

**Developing a 'Profile' of Emergent Personality Disorder Among  
Adolescents: The Effect on Treatment Outcomes in the IMPACT  
Adolescent Depression Study**

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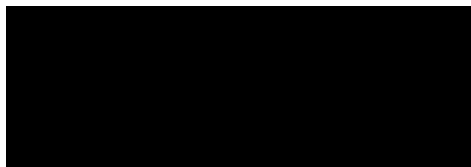
D.Clin.Psy Thesis (Volume 1), 2017

University College London

## **Doctorate in Clinical Psychology 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:**



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**Date:** 5<sup>th</sup> October 2017

## OVERVIEW

**PART I** is a systematic review of existing research investigating the relationship between peer victimisation in childhood and subsequent emergence of personality disorder (PD) in adolescence or adulthood. The quality of current evidence and key outcomes are considered, while potential mechanisms that could account for a peer victimisation-PD relationship are explored by drawing on theoretical models of PD development.

**PART II** comprises of an empirical study involving secondary analysis of data from the IMPACT Trial (Goodyer et al, 2016). The study focuses on the development of an emergent Personality Disorder ‘profile’ among adolescents with a diagnosis of major depression, and subsequently exploring whether this profile predicts depression treatment outcomes for young people in terms of depression severity, rates of recovery and withdrawal from treatment.

**PART III** is a critical appraisal of the research process overall. The experience of conducting the research is reflected upon, including how the project developed in the context of the author’s professional, intellectual and personal interests. Strengths and limitations of the research are considered as well as ideas for how it could be expanded in future.

## **ACKNOWLEDGEMENTS**

I wish to thank Professor Peter Fonagy for his unfailing support and encouragement, not only throughout the process of writing the thesis but through the entire course of my clinical training. For reading the numerous drafts, advising on statistics and for providing advice with a seemingly limitless stock of patience and mental endurance, I am very grateful. This research project would not have been possible without the support and collaboration of the IMPACT research team, who not only facilitated access to data and provided crucial advice but also inspired my interest in the project in the first place. Long before the thesis came into existence, I gained indispensable experience working directly on the trial with this group of inspirational and knowledgeable investigators, research assistants and clinicians. For this I am incredibly thankful. Of course, the greatest acknowledgement is due for the young people, their families and all the dedicated CAMHS clinicians involved in the IMPACT trial who ultimately made the research possible.

The work presented in this thesis is the result of a long professional, as well as personal, journey. I wish to thank my husband Steve for everything he has done to support me in this endeavour, including frequently having to fill the role of both parents to our son while I spent endless weekends trying to master SPSS. More generally, I am hugely grateful to my family and friends who, in many ways, have helped to keep me on track particularly over the past year.

Finally, I wish to thank Will Mandy, Tony Roth and staff at UCL for their tremendous support and flexibility over the past few years, as I gradually found my way towards becoming a clinical psychologist.

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## **PART I: LITERATURE REVIEW**

### **Peer Victimization in Childhood and the Emergence of Personality Disorder in Adolescence and Adulthood: A Systematic Literature Review**

## ABSTRACT

**AIMS:** A review of existing research investigating associations between peer victimisation in childhood and subsequent emergence of personality disorder (PD) in adolescence or adulthood. The quality of current evidence and key outcomes are considered, while potential mechanisms that could account for a peer victimisation-PD relationship are explored by drawing on theoretical models of PD development. Implications for anti-bullying policy and clinical intervention are considered.

**METHOD:** 15 studies were identified for review following a systematic literature search across major electronic journal databases in September 2015. Studies were evaluated narratively with reference to design, sample and setting characteristics, measurement of peer victimisation and PD, statistical analyses and risks of bias. Key findings were identified for each and an overall interpretation of results is presented. A discussion of the limitations of the current body of evidence is presented, along with future implications for research and clinical practice.

**RESULTS:** Existing research suggests that childhood experiences of peer victimisation are associated with greater risk of PD. The most prominent, and most commonly studied, associations appear to be with BPD. However, several methodological limitations and threats of bias are observable across all studies reviewed including non-representative sampling, heterogeneity and questionable validity of measurement of victimisation and PD and cross-sectional design. Peer victimisation may potentially increase risk of PD by impacting upon emotional regulation, mentalisation and reflective function capacity or by establishing a negative socialising context for young people.

**CONCLUSION:** Schools and other peer settings should target peer victimisation through comprehensive anti-bullying policies, awareness campaigns for children, families and teachers, facilitating a pro-social ethos and ensuring sufficient channels for reporting bullying incidents. Psychological support should be readily available and offered to victims and early monitoring/screening for mental health difficulties including personality pathology should be considered. Further prospective research is urgently needed to help establish the direction and nature of the victimisation-PD link.

**KEYWORDS:** peer victimisation, childhood, bullying, personality disorder, personality pathology.

## **1.0 INTRODUCTION**

Bullying is a serious and insidious phenomenon that affects many children's daily lives in a range of negative ways. It is typically defined as a form of aggression which involves the committing of intentional, repeated and persistent acts within a context of imbalance of power whereby the victim cannot effectively defend him or herself (Olweus, 2013). As such, bullying is often conceptualised as a 'systematic abuse of power' (Sourander, 2007) and its manifestation can be direct (e.g. physically hitting, kicking, spitting, verbally threatening or abusing) and indirect (e.g. isolating someone from the peer group, sending notes and messages, spreading rumours).

### **1.1 The Significance of Peer Victimization: Developmental Sequelae and Outcomes for Victims and Perpetrators**

The existing body of research on bullying in childhood and adolescence is in some respects substantial and longstanding, with a multitude of studies corroborating the finding that being a victim of bullying (i.e. subject to victimisation) and abusive peer relationships in childhood is generally associated with increased risk for a range of adverse psychiatric outcomes as well as psychosomatic, physical health difficulties (Arsenault, 2010). Specifically, studies suggest that being a victim of bullying in childhood is associated with increased subsequent internalising problems including depression, anxiety and psychotic symptoms, suicidal ideation and attempt, self-harm and behavioural difficulties as well as frequently having a detrimental effect on school adjustment, educational attainment and subsequent functioning within social contexts (Kumpulainen et al, 1998; Barchia & Bussey, 2010; Hawker & Boulton, 2000; Deater-Deckard, 2001; Arseneault et al, 2006). Some studies observe these effects into adolescence, whilst others also identify their potential impact further into adulthood (Kim-Cohen, 2003; Copeland et al, 2013; Sourander et al, 2007; Arseneault et al,

2010). Peer victimisation has also been shown to predict negative psychological outcomes for children independently of physical, sexual and emotional maltreatment, in situations where both have co-occurred (Sansen et al, 2014).

The importance of these findings is unequivocal, in light of reports that bullying is a relatively common occurrence both within schools and in cyberspace, taking a variety of forms, whether overt or covert, physical or relational. While there remain some commentaries depicting bullying merely as a descriptor for a common ‘rite of passage’ for many children and young people, the overall consensus seems to be that bullying is neither a normative nor a harmless experience, either for the victim or the perpetrator. Involvement in bullying as a ‘bully’ instigator has been found to correlate significantly with conduct problems in adolescence and adulthood, aggression and criminal thinking (Ragatz et al, 2011), while being a bully-victim (i.e. both a victim and a perpetrator of bullying) presents an even more complex profile of maladjustment. Bully-victim status has been found to correlate with lower self-esteem, high anxiety, increased levels of callousness (i.e. lower levels of remorse when committing antisocial acts) (Fanti et al, 2009) and with aversive social behaviour. Such negative psychological and behavioural outcomes have often been found to be more strongly associated for bully-victims than for bullies (Toblin et al, 2005; Ragatz, 2011).

Considering the potentially profound and deleterious impact of peer victimisation on the individual, their family, broader relations, education and health, the management and prevention of bullying has become an increasing concern for school and public policy in many countries across the globe, and it is acknowledged that anti-bullying programmes still have some way to go in terms of effectiveness. On the other hand, where bullying occurs and psychiatric difficulties *do* arise for the individual in the process or its aftermath, the accumulated research should help inform clinical case formulations and treatment interventions where required.

Regarding specific psychiatric consequences for victims of bullying in childhood, certain areas have been less extensively studied than others. One area that has only more recently received attention is the potential relationship between peer victimisation and emergence of personality disturbances in adolescence as well as adulthood. This is likely to reflect growing consensus around the validity of assessment, diagnosis and treatment of maladaptive personality traits in childhood and adolescence (Fonagy et al, ESCAP Congress 2015; Chanen & McCutcheon, 2013). Current research into possible links – or lack of – between peer victimisation and personality pathology in adolescence and adulthood is important for several reasons: firstly, it may help to further inform us on the developmental trajectories, aetiology and functional mechanisms of various personality disorders where currently gaps in knowledge exist. Secondly, this may subsequently enable better clinical attunement to individuals' early risk and protective factors to what are often debilitating conditions, and encourage early intervention where appropriate. Thirdly, it may help to further strengthen support for the prevention and management strategies tackling bullying in school and other relevant peer settings.

## **1.2 Pathways to Personality Disorder**

Investigating the developmental pathways to personality disorders, especially through a biopsychosocial lens, is an important endeavour in light of consistent findings that personality disorders typically have profound effects upon an individual's emotional, interpersonal and physical functioning and in many cases prove to be lifelong conditions that are challenging to treat clinically. 'Personality disorder' (PD) is an umbrella term which broadly refers to a pervasive and enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individual's culture, is inflexible, stable over time and leads to significant distress and functional impairment (APA, 2013). Several distinct

sub-types of PD are typically identified, characterised by particular patterns of symptomatology and presentation. Borderline Personality Disorder (BPD), for example, is typically characterised by unstable self-image, instability in personal relationships, significant impulsivity and affect dysregulation. The hallmark presentation of Narcissistic Personality Disorder, meanwhile, includes a pattern of grandiosity, need for admiration, and a lack of empathy towards others. Despite the distinguishing presentations and patterns observable among the different disorders, the majority of personality disorders generally have an onset in adolescence and are commonly manifested in an individual's maladaptive cognitive, affective and interpersonal functioning, as well as impulse control. Amongst adolescent populations, BPD has seen the greatest increase in empirical studies over recent years and it is suggested that this is reflective of the fact that BPD is likely to be most representative of the core psychopathology evident across all PDs (Sharp & Fonagy 2015, Higgitt & Fonagy 1992).

### *1.2.i Epidemiological Considerations*

Numerous longitudinal and cross-sectional studies, utilising both community and clinical samples, have reported a positive relationship between adverse traumatic childhood experiences such as child maltreatment (including sexual, emotional and physical abuse and neglect) and PD in adolescence and adulthood (Battle et al, 2004; Fergusson et al, 2008; Hengartner, 2013; Laporte & Guttman, 1996). There is well-established empirical support for an association between exposure to several childhood adversities such as parental loss and separation and placement in care and later PD diagnosis (Coid, 1999; Yen et al, 2002). However, other forms of childhood adversity have been less extensively studied, particularly those forms that some authors define as non-traumatic (e.g. Hengartner et al, 2013) including poverty, family and household dysfunction and school/peer problems (including bullying).



While, as noted, there is significant literature to support that the latter of these is common in the majority of Western communities, to the author's knowledge there has been no systematic review to date to summarise and evaluate findings with regards to potential associations between peer victimisation and the emergence of personality disorder. It is therefore both well-timed and appropriate that existing studies within this field are assessed in order to corroborate findings and help outline future directions for research, as well as highlighting current implications for clinical practice. It is presumed that identifying potential, specific effects of peer victimisation may strengthen the motivation for anti-bullying policy and victimisation management in schools, online and within communities at large, whilst identifying potential, specific precursors to the development of personality disorder may improve early intervention (including within schools) and subsequent treatment when difficulties arise for the individual in adolescence and adulthood.

### *1.2.ii Theoretical and Ontogenetic Considerations*

In light of the mounting recognition of potential etiological precursors to personality disorders, it is essential to consider the conceivable mechanisms underlying the development of personality disorder. Employing a biopsychosocial perspective within a broader framework of developmental psychopathology is particularly useful to this end, enabling an appreciation of the potentially complex interweaving of biological, psychological and socio-environmental factors and contexts involved in the development and presentation of personality pathology across the lifespan. It is possible that some of these mechanisms may be applicable across most (if not all) personality disorders, whilst some may be disorder-specific, but this is an important task if we hope to understand whether, how and why one might expect an association between peer victimisation and subsequent personality disorder in the first place.

Whilst several plausible models exist, due to space constraints the present paper predominantly draws attention to Marsha Linehan's Biosocial Theory of BPD (Linehan, 1993), Peter Fonagy's conceptualisation of borderline personality disorder within a framework of attachment and mentalisation (Fonagy, Target & Gergely, 2000) and John Gunderson's interpersonal hypersensitivity theory (Gunderson & Lyons-Ruth, 2008). These theories are explored in detail in section 4.0, but the overarching tenet of each requires mention here. It is also worthy of note that these theories, whilst focusing primarily on BPD, have been prioritized here due to the increasing suggestion that BPD is perhaps most representative of the core pathology associated with most PDs overall (Sharp & Fonagy, 2015).

Linehan's suggestion that BPD is likely triggered by a combination of temperamental vulnerabilities (emotional sensitivity) and one's experiences of a pervasively invalidating environment, particularly in the context of attachment relationships, is potentially relevant when considering one's 'peer' environment as well. Invalidation, it is argued, prevents the child from learning to understand, label or tolerate their own emotional reactions. This results in the commonly observed BPD characteristic of fluctuating between emotional inhibition and emotional lability. Peer victimisation typically poses a severely invalidating environment for young individuals. Repeated experience of victimisation, especially where a child presents with other vulnerability (such as heightened emotional sensitivity, or 'trait impulsivity' as subsequently elaborated by Crowell and colleagues), may serve to heighten emotional dysregulation over time, manifesting as BPD symptomatology (Linehan, 1993; Crowell et al, 2009).

An alternative theoretical conceptualisation of BPD has been proposed by Fonagy and colleagues, who suggested that some of its characteristics can be seen to originate from developmental pathology associated with the inhibition of 'mentalisation' and reflective

function. Both capacities, and more broadly a person's 'internal working model' of relationships and interaction, typically develop within the early attachment context. It could be argued that the traumatic experience of perpetual peer victimisation/bullying could impact upon an individual's mentalising capacity and reflective functioning since hostile peer relationships force an individual to turn away from other minds, as part of a defensive attempt to limit exposure to harmful psychosocial encounters. The network of interconnected minds that is usually required to maintain one's capacity to mentalise subsequently collapses (Fonagy, personal communication, 2016). Rather than being able to contemplate the range and variety of others' states of mind, the individual is led to assume malice and intentional harm. This may account for the commonly observed emotional instability and irritability in individuals with BPD.

A further alternative was suggested by Gunderson who identified 'interpersonal hypersensitivity' as a particularly significant (and biologically rooted) disposition for BPD. It could be argued that individuals who present with such hypersensitivity are more vulnerable to bullying, in that their experience will create a more powerful emotional reaction than it would for someone without such disposition; a parallel can be drawn here with Linehan's model which highlights emotional sensitivity. Over time, the interplay between stressful peer interactions and hypersensitivity in interpersonal terms may develop into disorganised and controlling interpersonal strategies, symptomatic of the typically contradictory interpersonal features of BPD (Gunderson & Lyons-Ruth, 2008).

One aspect that unites these and other approaches is that the development of personality disorder is indisputably seen as a process marked by bidirectional interaction and transaction between individuals' genetic/biological vulnerabilities and environmental conditions within which they function. Whilst this framework is generally accepted in recent literature, there has traditionally been an overarching tendency in personality disorder

research – and psychopathology models more broadly - to focus on the nature and influence of early attachment, parent/caregiver interactions and the family environment within the developmental trajectory of personality disorders. Yet relationships within one's social network provide a crucial social context for personality development. Moreover, ongoing relationships and personality co-develop over time, since individuals commonly both select and evoke relationships that accentuate their personality traits (Neyer & Lehnart, 2007).

There is increasing recognition of the influence of peer relationships on individuals' risk and resilience profiles, whereby positive peer relationships – particularly when characterised by close friendship and acceptance - can provide a protective buffer for a young person to help attenuate the effects of adverse experiences whereas, conversely, the absence of positive peer relationships and/or the presence of negative peer relationships is associated with negative outcomes including a range of internalising and externalising difficulties such as depression, anxiety and suicidal ideation (see Sansen, Iffland & Neuner, 2014 for summary).

