perhaps did not feel as invested or obliged to complete the study as they might have done if they had consented at baseline. A number of hypotheses could be made about the representativeness of the resulting sample. Firstly, the experience the individual had during the admission may have impacted on willingness to participate in research, i.e. those who had a more beneficial experience might be more likely to participate and vice versa. Secondly, given the age range of the sample, those who were not contactable could represent individuals who had moved away from home and/or gone to university potentially resulting in a loss to follow-up of those showing greatest functional improvement. When considering current symptomatology, Chanen et al. (2004) highlighted a concern that high attrition rates could be due to individuals with current symptoms of pathology not participating, therefore resulting in a failure to follow up more severe participants and an underestimation of stability of PD. Allot, Chanen and Yuen (2006) analysed the relationship between difficulty of following-up participants and level of pathology. They found the number of Axis I and Axis II diagnoses at 2 year follow-up was significantly related to how difficult it was to gain participation in the study (i.e. number of contact attempts made), but baseline levels of pathology were not. Based on this, they advised caution in

interpreting longitudinal studies with high attrition rates, as these studies are likely to underestimate rates of continuing disorder with the sample (Allot et al., 2006).

Another concern with the present study is the reliance purely on self-report measures and the impact of this on results. Morey and Hopwood (2013) reported that the use of self-report measures in longitudinal PD studies contributed to higher levels of stability being found. This difference may partly arise from the fact that a self-report measure is inherently a measure of how the individual views themselves as opposed to how they may be viewed by others (clinicians) and observed in behaviour (Morey & Hopwood, 2013). Other researchers have raised concerns about the reliability and validity of using self-report measures within PD populations and the impact that current symptomatology can have on reports (Widom et. al., 2009; Zanarini et al., 2000). As baseline assessments were conducted within a week of admission to the unit, these were reflective of symptomatology when individuals were 'at crisis point' in a very unstable period, potentially leading to an overestimation of PD traits and attachment difficulties at baseline. Conducting assessments once the participant was settled in the unit or re-administering questionnaires at discharge may have yielded different results.

This study relied on the MACI for assessment of PD traits within the sample. Although this is a well-established self-report measure of identifying PD traits within adolescents and relates to DSM-IV PD criteria (Baum et al., 2009; Pinto & Grilo, 2004), it is not a formal diagnostic tool. It may have been preferable to use a more formal diagnostic measure such as the Structured Clinical Interview for DSM-IV – Axis II (SCID-II) (First et al., 1995), which is seen as the 'gold standard measure' for diagnosing PDs (Lobbestael, Leurgans & Arntz, 2011). However, constraints on time and existing baseline data available did not permit this.

Finally, there are on-going debates within the field regarding the applicability of PD diagnostic criteria in the DSM-IV to adolescents, as they were originally developed for the purpose of diagnosing adults (Johnson et al., 2000; Levy et al., 1999; Tackett et al., 2009). Levy et al. (1999) call for criteria to be adapted to take into account developmental processes during adolescence and the interplay between these and the development of PD traits.

Despite the limitations highlighted above, the main strength of this study was that it was a two year follow-up study in a relatively under-researched population. This appears to be the first study to conduct more in-depth analyses of how attachment may relate to stability over time in PD traits during adolescents. Therefore, this should be considered as a pilot study into the relationship which suggests that further in-depth investigation may be warranted.

Future Research and Implications

There is clearly wide scope for future research in this area. The current study needs to be repeated with the limitations highlighted above addressed, most importantly using a larger sample, perhaps across multiple sites, to see if the findings still stand. With a sample of sufficient size, stability for individual PD types could be assessed and more variables could be considered (e.g. childhood trauma, mentalization) to enable a richer evaluation of key factors impacting on stability. This could also enable exploration of mediating and moderating relationships. The use of multiple sources of assessment (clinician rated measures of diagnosis, parent and peer rated measures of attachment, observational methods) would allow for an even more comprehensive understanding to be developed (Morey & Hopwood, 2013). It is conceivable that data from the existing large scale prospective studies (CIC) could be used to look at the link between attachment and stability in more

detail, although some studies have considered this to an extent (Crawford et al., 2006; 2009).

When considering the field of PD research as a whole, until recently research has focused on factors relating to the aetiology of PDs. However, given new conceptualisations of the stability of PDs, there is a clear need to increase research exploring the factors that contribute to the maintenance of PD traits (Lenzenweger & Cicchetti, 2005). This research should emanate from a developmental psychopathology perspective with consideration of the course of PDs during normative developmental processes and reviewing protective as well as risk factors across the life span (Lenzenweger & Cicchetti, 2005; Tackett et al., 2009). The role of genetic and neurobiological factors needs to be integrated within this research (Lenzenweger & Cicchetti, 2005).

In terms of clinical implications, the findings here demonstrate that PDs are not fixed from adolescence and so early identification and intervention is crucial in improving longer term outcomes. Clinicians should consider the importance of attachment patterns within assessments and interventions with adolescents with PD traits. Encouragingly, Rossouw and Fonagy (2012) recently found Mentalization Based Treatment for Adolescents (MBT-A) to be superior to TAU in adolescents with comorbid self-harm and depression. Results showed improved mentalization ability, reduced attachment avoidance and an improvement in emergent BPD symptoms and traits. MBT-A therefore could be beneficial in decreasing the likelihood of PD traits remaining stable into adulthood, by impacting on an individual's ability to mentalize and their attachment relationships.

Conclusions

To conclude, the present study sought to explore the stability of PD traits at a two year follow-up of adolescents admitted to an inpatient unit and to evaluate the role of attachment within this. Findings echoed previous research in that there was a decline in levels of PD traits within the sample over time. However, there was still a significant proportion of adolescents for whom PD traits endured. Higher overall quality of attachment and lower levels of peer alienation were found to be predictive of improvement over time. Unfortunately, due to limitations within the study, namely lack of power, these results cannot be considered valid and further research with larger samples is needed to replicate these findings. In general, there is a lack of research into factors contributing to the maintenance of PD traits throughout the life span, which needs to be addressed in future research.

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Part 3: Critical Appraisal

Introduction

This critical appraisal seeks to reflect on issues that arose during the conceptualisation and implementation of the research project. Key areas to be considered will be the strengths and limitations related to joining an established research trial, difficulties in conducting longitudinal research, the reliance purely on self-report measures, and discussion of some current issues within the field of Personality Disorder (PD) research as a whole. The intention is that these reflections will be beneficial for future researchers and will highlight theoretical concerns pertaining to PDs requiring further exploration.

Joining an established research trial

From the outset of this project, there were clear positives to joining an existing research trial. As outlined in Appendix E – Joint Working, there was a reciprocal relationship setup with the adolescent inpatient service, in that the trainees would conduct the service's follow-up recruitment and assessment for the Treatment As Usual (TAU) group for the Mentalization-Based Therapy (MBT) trial and, in return, would use the data for their respective studies. A clear benefit of this was that there was a strong investment from the research setting in supporting the project, including supervision and use of resources, which eased the recruitment process and facilitated information and knowledge sharing between trainees and clinicians/researchers within the service when necessary. Added to this, conducting the recruitment and data collection stages of the project jointly with another trainee was a positive experience. This allowed for a shared workload and collaborative problem solving around any issues, as well as mutual support, reassurance and encouragement throughout the project. I feel that having two trainees representing the individual needs of the Doctorate in Clinical Psychology projects in relation to

the wider trial enabled the necessary amendments to ethics proposals etc. to be sought more easily and generally resulted in a greater presence of the projects within research team and the service as a whole.

In effect, the research conducted here represents the second stage of a longitudinal study as the baseline data was collected prior to my involvement within the research. Obviously, given the timescales of the doctoral research, conducting a two year longitudinal study would not have been possible otherwise. A benefit of this was that ethical approval had already been obtained, allowing recruitment to begin without delay. However, this meant that the measures used were already fixed, thus somewhat shaping the possible remit of my study. For example, the use of the Millon Adolescent Clinical Inventory (MACI) (Millon, Millon, Davis & Grossman, 2006), a diagnostic screening or clinical assessment tool, meant that only self-reported traits of PD could be considered. If a more formal diagnostic tool had been used, such as the Structured Clinical Interview for DSM-IV – Axis II (SCID-II) (First, Gibbon, Spitzer, Williams, and Benjamin, 1997), this would have allowed consideration of actual diagnoses as opposed to traits. However, the MACI was normed for the age group in question, whereas the SCID-II was primarily constructed for use with adults, and the MACI suited the mode of data collection here, in terms of the brevity of completion and its suitability for completion by post, thus supporting its use. The measures used in the study are discussed further in the section on self-reports below.