A related notion that deserves consideration, however, is the possibility that different types of personality disorder may be differentially related to particular forms of peer experience in childhood and adolescence (and this may occur through various mechanisms). This includes the specific nature of one's involvement in peer victimisation, such as being a 'victim' of bullying, a 'bully-victim' (i.e. both a perpetrator and a victim) or 'bully-only'. The previously mentioned theories by Linehan, Fonagy and Crowell provide some explanation, for example, how persistent experiences of victimisation by one's peers in childhood may contribute towards the development of BPD. However, these mechanisms may not fully account for relationships between *all* forms of exposure to bullying/victimisation and *all* types of PD, if one considers their specific differences in terms of aetiology and presentation, alongside the common components that most PDs are thought to share.

Perhaps the most pertinent example is that of Antisocial PD (ASPD), which is typically characterised by a failure to conform to lawful and ethical behaviour, an egocentric, callous lack of concern for others, deceitfulness, irresponsibility, manipulateness and risk-taking (APA, 2013). ASPD is known to be strongly associated with antisocial behaviour and conduct problems in youth, yet the association with antisocial behaviour is not as significant a precursor of BPD, which is typically characterised by an unstable self-image, affective negativity including insecurity, depressivity and hostility, and risk taking (APA, 2013). Since the *perpetration* of bullying in childhood and adolescence can itself be conceptualised as a form of antisocial behaviour, one might feasibly postulate that being a persistent ‘bully’ or a bully-victim is more likely to be associated with ASPD than being a bullying ‘victim’ alone, that being a perpetrator is more likely associated with ASPD than with BPD, while being persistently targeted, isolated and invalidated as a *victim* will be more likely to be associated with BPD and other PDs not predicated on a history of antisocial behaviour and aggression or callousness towards others. Although the core focus of the present review is on the relationship between being a victim of bullying (rather than a bully) and subsequent personality pathology, it is nevertheless important to appreciate the potential complexity of any victimisation-PD relationship in light of these factors and to remain mindful of this when interpreting existing research in this field.

In the context of the above considerations, it seems highly appropriate to recognise the influence of peer relationships on personality development, including personality pathology, and to encourage a move away from ‘parent blaming’ towards more balanced perspectives on individuals’ psychological adjustment within their social context.

### **1.3 Current Study: Summary of Aims**

The core aim of this review is to evaluate existing, relevant studies on their capacity to address the following key questions:

1. Is the experience of bullying/peer victimisation associated with increased risk of emergent personality disorder in *adolescence*?
2. Does the experience of bullying/peer victimisation in childhood increase risk of personality disorder in *adulthood*?
3. Is the experience of bullying/peer victimisation in childhood associated with higher risk of developing any *particular* personality disorder in adolescence/adulthood?

The present review offers a narrative synthesis, rather than a meta-analytical approach, with particular attention given to the methodological strengths and limitations of currently available research, theoretical consideration of possible mechanisms of interaction between peer victimisation and PD, as well as subsequent directions for further investigation.

## **2.0 METHOD**

### **2.1 Literature Search Strategy**

The Cochrane Database of Systematic Reviews (CDSR), UCL e-resources and Google Scholar were initially searched in August 2015 to establish whether a comparable systematic review with relation to peer victimisation and personality disorder was already in existence.

No such systematic review was found.

The subsequent aim of the literature search was to identify all published studies investigating the relationship between peer victimisation/bullying in childhood and subsequent personality disorder of any variety, conducted at any time up to the point when the search was run on 16<sup>th</sup> September 2015. Studies were located using a systematic search

procedure across a number of electronic databases: PsychInfo, PubMed, MedLine, British Education Index (BEI) and the Education Resource Information Center (ERIC). The author liaised with research colleagues and a subject specialist librarian at UCL to acknowledge these databases as the most relevant to the current review type and topic. In order to ensure the quality and relevance of material utilised within the present review, unpublished material, material outside of peer-reviewed sources and grey literature was not referred to.

A manual search was also conducted on the citations and references within key papers yielded by the database search. This was to ensure that relevant authors and studies had not been inadvertently missed during the primary search.

## 2.2 Key Search Terms

Existing systematic reviews and literature pertinent to bullying and peer victimisation in schools, as well as reviews relevant to personality disorder in adolescence and adulthood, were consulted in order to aid the generation of key concepts and subsequent search terms. Where concepts occurred in broad and specific forms (e.g. ‘bullying’ versus ‘childhood bullying’, ‘personality disorder’ versus ‘borderline personality disorder’), the former was employed in the search strategy in order to enable a more comprehensive literature search.

The following search terms were subsequently used to conduct both ‘key word’ and subject heading searches across all the databases (Table 1):

**Table 1**

***Key Search Terms***

Concept	Key search terms
Bullying	Bullying, Victimization, Victimization, Peer Relations, School violence, Abusive Peer Relationships

Childhood	Youth, Child*, Childhood, Adolescen*, Adolescence, Adolescent Development, Predelinquent Youth, School-age, Juvenile, Teenage
Personality Disorder	Personality Disorder, PD, Psychopathology

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Across all databases, within each concept group as per table 1, the operator ‘OR’ was used to ensure all relevant terms were searched for (e.g. ‘bullying’ *or* ‘victimisation’ *or* ‘abusive peer relationships’ etc.). Searches 1 and 2 were then combined using the ‘AND’ conjunction first, to ensure the capturing of peer victimisation/bullying experiences *in childhood*, before combining this with search 3 (e.g. [search 1 and 2] AND [search 3]). This was done in order to avoid restricting personality disorder occurrence to any particular age group as well, otherwise longitudinal studies involving observations of emergent PD in late adolescence and adulthood might have been missed.

Truncation and wildcard functions were used where necessary and possible across each database (e.g. with the term ‘adolescen\*’ and ‘victimi?ation’) to ensure that differential spelling and endings to key root words were accounted for. Some variation can be observed in the characteristics of different databases (particularly between those that operate on different platforms such as Ovid or Ebsco) but generally all databases enable the broadening or narrowing of search terms using thesaurus or auto-explode options, and these were used in each case to ensure all relevant concepts and search terms were captured under each umbrella term. The complete search strategy for all databases used in this review is presented in Appendix A. A second, basic search was also run across databases – using a simple combination of the subject headings ‘bullying’ AND ‘personality disorder’ - to cross-check results against the more complex search strategy and ensure no key texts had been missed.



### **2.3 Search Limits**

To minimise the risk of omitting relevant studies, very few limits were applied to the literature search at the outset: these included limiting results only to human studies and papers presented in English language. A search was also subsequently conducted without these limitations to explore whether applicable studies had been published in other languages, but this did not yield any additional relevant results. At the point of the search, there was no limit set as to the date of publication or publication type, again to avoid restricting results at this stage.

### **2.4 Obtaining the Literature**

Following the systematic search, the vast majority of retrieved literature was in the form of research/scientific papers presenting original studies and these were obtainable via the database directly or online through other publication channels. They also included a short abstract available directly via the results page on the specific database.

Where literature was in the form of a book chapter, in most cases either a description of this or the chapter itself was available online. Where papers were not available via database or online, attempts were made to source the literature through the institutional library or contact was made with the authors directly to request access.

All search results were initially screened for relevance on the basis of title and abstract. All results considered relevant at this stage proceeded to further manual full-text screening whilst those deemed clearly irrelevant were excluded. Commentary pieces or review papers were subsequently segregated from papers that presented original research and the latter were selected for systematic review pending the satisfaction of the below inclusion/exclusion criteria.

## 2.5 Inclusion and Exclusion Criteria

Studies were eligible for the systematic review if they fulfilled the following minimum criteria:

- Included either a focused, specific investigation of an association between bullying/peer victimisation and personality disorder OR the association was investigated as part of a broader study of bullying outcomes or personality disorder correlates and is clearly discernible in its results
- Investigated an association between the experience of bullying as a ‘*victim*’ (including a ‘bully-victim’), as opposed to bully-perpetrator only, and personality disorder
- Were quantitative in nature
- Presented in English language (although not restricted to being conducted in English-speaking countries or populations)

Studies were excluded from the systematic review if they:

- Investigated broad associations between ‘abuse’ or ‘maltreatment’ and personality disorder without specifying bullying or peer victimisation within these categories
- Were Qualitative in nature or represented single case studies
- Were ultimately unobtainable either via database, the academic institution, online or from the author directly

The flowchart in Figure 1.0 provides a more detailed illustration of the process of identifying relevant literature through to selection of eligible studies for systematic review. Since each database was searched individually, identification and exclusion of duplicates did not occur until all databases had been searched and all relevant records from each database were imported into a citation management programme (Endnote).

A total of 1,507 records were identified via database search and additional citation search, of which 1,404 were excluded due to irrelevance. Once duplicates and unobtainable

resources were removed, 75 records proceeded to detailed, full-text screening. 15 studies were ultimately identified as appropriate and eligible for the current review. The search was re-run on 20<sup>th</sup> September 2016 to check for eligible studies published in the interim, but no additional records were found.

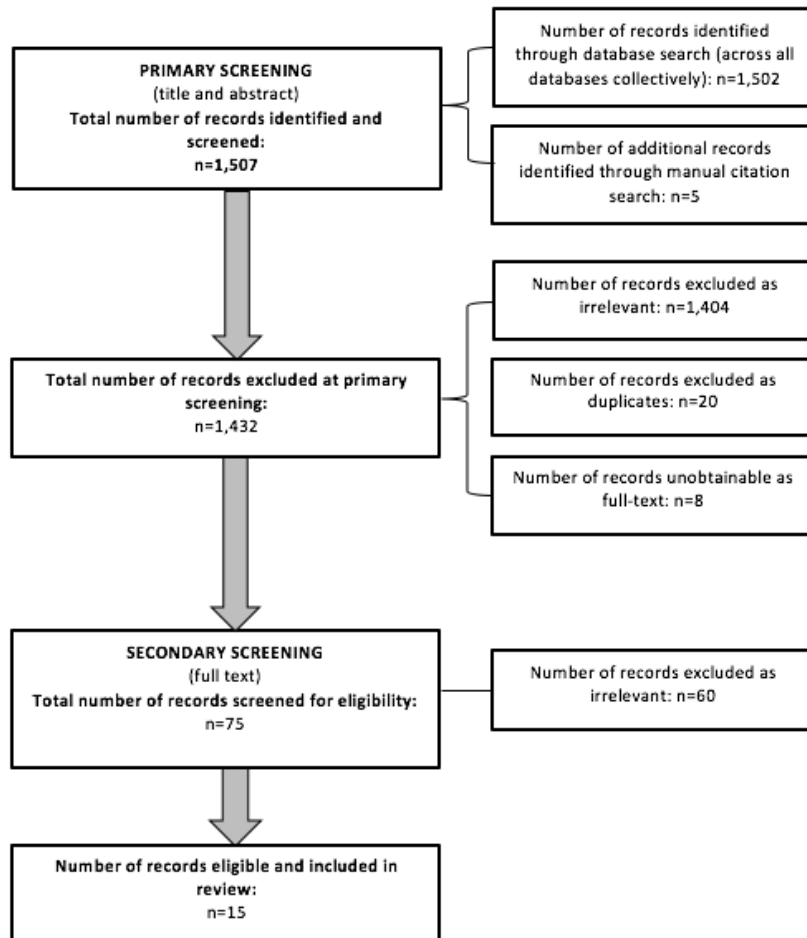
## **2.6 Statistical Comparisons**

Key statistical findings for each study are presented in Section 3: Results. To enable consistent comparison and commentary across studies, all effect sizes (ES) are reported both in the authors' original format (e.g.  $r$ , Odds Ratio, Chi Square etc.) as well as subsequently in Cohen's  $d$  (including confidence intervals at the 95% level) to provide a common metric of ES for discussion. Effect sizes were converted to Cohen's  $d$  by the review author using the *Cran.R-Project* software, 'compute.es' package, or manually where appropriate. 95% Confidence Intervals were subsequently calculated for all Cohen's  $d$  effect sizes except where statistically not possible due to lack of necessary data<sup>1</sup>.

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<sup>1</sup> For example, where original CI's for Odds Ratio or the number of subjects in PD/non-PD groups with regard to outcome variable were not provided by the study author.

**Figure 1.0 Systematic Literature Search Flowchart**



## **3.0 RESULTS**

### **3.1 An Overview of Study Attributes**

#### *3.1.i Sample Characteristics and Variety of Participants*

Table 2 (Appendix B) illustrates a detailed summary of key descriptive characteristics for all fifteen studies included in the present review. It is noticeable that the association between peer victimisation and personality pathology has been explored with significant diversity in terms of the type of participant populations used, including various age groups and settings (e.g. school-aged children, clinical PD populations, adult community samples, prisoner cohorts and generic outpatient attendees). Authors have reported a range of sampling methodologies, some involving stratified sampling gained from broader population samples (e.g. Hengartner et al, 2013), while others involved recruitment of participants presenting consecutively within a primary care setting (e.g. Sansone et al, 2010). Some samples relied on psychiatric referrals (Laporte et al, 2012) whilst others consisted of anonymous online survey respondents (Goodman et al, 2010, 2013). All studies involve non-randomised samples.

Sample sizes also vary significantly, with the smallest at 106 participants (53 sibling-pairs; Laporte et al, 2012) and the largest at 6050 (Wolke et al, 2012; sample based on the Avon Longitudinal Study of Parents and Children). Nine studies incorporate a comparative balance of male and female participants, while six studies involve single-sex samples only: Sansone et al, 2013 (female sample), Laporte et al, 2013 (female sibling sample), Sourander et al, 2007 (male sample), Goodman et al, 2010 (female sibling sample), Goodman et al, 2013 (male sibling sample) and Roberts et al, 2008 (male offender sample).

Geographically, of the fifteen studies five were conducted in the United States (Sansone et al, 2010; Sansone et al, 2013; Goodman et al, 2010; Goodman et al, 2013; Copeland et al, 2013), one in Hong Kong (Fung & Raine, 2012), one in Japan (Kawabata et

al, 2014), one in China (Zhu & Chan, 2015), three in the United Kingdom (Lereya et al, 2013; Wolke et al, 2012 and Roberts et al, 2008), one in Switzerland (Hengartner et al, 2013) and one in Finland (Sourander et al, 2012). Two studies did not report their location, other than describing it as an ‘urban setting’ (Laporte et al, 2012) and a ‘summer-camp setting’ (Natsuaki et al, 2009). The majority of studies involved samples which were local/regional, except Fung and Raine (2012) (whose sample included children from primary and secondary schools drawn from across Hong Kong), and Sourander et al, 2007 (whose sample was drawn from the ‘From a Boy to a Man’ male cohort study in Finland). Two studies (Goodman et al, 2010; Goodman et al, 2013) were web-based with potentially national U.S. reach: these involved anonymous parent surveys placed on the ‘National Education Alliance for Borderline Personality Disorder’ (NEA-BPD) website.

### *3.1.ii Bullying/Victimisation and its Subtypes*

There is significant heterogeneity among the reviewed studies in terms of how peer victimisation/bullying has been conceptualised and subsequently measured. Nine studies operationalised ‘peer victimisation’ as an overarching, single concept - reporting descriptions of varying scope - that participants either had or had not experienced in childhood (e.g. Sansone et al, 2010; Sansone et al, 2013; Sourander et al, 2007; Roberts et al, 2008; Goodman et al, 2010; Goodman et al, 2013; Hengartner et al, 2013; Natsuaki et al, 2009; Copeland et al, 2013). Of these, two studies also considered ‘bully-victim’ status as well as victim-only, i.e. participants who had both bullied others and been bullied themselves (Sourander et al, 2007; Copeland et al, 2013).