Another issue that arose in joining an existing trial related to discrepancies within the data already collected. Firstly, as discussed in the results section of the empirical paper, there was some missing data within the baseline assessments, namely with subscales of the Inventory of Parent and Peer Attachment (IPPA)

(Armsden & Greenberg, 1987; Laible et al., 2000), for which Missing Data Analysis was used. There were limited records regarding the process of assessment at baseline and it was unclear why this missing data situation had arisen. Secondly, in some cases, basic information and demographic data was not adequately recorded at baseline (e.g. length of admission, diagnosis), resulting in electronic records being searched to obtain this data, which was a lengthy and time consuming process. The diagnosis stated on the electronic records was used initially to assess suitability for inclusion in the study (e.g. those with psychosis or learning disability diagnosis were excluded). However, it was often unclear how this diagnosis had been derived and whether any formal assessment had taken place and sometimes numerous diagnoses had been recorded. Due to this uncertainty, the diagnoses were not used for any further part of the study.

I believe that some of these issues represent general difficulties when conducting longitudinal research in a busy clinical setting, where multiple researchers may be involved at different time points and have since left the service, as was the case in the current study. This has highlighted the importance of keeping good quality records at every stage of research, both relating to data collected and to thinking around decisions made, to enable future researchers using the data to understand fully what has already been done and why. It felt in the earlier stages that it took time to establish ownership over the project as a piece of independent research and to negotiate the boundaries between my study, the wider trial and the other trainee's research, partly because elements of the design and methodology were already fixed.

Longitudinal research

As emphasised in the discussion of the empirical paper, lack of power and attrition was one of the major difficulties with this study and resulted in the results not being considered valid. Loss of participants to follow-up is a common difficulty with longitudinal research, with reports of recruitment rates varying from 30% to 80% (Fischer, Dornelas & Goethe, 2001). This is a serious problem in that it creates potential bias within samples. In terms of mental health populations, this bias is prone to lead to more favourable outcomes in research as participants located more easily are likely to be those that are leading more stable lives with better outcomes (Fischer et al., 2001). Demographic variables such as low educational attainment, low socio-economic status, being younger and unmarried/divorced have been associated with higher drop-out rates in epidemiological adult studies (Fischer et al., 2001; Gustavon, Soest, Karevold & Røysamb, 2012). Similar variables have been associated with reduced initial participation of families in research (Pérez, Ezpeleta & Domenech, 2007). In exploring factors related to drop-out of adolescents in an epidemiological study of at-risk families, Pérez et al. (2007) found the risk of attrition was higher when adolescents had more significant life events, required extra support at school and were in contact with mental health services.

Interestingly, attrition rates within the longitudinal PD studies appear to be relatively low. The Children in the Community (CIC) study had retained 84% of their sample at follow-up at age 33 (Crawford, Shaver, Cohen, Pilkonis, Gillath & Kasen, 2006) and the Longitudinal Study of Personality Disorders (LSPD) had a 97% completion rate at follow-up during its first wave of data collection (1990-1997) (Lenzenweger, 2006). Sixty-six per cent of the Collaborative Longitudinal Personality Disorders Study (CLPDS) (Gunderson et al., 2011) and 91% of

participants in the McLean Study of Adult Development (MSAD) (Zanarini, Frankenburg, Hennen, Reich & Silk, 2006) completed 10 year follow-ups. From the two adolescent longitudinal studies (two year follow-ups) the retention rates varied between 61% for Levy et al. (1999) and 96% for Chanen, Jackson, McGorry, Allot, Clarkson and Yuen (2004).

The low attrition rates within these studies may be indicative of the large scale of the projects, therefore having increased resources to aid recruitment, which has been shown to improve retention rates (Allot, Chanen and Yuen, 2006; Pérez et al., 2007). Allot et al. (2006) found difficulty in following up participants was related to levels of Axis I and Axis II pathology at point of follow-up, but not at baseline, stressing the need to follow up as many members of a sample as possible to ensure that representativeness is achieved and those most severe are not missed.

As hypothesised previously, it could be that the retrospective model of recruitment within the current study partly contributed to the lower recruitment rate, as well as out of date contact details being an issue. If consent had been sought at baseline then individuals may have been expecting the service to contact them at some point and may have felt a degree of investment in continuing with the research. However, as potential participants were unaware of the study until they received a telephone call asking them to participate, this could have meant they were less likely to take part. For many, their admission to an inpatient unit was probably a very difficult experience and representative of a distressing period in their lives from which they may wish to distance themselves. Therefore, receiving a telephone call from the service 'out of the blue' could have brought back uncomfortable memories for some, reducing the likelihood of their participating. Also, as late adolescence is quite a transitional period with changes in educational/work settings, it could be that

participants were concerned about new people in their lives finding out about their admission if they saw correspondence from the service, again decreasing the likelihood of participation.

The decision to reimburse participants £10 for their time in completing the study appeared to have a significant impact on recruitment. Qualitatively, during telephone calls discussing the research adolescents became much more engaged once the offer of payment had been made. It is thought that the recruitment rates may have been even lower had this step not been taken. Although there could be some ethical concerns with regards to reimbursing participants for their time, in terms of coercion to participate, this is now a fairly widely used approach to increase participation in studies, particularly longitudinal ones (Borzekowski, Rickert, Ipp & Fortenberry, 2003). In focusing on adolescent research, Borzekowski et al. (2003) found that of 127 studies, 55% compensated participants for their time. Payment was not found to impact on the quality of data e.g. participants leaving questions blank or choosing multiple responses.

The small sample size limited the number of possible predictors it was acceptable to input into the statistical analysis. There were a number of other questionnaires for which data was obtained as part of the wider MBT trial which would have been theoretically relevant to include when considering change over time in PD traits and the link between this and attachment, such as previous trauma, self-concept and mentalization. However, this would have resulted in an even greater loss of power. A far larger research trial would be required to consider all these potential variables.

Self-report measures

This study, as is common in research, relied solely on the use of self-report measures. These have numerous benefits for researchers in that they are often easier to administer and take less time than interview measures. They can be completed by participants independently allowing different data collection methods, such as completion by post, as in this study. This, in turn, reduces resources needed, i.e. rooms to interview participants, which are often difficult to obtain in busy clinical settings. The result is less time-consuming for both participants and researchers and therefore less costly (Patel, Doku & Tennakoon, 2003).

However, there are a number of concerns with relying purely on self-report measures. As articulated in the discussion of the empirical paper, Morey and Hopwood (2013) found the mode of measurement (self-report versus clinician interview) impacted on the results of studies, with higher rates of stability in PDs being found with self-report questionnaires. The use of self-report measures for assessing personality disorder has been criticised by researchers. Studies have shown only a modest correlation between self-reports and informant reports of personality and with interview based measures (Klonsky, Oltmanns, & Turkheimer, 2002), with self-reports having a tendency to result in over-diagnosis (Hopwood et al., 2008). It is thought that self-report measures may be affected by current levels of symptoms, particularly in more acute hospital settings (Zanarini et al., 2000). Participants with PD may potentially lack the necessary insight to judge their own personality difficulties or may manipulate results in order to present in a certain way (e.g. cry for help) (Hopwood et al., 2008).

Westen and Shedler (2007) advocate for the use of clinician rated measures, stating these are beneficial as assessment can encompass internal states and

externally observed behaviours because clinicians are trained to have the skills to assess both. They assert that not all personality processes are accessible to the individual, due to their implicit nature or denial of difficulties, and therefore cannot be measured by self-report. Skilled clinicians are able to elicit information relevant to assessing personality difficulties, including ways of regulating emotions and interpersonal relationship styles etc., which allow in-depth conclusions to be drawn (Westen and Shedler, 2007).

With this in mind, I had hoped to use the Shedler-Western Assessment Procedure - II - Adolescent version (SWAP-II-A) (Westen, Shedler, Durrett, Glass & Martens, 2003) within the study as well. The SWAP-II-A is a 200 item Q-sort instrument for assessing adolescent personality pathology, providing both dimensional and categorical diagnosis across the personality disorder subtypes, to be completed by clinicians who have longitudinal knowledge of patient. It aims to harness the advantages of clinical knowledge and intuition, but uses these in a statistically and empirically validated tool (Shedler & Westen, 2007). Clinicians are required to sort 200 statements into categories based on how descriptive they are of the patient (not descriptive/irrelevant = 0 to highly descriptive = 7) and need to assign a specified number of items to each category.

The aim was that clinicians at the service would complete this retrospectively, based on their knowledge of participants while they were at the unit, therefore providing another baseline assessment of PDs. This would have allowed for exploration of the validity of using this measure retrospectively and also a comparison of levels of agreement between self-reported measures of PD in adolescents (MACI) and clinician rated ones (SWAP-II-A) (a potential study within its own right).