The other six studies identified specific sub-types of peer victimisation as well as a combined concept of peer victimisation in some, but not all, cases. For example, one study explored physical victimisation, social manipulation, verbal victimisation and attack on

property by peers as well as ‘total victimisation’ (composite score) (Fung & Raine, 2012). Another study distinguished between ‘physical victimisation’ and ‘relational victimisation’ but did not consider combined victimisation (Kawabata et al, 2014); similarly, Zhu & Chan (2015) distinguished ‘direct’ victimisation from ‘relational’ victimisation and analysed these independently for association with personality pathology. Laporte and colleagues (2012) stipulated peer victimisation to involve ‘physical and/or emotional abuse perpetrated by peers or dating partners’ but did not explore a general/combined experience of peer victimisation in their analysis. Lereya and colleagues (2013) considered ‘relational’ and ‘overt’ bullying, with participants being categorised as being victimised if either or both of these were endorsed. They also considered chronicity of victimisation (i.e. if it occurred infrequently/sporadically or was stable/long-term), but these were not assessed independently in the context of possible association with personality pathology. Another study explored both overt and relational aspects of victimisation, with participants categorised as having been victimised if either of the sub-types were endorsed frequently/very frequently; additionally, a ‘combined victimisation’ concept involved experiences of both relational and overt bullying, and chronicity was also assessed (Wolke et al, 2012).

### *3.1.iii Personality Pathology and its Subtypes*

The studies included in the present review present significant diversity in terms of which personality disorder(s) or aspects of personality pathology they focused on as outcomes. Some studies concentrated exclusively on a single personality disorder type: for example, eight studies focused on Borderline Personality (Sansone et al, 2010; Sansone et al, 2013; Kawabata et al, 2014; Laporte et al, 2012; Lereya et al, 2013; Wolke et al, 2012; Goodman et al, 2010; Goodman et al, 2013). Two studies focused exclusively on Antisocial Personality (Copeland et al, 2013; Sourander et al, 2007). One study focused on Borderline Personality

and Antisocial Personality (Zhu & Chan, 2015); one focused on Schizotypal Personality (Fung & Raine, 2012); one focused on Paranoid Personality (Natsuaki et al, 2009). Two studies considered a whole range of personality disorders (ten in total, based on DSM-5 subtypes) (Hengartner et al, 2013; Roberts et al, 2008).

Notably, the studies also vary significantly in their operationalisation of personality pathology among participants. Some considered the presence or absence of personality disorder (PD) categorically, based strictly on formal diagnostic information about the participant: this included those studies where ‘PD’ participants were sourced from clinical samples/ psychiatric referrals confirming particular pre-existing PD diagnoses (e.g. Laporte et al, 2012; Goodman et al, 2010; Goodman et al, 2013; Sourander et al, 2007 using registry information including psychiatric diagnoses). Additional screening research measures were then subsequently conducted to confirm this. Other studies did not involve clinical samples but still adopted measures of PD which enabled categorical PD diagnosis according to DSM-IV symptom thresholds (e.g. Hengartner et al, 2013; Lereya et al, 2013; Natsuaki et al, 2009; Wolke et al, 2012; Copeland et al, 2013; Roberts et al, 2008). Other studies were less diagnosis-driven and instead investigated dimensionally the presence and extent of particular PD ‘traits’, features or symptomatology among participants (e.g. Sansone et al, 2010; Sansone et al, 2013; Kawabata et al, 2014, Fung & Raine, 2012; Zhu & Chan, 2015).

## **3.2 Methodological Considerations**

### *3.2.i Design*

Of the fifteen studies reviewed, the majority (nine) were cross-sectional in nature, measuring both the predictor variable (peer victimisation) and outcome variable (PD) at one time-point, using retrospective reports. Notably, it emerged upon review that one of the studies included in the present paper – Zhu and Chan, 2015 – conceptualised the association between



victimisation and personality pathology in the opposite direction to the remaining 14 studies: that is, the authors focused on correlates of *peer victimisation* with a view to identifying potential predictors of experiencing victimisation, including PD, rather than vice versa. This study was nonetheless included in the present review as a point of interest, since the possibility of an association between the two variables is still potentially informative: the cross-sectional nature of the study means that the existence of an association between victimisation and PD does not assume directionality of the association or causality, as the authors themselves note.

A considerable limitation of the cross-sectional studies is that they only permit exploration of the presence or absence of an association between victimisation and personality pathology; they cannot establish either the direction of the relationship or causality between the two variables. Any associations observed are necessarily a time-specific snapshot of co-occurrence and provide no information on change over time in the presentation of either variable, which would be necessary to ascertain which phenomenon is really impacting upon the other.

Some longitudinal, prospective studies are observed in the present review and this is typically a more useful design for investigating the directional relationship between variables such as victimisation in childhood and subsequent emergence of personality pathology. It enables observation of change over time and, with sufficient baseline information, can provide some insight into chronological emergence of particular outcomes. For example, Wolke and colleagues (2012) utilised a participant sample drawn from the Avon Longitudinal Study of Parents and Children (ALSPAC) cohort study, where measures were used to collect data about children on a wide range of variables across time. Experience of peer victimisation (as well as bully- and bully-victim status) was reported by multiple informants (child, parent and teacher) at three time points in total between child ages 4-10. BPD symptoms were

assessed at age 11.7, while a number of factors were measured at baseline in childhood, including various axis-I disorders, maladaptive parenting and multiple family risk factors.

Lereya and colleagues (2013) conducted a very similar study, also utilising a sample drawn from the ALSPAC cohort study. As above, peer victimisation was reported at three time points in total involving multiple informants, along with a number of other adverse childhood experiences including pre-school experience of maladaptive parenting, domestic violence, depression and externalising behaviour difficulties. Multiple outcome variables were assessed, of which one was borderline personality disorder (the primary outcome of interest in this study was self-harm in adolescence).

However, despite an overarching prospective, longitudinal design in both the Wolke and Lereya studies, a crucial caveat relevant to the present review was that borderline personality disorder in each study was itself only measured at one point, even though victimisation was measured longitudinally. BPD was assessed at 11.7 years, with no baseline (7-10 years) measure available. Therefore, it is not possible to make inference about whether victimisation led to the emergence or increase in BPD symptomatology, as baseline presence/severity of BPD is not available for comparison. The same limitation applies to the study of Paranoid PD by Natsuaki and colleagues (2009). It is possible that the lack of childhood baseline measures to some extent reflects the fact that it is only a relatively recent development that personality pathology assessment in childhood and adolescence is considered to be clinically informative and appropriate.

A more robust methodology in this respect was employed by Copeland and colleagues (2013) who assessed childhood psychiatric disorders/status, family hardship factors and peer victimisation annually with children between the ages of 9 and 16 (using child and parent-report), as well as adult psychiatric outcomes between the ages of 19-26, using self-reports. Similarly, in their longitudinal evaluation of a sample of males in Finland

(based on the 'From a Boy to a Man' nationwide cohort study) Sourander and colleagues measured peer victimisation at age 8, using child, parent and teacher reports while antisocial personality disorder (ASPD) was assessed at two time points, based on mental and physical health information gained from military registry records (which include all psychiatric diagnoses for each individual) (Sourander et al, 2007). This enabled some insight into change in PD status in the interim period. More importantly, the study also included baseline screening measures (the authors note parent and teacher-reports on Rutter questionnaires for screening children's emotional and behavioural problems) to ascertain a range of children's psychiatric symptoms and/or presence of psychiatric disturbance at baseline (age 8). Whilst there was no specific ASPD measure at baseline, it is probable that clinically significant personality disturbance would have been identified using the screening instruments at this time point. The utility of a longitudinal design such as this one, with baseline and follow-up measures of psychiatric symptoms, more readily enables conclusions about the chronology and direction of a relationship between peer victimisation and subsequent personality disturbance.

Kawabata and colleagues (2014) also adopted a longitudinal approach, assessing peer victimisation and BPD features at two time-points among children aged 9-11. However, the time interval of data collection was only 6 months, therefore it was relatively short-term in follow-up which does not facilitate conclusions about longer-term predictors of BPD in adolescence and/or early adulthood.

Despite longitudinal studies being typically more robust in terms of identifying predictors rather than mere correlates of outcome (PD in this case), it is evident from the current review that, even within this group, researchers adopt a range of approaches of which some may be more advantageous than others. Short time-intervals between data collection

points and lack of baseline measures for outcome variables are significant limitations with regard to establishing temporal associations between victimisation and personality pathology.

### *3.2.ii Data Gathering: Source of Informants*

Almost all studies relied heavily on self-report measures regarding childhood experiences of peer victimisation. Seven studies used self-report measures exclusively; four studies additionally sought teacher-reported information; six studies included parent-reported data alongside self-reports. Only three studies had data from all three informants (child/participant, parent and teacher) (Lereya et al, 2013; Wolke et al, 2013; Sourander et al, 2007). One study collected victimisation data from camp-counsellor observation/report only (Natsuaki et al, 2009) while two studies relied only on parent-report (Goodman et al, 2010; Goodman et al, 2013). With regard to data on personality pathology, self-report was even more dominant, with twelve of the fifteen studies employing exclusively self-report measures for the outcome variable. One study considered official data on psychiatric status of all participants available via military registry (Sourander et al, 2007); two studies gained PD information from parent-report only, albeit it required their confirmation of the participant's clinical BPD diagnosis (Goodman et al, 2010; Goodman et al, 2013). In total, seven studies used exclusively self-report for *both* peer victimisation and personality pathology measures; two studies used exclusively parent-report for both measures. This presents a potential risk that the presence and strength of associations between victimisation and PD may, at least in some part, be a consequence of shared variance due to the influence of a single respondent. Lack of triangulation in the majority of studies may also increase the impact of informants' recall bias on the outcome data.

### *3.2.iii Measurement of Predictor and Outcome Variables*

With the exception of one study (Zhu & Chan, 2015), all studies considered bullying/peer victimisation as the independent, predictor variable whilst personality pathology was the dependent, outcome variable. The definition – and measurement – of each, however, varied significantly across studies, to the extent that only two authors used the same measure or scale for the bullying/victimisation variable (see below) (Wolke et al, 2012 and Lereya et al, 2013).

Similarly, for PD, studies utilised a host of different measures with only two studies seeing any overlap: The Childhood Interview for DSM-IV Borderline Personality Disorder [UK Version] (CI-BPD-UK) was used in the Lereya and Wolke studies (Lereya et al, 2013 and Wolke et al, 2012).

With regard to bullying/victimisation, several studies exclusively used simple questions embedded within a broader survey to capture participants' experiences of victimisation: this included binary questions such as whether one had been bullied (yes/no), for how many years and by how many bullies (Sansone et al, 2010, 2013) or in some cases a single question on bullying requiring yes/no endorsement (Goodman et al, 2010, 2013). Hengartner and colleagues also used binary yes/no questions including 'were you frequently physically assaulted in school/insulted in school/excluded or ignored?' (Hengartner et al, 2013), embedded within a broader child adversity questionnaire devised by the authors themselves. A similar approach was used by Roberts and colleagues (2008) and Sourander and colleagues (2007).

The remaining eight studies used formally validated measures (see table 3 Appendix C for full summary) although there was significant variation in type: Copeland and colleagues, for example, collected information on victimisation and bullying as part of a general Child and Adolescent Psychiatric Assessment (CAPA) interview; Natsuaki and

colleagues used an *observer-reported* questionnaire (Mt. Hope Family Center Bullying-Victim Questionnaire) while both Wolke and Lereya used the Bullying and Friendship Interview Schedule for child self-report and a single item from the Strengths and Difficulties Questionnaire for teacher/parent reports). Some authors incorporated more detailed, victimisation-focused measures, including the Multidimensional Peer Victimization Scale (Fung & Raine, 2012), Children's Social Experience Questionnaire (Kawabata et al, 2014), Juvenile Victimization Scale and Relational Aggression Scale (Zhu & Chan, 2015) to measure aspects of both direct and indirect peer victimisation.

Laporte's use of the Childhood Trauma Interview (CTI; Fink, Bernstein, Handelsman, Foote, & Lovejoy, 1995) to capture peer victimisation is somewhat problematic; even though the CTI has been well validated (Fink et al, 1995), peer victimisation in the present study was coded 'when physical, emotional or sexual abuse by peers *or dating partners*' occurred, considering duration, frequency and severity. The inclusion of partners in the same category potentially precludes the possibility of disentangling victimisation at the hands of peers *per se*. It is also questionable whether the measure, in focusing on six types of trauma, is conceptually appropriate for assessing 'bullying'.

There was significant inconsistency among the presented studies in their provision of a definition of terms – 'bullying' or 'victimisation' – to participants completing the specified self-report measures. Only one study specifically reports providing a definition of terms to its informants (Natsuaki et al, 2009) whilst others do not report this. For some, particularly those using validated, bullying-specific measures, items contained within scales were arguably self-explanatory and additional definition of terms was not necessary.

There was great variability with regard to measurement of Personality Disorder across studies. Only one study (Roberts et al, 2008) used the SCID-II which is widely used for diagnostic purposes of axis-II disorders. A few other studies also used diagnostic interview

methods, including the Diagnostic Interview for Personality Disorders (DIPD-IV) (Laporte et al, 2012) and the Childhood Interview for DSM-IV Borderline Personality Disorder (CI-BPD-UK) (Lereya et al, 2013; Wolke et al, 2012). One study gained information on all participants from military registry records which included all formal psychiatric diagnoses including personality disorder (Sourander et al, 2007) with no further PD-measures used by the researchers themselves. The remaining studies, ten in total, all used self-report scales incorporating different types of PD symptomatology, some of which enabled categorical PD diagnostic scores (Hengartner et al, 2013) but most yielded mainly dimensional trait-scores on various personality features. Goodman and colleagues, in both of their studies, incorporated a BPD screening instrument into an internet survey and requested informants (parents) to confirm that their child had also had a formal BPD diagnosis. The accuracy and reliability of this method is unfortunately unknown and difficult to test.

An overview of all fifteen studies raises serious questions about the adequacy of measurement of key variables as well as outcomes. It indicates a potential lack of construct and content validity with regards to peer victimisation/bullying, in light of the various measurement strategies used (nearly half of them using single questions and self-constructed surveys). The widespread lack of providing a definition of bullying is problematic in leaving participants – in most cases children and adolescents – to subjectively interpret the concept. However, some have argued that providing a definition could trigger reactivity with the potential to discourage honest disclosure (Evans et al, 2014). It seems probable though that this is more likely to be the case for disclosures of bullying acts/behaviour, which is socially-stigmatised, rather than disclosures of victimisation experiences. The variation between studies in terms of measuring PD is also of major concern: firstly, it is clear that the majority of studies had no clinically-based assessment of this variable and relied heavily on self-report which involves risk of subjectivity and bias. It has been noted, for example, that the PDQ-4

has rather low predictive power as a screening measure for personality disorder and is inclined to produce a high rate of false positive results (de Reus et al, 2013). Secondly, it is potentially challenging to compare PD outcomes given that some studies looked at PD in categorical, diagnosable terms and utilised clinical samples, whilst others looked more broadly at personality pathology features on a continuum and utilised community samples.

### *3.2.iv Statistical Control for Covariance*

Several factors could impact upon the relationship between peer victimisation and subsequent personality disorder. For example, mental health status (other than PD), socioeconomic and other environmental circumstances, age, sex and gender, family structure and functioning, attachment difficulties, other adverse childhood experiences, to name a few.

Details of statistical analyses are presented for each study in Table 3 (Appendix C). A number of studies in the present review did not report any statistical control for covariance or confounding factors (Sansone et al, 2010, 2013; Fung & Raine, 2012), whilst others demonstrated limited statistical controls: Goodman and colleagues, for example, who conducted a survey among parents about sibling pairs (one of whom had BPD) gained information on a number of demographic and developmental items (ranging across developmental epochs for both siblings) but controlled mainly for household income and age. There was no reported control for current demographic differences between siblings, for parental psychopathology, whether siblings were full- or half-siblings, raised together or apart, nor for childhood adverse experiences of abuse and trauma, which have been found in other research to increase risk of BPD (Grilo et al, 2002; Lobbestael et al, 2010). Lack of, or limited, control for possible covariates leads to inevitable difficulty in being able to draw truly meaningful conclusions about the nature of the victimisation-PD relationship per se. In the absence of consideration of mediating or moderating factors, we cannot reliably ascertain



whether the relationship is either truly unique or clinically significant, or whether another phenomenon might explain the link.

Several studies demonstrated greater robustness by measuring other predictor variables and potential confounding factors, in addition to bullying/victimisation, for which they later report statistical control when assessing victimisation-PD associations. This includes Hengartner and colleagues who looked at other childhood adversity including maltreatment, conduct problems at school, poverty, family conflict and substance abuse (Hengartner et al, 2013). Others also looked at family separation and loss (Laporte et al, 2012), domestic violence and maladaptive parenting and depression (Lereya et al, 2013; Wolke et al, 2012), other psychiatric symptoms such as hyperactivity, conduct and emotional difficulties (Sourander et al, 2007), child IQ (Wolke et al, 2012), school type/environment (Zhu and Chan, 2015), ethnicity and comorbid personality disorders (Roberts et al, 2008).