However, the SWAP-II-A can take around 40 minutes to complete per participant and even longer when the clinician is unfamiliar with the process. It became apparent during the course of the study that the demands on clinicians' time were too great to allow for completion of this measure. The service has started using it for current participants in the MBT group of the trial, but to complete it retrospectively for those in the TAU group, when there would be no clinical utility for clinicians, was not a priority. Added to this, as this version of the measure is relatively new, the scoring programme for it is not yet publicly available. Contact was made with the developers of the SWAP in American and they did agree to aid with scoring of the measure. However, they were often slow to respond to correspondence and there was a concern that, even if the measures were completed, the results would not be obtained in sufficient time for inclusion in this study. Therefore the decision was taken to abandon the use of this measure within the study, resulting in the research relying solely on self-reports.

In relation to the other main construct in this study, there have also been criticisms of the use of self-report measures for attachment. Ainsworth's attachment theories were based on the notion that attachment is an internal working process that is partially unconscious and therefore not conducive to the use of self-report (Wilson & Wilkinson, 2012). However, reviews have shown that there is no empirical basis for this criticism and that self-report measures of attachment are able to assess the unconscious and implicit nuances of attachment processes in a reliable and valid way (Shaver & Mikulincer, 2004; Wilson & Wilkinson, 2012). Within the context of this study, I wonder whether, due to the recency of admission, self-reports of attachment at baseline were likely to be more negative and reflective of potential transient relationship difficulties (e.g. strain put on adolescent-parent relationship due to

admission, experienced rejection by peers due to admission) rather than of more longitudinal attachment bonds. The IPPA only measures attachment within specific relationships and perhaps the use of a measure that allowed for assessment of more global working models of attachment, which have been shown to overarch relationship specific attachments, would have also be beneficial (Overall, Fletcher & Frissen, 2003; Shaver & Mikulincer, 2004).

Wider issues in PD research

From conducting both the research study and the literature review a number of issues within PD research/diagnosis at present became apparent. It is beyond the scope of this appraisal to review these fully here and I would refer readers to Morey and Hopwood (2013), Shedler and Western (2007) and Skodol (2012) for more in-depth discussion. From Morey and Hopwood's (2013) review, it is evident that the way PD is conceptualised and measured within research has a significant impact on the outcomes found and therefore knowledge and consideration of the following points are crucial when designing and implementing research studies. Firstly, there is inconsistency within the current literature with the use of categorical vs. dimensional methods of diagnosing PDs. This is related to fundamental differences in the conceptualisation of PD as to whether it is seen as on a continuum with 'normal' personality traits and representative of the extreme ends of these, a dimensional approach, or whether it is seen as something markedly different and separate from 'normal' personality and thereby 'abnormal', a categorical approach, as in the current DSM-IV system (Morey & Hopwood, 2013; Skodol, 2012; Tackett, Balsis, Oltmanns & Krueger, 2009).

Skodol (2012) criticised the DSM-IV approach for proposing arbitrary cut-offs (e.g. meeting five out of nine symptoms), without any sound empirical bases. A

proposal was put forward for the new DSM-5 to be based on a hybrid dimensional-categorical approach. This stipulated the retention of six specific PD subtypes (antisocial, avoidant, borderline, narcissistic, obsessive-compulsive, and schizotypal) and inclusion of PD Trait Specified (PDTS) (for those not meeting any subtype), to be based on meeting certain criteria (categorical). Guidelines allowing for assessment of levels of personality functioning and pathological personality traits (five board trait domains, with nine more specific facets for each domain) using a continuum approach (Skodol, 2012) were also proposed. However, this model was not accepted for the final version of the DSM-5 published in May 2013 as it was deemed too complex for clinical practice (American Psychiatric Association, 2013). Instead, the original categorical system with the same ten personality disorder diagnoses was retained. It has been included in Section III of the DSM-5, which stipulates areas for further research (American Psychiatric Association, 2013). The combining of the Axis I and Axis II diagnoses into one category in the DSM-5 will hopefully be helpful in dispelling the myth that PDs are distinctly different from other mental health disorder and perhaps, in time, reduce the stigma associated with these diagnoses.

Secondly, another difficulty, which partly arises from the categorical approach, is the high rates of comorbidity between different types of PD. Shedler and Westen (2007) state that individuals meeting criteria for a PD, will often fit the criteria for between four and six different PD types, evidencing a lack of discriminant validity. In addition, there is huge heterogeneity with individuals diagnosed with a certain type of PD. For example, it has been found that there are 256 possible ways to meet the criteria for BPD within the DSM-IV (Johansen, Karterud, Pedersen, Gude & Falkum, 2004). Interestingly, in reviewing the research

literature, the issues with comorbidity and heterogeneity do not often appear to be discussed. There is a tendency in studies to combine PDs when conducting research, either referring to the different PD Clusters or to any PD. This made it difficult to summarise results across studies in the literature review when attempting only to focus on borderline PD. The combining of PD diagnoses in research may partly reflect the difficulty of analysing so many different subtypes when sample sizes are limited (as was the case with this study) and also the problem of how to categorise participants when they meet criteria for more than one type of PD. Again, it was generally hoped that changes could be made to the diagnostic criteria in the DSM-5 aimed at reducing the comorbidity and heterogeneity (Skodol, 2012). Since this has not come to fruition it is unclear how this may be resolved. Researchers clearly have an important role in exploring and documenting these issues within their samples to support potential diagnostic changes in the future.

Another concern raised regarding diagnoses is that the diagnostic criteria for PD have been developed for use with adults and have never been adapted for adolescents or children. Exploration is needed of whether criteria should be modified to make them more applicable for use with this younger age group, particularly given the increase in research and diagnoses of PD during adolescence (Johnson, Cohen, Kasen, Skodol, Hamagami & Brook, 2000). Levy et al. (1999) stated that it is unlikely that the presentation of PD in adolescence would directly mirror its presentation in adulthood, particularly given the vast number of developmental processes during this period. They stress the need for a developmental psychopathology approach, taking into account typical development and how and why those in the prodromal stages of PD diverge from this course.

Tackett et al. (2009) reviewed differences in the development of PD at each stage of adolescence in relation to normative development.

Finally, a frustration that arose when reviewing the literature was the number of studies that used the same sample. In the literature review, 11 of the 39 studies shared samples. Obviously, the majority of these were longitudinal studies where multiple publications over time are necessary. However, it is still worth reflecting on the size of the overall sample pool on which PD studies are based and that this is not as large as it may seem from the number of studies published in the field, thus impacting on the potential generalizability of our knowledge of PD. It was not always made clear when multiple publications had drawn from one sample and inferences had to be made from comparison of sample sizes, procedures and authors. It is important that researchers clearly state when a sample has been used previously to allow for assessment of the impact of the findings given what is already known from that sample. Additionally, as is often the case with research, the majority of the studies have been conducted in America and little thought has been given the applicability of findings to different countries.

Conclusions and Recommendations

This appraisal has presented some of my reflections on the process of conducting a longitudinal research project on the stability of PDs and attachment in an adolescent population. I have discussed the strengths and limitations of joining an existing research project and how this can an impact on the nature of the study produced. These may be important points for future researchers to consider when embarking on collaborative research. The difficulties with conducting longitudinal research have been highlighted, including further exploration of potential reasons for the low response rate within this study. Research has shown that high attrition rates

are likely to bias study outcomes, leading to an underestimation of pathology as participants who are more severe are more likely to dropout (Allot et al., 2006; Pérez et al., 2007). In hindsight, perhaps more consideration of how to meet the response rate required to achieve sufficient power given the finite sample for recruitment (50 out of 61, 82%) would have been helpful at the outset of the study. However, as baseline data had already been collected, I am uncertain as to what could have been done differently to improve this.

Another limitation to this study was the reliance of self-report measures. I have considered the concerns in relation to this for both the assessment of PD and of attachment and also outlined steps that were taken to attempt to use a clinician rated measure. Self-reports are heavily relied on in research and future studies may benefit from incorporating multiple sources of assessment where possible, particularly when researching PDs (Morey & Hopwood, 2013).

Finally, I have presented an overview of some concerns or shifts within the area of PD at present, namely the approach taken to diagnosis, comorbidity and heterogeneity, applicability of diagnostic criteria to adolescence, and the generalizability of samples used in research. It is perhaps disappointing that the new DSM-5 has conceivably fallen short of addressing some of these points by making no changes to the diagnostic criteria and hopefully amendments will be made in the future. My understanding of these issues has developed throughout the course of this research and I think that having a clearer idea of them during the design stages of the project would have been beneficial. I hope that by presenting them here, future researchers will be able to consider the implications of these when conceptualising and conducting their research.