### **3.3 Key Outcomes**

The key statistical analyses, outcomes, effect sizes (Pearson's  $r$  and/or Cohen's  $d$  where possible) and relative confidence intervals for all studies are presented in Table 3 (Appendix C).

#### *3.3.i Findings from Prospective Studies*

Overall, there is consistent indication that experience of peer victimisation in childhood is associated with various types of personality pathology in adolescence and adulthood.

Persistent ('stable') victimisation in childhood was found to predict BPD in adolescence by Lereya and colleagues (2013), although the effect was small. Wolke and colleagues (2012), who used a larger sample from the same cohort as Lereya (from the ALSPAC study) found that experience of any peer victimisation – whether chronic/stable or infrequent – significantly

predicted BPD, although the effect again was relatively small; a moderate effect size was found for association between (child-reported) stable peer victimisation in childhood and BPD in adolescence whereby children who experienced chronic victimisation were at highly increased risk of BPD symptoms. Experience of both overt and relational bullying was most strongly linked with BPD in adolescence and a relatively large effect was noted here.

Importantly, in both Lereya and Wolke studies, substantial statistical control of possible confounders was used (including baseline axis I symptomatology) and the strength of association between victimisation and BPD was not significantly altered by doing so. The authors note that this is suggestive of a causal victimisation-BPD relationship. Unfortunately, given that BPD symptomatology in both studies was only measured at follow-up (age 11.8 years), there is no information on possible personality disturbance prior to experiences of victimisation: it could be that early borderline personality features may be a precursor of peer victimisation rather than – or as well as – a consequence.

Physical victimisation in childhood was significantly associated with Borderline Personality features in the Kawabata study, with a medium effect size (Rosenthal, 1994). Moreover, children who experienced physical victimisation showed an increase in BPD symptoms during the follow-period. It should be noted however that the time-frame of this study was very brief (6 months between baseline and follow-up measures on both bullying and BPD), and BPD was measured via child questionnaire self-report only (unlike bullying) which risks response bias and is insufficient to establish clinically significant borderline pathology. The advantage of this study over others presented in this review however is that borderline features were measured at both time points, enabling at least some evaluation of personality change/stability over time relative to victimisation experiences.

Sourander and colleagues, who looked not only at victimisation but also ‘bully-victim’ as well as ‘bully’ experiences, found that being a ‘bully-victim’ in childhood

significantly predicted Antisocial Personality Disorder (ASPD) in adulthood, and that the association remained significant even after controlling for some potential confounders such as parental educational level and psychiatric symptoms at baseline, including mood and anxiety disorders, substance abuse and psychosis. Subsequent calculation suggests a large effect. Interestingly, the authors found no relationship between being a ‘victim-only’ of bullying and subsequent ASPD. This suggests that factors involved in bullying *perpetration* (which in itself is a type of antisocial behaviour) such as aggression towards peers or interpersonal difficulties, or the interaction of these with victimisation experience, are potentially more salient towards increased risk for this particular type of personality disorder. A notable limitation of this study, however, is that psychiatric comorbidity in the follow-up period was not directly assessed by clinicians or the research team and instead relied on the original military registry records having been kept up to date. Inaccuracies in recent diagnoses or changes in health status may have compromised the results.

Copeland and colleagues also focused on ASPD as an outcome. They reported no significant relationship between being a ‘victim’ of bullying or a ‘bully-victim’ in childhood and ASPD in young adulthood. A notable strength of this study was that childhood psychiatric status was rigorously assessed (annually, between ages 9-16) as part of the Child and Adolescent Psychiatric Assessment conducted with both children and parents/caregivers, as well as adulthood psychiatric status. Victimisation was assessed as part of the childhood assessment and involved multiple informants, but one limitation is that it did not distinguish between overt and covert victim experiences, which may have differential impact on the development of personality pathology.

One study found no significant relationship between childhood experiences of peer victimisation and later paranoid personality symptoms in adolescence (Natsuaki et al, 2009). The absence of an association, however, should be viewed within the context that

victimisation was measured (via observation) at baseline (age 9-12), whilst PPD was assessed only at follow-up (age 15). As such, whilst the study was conducted over a number of years, there was no baseline assessment of psychiatric status for comparison (the authors assessed and controlled for child maltreatment and childhood externalising behaviour problems but no further assessments were reported). PPD was also measured at symptom-level rather than diagnostically, because most participants did not reach criteria for diagnosis at follow-up. Thus, the clinical utility of the results in terms of identifying risks for PPD per se are limited.

### *3.3.ii Findings from Cross-Sectional Studies*

All nine cross-sectional studies reported statistically significant relationships between peer victimisation in childhood and various forms of personality pathology. Sansone and colleagues found moderate positive associations between being bullied and BPD (indicated by scores on the PDQ-4 that exceeded the threshold) as well as on the Self-Harm Inventory for their female sample (Sansone et al, 2013). This echoed the results of their earlier study (Sansone et al, 2010), which involved both males and females, although the effects were substantially larger in the mixed-sample study. Both studies have significant methodological limitations including non-representative samples and lack of statistical control for possible confounders (including comorbid psychiatric diagnoses, socioeconomic status, age). Peer victimisation measurement was also limited in being mainly dichotomous and not specifying a timeframe for bullying experiences. Finally – as is the case for all studies presented in this section – the cross-sectional, retrospective design curtails the possibility of making any conclusions about causality of peer victimisation experiences in the development of PD as well as the true direction of the reported relationship.

One study focused on schizotypal personality (Fung & Raine, 2012). The authors found a moderately strong, significant relationship between peer victimisation and overall

schizotypy, as well as significant associations between particular types of victimisation and aspects of schizotypal features: most notably, verbal forms of peer victimisation and social manipulation were most strongly associated with schizotypy. The study also identified 'high' versus 'low' victimisation differences, whereby those participants with the highest victimisation scores presented with more than double levels in schizotypy scores. A severe limitation of the study was the use of a single respondent via self-report measures on both victimisation and PD. There is some risk of social desirability bias in the results, particularly in light of the nature of the sample population (children aged 9-15, based in school). However, a very large sample (3,000+) was used which is an advantage in terms of representativeness and power to detect significant effects where those exist.

Hengartner and colleagues investigated ten PD types in their study and found statistically significant associations between peer victimisation and every PD. The strongest associations were evident for borderline and schizotypal PD; however, these were still quite small effects overall. Victimisation was self-reported, as was PD; the former was assessed in a dichotomous manner where participants were asked to respond yes/no and subjectively interpret the word 'frequently' in doing so. Thus, there are significant methodological limitations here which may introduce a degree of bias to the results. An advantage of the study, however, is that it provides some insight into a number of different PD forms, enabling some comparison across these.

Laporte and colleagues, using a sibling-pair design, also found a significant positive relationship between peer victimisation and BPD, however only for physical forms of peer victimisation (not emotional peer abuse). The observed effect was moderate. It should be noted however that the small sample size and constitution (in not looking at any additional siblings outside of each pairing) may have introduced sampling bias into results, particularly given that non-BPD siblings also reported significantly higher rates of peer abuse compared

to population norms (as the authors note). Potentially significant confounders, such as non-shared environmental factors, were not controlled for.

Zhu and Chan (2015) who focused on borderline and antisocial PD, found significant associations between borderline ‘traits’ and lifetime-prevalent direct and relational victimisation after controlling for factors such as family SES, gender, school type, substance use, attachment style and mood problems, but no significant relationship between antisocial personality and either form of victimisation. It should be noted that the odds ratios were small despite being significant. Due to insufficient information, it was not possible to calculate Cohen’s *d* effect sizes for comparison.

Further corroboration for a peer victimisation-BPD link can be observed in the two studies by Goodman and colleagues (2010, 2013) where a significant, positive relationship was reported both for the male sibling and female sibling samples. Subsequently calculated effect sizes indicate a medium effect, slightly greater in the female study. However, important methodological constraints must be noted here: firstly, given the web-based nature of measures, the samples in both studies may be non-representative of the broader community (or clinical) population and there is risk of uptake bias in the results. Secondly, information is based on parental-report, but there was no control for parental psychopathology, nor for current siblings’ demographic differences in adulthood. The retrospective design increases potential for recall bias, particularly in questions about early childhood development and victimisation, the latter of which is not triangulated with either child or teacher reports.

Lastly, a study conducted among a male prison population highlighted significant associations between self-reported peer victimisation in childhood and avoidant, histrionic, borderline and antisocial PD as assessed via SCID-II (Roberts et al, 2008); these effects were observed after controlling for age, ethnicity, axis I disorders and comorbid personality

disorders. The strongest relationship was noted with histrionic PD (subsequent calculations indicate a moderate to large effect), followed by Avoidant and Borderline, but both of these were relatively small effects. The results should be evaluated with caution, given that the sample is not representative; rates of personality disorder are typically disproportionate in prison settings (the authors themselves note that, in their study, there was a prevalence rate of 72.9% for any kind of PD). Reliance on self-report of early childhood experiences (including peer victimisation) as well as self-recall of childhood temperament is problematic as it may involve recall bias as well as conflation of such factors, which can be difficult to distinguish (Roberts et al, 2008).

In summary, there does appear to be some indication in the reviewed body of research of a link between peer victimisation in childhood and potential emergence of personality disorder, but this must be viewed in the context of several methodological limitations and subsequent threats of bias across all studies: this includes largely non-representative sampling, heterogeneity and at times questionable validity of measurement with respect to peer victimisation as well as personality pathology (particularly where based exclusively on self-report), predominantly cross-sectional/retrospective designs, and lack of statistical control for possible confounders.

The most prominent associations are evident with BPD, but schizotypal and histrionic PD have also been highlighted against a backdrop of being bullied. The implications of these findings are discussed below, as well as potential mechanisms that may help to elucidate the relationship.

## **4.0 DISCUSSION**

### **4.1 Addressing Key Questions: Quality and Adequacy of Current Evidence**

The core aim of the present review has been to identify and evaluate the quality and adequacy of evidence geared towards addressing the following questions: is the experience of bullying/peer victimisation associated with increased risk of emergent personality disorder in either *adolescence or in adulthood*; and is the experience of bullying/peer victimisation in childhood associated with higher risk of developing any *particular* personality disorder in adolescence or adulthood?

Fifteen studies were identified and their overall appraisal is suggestive of at least some association between peer victimisation (i.e. being bullied) in childhood and subsequent personality disturbance, particularly BPD. Of those studies that considered other bullying statuses, being a ‘bully-victim’ was found to be significantly associated with ASPD by some authors (Sourander et al, 2007) but not others (Copeland et al, 2013), although the odds of ASPD were notably higher for this group by comparison to victim-only status across the studies. In any case, however, it is clear that the evidence base at this stage is still relatively small, with significant methodological limitations that undermine its quality and current adequacy to answer the key questions. Perhaps most notably, the dominance of cross-sectional, retrospective research in this field impedes the possibility of drawing conclusions about the victimisation-PD relationship: we cannot, at this point, infer causality of personality pathology due to early peer victimisation experiences, nor indeed the directionality of the link. The prospective studies, while methodologically superior, also faced limitations due to lack of direct assessment of PD or related precursors at baseline and short-follow up periods, preventing conclusions about long-term, adulthood outcomes. One challenge, potentially, has been a historical hesitancy with regard to clinically assessing or diagnosing personality disorders in youth, which means that gathering information for prospective research has proven to be difficult.



It is nevertheless encouraging that existing research has involved both community as well as clinical populations, and that some of the reviewed studies have included very large samples, as these conditions typically facilitate more representative findings. While no firm conclusions can be drawn at this stage, there is clear indication that further research is warranted to further explore the relationship between being bullied and personality disturbances, given the suggested prevalence of peer victimisation across various settings and the profound impact that personality disorder can exert on an individual's emotional, interpersonal and physical functioning.

#### **4.2 Theoretical Considerations: How Might Peer Victimisation lead to Personality Disorder?**

At the outset of the present review, three prominent models of BPD development were presented. This was done on the proviso that these models may be appropriate in helping to explain the developmental pathways of other PDs as well, particularly in light of increasing recognition that most PDs present significant amounts of shared core pathology and that BPD is often cited as a prototypical personality disorder in this respect. In this section, these models are revisited in more detail in order to consider potential mechanisms and explanations behind the association between peer victimisation and subsequent personality pathology, as indicated by the present review. It is important to note that the reviewed research most notably points towards a relationship between being bullied and BPD, thus a discussion of these models is all the more warranted; however, additional mechanisms are also addressed that may help to clarify how experiences of being bullied could link to personality disorder. Finally, within the context of some (albeit mixed) evidence for a relationship between being a 'bully-victim' and ASPD specifically, some initial thoughts are given to the nature of this association.

#### *4.2.i Peer Victimisation Constitutes an Invalidating Environment and ‘High Risk’ Interpersonal Transactions*

The biosocial theory of BPD (Linehan, 1993) proposes that BPD is characterised by dysregulation across all aspects of emotional responding: this is broadly conceptualised to include emotion-linked cognitive processes, facial reactions, action urges, biochemistry and physiology (cf. in Crowell et al, 2009). It is argued that emotional dysregulation arises in the process of interaction between an individual’s biological vulnerabilities with particular environmental conditions during early development, and leads to dysfunctional response patterns during emotionally challenging situations (Crowell et al, 2009). This dysfunctional response can include increased emotional sensitivity to stimuli, a sense of dysregulation of intense emotional experience, and a slower return to ‘baseline’ following heightened emotional response. One of the key environmental influences on the development of BPD that Linehan suggested was an individual’s development within an ‘invalidating context’, whereby it is regularly communicated to the child that their emotional expressions are unwarranted and unjustified, and that emotions should be dealt with internally and privately without support from the parent/carer. The outcome of this is that it prevents the child from learning to understand or tolerate their own emotional reactions, and the child does not learn how to label their emotional experience. This results in the commonly observed BPD characteristic of fluctuating between emotional inhibition and emotional lability.

Linehan’s original biosocial theory has since been revisited and extended by Crowell and colleagues, who draw on more recent scientific understanding of the potential biological correlates of BPD and consider the interplay between these and environmental stressors<sup>2</sup>.

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<sup>2</sup> This includes deficits in the functioning of the central serotonin system (5-HT), hypodopaminergic functioning and vasopressin; they also note that emotional lability/affective instability appears to be associated with deficits in the cholinergic and noradrenergic systems and heightened hypothalamic pituitary-adrenal axis responding, which occurs predominantly in interaction with environmental factors including experience of maltreatment in youth and, more broadly, with prolonged exposure to stress.

From a psychosocial viewpoint, the authors note that - as well as emotional dysregulation - early *trait impulsivity* is also a significant vulnerability factor for BPD, and for emotional dysregulation itself. They suggest that this is likely to exacerbate risk during development due to its evocative impact on interpersonal relationships and social functioning, subsequently impairing healthy emotional development (Crowell et al, 2009). The authors review other significant psychosocial risk factors for BPD including family psychopathology, disrupted attachment relationships and child maltreatment; the latter of these in particular, they point out, undeniably contributes to one's environment being a highly invalidating developmental context, to follow Linehan's terminology; however, it should be noted that the general consensus in current literature is that a history of abuse and maltreatment is neither sufficient nor necessary for the development of BPD (Zanarini et al, 1997; Crowell et al, 2009).

The model of BPD that Crowell and colleagues ultimately present is comprehensive and relatively complex, but can be summarised as follows: they propose that the child's early biological vulnerabilities influence her temperament (e.g. negative affectivity, impulsivity, elevated emotional sensitivity), which in turn affects their environmental context via transaction with the caregiver; this transaction can be described as 'high risk' if the caregiver's contribution within the transaction includes invalidation of the child's emotions, negative reinforcement of aversive emotional expression, ineffective parenting (either due to poor 'fit' given the child's temperament and/or lack of family resources). In interaction, these two factors - i.e. impulse control deficits and emotional sensitivity plus environmental reinforcement of emotional lability (especially by the caregiver) - result in increased risk for psychopathology for the child, as well as heightening emotional dysregulation. The child's capacity to process information is impaired, as is her ability to control mood-dependent behaviour. If this process is repeated over time, through numerous transactions of this type

with the caregiver, there is an increased risk for negative outcomes including social isolation, problematic peer relationships, hopelessness and low self-efficacy, emotional vulnerability, and oscillation between impulsive behaviour (including self-injury) and withdrawal, which collectively fit the profile for BPD diagnosis.