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Appendices

Appendix A

Criteria for Critical Appraisal of Studies (Young & Solomon, 2009)

Criteria for Critical Appraisal of Studies (Young & Solomon, 2009)

Reference
Young, J. M., & Solomon, M. J. (2009). How to critically appraise an article. *Nature Clinical Practice: Gastroenterology & Hepatology, 6*, 82-91.

Appendix B

Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement (Von Elm et al., 2008)

STROBE Statement - (Von Elm et al., 2008)

Appendix C

Table of Summary of Studies in Literature Review

Table of Summary of Studies in Literature Review

Reference	Design	Measures		Participants	Key Findings
		Trauma	BPD		
Afifi et al. (2011)	CS	CTQ (subset)	AUDAD IS-IV	National Sample (n=34,653) 8.1% of sample reported EA, 9.4% reported EN Of those with BPD (n not stated) 28.6% reported EA and 21% reported EN	Odds Ratio analysis showed EA and EN to be significantly predictive of a diagnosis of BPD as well as other Cluster A and Cluster B disorders.
Battle et al. (2004)	CS	CEQ-R	DIPD-IV	Patients Any PD (n=517) (of which BPD n=214) v.s. MDD (n=83) Any PD - EA=54%, VA=56%, N=68% BPD - EA=66%, VA=65% N=90% MDD - EA=30%, VA=39%, N=68%	Higher means scores on EA, VA and N in the BPD group than MDD group. Chi-squared tests showed sig. higher levels of all three when comparing all PDs to MDD (did not do separate analysis of BPD)
Bellino et al. (2005)	CS	CEQ-R	SCID-II	Outpatients BPD&MDD (n=45) v.s. MDD (n=74) BPD&MDD - V/EA=60% MDD - V/EA=35%	Chi-squared showed sig. higher levels of V/EA in comorbid BPD&MDD group than in MDD alone group.
Bierer et al. (2003)	CS	CTQ	SCID-II	PD Outpatients (n=182, of which BPD = 71) In whole sample EA=59.9%, EN=57.1% (did not give % for just BPD)	Found a trend correlation between BPD and EA but not with EN
Bornovalova et al. (2010)	CS	CTQ – (EA Subset)	SCID-II	Substance Users (n=382) Used latent class analysis to put into 4 BPD groups: Class 1 - no BPD=40.2%, EA mean=1.59 Class 2 = 25.3%, EA mean=1.83 Class 3 = 26.97%, EA mean=2.34 Class 4 - high BPD = 7.71%, EA mean=2.93	ANCOVA (gender and income controlled for) found significant differences between the classes with LSD Contrasts showing Class 4 to be significantly higher than others and Class 3 to be significantly higher than class 1 and 2

Bradley et al. (2005)	CS	CDF	Clinician rating and SWAP-200	Patients via Clinician reports (n=524, 26.6% meet criteria for BPD) For whole sample mean scores for family stability=4.18, family warmth=3.05, relationship with parents=5.75 (all on 7 point scale)	Significant correlations between BPD diagnosis and all three family environment measures, shown to be independent predictor in regression analysis and family environment was showed to partially mediate the relationship between BPD and SA and PA
Carlson et al. (2009)	LP	CpS	SCID-II	At risk sample (based on poverty, young single mother and mothers educational attainment) (n=162) followed from birth to 28. BPD-mean score on SCID symptom count at age 28=0.46 SD=1.16 (on 0-7 point scale) Composite rating of maltreatment mean (54months to 18 years)=0.32, SD=0.83, range=0-4	In early childhood (data collected between 0 and 5 years old) maltreatment, attachment disorganisation, maternal hostility, maternal boundary dissolution and life stress were all found to significantly correlate with BPD symptoms at age 28. As was parent-child relationship disturbance (measured at age 13 via observation of interactions) and a composite score of family disruption from 1 to 18 years. Self-representation in middle childhood found to mediate relationship between attachment disorganisation in infancy and BPD symptoms in adulthood
Crawford et al. (2009)	LP - CIC	CpS	SCID-II	Community Sample (n=766) (CIC study, 0-33 years) 37 in whole sample met legal criteria for SA, PA or N 35 had maternal separation before age 5	Participants with early separations (especially those for reasons other than illness) had significantly higher levels of BPD symptoms in adulthood and that the decline in these symptoms between 22 and 30 was slower than in those without maternal separations.
Driessen et al. (2000)	CC	CTQ	SCID-II	BPD patients (n=21) vs. Healthy Controls (n=21) BPD - PA&EA mean=3.2 (0.8), EN=3.5 (0.6) Controls - PA&EA mean=1.5 (0.7), EN=2 (0.5)	Mann-Whitney U showed significant difference between the two groups for both combined PA&EA scores and EN scores. Significantly smaller hippocampal and amygdala volume found in BPD compared to controls. Difference only found to negatively correlate with trauma scores when groups analysed together.

Giesen-Bloo & Arntz (2005)	CC	STI	SCID-II	BPD Patients (n=14) v.s. Cluster C Patients (n=14) v.s. Axis I Patients (n=19) v.s. Healthy Controls (n=21) Mean trauma composite scores: BPD M=93.73 (30.72) Cluster C M=56.21 (39.27) Axis I M=42.28 (23.10) Control M=29.25 (23.19)	ANOVA showed significant differences between groups with BPD patients having the highest mean scores. Also found BPD patients to perceive the world as more malevolent, that they have less luck and lower levels of self-esteem compared to other groups. However, these were found to be independent of the experience of trauma.
Goodman et al. (2003)	CS	CTQ	SCID-II	BPD Patients (n=61) vs. Other PD (n=112) Mean Emotional abuse scores: BPD M=15.9 (5.01) Other PD M=13.5 (5.4)	Correlated CTQ with affective measures instead of SCID. Found significant correlation between emotional abuse and affective liability and affective intensity in the other PD group but not the BPD group.
Gratz et al. (2008)	CS	CTQ	SCID-II	Substance Users (n=76) 31.6% (n=24) of sample met criteria for BPD 26.3% (n=20) of whole sample reported a history of EA Overall childhood maltreatment mean for BPD sample =63 (24), for non-BPD = 43.84 (14.98)	Substances Users with BPD reported significantly higher levels of childhood maltreatment than those without BPD. Emotional dysregulation fully mediated the relationship between childhood maltreatment and BPD diagnosis, but not BPD symptom count.
Gratz et al. (2011)	CS	CTQ – (EA subset)	CPNI	Children ages 11-14 (n=225) BP features M=15.05 (4.14) (scale range 9-36) 9.3% of sample reported moderate to severe history of EA	Found a significant correlation between with EA and BP features. A hierarchical regression model showed that EA accounted for unique variance in BP features above personality traits (e.g. impulsivity affective dysfunction)
Grover et al. (2007)	CS	CTQ	SCID-II	Community Sample (n=61) Abused - n=28 Non-abused n=33 NB: no significant differences found in symptom levels between those who reported only EA compared to those who reported SA or PA	BPD symptoms higher in abused group than non-abused group.
Gunderson et al. (2006)	LP	CEQ-R	DIPD	BPD Patients - 2 year follow up of outcomes (n=160) Levels of abuse in sample not given	Abuse and neglect were not significant predictors of no. of BPD criteria met at 2 year follow up.

Heigeland & Torgersen (2004)	LR	Rating of hospital records	SIDP-IV	Adolescent inpatients followed up in adulthood (n=132) BPD n=25, no BPD, n= 107 Total trauma scores: BPD M=7.12 (4.19) No BPD M=4.91 (3.32)	Significant difference between BPD and no BPD on total scores and abuse and environmental instability scores.
Horesh et al. (2008)	CC	CTQ	DIB	Adolescence patients (n=59) BPD n=20, MDD n=19, Healthy controls n=20 Scores for EA and PA combined: BPD M=56.2 (13.3) MDD M=49.9 (12.79) Control M=40.5 (10.7)	ANOVA showed there to be a significant difference between both clinical groups and control group, but not between the MDD and BPD groups.
Huang et al. (2012)	CS	CECA-Q	SCID-II	Patients (study in China) (n=382) BPD n=203, Other PD n=109, Non PD n=70 Mean scores for Emotional Antipathy by parent: BPD - Mother M= 20.55 (5.98), Father M=21.99 (6.27) Other PD - Mother M= 17.98 (5.61), Father M= 18.39 (5.43)) No PD - Mother M=16.96 (5.14), Father M=18.80 (5.57)) Mean scores for Neglect: BPD - Mother M=19.16 (5.55), Father M=21.89 (5.96) Other PD - Mother M=16.47 (4.94), Father M=19.25 (5.86) No PD - Mother M=15.97 (4.30), Father=19.84 (5.62)	Significant higher levels of emotional antipathy and neglect from both parents in the BPD groups compared to other PD and no PD groups.
Johnson et al. (2000)	LP - CIC	CpS	PDQ	Community sample n = 738 followed up from infancy to early twenties Prevalence in sample of any PD 24.4% (n=180), prevalence of BPD 2.8% (n=21)) Prevalence in sample of any neglect 12.9% (n=95), prevalence of EN 2.8% (n=21)	Found that emotional, physical and supervision neglect were associated with an increased risk for PDs in early adulthood, when controlling for age, gender, physical and sexual abuse. However, only supervision neglect was found to be a significant predictor of BPD symptoms in early adulthood and not emotional or physical neglect.
Johnson et al. (2001)	LP - CIC	CpS	PDQ	Community sample n = 793 followed up from infancy to early twenties Verbally abused - n=78, not verbally abused - n=715 Prevalence of BPD in abuse group = 7.7% (n=6) Prevalence of BPD in not abused group = 1.8% (n=13)	Found experience of verbal abuse to be significantly predictive of BPD symptoms in adulthood, when PA, SA and other neglect controlled for.