Against this background and in the context of the present review's findings that childhood experiences of victimisation are associated particularly with BPD in adulthood, a possible hypothesis is that in early and middle childhood, a time during which peer relationships become more central and significant to one's social development and functioning as well as identity formation, it is plausible that the experience of peer victimisation (which may involve emotional and/or physical abuse) itself constitutes a multitude of 'high risk' transactions for the child at a time where an increasing proportion of the child's interactions and communication are within the peer group setting. This is particularly the case for children who may harbour early biological vulnerabilities such as elevated emotional sensitivity and/or impulsivity. It is also possible that these biological dispositions are themselves risk factors for becoming a victim of bullying (Wolke, Schreier, Zanarini & Winsper, 2012; Crowell et al, 2009). Peer victimisation provides a highly invalidating context, for example through peers' expression of hostility towards the child (both direct and indirect), mockery of the child's emotional responses or negative reinforcement of extreme emotional expression (e.g. the child has to respond adversely to get help or support from others in the face of victimisation). Repeated experience of victimisation, where the child's vulnerability is met with an invalidating environment, may serve to heighten emotional dysregulation over time. It is important to note, however, that other risk and protective factors (including the child's family environment, caregiver interactions, other social relationships etc.) require consideration in terms of their potential

mediating role in the association between victimisation and subsequent development of personality pathology.

#### *4.2.ii Peer Victimisation Inhibits Mentalisation and Reflective Function*

An alternative theoretical conceptualisation of personality disorder has been proposed by Fonagy and colleagues, who postulate that some characteristics of borderline personality disorder can be seen to originate from developmental pathology associated with the inhibition of *mentalisation* and *reflective function* (Fonagy, 2000). Broadly, these concepts refer to the capacity to think about mental states in oneself and in others, and they are significantly related to making sense – and finding meaning – in the behaviour of others and one's own. The authors suggest that this capacity is hindered when a child's early attachment relationship with his primary caregiver does not offer a secure space for the child to explore the mind of the caregiver and learn about mental states in the self and others. It is suggested that this might occur most commonly in circumstances of early trauma experiences such as abuse, as the child may defensively inhibit their capacity to 'mentalise' about their caregiver's wish to intentionally harm him. This leaves the individual operating on inaccurate impressions of others' feelings, thoughts and intentions, which renders them highly vulnerable particularly in intimate relationships which are emotionally charged attachment relationships (Fonagy, 2000).

The absence, or deficit of, mentalisation and reflective function prevent an individual from being able to think about (the representation of) reality with various alternatives at their disposal; they are unable to think about the possibility of themselves or another holding a false belief, for instance in terms of the other's desire or intention towards them. When the actions of others are unexpected, usually mentalisation provides a 'buffer' that allows one to think about various hypotheses to account for those actions, without automatically

concluding that it was malicious in its intent. When no such mentalising buffer is available, one's 'internal working model' that develops within the early attachment context and trauma experience leads the individual to assume that malice and intentional harm are probable.

Fonagy suggests that this may account for the commonly observed emotional instability and irritability in individuals with BPD. Other symptoms frequently observed include impulsivity; Fonagy proposes that this may be due to an individual's lack of awareness of their own emotional states, related to the absence of their symbolic representation in the individual – this representation, it is argued, would normally be achieved within the context of a secure child-caregiver attachment, whereby the caregiver appropriately recognises and external 'mirrors' the child's internal emotional states, enabling the child to internalise representations of himself as an intentional, thinking, feeling agent. (For a more detailed exploration of BPD symptomatology and specific association with mentalisation deficits see Fonagy, 2000).

Fonagy's model of BPD could help to elucidate the possibility of an association between victimisation and subsequent development of the disorder, if we consider how the experience of perpetual peer victimisation/bullying could impact upon an individual's mentalising capacity and reflective functioning. Given that the skill of mentalising is maintained through an interpersonal and intersubjective process, hostile peer relationships force an individual to close off others' minds, as part of an adaptive, protective attempt to limit exposure to harmful psychosocial encounters. There is potentially substantial overlap between this theoretical stance and Linehan's approach, since emotional dysregulation will compromise one's ability to mentalise; meanwhile, one's tendency – and vulnerability – to misperceive interpersonal situations through impaired mentalisation can readily generate emotional arousal.

#### *4.2.iii Peer Victimisation Experiences Interact with Interpersonal Hypersensitivity and Increase Likelihood of Disorganised Interpersonal Strategies*

A theoretical stance presented by John Gunderson in his interpersonal hypersensitivity' model of BPD may shed further light on how being bullied could increase risk for subsequent personality disorder (Gunderson & Lyons-Ruth, 2008). Gunderson suggests that individuals with BPD typically exhibit interpersonal styles characterised by a paradoxical combination of an intense need for closeness and attention with an intense rejection sensitivity and fear of abandonment. The latter of these aspects in particular, he notes, is generally accepted as the more pathogenic component of the BPD patient's interpersonal-style (since need for closeness and attention are considered to be adaptive and typical from an evolutionary perspective). Gunderson refers to this as the 'interpersonal hypersensitivity phenotype', noting that research indicates a relatively high rate for its heritability (e.g. 0.48, Jang et al, 1996). Such findings would suggest that interpersonal hypersensitivity represents a genetically rooted disposition – or risk factor – rendering some individuals more vulnerable to developing BPD in adulthood. According to Gunderson, individuals with interpersonal hypersensitivity are more vulnerable to criticism and other interpersonal confrontation or negatively perceived social encounter. It plausibly follows that they would also be more vulnerable to bullying; that is, where bullying occurs, their experience of victimisation is likely to create a much more powerful emotional reaction than it would for someone without a disposition for interpersonal hypersensitivity. The interplay of the stressful peer interactions with the individual's hypersensitivity may, over time, develop into disorganised and controlling interpersonal strategies that in turn provide the springboard for the typically contradictory interpersonal features of BPD to arise (Gunderson and Lyons-Ruth, 2008).

#### *4.2.iv Peer Victimisation is a Negative Socialising Context*

Another explanation linking peer victimisation and PD could be that early experiences of being bullied provide a negative socialising context, by facilitating observation and assimilation of hostility between peers, direct and indirect aggression as means of expressing emotion, and enmeshed peer networks. Fung and Raine, in considering schizotypal PD more specifically, also note that children who are verbally, physically and socially victimised could be reasonably expected to reactively develop paranoid ideation, social anxiety, hypersensitive forms of self-referential thinking and hostile attribution bias, all of which feature across the three major factors of schizotypal personality (Fung & Raine, 2012). This could possibly be explained by hypervigilance for threatening stimuli in social situations. It could also be that persistent difficulties and ruptures in important peer relationships place constraints on a child's subsequent trust towards others, casting doubt over loyalty and exacerbating fear of abandonment. In some ways, these may be similar processes to those known to occur in the case of early childhood maltreatment and subsequent BPD (Battle et al, 2004).

Being a 'bully-victim' might also provide a negative socialising context, but perhaps one with particularly striking consequences for the individual given the dual nature of this status. Sourander and colleagues found that being a 'bully-victim' was significantly associated with ASPD (Sourander et al, 2007), and it could be argued that the impact of being a perpetrator at the same time as a victim may lead to conditions that make the further propagation of antisocial behaviour, as well as hostility and undermined trust towards others, much more probable than otherwise. Existing research, not focused on PD as an outcome specifically, consistently points towards particularly suboptimal outcomes in terms of psychological and social functioning for the bully-victim group by comparison to bully- or victim-only (e.g. Haynie et al, 2001). For instance, bully-victims generally display higher rates of problematic behaviour and aggression, lower self-control and social competence (which will impact their ability to form positive, nurturing and protective friendships), and



are involved in more deviant peer groups where aggression and violence is considered to be an acceptable behaviour. Where such behaviour is reinforced by one's group membership, bully-victims may be at particular risk of developing and continuing antisocial behaviour into adulthood (Haynie et al, 2001). Furthermore, through a combination of persistent experiences of intimidating others, using aggression in order to express emotion as well as make gains and have one's needs met, at the same time as feeling victimized oneself and facing continuous invalidation and marginalisation by one's contemporaries, a clearly suboptimal environment is set for the development of pro-social interpersonal skills. It is also likely that the negative socialising context begins prior to - and extends beyond - the peer group for the individual. There is significant research to suggest that young people with conduct problems and antisocial behaviour are more likely to experience harsh parenting within their home environment, including hostile, authoritarian discipline, permissiveness and neglectful caregiving and explicit modelling of violence by caregivers (Pinquart, 2017), which is likely to contribute towards the development as well as reinforcement of similar behaviour for the child through genetically and environmentally interactive pathways.

#### *4.2.v A Question of Directionality*

There is significant conceptual overlap between several of the above-mentioned theories, even where the core focus of specific orientations might vary. One aspect that unites Linehan, Fonagy and Gunderson's models is that the development of personality disorder is seen as a process marked by bidirectional interaction and transaction between individuals' genetic/biological vulnerabilities and environmental conditions within which they function. It is important to acknowledge that such environmental influences clearly include, but are not limited to, the nature of early attachment and interaction style with the caregiver; other

environmental conditions are likely to be implicated (including the peer context) and be of varying salience, depending on one's developmental stage.

The fundamental importance of adopting a multi-level perspective when considering developmental pathways to personality disorder is underscored when we consider the multifinality of outcomes for individuals who may face similar environmental or biological risk factors (e.g., disorganised attachment in infancy is neither sufficient nor necessary for the emergence of borderline personality disorder, neither is experience of trauma and/or maltreatment in childhood - Zanarini et al, 1997). Similarly, when considering personality development more broadly, evidence strongly suggests that temperamental factors as well as a plethora of life experiences, as opposed to predominantly early parent-child interaction, appear to be related to individual differences in personality continuity and change over the life-course, including into older age (Caspi & Roberts, 2001). Relationships within one's social network provide the social context for personality development; moreover, ongoing relationships and personality co-develop over time, since individuals commonly both select and evoke relationships that accentuate their personality traits (Neyer & Lehnart, 2007).

With this latter point in mind, the association between peer victimisation and PD may be even more complex: it is possible, as some authors have suggested, that the interpersonal difficulties and emotional dysregulation typically seen in BPD are not the outcome of being bullied, but potentially a precursor, if not a cause, of it (Wolke et al, 2012). It could be, for example, that high sensitivity, emotional instability, impulsivity, disruptive or chaotic behaviour, poor mentalising or eccentricity in childhood might trigger certain hostile reactions among peers, rendering this group of children particularly vulnerable to bullying (Malt et al, 2015; cf. in Monsvold).

Finally, it is also possible that peer victimisation and PD are not strictly causal in either direction, but rather that these experiences do merely covary as a function of being

similarly associated with other, adverse experiences or dispositions in childhood, such as neglect or abuse (Sansone, 2010). However, given some of the research here which utilised robust controls to minimise such covariance effects and nonetheless found notable links between bullying experiences and PD, this is perhaps the least plausible, and the least informative, explanation.

### **4.3 Limitations**

The present review included fifteen quantitative studies, identified via a systematic literature search. However, a number of additional papers which were identified as potentially relevant were unobtainable at the time of the search (either through being unavailable as full-text or because the original author was not contactable), which could influence the overall findings highlighted here and the general theme emerging from results. One paper was initially identified as relevant because it investigated the relationship between victimisation at school and personality disorder, however it was subsequently excluded from the review as bullying in this study was perpetrated by teachers as opposed to peers (Monsvold et al, 2011).

Qualitative research and single case-studies were not included in the review, which is a potential limitation in terms of scope. However, the systematic database search did not identify any such studies. Similarly, studies in languages other than English were not included, but the systematic search did not identify relevant non-English studies at the time when search limits were not applied.

The literature search was performed primarily on peer-reviewed academic journals via database search; grey literature including government or academic reports, working papers, white papers or other articles were not included in the systematic search. However, this was done to help ensure the highest quality of data being included in the review. It may

be that a broader search incorporating additional materials could enrich the data and identify qualitative accounts and case studies that were not found upon database search.

#### **4.4 Implications and Future Recommendations**

With a gradual shift towards the acceptability of earlier assessment of personality difficulties, there is scope for more robust, prospective research in this arena in future. Given the number of potential environmental, familial and biological factors that have been linked with development of PD, future studies investigating peer victimisation as a predictor of PD must also adequately control for such confounders. More research that incorporates formal, validated measures of peer victimisation is needed, and ideally information on PD status should be aggregated through involvement of multiple informants (e.g. participant self-report, clinician-report, parent/carer). Further research focusing on a range of specific personality disorders would also be valuable, to help decipher whether peer victimisation has any unique effects in the lifetime trajectories of these.

Existing research could be further enriched by qualitative studies exploring young people's experience of being bullied in childhood, their perspective on living with a personality disorder and how they make sense of its impact on their lives. Such research was not included in the present review because none was identified during the literature search. Notwithstanding the limitations of the studies included in this review, there does appear to be broad consensus in the evidence about potentially significant links between being bullied by peers and presenting with PD or relevant symptoms. This association should not be taken lightly; it should be seen as further indication that being bullied can have a potentially far-reaching and long-term impact on its victims, one that may even penetrate the very fabric of their identity and influence their subsequent social relationships. It should strengthen the message that we need to target peer victimisation in schools and other peer settings, via

adequate anti-bullying policies, pro-social training programmes for pupils and awareness-raising among young people, parents, carers, teachers and the broader community. Schools must implement and encourage clear, safe and supportive channels for reporting incidents of bullying to staff to ensure that no child is left feeling vulnerable, isolated and suffering in silence. Clinically, children and adolescents who are already victims, or at risk of, peer bullying, should have available to them appropriate, non-pathologising psychological support as well screening (perhaps within school settings as well as in the community) to monitor for subsequent or coexisting mental health difficulties. Finally, the review findings should hopefully challenge any remaining sentiment that bullying is a mere rite of passage, for it is clearly neither normative, nor harmless, to those who experience it.

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## **PART II: EMPIRICAL PAPER**

**Developing a 'Profile' of Emergent Personality Disorder among Adolescents: The Effect  
on Treatment Outcomes in the IMPACT Adolescent Depression Study**

## ABSTRACT

**AIMS:** Existing research supports the validity and importance of Personality Disorder (PD) assessment, diagnosis and its clinical management in adolescence. In adult populations, PD comorbid with depression has been shown to negatively impact on depression treatment outcomes, yet similar research among adolescents is scarce. Based on data from the adolescent depression IMPACT trial, the present study aimed to construct an emergent ‘PD profile’ to indicate the extent of personality pathology among adolescents. The profile was used to investigate whether emergent PD predicted depression treatment outcomes. It was hypothesised that greater scores on the PD profile would be associated with greater depression severity at follow-up (post-treatment), higher rates of treatment drop-out and lower likelihood of recovery from depression. An interaction with treatment modality was also predicted whereby CBT may be associated with better outcomes than STPP due to its simpler, goal-oriented focus and structure and lower likelihood of entanglement with complex difficulties around PD.

**METHOD:** A range of measures used in the IMPACT trial were explored at item level. Forty items were identified as thematically relevant to PD. Based on a total sample of 449 subjects, exploratory factor analysis was conducted to identify core latent constructs towards the PD profile. A two-factor profile was taken forward for subsequent analyses. Multiple regression analyses were performed to test for the key hypotheses.

**RESULTS:** PD profile scores did not predict depression severity post-treatment, nor withdrawal from treatment or likelihood of recovery. There was no difference between CBT and STPP with regards to depression outcomes post-treatment.

**CONCLUSION:** The findings from this study were considered in the context of its methodological limitations and cannot be extrapolated beyond the current sample without further validation of the PD profile. Overall, it was encouraging that emergent PD features did not appear to be an obstacle towards positive treatment response for the majority of adolescents in the study. However, a large proportion of young people were still above clinical threshold for depression following treatment, and possible reasons for this were contemplated. Predominantly, the proposal of an underlying propensity for psychopathology in recent literature was considered and whether this may be a better predictor of treatment non-response than PD. Developments for future research are suggested.