Joyce et al. (2003)	CS	PBI and clinical interview	SCID-II	MDD Outpatients (n= 180) 17% (n=30) met criteria for BPD Moderate or Severe abuse and/or neglect - n=109	Increasing levels of abuse and/or neglect significantly increased the probability of having a diagnosis of BPD and also avoidant PD. Also looked at borderline temperament (novelty seeking and harm avoidance), childhood/adolescent depression, conduct disorder, hypomania and drug and alcohol misuse and found these to increase risk of BPD too.
Joyce et al. (2006)	CS	PBI and clinical interview	SCID-II	MDD outpatients (n=335) 25.8% of sample met criteria for BPD 138 had experienced moderate abuse or neglect (18.8% of which had BPD 38 had experienced severe abuse or neglect (33% of which had BPD	Experience of abuse and/or neglect was significantly predictive of a BPD diagnosis. Also found a significant association between BPD and the 9-repeat allele dopamine transporter (DAT1).
Kingdon et al. (2010)	CS	CTQ	SCID-II	Patients (n=111) BPD n=59, Schizophrenia n=33, Comorbid n=19 % of severe EA in each group: BPD - 92%, Schizophrenia - 43%, Comorbid - 82% % of severe EN in each group: BPD - 26%, Schizophrenia - 63%, Comorbid - 78%	EA and EN significantly higher for BPD and Comorbid group than Schizophrenia group
Lange et al. (2005)	CC	TAQ	SCID-II	BPD inpatients (n=17) v.s. Healthy Controls (n=19) Mean Levels of neglect: BPD - M=5.4 (1.3) Controls - M=3.1 (0.7)	T-tests showed significantly higher level of neglect in the BPD group. BPD had reduced glucose metabolism in right-sided ventromedial temporal and left sided parietal/posterior cingulate cortices.
Laporte & Guttman (2001)	CC	FIPE	DIB-R	BPD Patients (n=34) v.s. Anorexia Patients (n=34) v.s. Healthy Controls (n=33) Percentage of verbal abuse in sample: BPD - 71%, Anorexia - 35%, Control - 39%	Chi-squared analysis showed significantly higher levels of verbal abuse, as well as sexual and physical abuse in the BPD than other groups. Reports of abuse were corroborated by parents, thus providing support for reliability of retrospective reports of abuse.
Laporte et al. (2011)	CC	CTI	DIB-R	BPD Patients (n=56) and their Sisters (n=56) Percentage of EA in sample: BPD - 76.8%, Sisters - 53.4%	T-tests showed significantly higher levels of EA in BPD group compared to their sisters. Only three sister pairs were concordant for BPD, despite reporting broadly similar experiences of maltreatment. Provides support for reliability of retrospective reports of abuse.

Lobbestael & Arntz (2010)	CC	ITEC	SCID-II	Patients (n=147) BPD (n=45) v.s. ASPD (n=21) v.s. Cluster C (n=46) v.s. Healthy Controls (n=36) Median Levels of trauma in sample: BPD - Med=44.42, ASPS - Med=24.15, Cluster C - Med=29.69, Controls - Med=4,25	Found a significant difference between BPD and Cluster C and Control group for levels of trauma but not with ASPD group. Looked at emotional reactivity to abuse-related stress (via reaction times following abuse-related film clip). They found those with BPD scored higher on self-reported negative affect, maladaptive schema modes and physiological hyper-reactivity.
Lobbestael et al. (2005)	CS	VBG	SCID-II	Patients (n=48) BPD (n=16) v.s. APD (n=16) v.s. Healthy Controls (n=16) Mean levels of EA in sample: BPD - M= 44.5(12.86), APD M=43.56 (14.73), Controls - M=5 (6.74)	Found a significant difference between BPD and Control group but not APD group. The same was found for SA and PA. Also found high levels of maladaptive schemas in both PD groups
Machizawa-Summers (2007)	CC	CTQ	BSI	Outpatients (n=90) (study in Japan) BPD (n=45) v.s. Non-BPD (n=45) Mean EA in groups: BPD - M=15.38 (5.33), Non-BPD - M= 9.49 (4.38) Mean EN in groups: BPD - M=16.78 (4.45), Non-BPD - M=11.62 (4.59)	MANOVA showed significantly higher levels of EA and EN in BPD group, as well as lower levels of maternal and paternal care and higher levels of maternal and paternal overprotection. Regression analysis showed EA, EN and paternal overprotection to be significant independent predictors of BPD diagnosis but not SA or PA. Regression analyses showed EA, EN and parental overprotection sig, independent predictors
Rogosch & Cicchetti (2005)	CC	Official Records	BPD precursors composite derived from a number of scales	Adolescents (n=360) Maltreated (n=185) v.s. Non-maltreated (n=175) Percentage of high BPD precursors: Maltreated - 23.2% Non-maltreated - 9.1%	T-test showed significantly higher levels of BPD precursors in maltreated compare to non-maltreated group. Attentional networks and processes of alerting, orienting and conflict were not found to mediate this relationship, implying additional biological/cognitive precursors to BPD.

Sieswerda et al. (2006)	CC	VBG	SCID-II, BPD Checklist	BPD Patients (n=16) v.s. Cluster C PD (n=18) v.s. Axis I (n=16) v.s. Healthy Controls (n=16) Mean EA scores: BPD - M=32 (18), Cluster C PD - M=19 (17), Axis I - M=16 (16), Control - M=6.4 (11)	Significant difference between groups on EA mean, with BPD group having highest mean score. BPD showed hypervigilance for both positive and negative schema related cues in an emotional stroop task. They were particularly biased towards negative ones, as were Axis I disorders. Discussed in relation to hypervigilance to emotional cues.
Specht et al. (2009)	CS	CTQ	SCID-II	Incarcerated Women (n=117) 35% of sample met criteria for BPD	Significant correlation with BPD severity and EA and lack of emotional support from parents but not EN. Regression analysis showed lack of emotional support to be a significant independent predictor of BPD diagnosis. When considering schema modes, They found both disconnection/rejection and impaired limits to fully mediate the relationship between childhood trauma and BPD severity.
Tyrka et al. (2009)	CS	CTQ	SCID-II	Community sample (n=105) Adults with history of child abuse (n=70) v.s. those with no history (n=35)	Experience of EA (as well as SA and PA) was associated with elevated symptoms of BPD, as well as other PDs. No significant difference depending on type of abuse.
Weniger et al. (2009)	CC	TAQ	SCID-II	BPD Patients with a history CA with PTSD (n=10), BPD Patients without a history CA with PTSD (n=14), Health Controls (n=25) Mean levels of Neglect: BPD w PTSD - M=5.7 (1.6), BPD w/o PTSD - M=5.1 (1.4), Control - M=2.9 (0.9)	Found significant difference in levels of neglect (as well as SA and PA) between groups, with BPD groups having higher means. Those with BPD had significantly smaller amygdala and hippocampal volumes, but this did not differ in BPD patients depending on whether PTSD was present or not.
Widom et al. (2009)	LP	Official Court Record	DIPD-R	Children followed up in adulthood (n=896) Abused (n=500) v.s. Matched Controls (n=396) Prevalence of BPD in sample: Abused group - 14.9%, Match Controls - 9.6%	Odds Ratio analysis showed experiencing abuse in childhood to be significantly predictive of a diagnosis of BPD in adulthood. Also found that those abused were more likely to be unemployed, have lower education, to have never been married or to be divorced/separated and to be at greater risk of MDD and PTSD