## 1.0 INTRODUCTION

In the past decade, evidence has grown to support the validity and importance of Personality Disorder diagnosis and its clinical management not only in adult populations, but also in adolescence. Several precursors of personality pathology are usually already emergent at this stage which echoes the fact that personality disorders are, by definition, chronic rather than discrete, typically with an early onset and persistence into adulthood (Paris, 2003). While there is some variability reported in the course and prognosis of different personality disorders over time, studies consistently show that essentially all are associated with significant impairment in terms of an individual's emotional, interpersonal and physical functioning, and in many cases prove to be lifelong conditions that are challenging to treat clinically (Skodol et al, 2002). Within a context of potentially devastating implications, early assessment and intervention is highly relevant and justified.

Furthermore, adult-population research points towards the pervasive comorbidity of personality disorders with other psychiatric conditions such as major depression, as well as identifying that such comorbidity is associated with generally poorer outcomes (Newton-Howes, Tyrer, & Johnson, 2006). Similar research, however, remains sparse for adolescent populations, despite broad agreement that major depression among young people is associated with short- and long-term adverse consequences for individuals, their familial and social groups and the wider society, particularly without appropriate and timely therapeutic intervention.

The present study aims to contribute towards addressing this gap, by investigating whether the presence of emergent personality pathology in adolescents with concurrent major depression is likely to adversely affect their depression treatment outcomes. Drawing on data gathered across multiple outcome measures in the national IMPACT trial (Goodyer et al, 2016), the study specifically strives to construct a 'profile' of measurable, emergent

personality psychopathology symptoms among adolescents receiving therapy for major depression across three treatment modalities (CBT, Short-term Psychoanalytic Psychotherapy and a standard Brief Psychological Intervention). It is anticipated that young people who demonstrate higher levels of symptoms within this profile will have worse treatment outcomes overall. Worse outcomes are conceptualised in terms of a) greater severity of self-reported depression symptoms at follow-up b) higher rate of treatment drop-out and c) lower rates of recovery from depression according to typical clinically significant thresholds on validated self-report measures. It is anticipated that CBT might have superior outcomes to STPP, since it is typically more goal-oriented, focused on current difficulties and less likely to involve therapeutic elements that pertain to, and can become entangled with, complex personality issues. The implications of this for further research, as well as for clinical assessment/intervention with adolescents with major depression and complex presentations, are subsequently considered.

### ***1.1 Conceptualisation of Personality Disorder and its Core Components***

At the outset, it is essential to define our current understanding of ‘personality disorder’ and its core components, in order to inform the development of an emergent personality disorder profile in adolescents in the present study.

‘Personality disorder’ (hereafter referred to as PD) broadly refers to a “pervasive and enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individual’s culture, is inflexible, stable over time and leads to significant distress and functional impairment” (APA, 2013). The current diagnostic approach in the DSM-5 identifies at least ten distinct sub-types of PD, which are assigned to one of three major ‘clusters’ (Cluster A: Odd, bizarre, eccentric, Cluster B: Dramatic-erratic; Cluster C: Anxious-fearful). The various types of PD are characterised by particular patterns of

presentation. However, all appear to share important features that are fundamentally manifested in individuals' maladaptive *cognitive, affective* and *interpersonal* functioning (including dysfunctional ways of perceiving and interpreting self, other people and events; markedly intense, labile or otherwise maladjusted emotional response) as well as impaired impulse control (APA, 2013).

In response to a growing body of research highlighting the limitations of a categorical approach to PD diagnosis (including high rates of comorbidity between various PDs) and support for a dimensional conceptualization of personality pathology instead, an alternative model of PD has also been proposed in the DSM-5. Here, PDs are characterized by moderate to severe impairments in personality *functioning* ('Criterion A': including impairments in self and interpersonal domains, reflected in disturbances of identity and self-direction, empathy and intimacy) and pathological degrees of personality *traits* (Criterion B: including specific trait facets across domains of Negative Affectivity, Detachment, Antagonism, Disinhibition, and Psychoticism). A matrix illustrating these domains and their facets can be seen in Appendix 1. These traits are seen to exist on a continuum (with opposing poles at either end), whereby all individuals – both those with suspected personality pathology and those without – would fall somewhere along the spectrum. Those at the extreme ends are likely to exhibit disordered personality symptoms. For PD diagnosis to be considered, judgement must therefore be made about an individual's degree of particular traits, rather than their presence or absence.

The dimensional approach to PD is more intuitive in the context of findings that abnormal personality is highly related to normal personality functioning, which itself is usually considered in continuous terms (Lawton et al, 2011). There is substantial evidence supporting the notion that extreme levels of core personality traits – drawing on the extensively-validated Five-Factor Model (McCrae & Costa, 1996) – contribute to, if not



constitute, the maladaptive nature of PD. For example, Lynam and colleagues were able to demonstrate that the Five-Factor Model can be used to depict comprehensive, dimensional prototypes for all DSM-IV PD categories (Lynam & Widiger, 2001). In their study, BPD for instance was characterised by high scores on all Neuroticism facets except for self-consciousness; low scores on Agreeableness, particularly on the compliance facet; and low scores on Conscientiousness, particularly on the facet of deliberation. The highest scores for particular facets were for Anxiousness, Angry Hostility, Depressiveness, Impulsiveness and Vulnerability, Feelings and Actions. There is also some evidence that two personality dimensions in particular - high neuroticism and low agreeableness - seem to be associated with nearly all PDs (Lawton et al, 2011).

In summary, there is a growing evidence base in support for a dimensional perspective on personality pathology, from which a number of specific ‘core components’ emerge that seem to generalize at least in some part across most PDs: as highlighted in the DSM-5 and the afore mentioned literature, this includes an increased degree of traits such as negative affectivity (high neuroticism, instability of emotion, depressivity, anxiousness), detachment (including withdrawal or avoidance of personal/emotional attachments, suspiciousness); antagonism (low agreeableness, as well as manipulateness, callousness and hostility), disinhibition (impulsivity, risk-taking and self-injurious behaviour) and psychoticism (may include odd/eccentric beliefs as well as lack of lucidity of thought manifest through depersonalization or derealisation, or disturbed self-image). What appears to lay at the heart of severe personality pathology is the fundamental disturbance that these aspects inflict upon the way in which individuals’ experience themselves and their interpersonal worlds, characterised by emotional dysregulation, confusion and volatility.

## ***1.2 Emerging Personality Pathology in Adolescence: The Validity and Importance of Assessment***

Amongst adolescent populations, Borderline Personality Disorder (BPD) has seen the greatest increase in empirical studies over recent years and it is suggested that this is reflective of the fact that BPD is likely to be most representative of the core psychopathology evident across all PDs (Sharp & Fonagy 2015, Higgitt & Fonagy 1992). BPD is a complex psychiatric condition, characterised by affective instability, impulsivity, interpersonal problems, cognitive and identity disturbances and suicidality (APA, 2000; Crick et al, 2005; Bateman & Fonagy, 2009).

While decipherable symptoms of BPD are likely to first emerge during adolescence (Bradley et al, 2005; Westen & Chang 2000; Stepp, 2012; Bateman & Fonagy 2009), and existing research suggests that self-harm - which is characteristic of BPD - appears to have an adolescent onset in two-thirds of all BPD cases (Zanarini et al, 2006), diagnosis of this disorder, or any PD for that matter, among young people has long been controversial. Historically there has been resistance in doing so prior to adulthood due to the argument that key developmental aspects including personality traits, emotional self-regulation and autonomous behavioural control are not stable until then (Griffiths, 2011). On the other hand, early detection and treatment intervention appropriately matched to diagnosis is likely to improve the adulthood outcomes of individuals who experience any PD and associated impairment in youth (Sharp et al, 2012). Recent evidence suggests, for example, that BPD symptoms during university/college years are associated with poor social and academic outcome (Bagge et al, 2004), and with compromised adult functioning including lower occupational attainment (Winograd et al, 2008), as well as poorer outcomes in intimate and social relationships (Stepp, 2012).

There is growing evidence to suggest that BPD can be meaningfully and accurately diagnosed during adolescence (Miller et al, 2008) and also that the stability and reliability of BPD symptoms and personality features is relatively high both pre- and post-18 years of age (Clark, 2009; Cicchetti & Crick, 2009). Research suggests that while there is some decline in the mean level of BPD traits from adolescence into young adulthood, the rank-order stability of these traits is high over this period and reflects the stability of BPD traits found during adulthood (Bornovalova et al, 2009; Lenzenweger, 1999). Furthermore, existing evidence estimates the cumulative prevalence for BPD at age 14 and age 16 to be 0.9% and 1.4%, respectively (Johnson et al, 2008), with significantly higher prevalence in clinical samples ranging between 11% and 50% in some psychiatric populations (Chanen et al, 2004). With these considerations in mind, there are strong grounds to propose that identifying emergent BPD markers (which may well constitute markers of other PDs at this stage) in adolescence is feasible as well as relevant both to the immediate and long-term outcomes of youth, and to the trajectory of their treatment: thus, it is important to look at it more closely.

### ***1.3 Personality Disorders and Major Depression: Implications for Treatment and Outcomes***

In adult populations, existing research indicates high concurrence between PDs and an array of affective disorders, with major depression being possibly the most ubiquitous (Biskin & Paris, 2013). According to Grant and colleagues, approximately 75% of individuals with a lifetime BPD diagnosis meet criteria for a lifetime mood disorder (Grant et al, 2008). Other studies suggest rates as high as 96% of patients with BPD having a mood disorder during their life, with lifetime depression reported at 71% to 83% (McGlashan et al, 2000; Zanarini et al, 1998).

In the context of significant comorbidity rates between PDs and major depression, the former (especially BPD) have received increasing albeit still limited attention in research aiming to explore their influence and prognosis for the course and outcome of such disorders (Grilo et al, 2000; Mulder et al, 2002; The Collaborative Longitudinal Personality Disorders Study – Grilo et al, 2005; Skodol et al, 2011). Such research generally points to the conclusion that BPD is associated with adverse effects including significantly slower remission from depression and faster relapse for patients, by comparison to those without BPD (Grilo et al, 2005). Other research has more specifically investigated the potential effects of BPD comorbidity on treatment outcomes. A meta-analysis conducted by Newton-Howes and colleagues, notably, concluded that personality disorders are associated with poorer outcomes in treatment studies of adult depression (Newton-Howes, Tyrer, & Johnson, 2006). However, there is also research that is less conclusive. A systematic review by Mulder suggested that studies yield mixed results, noting that whether or not personality pathology significantly worsens outcome in patients with major depression depends largely on study design, including how personality pathology is measured (therefore also the prevalence rates). The author also notes that depressed patients with personality pathology appear less likely to receive adequate treatment in uncontrolled studies. It is also possible that depression characteristics, such as chronicity and severity, may be directly related to personality pathology and this should be – but often is not – controlled for in trials if we are to accurately assess the unique contribution of personality pathology to treatment outcomes (Mulder, 2002). In his review, Mulder concludes that in general, the best-designed studies reported the least effect of personality pathology on depression treatment outcome (Mulder, 2002).

Parallel research, investigating potential moderating and/or mediating effects of PDs on the course of major depression and other psychopathology, among adolescents remains somewhat limited. Sharp and colleagues conducted a trial to investigate the incremental

validity of BPD relative to major depression at predicting suicidal ideation and deliberate self-harm among adolescents, finding support for this (Sharp et al, 2012) but further longitudinal studies are urgently needed. Even more limited is the current body of research with respect to the possible impact of PD comorbidity on adolescent *treatment* outcomes for major depression. Such research would be highly warranted given that it could help to elucidate not only prognosis for young people's longer-term functioning, but also potentially reveal important considerations to bear in mind when planning treatment intervention for adolescents with comorbid PD-MDD presentations. One such consideration may be whether particular treatment protocols could fare better or worse overall for individuals with PD-concurrent depression in terms of initial treatment uptake, duration of clients' engagement with the treatment and ultimately the therapeutic effectiveness, improved mood and enhanced quality of life.

The importance of knowing what is likely to work best for whom amidst such complex difficulties in adolescence is clear. Firstly, it is estimated that 1 in 10 referrals to child psychiatrists in Child and Adolescent Mental Health Services (CAMHS) involve significant depressive conditions (Goodyer et al, 2011), and that at least 30% of adult affective disorders including depression start in adolescence (Keenan-Miller et al, 2007). Thus, the demand for the most cost-effective early intervention is potentially very large. Secondly, we know that major depression among adolescents entails significant adverse outcomes and costs for the individuals concerned, their families, friends, the wider society, as well as for the NHS including increased risk of depression relapse in adult life (Harrington & Dubicka, 2001; Lewinsohn et al, 1999), non-affective disorders (Lewinsohn et al, 1999) suicidal behaviour (Fergusson et al, 2007), alcohol dependence (Crum et al, 2008) and unemployment (Fergusson et al, 2007). Therefore, it is imperative to strive for not only the most effective but also the most acceptable treatment for young people (one that they are

most likely to engage with), taking into account what is likely to work best for those with comorbid emergent personality disorder. Refining the evidence base and clinical guidelines with regards to this is likely to help limit the costs associated with depression both for the individual, their family and for public health in the long term.

Within this context, an area of particular interest and the focus for the present study is the initial development of a 'profile' of emergent PD symptomatology among adolescents with moderate to severe depression. The profile aims to embrace the core components of PD, and will be constructed through a compilation of items from a range of outcome measures used in the IMPACT Trial that are considered to reflect these components (Goodyer et al, 2016). The study involves secondary analysis based on this data.

The above endeavour leads onto the following question:

*Q. Can an emergent PD profile be used to predict adolescent individuals' adherence and response to currently available treatment for major depression (when controlling for severity) in the IMPACT study?*

Drawing on existing adult population literature, it is hypothesised that adolescents who score highly on the emergent-PD profile will have worse depression treatment outcomes than those with lower scores. Specifically, they are expected to a) show greater depression severity post-treatment compared to low-PD scorers, even when controlling for initial severity; b) be less likely to engage with treatment/more likely to drop out before completing the treatment course and c) show lower rates of recovery from depression than low PD-scorers at the final post-treatment follow-up. It is also predicted that there may be an interaction effect between treatment modality and PD. CBT may have better outcomes with regards to depression severity than STPP, due to being oriented towards a specific, simple

goal, being structured around current difficulties and thus less likely to become entangled with complex personality issues.

#### **1.4 *Improving Mood with Psychoanalytic and Cognitive Therapies (IMPACT Trial):***

##### ***Background and Considerations of Personality Pathology***

The IMPACT study (Goodyer et al, 2016) is a randomised-controlled, pragmatic effectiveness superiority trial designed to investigate whether specialised psychological treatment reduces the risk for relapse in adolescents with moderate to severe unipolar depression, compared to a brief psychological intervention akin to standard care. The trial specifically compared the effectiveness of three therapeutic interventions in the treatment and relapse prevention of adolescent depression over a period of 86 weeks: Cognitive Behavioural Therapy (CBT), Short Term Psychoanalytic Psychotherapy (STPP) and Brief Psychological Intervention (BPI). The trial also involved intricate health economics analysis to investigate the cost-effectiveness of specialised treatment for major depression in adolescence and analyses were conducted on an intention-to-treat basis. It is a, multi-centre, NHS-based trial which recruited young people (aged 11-17) from 15 CAMHS services across three UK regions. Detailed primary findings from the IMPACT study were recently published and are available in *The Lancet* (Goodyer et al, 2016). In brief, the authors found no evidence for the superiority of CBT or short-term psychoanalytical therapy compared with a brief psychosocial intervention in maintenance of reduced depression symptoms 12 months after treatment. Short-term psychoanalytical therapy was as effective as CBT and the team concluded that these, along with brief psychological intervention, offered more choice of therapy for adolescent patients attending routine CAMHS clinics.

The IMPACT study protocol included one validated measure of borderline personality disorder specifically – the Zanarini Rating Scale for Borderline Personality

Disorder (ZAN:BPD) (Zanarini et al, 2003). This is a structured clinical interview which was originally administered by the research team at baseline and 52 or 86 week follow-ups and is an indicator of presence/absence of BPD and its severity. However, the present study does not focus on this particular measure in terms of its relationship to subsequent depression treatment outcomes. This is because preliminary screening analyses of the data revealed significant inconsistency in how the measure was administered, whereby subjects completed the measure at differing time points, some completing it once (not necessarily at baseline) while others at two or three time points. This rendered comparison across subjects difficult, not least because of the possibility that some of the variability among subjects may have been attributable not to their personality pathology symptoms, but by the fact that many were already in – or even after - therapeutic treatment in the trial at the point of completing the interview. For those who completed the measure at two or more intervals including baseline as per original study protocol, some subjects had inconsistent outcomes in terms of presence of BPD across the two time points (there were two cases of direct opposite diagnoses with one interval stipulating definite presence and one absence of BPD). Missing value analysis indicated 34-38% missing data for the ZAN:BPD overall and at item-level at baseline, which was considered not acceptable for further analysis in the current study. The option to remove cases with missing data in the ZAN:BPD diagnosis was not viable as this would have compromised the overall sample size as well as potentially introducing bias into any findings. Finally, out of the whole eligible sample in the IMPACT study (n=465), only 10% subjects had either a possible or definite diagnosis score at baseline on the ZAN:BPD measure, which is significantly lower than would be expected in a clinical adolescent population. Whilst this alone is not reason enough to question its reliability, it nevertheless raises some concern. In light of all of these considerations, the decision was made not to use the ZAN:BPD measure



as the emergent-PD indicator and to instead construct a composite PD-indicator (i.e. the emergent PD-profile) in the present study.

## **2.0 METHOD**

The present study involves secondary analysis of data from the IMPACT Trial (Improving Mood with Psychoanalytic and Cognitive Therapy), collected between 2010-2013. Part 2.1 below summarises the methodology utilised within the IMPACT Trial (Goodyer et al, 2016) to facilitate the interpretation of the present study (part 2.2) in its context. The author of the current study was involved in data collection and later local trial coordination as part of the IMPACT research team between 2010-2012.

### **2.1 *IMPACT Study***

#### **2.1.i *Study Design and Participants***

IMPACT was a multi-centre, pragmatic, single-blind, randomised controlled superiority trial conducted across three regions of England: East Anglia, North London, and the North West of England. Adolescents (aged between 11–17 years with a diagnosis of DSM IV major depressive disorder) were recruited from 15 routine NHS CAMHS clinics. The adolescents entered into this trial represented a clinical population and had high numbers of symptoms as well as concurrent personal impairments.

Exclusion criteria included generalised learning difficulties, pervasive developmental disorder, pregnancy, current use of another medication that could interact with an SSRI, current substance or alcohol abuse disorders, previous completion of one of the study treatments, and a primary diagnosis of bipolar disorder, schizophrenia, or eating disorders. The study was approved by the Cambridgeshire 2 Research Ethics Committee (reference

09/H0308/137) and local NHS provider trusts. All patients and their parents gave written informed consent (Goodyer et al, 2016).

### *2.1.ii Procedures*

#### ***Recruitment of Participants***

CAMHS clinicians across the services involved in the trial completed initial IMPACT screening forms for young people aged 11-17 at the point of assessment. Upon consent, adolescents meeting basic inclusion criteria and not meeting exclusion criteria were forwarded to the research team and subsequently contacted by research assistants who provided further information about the trial. Baseline assessments were conducted for all consenting subjects (either within CAMHS settings or as home visits), typically involving both a young person and parent/carer assessment completed by two research assistants. Baseline assessment was a requisite to corroborate diagnostic threshold for major depressive disorder, severity, as well as comorbidity and risk assessment. Once eligibility for the trial was confirmed and with informed consent from both the young person and parent/carer (where required), subjects were randomised to one of the three interventions by the Trial Manager (Goodyer et al, 2016).

#### ***Randomisation and Masking***

Patients were randomly assigned via a web-based randomisation service to receive either CBT or short-term psychoanalytical therapy versus the brief psychological intervention. Randomisation was done by the trial coordinator, with stochastic minimisation by age (11–13 years vs 14–15 years vs 16–17 years), sex, self-reported depression sum score ( $\leq 29$  vs 30–39 vs 40–49 vs  $\geq 50$ ), and region (East Anglia vs North London vs North West England). In view

of the nature of the interventions, patients and clinicians were aware of group allocation, but allocation was concealed from outcome assessors.

470 young people were randomized in total, to receive the brief psychosocial intervention (n=158) versus CBT (n=155), or short-term psychoanalytical therapy (n=157). Five patients withdrew before starting treatment and their data was deleted upon request; the remaining 465 participants comprised the intention-to-treat population, with 392 (84%) patients who provided one or more self-reported depression symptom scores at weeks 36, 52, and 86 available for primary analysis of depression outcomes (Goodyer et al, 2016).

### ***Data Collection and Measures***

Assessments were conducted at baseline, 6, 12, 36, 52 and 86-week follow-up intervals from start of treatment and the same assessors were used at each interval wherever possible for reasons of consistency and familiarity for subjects and their families. The format of assessments involved a combination of structured interviews and self-report measures, lasting approximately 2 hours. Figure 1.0 below illustrates which measures were administered at which assessment time-point. In addition, the Kiddie Schedule for Affective Disorders and Schizophrenia—Present and Lifetime (K-SADS-PL) was administered at all time points (Goodyer et al, 2016). A full glossary of measures is available in Appendix 2.

**Figure 1.0 Schedule of Assessments in the IMPACT Study**

Data Collection Instrument	baseline (1)	baseline parent (2)	6 weeks (3)	6 weeks parent (4)	12 weeks (5)	12 weeks parent (6)	36 weeks (7)	36 weeks parent (8)	52 weeks (9)	52 weeks parent (10)	86 weeks (11)	86 weeks parent (12)	extra (13)	extra parent (14)
Subject List	✓													
AIM - adolescent integrative measure					✓	✓			✓	✓	✓	✓	✓	✓
APQ - Alabama Parenting	✓	✓			✓	✓	✓	✓	✓	✓			✓	✓
CA-SUS - service use schedule		✓		✓		✓		✓		✓		✓	✓	✓
CGI	✓		✓		✓		✓		✓		✓			
DEEP - domain expression emotion profile			✓											
Demographic Questionnaire		✓												
DES-IV	✓				✓				✓					✓
DEQ - depressive experiences questionnaire	✓								✓		✓			✓
DSC	✓				✓				✓		✓			✓
EQ5D	✓		✓		✓		✓		✓		✓			✓
FAD - family assessment device	✓	✓			✓	✓			✓	✓				✓
FQ - friendship questionnaire	✓	✓			✓	✓			✓	✓				✓
HoNOSCA - health of nation outcome scales	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
LEQ	✓				✓				✓					
LEQ Parent		✓				✓				✓				
NEO - five factor questionnaire	✓	✓							✓	✓	✓			✓
RRS - ruminative responses scale	✓		✓		✓		✓		✓		✓			✓
Rtshia Risktaking Selfharming	✓		✓		✓		✓		✓		✓			✓
SCL90 - symptom checklist		✓		✓		✓		✓		✓		✓	✓	✓
Wais Working Alliance Inventory			✓	✓	✓	✓	✓	✓						✓
WAI-S Therapist			✓	✓	✓	✓	✓	✓						
YPQ - young person quest	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
ZAN - borderline personality disorder	✓								✓		✓			✓

(Source: REDCap Database, IMPACT Research Team)

### **Therapeutic Interventions**

Concurrent with research assessments/data collection, subjects received therapeutic intervention within CAMHS settings. All treatments were manualised; a full description of the treatment manuals can be found in Goodyer and colleagues (2016) including theoretical and practical differences. Short-term psychoanalytical psychotherapy consisted of a schedule of 28 sessions over 30 weeks, with parents or carers offered up to seven additional sessions by a separate parent therapist. The techniques of this intervention are based on close and detailed observation of the relationship the child or young person makes with their therapist. The therapist introduces the therapeutic task to the young person as one of understanding feelings and difficulties in their life. The therapist is non-judgmental and enquiring, and

conveys the value of self-understanding. Therapists were CAMHS clinicians with child and adolescent psychoanalytical psychotherapy training (Goodyer et al, 2016).

CBT was based on the classic form originally developed for adults with depression. The intervention was adapted for the study to include parental involvement, focused on engagement in therapy, and emphasised the use of behavioural techniques. The focus of CBT is to identify the behaviours and information processing biases that maintain depression and low mood, and to amend these through a process of collaborative empiricism between the therapist and patient. CBT in this study involved a programme of up to 20 sessions over 30 weeks. CBT therapists were routine CAMHS clinicians and were either clinical psychologists or other clinicians who had received post-qualification training in CBT (Goodyer et al, 2016).

The brief psychosocial intervention (BPI) was derived from the routine specialist clinical care delivered in the ADAPT trial (Goodyer et al, 2008) and tailored on the basis of findings suggesting this intervention might be clinically effective. Emphasis in the BPI intervention was on the importance of psychoeducation about depression, in addition to action-oriented, goal-focused, and interpersonal activities as therapeutic strategies. Neither self-understanding nor cognition change are components of the programme. The programme consisted of 12 individual sessions, including up to four family or marital sessions delivered over 20 weeks. Therapists were drawn from routine CAMHS clinics.

For all three intervention groups, liaison with external agencies such as teachers or social care services occurred where appropriate. All therapy sessions were audiotaped. A computerised randomisation procedure was used to select tapes stratified by age, treatment, and whether obtained early (two to four sessions) or later (after four sessions) in the therapy. Randomisation was done with the Comparative Psychotherapy Process Scale and the Brief Psychosocial Intervention scale. Independent raters rated each treatment session from the three treatment modalities to assess treatment fidelity and differentiation.

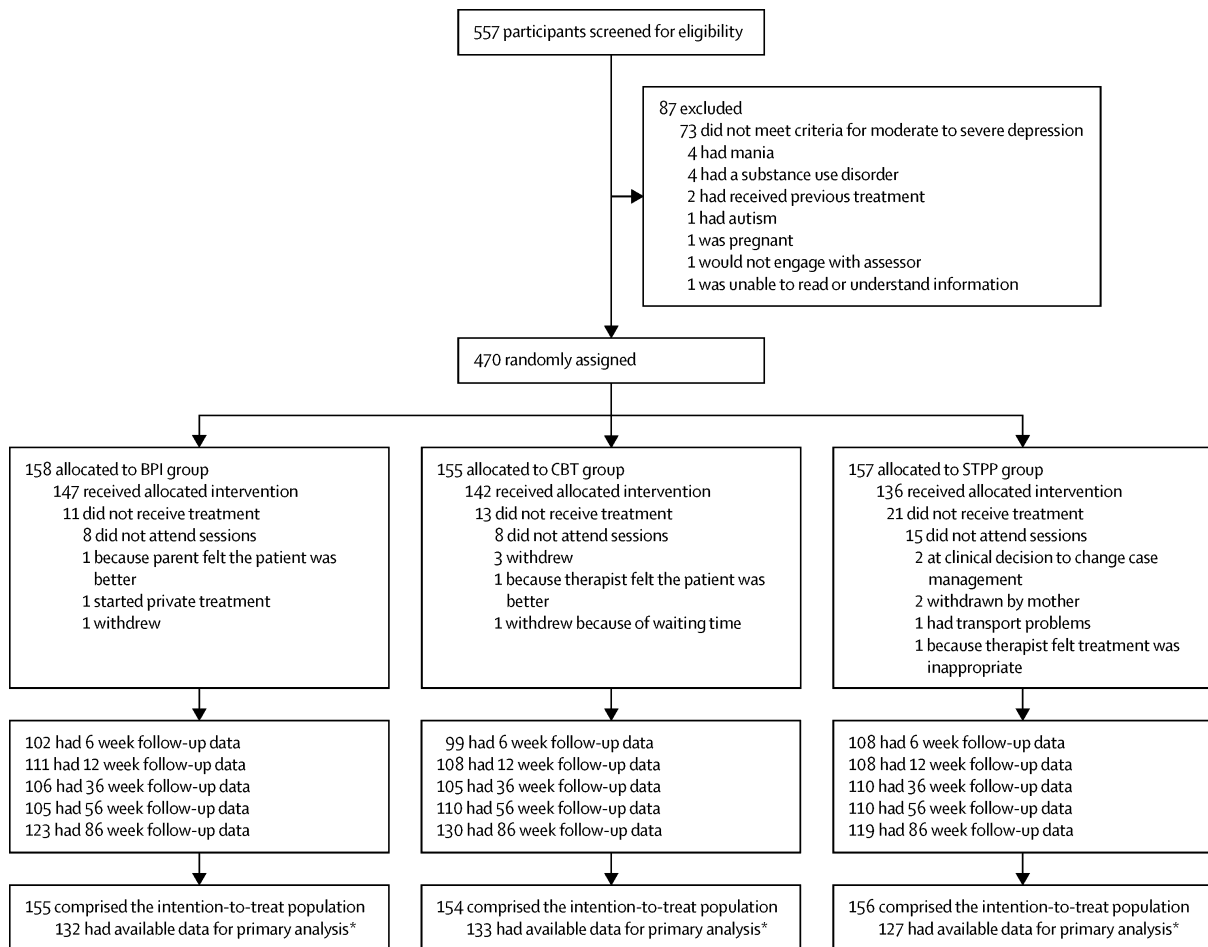
In accordance with NICE guidelines, fluoxetine could be added if clinicians deemed that combination therapy might accelerate the time to remission. A test dose of 10 mg was given for 48 hours, followed by 20 mg as a single dose. If no improvement was shown within 2–4 weeks, the dose could be adjusted upwards to a maximum of 60 mg (Goodyer et al, 2016).

### *2.1.iii Outcomes and Analyses*

The primary outcome in the IMPACT study was self-reported depression symptoms at weeks 36, 52, and 86 following randomisation (i.e. at the end of treatment), as measured with the Mood and Feelings Questionnaire (MFQ). Secondary outcomes were self-reported sum scores on the Revised Children's Manifest Anxiety Scale (RMAS), the revised Leyton Obsessional Inventory (LOI) for adolescents, and the Health of the Nation Outcome Scales for Children and Adolescents—a measure of overall current psychosocial impairment. A brief self-reported antisocial behaviour checklist based on DSM IV criteria for conduct disorder was used as a binary (none, one or more) measure of antisocial behavioural symptoms. Presence of major depressive disorder was also measured over time by use of the Kiddie-Schedule for Affective Disorder and Schizophrenia. The study was not powered to test a specific diagnosis hypothesis. Two additional clinical measures were assessed: the Columbia Suicide Inventory and the self-report Risk and Self Harm Inventory. Economic measures included the Child and Adolescent Service Use Schedule, for collection of service and other resource use data, and the EuroQol 5D questionnaire 3-level measure of health-related quality of life, for calculation of quality-adjusted life-years (QALYs). The primary analysis population comprised 392 (84%) patients who provided one or more self-reported depression symptom scores at weeks 36, 52, and 86 (Figure 2.0). All analyses were

conducted on an intention-to-treat basis. A detailed description of the statistical analysis plan can be found in the original paper (Goodyer et al, 2016).

**Figure 2.0** *IMPACT Study: Profile*



Source: Goodyer et al, 2016

## 2.2 Current Study

### 2.2.i Ethical Considerations

The current study did not require additional ethical approval to that gained for the IMPACT Trial (reference 09/H0308/137). All analyses were secondary in nature and conducted using the existing dataset obtained under the original study protocol.

### *2.2.ii Procedures*

All statistical analyses were conducted using SPSS v24. The overall approach involved firstly identifying items from across a range of IMPACT measures that were theoretically linked to personality disorder; secondly, running an exploratory factor analysis on these items to identify possible markers of PD (i.e. latent variables that might represent core PD components and form a 'profile' of emergent PD in the present sample), and finally conducting statistical analyses including regression, comparison of means and tests for association to explore whether young people who scored highly on these markers had lower adherence to, and worse outcomes in, depression treatment.

#### ***Item Identification***

At the outset, all young person self-report measures used within the IMPACT study at baseline were qualitatively reviewed at item-level, to consider their relevance to emergent personality disorder symptomatology and its core components. Items from some measures which were originally considered thematically relevant were subsequently disregarded where the measure had been discontinued early on in the IMPACT trial. This included items on the NEO:FFI, DSC, DES, FAD, Friendships Questionnaire and the LEQ measures. Items from the SCL:90, although thematically relevant, were not considered as this was a measure of parental/carer symptomatology rather than that of the young person. As noted previously, items from the ZAN:BPD were also dropped due to the unreliability of data gathered using the measure in this trial. Furthermore, at initial screening this measure had 38% missing data overall. Finally, items from the MFQ scale were also not used (although sixteen were potentially relevant to PD), since total MFQ scores would ultimately be used as a primary outcome indicator of response to treatment/reduction of depressive symptoms and their use in constructing a PD profile could result in inflated correlations.