Wingfield et al. (2011)	CC	ETI	SCID-II and BSL	Patients (n=214) BPD (n=59) v.s. MDD (n=47) v.s. Healthy Controls (n=108) Mean EA Scores: BPD - M= 203.4 (146.3), MDD - M=192.2 (140.7), Controls - M=36.3 (71.0)	ANOVA showed there was a significant difference between groups on EA, (as well as PA and SA). Bonferroni post hoc analysis showed a significant difference between the clinical groups and control group, but not between the MDD and BPD group for EA and PA. The BPD group had higher levels of SA than the MDD group.
Zanarini et al. (2000)	CS	CEQ-R	DIB-R, DIPD-R	Inpatients (n=467) BPD (n=358) v.s. Other PD (n=109) Percentage of EA in sample: BPD - 33%, Other PD - 18.3% Percentage of biparental neglect BPD - 77.1%, Other PD - 55%	Chi-squared tests showed the BPD group to have experienced significantly higher levels of EA and biparental neglect than the Other PD group.
Zanarini et al. (2006)	LP	CEQ-R	DIB-R	BPD Patients - 10yr follow-up of outcomes (interviewed at 2,4,6,8 and 10 years) (n=290) Mean abuse score (inc. EA and PA) - M=7.3 (5.3) Mean neglect score (inc. EN and PN) - M=14.7 (11.0)	Hazard Ratio's showed that experience of abuse and neglect were predictive of significantly longer time to remission over 10 year follow-up, as was SA, along with having a family history of mood disorder or substance misuse difficulties and a number of temperament variables.
Zhang et al. (2012)	CS	CTQ	PDQ SCID-II	Patients (n=162) (in China) BPD (n=80) v.s. Narcisstic (n=38) v.s. Histrionic (n=30) v.s. Antisocial (n=14) Mean scores for EA: BPD - M=9.94 (3.92), Narcisstic - M=8.79 (4.04), Histrionic - M=7.03 (2.71), Antisocial - M=8.43 (2.31) Mean scores for EN: BPD - M=15.08 (5.28), Narcisstic - M=12.76 (5.22), Histrionic - M=11.53 (4.45), Antisocial - M=10.93 (3.58)	Chi-squared tests the BPD group to have significantly higher scores for EA and EN than all other Cluster B diagnosis. However, in a wider sample of all PDs (n=1402) a regression analysis also showed that EA and EN were significantly predictive of both Cluster A and Cluster B PDs.

Notes. M = Mean ( ) = Standard Deviation
EA = Emotional Abuse, EN = Emotional Neglect, N= Neglect (inc. emotional and physical), PA = Child Physical Abuse, SA = Child Sexual Abuse, VA = Verbal Abuse. BPD = Borderline Personality Disorder, MDD = Major Depressive Disorder, PD = Personality Disorder.
Design: CS = Cross-Sectional, CC = Case-Control, LP = Longitudinal Prospective, LR = Longitudinal Retrospective, CIC = Children In the Community study.
Trauma Measures: CDF = Clinical Data Form (clinical rated measure), CEQ-R = Childhood Experiences Questionnaire – Revised, , CECA-Q = Childhood Experiences of Care and Abuse Questionnaire, CpS = Composite Score based on variety of assessments (official records, self-report, maternal interviews),CTI = Childhood Trauma Interview, CTQ = Childhood Trauma Questionnaire, ETI = Early Trauma Inventory, FIPE = Family interview for protectiveness and empathy, ITEC = Interview for Traumatic Events in Childhood, PBI = Parental Bonding Instrument, STI = Structured Trauma Interview, TAQ = Traumatic Antecedents Questionnaire, VBG = Structured Childhood Trauma Interview (Dutch).
BPD measures: AUDADIS-IV = Alcohol Use Disorder and associated disabilities diagnostic interview schedule, BPD Checklist = Borderline Personality Disorder Checklist, BSI = Borderline Syndrome Index, BSL = Borderline Symptom List, CPNI-BP = Coolidge Personality and Neuropsychological index for Children – Borderline Personality subscale (completed by care-giver), DIB (-R) = Diagnostic Interview for Borderlines (-Revised), DIPD (-R) = Diagnostic Interview for DSM-IV Personality Disorders (-Revised), PDQ = Personality Diagnostic Questionnaire, SCID-II = Structured Clinical Interview for DSM-IV - Axis II, SIDP-IV = Structured Interview for DSM-IV Personality, SWAP-200 = Shedler-Westen Assessment Procedure – 200.

Appendix D

Quality of Studies Analysis

Quality of Studies Analysis

Table D1
Overall Quality of Studies Analysis Results

Study	1. What type of research question is being asked? - is it clearly defined	2. Was the study design appropriate for the research question?	3. Did the study methods address the most important potential sources of bias? [a]	4. Was the study performed according to the original protocol?	5. Does the study test a stated hypothesis?	6. Were the statistical analyses performed correctly?	7. Do the data justify the conclusions?	8. Are there any conflicts of interest? If so are they stated	Total
Afifi et al. (2011)	1	1	1	1	1	1	1	1	8
Battle et al. (2004)	1	1	1	1	1	1	1	0	7
Bellino et al. (2005)	1	1	1	1	0[b]	1	1	0	6
Bierer et al. (2003)	1	1	1	1	1	1	1	1	8
Bornovalova et al. (2010)	1	1	0.5	1	1	1	1	1	7.5
Bradley et al. (2005)	1	1	1	1	1	1	1	1	8
Carlson et al. (2009)	1	1	1	1	1	1	1	1	8
Crawford et al. (2009)	1	1	1	1	1	1	1	1	8
Driessen et al. (2000)	1	1	1	1	1	1	1	1	8
Giesen-Bloo & Arntz (2005)	1	1	1	1	1	1	1	0	7
Goodman et al. (2003)	1	1	1	1	1	1	0[d]	1	7
Gratz et al. (2008)	1	1	1	1	1	1	1	0	7
Gratz et al. (2011)	1	1	0.5	1	1	1	1	1	7.5
Grover et al. (2007)	1	1	0	1	1	1	1	1	7
Gunderson et al. (2006)	1	1	1	1	1	1	0[e]	1	7
Heigeland & Torgersen (2004)	1	1	1	1	1	1	0[f]	1	7
Horesh et al. (2007)	1	1	1	1	1	1	1	0	7
Huang et al. (2012)	1	1	1	1	1	1	1	0	7

Johnson et al. (2000)	1	1	1	1	1	1	1	1	8
Johnson et al. (2001)	1	1	1	1	1	1	1	1	8
Joyce et al. (2003)	1	1	0	1	1	1	1	1	7
Joyce et al. (2006)	1	1	0	1	1	1	1	1	7
Kingdon et al. (2010)	1	1	1	1	1	1	1	1	8
Lange et al. (2005)	1	1	0.5	1	1	1	0[g]	1	6.5
Laporte & Guttan (2001)	1	1	1	1	1	1	1	1	8
Laporte et al. (2011)	1	1	1	1	1	1	1	1	8
Lobbestael & Arntz (2010)	1	1	1	1	1	0[c]	0[ce]	1	6
Lobbestael et al. (2005)	1	1	1	1	1	1	1	1	8
Machizawa-Summers (2007)	1	1	1	1	1	1	1	1	8
Rogosch &Cicchetti (2005)	1	1	1	1	1	1	1	1	8
Sieswerda et al. (2006)	1	1	1	1	1	1	1	1	8
Specht et al. (2009)	1	1	1	1	1	1	1	1	8
Tyrka et al. (2009)	1	1	0.5	1	1	1	1	1	7.5
Weniger et al. (2009)	1	1	1	1	1	1	1	1	8
Widom et al. (2009)	1	1	1	1	1	1	1	0	7
Wingfield et al. (2011)	1	1	1	1	1	1	1	0	7
Zanarini et al. (2000)	1	1	1	1	1	1	1	1	8
Zanarini et al. (2006)	1	1	1	1	1	1	1	1	8
Zhang et al. (2012)	1	1	1	1	1	1	1	1	8
No. of 1's	39	39	32	39	38	38	34	31	-
No. of 0's	0	0	3	0	1	1	5	8	-
No. of 0.5's	-	-	4	-	-	-	-	-	-

Note: Coding: 1 = yes, 0 = no or unable to determine
[a] Question 3: 1 = more than half met, 0.5 = half or fewer met, 0 = none met, see further tables below.
[b]Bellino et al. (2005) - failed to state a clear hypothesis. The article was presented in the journal as a 'brief communication' , so this could be due to brevity of reporting rather than failings in methodology.
[c] Lobbestael and Antz (2010) - only gave median descriptive statistics, which are perhaps not as informative as having mean scores, and did not adjust for number of statistical tests they performed at the expense of Type I error. However, the authors did note this in the limitations of the study.
[d] Goodman et al. (2003) – exclusion criteria (substance and medication free) meant sample may not be generalizable.
[e]Gunderson et al. (2006) and Lobbestael and Arntz (2010) – failure to measure other potentially confounding variables
[f]Heigeland and Torgersen (2004) - low power due to small numbers of BPD in sample
[g]Lange et al. (2005) – only compared with brain scans from healthy, non-abused controls

Table D2
Cohort Studies - Question 3: Did the study methods address the most important potential sources of bias?

Study	1. Is the study Prospective or Retrospective?	2. Is the cohort representative of a defined group or population?	3. Were all important confounding factors identified?	4. Were all important exposures and/or treatments, potential confounding factors and outcomes measured accurately and objectively in all members of the cohort?	5. Were there important losses to follow-up? 1 = no, 0 = yes	6. Were participants followed up for a sufficient length of time?	Score given [a]
Carlson et al. (2009)	Prospective	1	1	1	1	1	1
Crawford et al. (2009)	Prospective	1	1	0	1	1	1
Gunderson et al. (2006)	Prospective	1	0	1	1	1	1
Heigeland & Torgersen (2004)	Retrospective	1	1	0	0	1	1
Johnson et al. (2000)	Prospective	1	1	1	1	1	1
Johnson et al. (2001)	Prospective	1	1	1	1	1	1
Widom et al. (2009)	Prospective	1	1	1	1	1	1
Zanarini et al. (2006)	Prospective	1	1	1	1	1	1
No. of 1's	-	8	7	6	7	8	8
No. of 0's	-	0	1	2	1	0	0

Note: Coding: 1 = yes, 0 = no or unable to determine [a] Score given: 1 = more than half met, 0.5 = half or fewer met, 0 = none met.

Table D3
Case Control Studies - Question 3: Did the study methods address the most important potential sources of bias?

Study	1. Were the cases clearly defined?	2. Were the cases representative of a defined population?	3. How were the controls selected and were they drawn from the same population as the cases?	4. Were study measures identical for cases and controls?	5. Were study measures objective or subjective and is recall bias likely if they were subjective? - possible bias = 0	Score Given[a]
Driessen et al. (2000)	1	1	0	1	0	1
Giesen-Bloo & Arntz (2005)	1	1	1	1	0	1
Gratz et al. (2008)	1	0	1	1	0	1
Horesh et al. (2007)	1	1	1	1	0	1
Lange et al. (2005)	0	1	0	1	0	0.5
Laporte & Guttan (2001)	1	1	0	1	0	1
Laporte et al. (2011)	1	1	1	1	0	1
Lobbestael et al. (2005)	1	1	0	1	0	1
Machizawa-Summers (2007)	1	1	1	1	0	1
Rogosch &Cicchetti (2005)	1	1	1	1	1	1
Sieswerda et al. (2006)	1	1	1	1	0	1
Weniger et al. (2009)	1	1	1	1	0	1
Wingfield et al. (2011)	1	0	0	1	0	1
No. of 1's	12	11	8	13	1	12
No. of 0's	1	2	5	0	12	0
No. of 0.5's	-	-	-	-	-	1

Note: Coding: 1 = yes, 0 = no or unable to determine
[a] Score given: 1 = more than half met, 0.5 = half or fewer met, 0 = none met.

Table D4
Cross Sectional Studies - Question 3: Did the study methods address the most important potential sources of bias?

Study	1. Was the study sample clearly defined?	2. Was a representative sample achieved (e.g. was the response rate sufficiently high)?	3. Were all relevant exposures, potential confounding factors and outcomes measured accurately?	4. Were patients with a wide range of severity of disease assessed?	Score Given[a]
Afifi et al. (2011)	1	1	0	1	1
Battle et al. 2004	1	1	0	1	1
Bellino et al. (2005)	1	1	0	1	1
Bierer et al. (2003)	1	1	0	1	1
Bornovalova et al. (2010)	1	1	0	0	0.5
Bradley et al. (2005)	1	1	0	1	1
Goodman et al. (2003)	1	1	0	1	1
Gratz et al. (2011)	1	0	1	0	0.5
Grover et al. (2007)	1	0	0	0	0
Huang et al. (2012)	1	1	0	1	1
Joyce et al. (2003)	1	0	0	0	0
Joyce et al. (2006)	1	0	0	0	0
Kingdon et al. (2010)	1	1	0	1	1
Lobbestael & Arntz (2010)	1	1	0	1	1
Specht et al. (2009)	1	1	1	0	1
Tyrka et al. (2009)	1	0	0	1	0.5
Zanarini et al. (2000)	1	1	0	1	1
Zhang et al. (2012)	1	1	0	1	1
No. of 1's	18	13	2	12	12
No. of 0's	0	5	16	6	3
No. of 0.5's	-	-	-	-	3

Note: Coding: 1 = yes, 0 = no or unable to determine [a] Score given: 1 = more than half met, 0.5 = half or fewer met, 0 = none met.

Appendix E

Outline of Joint Working

Joint working

As stated previously, this project was conducted as part of a wider trial considering the effectiveness of Mentalization Based Treatment (MBT) for adolescents with comorbid diagnoses of BPD and depression. It was also carried out in conjunction with another Doctorate in Clinical Psychology Trainee, Zoe Given-Wilson, who was considering predictive factors in relation to on-going self-harm in adolescence. The bullet points below outline the nature of the relationship between the current study and these two projects.

MBT Trial

- A reciprocal relationship was established with the service where the MBT trial was being performed, in that trainees would conduct the follow-up recruitment for their TAU sample and in return be able to use data for their doctoral research project.
- Supervisors agreed that trainees' research projects were covered under ethical approval already granted for the MBT trial and therefore a new ethical application was not required. An amendment to add the trainees to the research project and to pay participants was granted (see Appendix F)
- Trainees were able to use resources available to all MBT trial researchers in order to facilitate recruitment (e.g. electronic records, copyrighted questionnaires, and postage paid envelopes) and were given honorary contracts at the service.
- Trainees worked largely independently from the MBT trial which was focused on prospective recruitment of the MBT treatment group. However, regular research meetings were attended in order to update on progress.
- Data was recorded on the research trials existing SPSS database and copied onto on separate databases for each trainees' individual use.
- Supervision was provided from the external supervisor (head of MBT research trial) regarding any clinical risk issues and methodological concerns relating to the wider trial.

Work in conjunction with other trainee (Given-Wilson, 2013)

- Recruitment, data collection and data recording for the current study were conducted jointly with another trainee sharing the same sample. Both trainees were equally involved in each of these stages.
- All theoretical conceptualisation, data analysis and write-up were done completely independently and the focus of the studies was different, with Given-Wilson (2013) focusing on predictors of continuing self-harm.

References

Given-Wilson, Z. (2013). The role of attachment in predicting repeated nonsuicidal self-injury among clinical adolescents: A two-year longitudinal study. *Unpublished manuscript.*

Rossouw, T. Fonagy, P., & Eparu, I. (unpublished). Mentalization-based treatment for young people with co-morbid depression and symptoms of emerging personality disorder. *On-going research.* North East London NHS Foundation Trust.

Appendix F

Ethical Approval and Amendment Letters

Ethical Approval Letter

National Research Ethics Service
East London REC 3
REC Offices
Block A, South House
Royal Free Hospital
Pond Street
London
NW3 2QG

Telephone:

27 January 2011

Ms Sue Boon

Dear Ms Boon

Study Title: **Mentalization – Based Treatment for young people presenting with co-morbid depression and symptoms of emerging personality disorder: A prospective 2-year controlled trial.**

REC reference number: **10/H0701/123**

Thank you for your letter of 22 December 2010, responding to the Committee's request for further information on the above research and submitting revised documentation.

The further information was considered by a sub-committee of the REC at a meeting held on 27th January 2011. A list of the sub-committee members is attached.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised, subject to the conditions specified below.

Ethical review of research sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

The Committee has not yet been notified of the outcome of any site-specific assessment (SSA) for the non-NHS research site(s) taking part in this study. The favourable opinion does not therefore apply to any non-NHS site at present. I will write to you again as soon as one Research Ethics Committee has notified the outcome of a SSA. In the meantime no study procedures should be initiated at non-NHS sites.

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

For NHS research sites only, management permission for research ("R&D approval") should be obtained from the relevant care organisation(s) in accordance with NHS research governance arrangements. Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at http://www.rdforum.nhs.uk.

Where the only involvement of the NHS organisation is as a Participant Identification Centre (PIC), management permission for research is not required but the R&D office should be notified of the study and agree to the organisation's involvement. Guidance on procedures for PICs is available in IRAS. Further advice should be sought from the R&D office where necessary.

Sponsors are not required to notify the Committee of approvals from host organisations.

It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	*Version*	*Date*
GP/Consultant Information Sheets	2 9Appendix G)	22 December 2010
Covering Letter	1	
Letter from Sponsor	1	29 September 2010
Investigator CV		
Response to Request for Further Information		22 December 2010
Participant Information Sheet: Prospective Group - Parents	2	22 December 2010
GP LETTER FOR PROSPECTIVE AND RETROSPECTIVE STUDY PARTICIPANTS	1	07 October 2010
MACI question booklet		
REC Application Form Changes Summary		
Referees or other scientific critique report		23 September 2010
Adult AQ age 16+		
Protocol	1	
Participant Information Sheet: Prospective group- Young People	2	22 December 2010
Investigator's Brochure		
REC application	1	
Participant Information Sheet: Retrospective Group- Participant	2	22 December 2010
Participant Information Sheet: Retrospective Group- Parent	2	22 December 2010
Participant Consent Form: For Retrospective Groups	2	22 December 2010
Participant Consent Form: For Prospective Groups	2	22 December 2010
GP letter for inpatients	1	07 October 2010
Assessment tools		
Adolescent AQ aged 12-15 years		

Please note the following typo errors:

Appendix E, Version II, dates 22/12/2010, page 1: 'hour and a halve' should be 'half'.

Appendix F: Point 4 of consent form: 'understand that all data are collected in the research'. Please delete 'are'

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Now that you have completed the application process please visit the National Research Ethics Service website > After Review

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

The attached document *"After ethical review – guidance for researchers"* gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

We would also like to inform you that we consult regularly with stakeholders to improve our service. If you would like to join our Reference Group please email referencegroup@nres.npsa.nhs.uk.

10/H0701/123	**Please quote this number on all correspondence**

With the Committee's best wishes for the success of this project

Yours sincerely

Revd Dr Joyce Smith
Chair

Email:

Enclosures: List of names and professions of members who were present at the meeting

"After ethical review – guidance for researchers"

East London REC 3

Attendance at Sub-Committee of the REC meeting on 27 January 2011

Committee Members:

Name	*Profession*	*Present*	*Notes*
Miss Elaine Mason	Retired Pharmacist	Yes	
Revd Dr Joyce Smith	Chair - Clergy/Consultant Dentist	Yes	
Dr Elizabeth Webster	General Practitioner	Yes	

Ethical Amendment Letter

NHS
Health Research Authority

NRES Committee London - East

HRA
Research Ethics Committee (REC) London Centre
Ground Floor
80 Skipton House
London Road
London
SE1 6LH

Tel: 020 3311 7254

03 September 2012

Ms Sue Boon

Dear Ms Boon

Study title:	**Mentalization – Based Treatment for young people presenting with co-morbid depression and symptoms of emerging personality disorder: A prospective 2-year controlled trial.**
REC reference:	**10/H0701/123**
Protocol number:	**n/a**
Amendment number:	**1**
Amendment date:	**20 August 2012**

Thank you for your letter of 20 August 2012, notifying the Committee of the above amendment.

The Committee does not consider this to be a "substantial amendment" as defined in the Standard Operating Procedures for Research Ethics Committees. The amendment does not therefore require an ethical opinion from the Committee and may be implemented immediately, provided that it does not affect the approval for the research given by the R&D office for the relevant NHS care organisation.

Documents received

The documents received were as follows:

Document	Version	Date
Notification of a Minor Amendment	1	20 August 2012

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

10/H0701/123:	Please quote this number on all correspondence

Yours sincerely

Mr Jay McGregor
Committee Co-ordinator

E-mail:

Copy to: *Prof Martin Orrel, NELFT*
Ms Sue Boon

Appendix G

The Inventory of Parent and Peer Attachment (Short Version) (IPPA)

IPPA - Parent

Instructions

In the following section, please indicate whether the following sentences about **your parents** are *Almost Never or Never true, Seldom true, Sometimes true, Often true or Almost Always or Always true* for you.

Please circle one answer each time.

1.	I tell my parents about my problems and troubles.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
2.	My parents help me to understand myself better.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
3.	If my parents know I am upset about something, they ask me about it.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
4.	My parents have their own problems, so I don't bother them with mine.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
5.	My parents respect my feelings.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
6.	When I am angry about something, my parents try to understand.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
7.	I wish I had different parents.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
8.	My parents accept me as I am.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
9.	I don't get much attention at home.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
10.	I get easily upset at home.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
11.	I feel silly or ashamed when I talk about my problems with my parents.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
12.	I feel angry with my parents.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True

Thank you very much for completing this.

IPPA - Peer

Instructions

In the following section, please indicate whether the following sentences about **your friends** are *Almost Never or Never true, Seldom true, Sometimes true, Often true or Almost Always or Always true* for you.

Please circle one answer each time.

1.	My friends support me to talk about my worries.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
2.	My friends care about the way I feel.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
3.	I tell my friends about my problems and troubles.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
4.	I like to get my friends' opinions on things I am worried about.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
5.	My friends listen to what I have to say.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
6.	My friends are good friends.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
7.	I wish I had different friends.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
8.	When I am angry about something, my friends try to understand.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
9.	I get upset a lot more than my friends know about.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
10.	I do not feel I belong when I am with my friends.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
11.	My friends get annoyed with me for no reason.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True
12.	I feel silly or ashamed when I talk about my problems with my friends.	Almost Never or Never True	Seldom True	Sometimes True	Often True	Almost Always or Always True

Thank you very much for completing this.

Appendix H

Consent Form and Information Sheet for Participants

Consent Form

Consent for research at [The Unit]

Name of project: A research project to see if the treatment plan at [The Unit] is effective in helping young people who suffer from depression and emotional and relationship difficulties to improve.

1 I/we have read and understand the information sheet and have had the opportunity to ask questions.

2 I/we understand that my/our General Practitioner will be notified of my/our participation in this study.

3 My/Our participation is voluntary and confidential and I/we are free to withdraw at any time, without giving any reason, without my treatment or legal rights being affected.

4 I/we understand that all data are collected in the research will be destroyed at the end of the research.

5 I/we understand that any publication resulting from the research will not identify me or my family in any way.

6 I/we agree to take part in the above study.

Name of young person:_____Date:_____Signature:_____

Name of parent/guardian:_____Date:_____Signature:_____

Name of researcher:_____Date:_____Signature:_____

Information Sheet

INFORMATION ABOUT RESEARCH PROJECT

Why have I been invited?

All young people who have presented to [The Unit] in the past few years with feelings of depression, a history of difficulties in the way they cope with their lives, the way they feel about themselves and their friendships will be invited to participate in this research. The study has been reviewed by the East London 3 Research Ethics Committee.

Steps of the research

1 How will I be participating in this research project?

You might recall that when you first came to [The Unit] you participated in the normal assessment phase in [The Unit], which involved meeting some of the professionals, as well as completing 3 packs of questionnaires. Should you decide you are happy to participate in this project we will be using information from these questionnaires you completed during your stay, and we will also ask you to complete again a part of those now, and some in about a year's time. The questionnaires you will be asked to complete now and in a year will take you approximately 1 hour to finish at each time point.

In order to allow us to use the information in the research, you will also need to sign a consent form. If you are under the age of 16, one of your parents will be asked to decide whether they consent to your participation, and will need to sign the consent form too.

2 How confidential is this?

As part of the research project, the information gathered will be given an anonymous identity (a code instead of your name), so that it is not recognisable as yours.

3 What happens with the data?

Nobody other than the researchers would have access to the data. If the research gets published, it would not contain any names or identifiable material. All the data will be destroyed once the research project has been completed.

4 What if I agreed to the research and then change my mind?

Participation in the research is voluntary. Any participant can decide to opt out of the research at any stage.

5 Will participation in this project affect treatment that I might be getting now or in the future?

No. If you have a treatment plan at the moment, it will stay the same. Participation in the project will not affect any treatment you might receive in the future either.

6 What if the research stirs up feelings inside me?

If you find that the research stirs up feelings inside you, please discuss this with the person assessing you.

7 Will my GP be informed that I agreed to participate in the research?

Yes we will let your GP know.

PLEASE KEEP IN MIND THAT YOUR PARTICIPATION IS VOLUNTARY AND YOU CAN CHOOSE TO WITHDRAW FROM THE STUDY AT ANY STAGE.

IF YOU HAVE ANY QUESTIONS, PLEASE CONTACT

[The Researcher] **ON** [Phone Number]