46 relevant items were identified for further review from across these measures within the IMPACT dataset. These items were chosen in light of the theoretical understanding of core components of personality disorder, as presented in Table 1.

### ***Item Screening***

Descriptive statistics (mode/median, frequencies and percentages and bar charts) were used to conduct preliminary screening on these items in terms of variability and skewness (Table 2). Items which were significantly skewed and/or showed little variability (i.e. items where >80% respondents selected the same value) were removed as these were considered to have little discriminant power across the sample. A total of six items were removed.

40 items were ultimately selected for statistical analysis (and their origins in terms of scale/measure) as summarized in Table 3 which includes scales of measurement for each item. The scale was Likert-type in all cases, although there was some variation in the number of points and groupings that were used. To ensure that low scores represented low symptomatology and high scores corresponded to high symptomatology across all selected items, some variables were reverse coded where necessary (RSES 1, 6 and 9). Standardised scores were then calculated for all items and saved as new variables in the dataset.

### ***Missing Data Analysis***

With a total sample of 465 subjects with IMPACT baseline data available at the outset, valid and missing data was assessed for all subjects across each of the selected 40 items (using the compute function and NMISS operator to calculate the total number of missing values per subject). Subjects who had less than 50% available data across these items (i.e. missing data on 21 or more items) were excluded from further analysis. This threshold for acceptable missing data at unit-level follows that utilized in other analyses using the IMPACT dataset

and was agreed with the broader research team. The remaining sample in the current study consisted of 449 subjects.

Of the 40 items, three (DEQ 5, 12, 17) had 14.5% missing responses, while two items (RRS 27, 34) had 9.8% missing responses; this was also the case for all other items (not selected for further analysis) across the DEQ and RRS measures respectively when missing values were assessed across entire scales rather than at individual item level. This suggests that these measures may have missing data because they were not administered to all subjects at baseline, rather than due to responses to specific items being intentionally or accidentally omitted. Three other items (all on the RSES scale) had around 1% missing responses, all other items (n=32) having no missing data. The retention of the selected 40 items was considered acceptable in light of the above.

Patterns of missing data at item-level were further explored using the Missing Value Analysis module in SPSS. Little's MCAR test revealed that the data was not missing completely at random (Chi-Square = 1066.642, DF = 257, Sig. < .001) (Little & Shenker, 1995). Prediction and imputation of estimated values was therefore conducted, using the linear regression method. Items for which missing value estimates were required included DEQ 5, 12 and 17; RRS 27 and 34; and RSES 1, 6 and 9. This was considered superior to listwise and pairwise deletion of missing values in subsequent factor analysis, as these methods are widely known to result in biased and/or inefficient estimates and in significant loss of data (Dong & Peng, 2013). For all subjects, standardised scores were generated for all items in the complete dataset.

### *2.2.iii Design and Statistical Analyses*

The initial plan was to firstly conduct an exploratory factor analysis (EFA) on the basis of the 40 items on half of the sample (n=225) followed by Confirmatory Factor Analysis (CFA) on

the remaining half of the sample (n=224) to verify the factor structure. The division of the dataset into two random subsamples for this purpose was completed using the compute function in SPSS and `rv.uniform(0,1)` operator to create a random number variable and subsequently using the median split approach to create two comparable groups.

Subsequent analyses indicated that, based on a sample of 225, it was not possible to converge onto a solution in exploratory FA. The correlational matrix was inspected in detail at the outset; once items with excessively high correlations (risking multicollinearity) and items with mainly low correlations ( $r < .3$ ) were removed in line with common recommendations (Field, 2013), and following analysis of sampling adequacy to obtain individual item Kaiser-Meyer-Olkin (KMO) values before proceeding with FA (KMO  $> .5$  required as a minimum), the removal of items that did not meet these criteria rendered factor analysis on the sample no longer viable. Therefore, ultimately the statistical analysis strategy changed towards completing an exploratory factor analysis on the entire sample (n=449).

The chosen method of extraction was principal axis factoring (PAF). Decisions regarding how many factors to extract and which factors to retain were initially guided by parallel analysis (eigenvalue Monte Carlo simulation) and by observing the scree plot. Parallel Analysis is generally considered to be the most accurate factor-retention method, and superior to merely using the Kaiser-Guttman criterion which postulates retaining any factors whose eigenvalue is greater than 1. (Matsunaga, 2010). The analysis was conducted using SPSS syntax available at <https://people.ok.ubc.ca/briocconn/nfactors/nfactors.html>.

Orthogonal (varimax) rotation was used in order to facilitate easier interpretation of factors, and to avoid multicollinearity which could be problematic in subsequent regression analyses. Factor loadings below .4 were not presented as, given the moderate sample size, loadings below this would not typically be considered significant (Field, 2013).

Factors that were extracted were assumed to represent latent variables that tap into aspects of PD (i.e. the PD profile). Analyses were subsequently conducted to explore whether higher scores on these markers predicted poorer outcomes. Primary outcomes were severity of depression symptoms at follow-up (measured by MFQ scores at 86-week follow up/t5), treatment drop-out and recovery status. The latter was measured on the basis of MFQ scores at follow-up, whereby scores above 27 were indicative of ongoing, clinically significant depression post-treatment (non-recovery) and scores below the threshold represented recovery (Wood et al, 1995). Analyses involved multiple regression, independent t-tests and Chi Square tests for independence. Follow-up data on the primary outcome (MFQ scores at 86-week follow-up, indicative of depression symptoms post-treatment) was available for 343 subjects.

#### *2.2.iv Power Analysis*

Power calculations were conducted using G\*Power. A priori analysis for multiple regression (fixed effects, mixed effects and interactions) with covariates of sex, age, SSRI prescription, baseline MFQ, and the independent variables PD Profile Score, CBT and STPP allocation plus two interaction terms (PD\*CBT/PD\*STPP), was used to determine the required sample. At alpha level .025 (adjusting for multiple testing with Bonferroni correction) and power at .90, a minimum required sample of 386 was required to detect a medium effect size ( $f=.25$ ). Post-hoc analysis indicated that, with the actual available sample of 342 and alpha level .025, the study was powered at .85 to detect a medium effect size.

Power analysis for a chi-square test was conducted using the same parameters of alpha at 0.025 level, power of 0.90, and 1 degrees of freedom. A minimum sample size required to detect a medium effect size ( $w=0.25$ ) was 199. Post-hoc analysis based on the

available sample (342) indicated that the study was powered at .99 to detect a .25 effect size and at .80 to detect a small effect size ( $w=.17$ ).

### **3.0 RESULTS**

#### **3.1 *Descriptive Statistics***

Frequencies and descriptive statistics including central tendency and skewness observations for items at initial screening ( $n=46$ ) are presented in Table 2. Six items had very limited variability in responses, whereby  $>80\%$  responses fell within the same value/category. These items were excluded, leaving 40 items for subsequent Factor Analysis.

Subject demographic data as well as treatment allocation is presented in table 4. There were 335 females in the sample and 114 males (75% and 25% respectively), which is roughly correspondent to prevalence rates of major depression among adolescent girls versus boys in clinical population studies (Thapar et al, 2012). The vast majority of subjects considered themselves White British (78%). 20% of young people had had anti-depressant medication (SSRI) prescribed prior to trial entry. Randomisation groups were highly comparable, with 33.6% of subjects in the BPI and STPP arms each, and 32.7% in the CBT intervention.

#### **3.2 *Exploratory Factor Analysis (EFA): Identifying and Developing an Emergent PD Profile***

Preliminary analysis involved inspecting the correlation matrix between all 40 items. Very high correlations ( $>.9$ ) were observed among some of these and six items were subsequently removed to reduce risk of multicollinearity<sup>3</sup>. The anti-image matrix was also assessed for

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<sup>3</sup> Removed items included item 4 (RTSHIA), 61 (RCMAS), 73 and 76 (Behaviours Checklist) and items 1 and 9 (RSES).

measures of sampling adequacy. Individual item Kaiser-Meyer-Olkin (KMO) values were below the typical acceptable limit of .5 on 11 items<sup>4</sup> and these were also removed.

A principal factor analysis with orthogonal rotation (varimax) was then carried out on the remaining 23 items. The observed communalities for all items were good, with the exception of one item (RTSHIA 1, communality = 0.02) and this item was excluded before re-running the analysis again on 22 items. The KMO test measure verified sampling adequacy for the analysis (KMO = .64). Residuals were observed to assess the goodness of fit of the model. There were 60 (25.0%) non-redundant residuals – differences between observed correlations and correlations reproduced based on the model - with absolute values greater than 0.05. This suggested a relatively good fit.

Parallel Analysis was conducted to compare the eigenvalues for factors in the raw data with corresponding random data eigenvalues; this can help to determine which ones occur beyond chance in the current sample. Five factors emerged with eigenvalues above Kaiser's criterion of 1 and twelve factors in total had eigenvalues occurring beyond chance (i.e. greater than eigenvalues for random data). However, it is common for parallel analysis to yield statistically viable factors where several may be fairly negligible particularly in larger samples (Buja & Eyuboglu, 1992). The scree plot was therefore also consulted. The plot was somewhat ambiguous, suggesting the retention of two, four or five factors.

Five factors were initially retained (output for the scree plot can be seen in Appendix 3). In combination, these accounted for 67.3% of the variance. However, the rotated factor matrix revealed that eight items had substantive cross-loadings onto at least two factors. These items (RTSHIA 11, 12, 13, 16, 17 and 25; DEQ 12 and DEQ 17) were removed and the analysis re-run again. Following this procedure, sampling adequacy remained acceptable (KMO = .60) and residuals were comparable to the previous analysis (26%). The revised

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<sup>4</sup> Removed items included RRS 27, RRS 34, RTSHIA items 2, 3, 5, 7, 8, 9, 26, 27 and 28

scree plot suggested two or four factors to retain. Total variance output indicated that four factors in combination accounted for 62% of the variance and these were looked at further in the rotated factor matrix. Table 5 shows the four-factor substantive loadings after rotation (i.e. absolute value above .4). Eigenvalues and variance explained by each factor are also presented.

Statistically, two dominant factors are evident here with five item loadings above .4 each and each independently accounting for approximately 20% of the total variance. The clustering pattern on these factors strongly indicates factor 1 to represent underlying *negative affectivity*, with items tapping into low self-esteem, perceived loneliness and depressivity and interpersonal antagonism. The second factor reflects the construct of *self-harm*, with items portraying a range of predominantly direct and visible self-mutilating behaviours. Factor 3 has only one substantive item-loading (pertinent to what might be labelled as ‘perceived *insecurity around relationship changes*’), and Factor 4 had two item-loadings, together indicative of *suicidality*. Since a minimum of three high loadings is typically recommended for a factor to solidly represent a latent construct (Field, 2013), it appeared that factors 3 and 4 – although conceptually relevant to PD - did not meet these statistical criteria therefore the analysis was re-run again to extract two factors only. Rotated factor loadings are shown in table 5a. Reliability analyses were run separately for each factor. Negative Affectivity (Factor 1) and Self-Harm (Factor 2) both had good internal consistency,  $\alpha = .80$  and  $\alpha = .78$  respectively. The two-factor solution and subsequent PD profile were deemed preferable to the four-factor solution given adequate item loadings onto each factor and good internal consistency across both factors.

The two-factor PD profile score (PD profile score – 2) was computed by summing the standardised factor scores for Factors 1 and 2 for all subjects. Subjects with higher scores were considered to present with more severe emergent PD features.

A simple correlation was conducted between PD profile scores and the ZAN:BPD, in an effort to consider external validity of the newly formed profile. Standardised sum scores on the ZAN:BPD at baseline were computed for all subjects ( $M=13.42$ ) for the analysis. This was done by summing items 1-9 in the measure to enable a continuous score, rather than having to use the categorical diagnostic item 10. The correlation was extremely small ( $r=.08$ ) which may indicate suboptimal external validity for the profile. However, this was necessarily considered in the context of questionable reliability of the ZAN:BPD measure itself as used in the IMPACT study, as discussed.

### ***3.3 Does the Emergent PD Profile Predict Depression Treatment Outcomes in Terms of Depression Severity?***

Multiple regression analysis with forced entry method was used to examine whether PD profile scores could predict severity of depression at follow-up (i.e. post-treatment MFQ scores). In this procedure, the dependent variable was MFQ score at follow-up (t5), indicating depression severity once treatment had finished. The independent variables were the PD profile score (predictor) from the EFA, baseline MFQ scores (i.e. depression severity at the outset) and SSRI prescription prior to trial entry, age and sex. Sum MFQ scores at baseline and follow up points were obtained for all subjects in the study<sup>5</sup>. Descriptive statistics for MFQ scores are presented in Table 6. 342 subjects had available MFQ scores at the final follow-up (Min= 0, Max=63 [out of 66],  $M=22$ ,  $SD=16$ ) and constituted the sample for the regression analysis.

Basic descriptive statistics and regression coefficients are shown in Table 8. The overall model was significant and in conjunction the predictors explained 10% of the

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<sup>5</sup> Summed data was obtained from the IMPACT research team. Scores were summed based on the 3-level scale which is precedent in existing literature (i.e.: all participants who responded as a '3' were recoded to a 2, so scale for each item is from 0-2). Also, the sum scores were generated based on available MFQ data at each wave as long as at least 50% of the items were completed



variance in follow-up MFQ scores (depression severity) ( $R^2 = .099$ ,  $F(5, 332) = 7.34$ ,  $p < .001$ ). PD profile score did not significantly predict follow-up MFQ scores ( $\beta = .05$ ,  $p = .307$ ). The only predictors that explained a significant proportion of unique variance in follow-up MFQ scores were the baseline MFQ score ( $\beta = .23$ ,  $p < .001$ ) and gender ( $\beta = .16$ ,  $p < .01$ ). A positive correlation between baseline MFQ and follow-up MFQ scores ( $r = .25$ ,  $p < .001$ ) suggests that greater depression severity at the outset is associated with worse depression outcomes at follow-up post treatment. Females had significantly higher MFQ scores at follow-up than males ( $M = 24$ ,  $SD = 15.50$  and  $M = 17$ ,  $SD = 15.23$  respectively), which may be indicative of greater severity of depression in adolescent girls in this sample even after treatment finished ( $t = -3.64(340)$ ,  $p < .001$ ).

Existing literature suggests that the prevalence of PDs among clinical adolescent populations is significant. Around 30% has been cited by some (Zimmerman et al, 2005), Chanen and colleagues reported 11% in adolescent outpatients and as high as 49% in inpatients (Chanen et al, 2007). Tyrer and colleagues similarly cite research pointing towards about a quarter of patients in primary care and 50% in psychiatric outpatient settings meeting criteria for BPD (Tyrer et al, 2015). In light of these figures, one aspect of interest in the present study was to examine more specifically whether subjects scoring in the top quartile on the PD profile score (thus, those who could be considered to have the most 'PD-like' symptoms) may differ in depression treatment outcomes compared to those scoring lower on the PD profile. Cases were ranked and grouped by percentile and an independent t-test was then done to compare the top 25% PD profile scorers with the remaining subjects. The lower-PD profile group showed slightly higher mean MFQ scores at follow-up ( $M = 23$ ,  $SD = 15.46$ ) than the higher-PD profile group ( $M = 21$ ,  $SD = 16.41$ ), but this was not statistically significant,  $t(340) = -1.04$ ,  $p = .300$ .

### ***3.4 Are High Scores on the Emergent PD Profile Associated with Depression Treatment Drop-Out?***

Using percentile groupings on the PD profile, a Chi Square analysis was conducted to investigate whether subjects scoring in the top quartile on the PD profile score (i.e. ‘high’ scorers) were more likely to withdraw from treatment than others. Assumptions of independence and expected frequencies met.

Within the ‘low’ PD-profile group, 13.7% subjects withdrew from treatment (86.3% remained in treatment for its duration). Within the ‘high’ PD-profile subjects, drop out was slightly higher at 17.7% (82.3% remained in treatment for its duration). However, the proportion of subjects who remained in treatment and those who withdrew was not significantly different between the high or the low-PD profile groups, suggesting that the relation between high PD profile scores and treatment drop-out was not significant,  $\chi^2 (1) = 1.08$ ,  $p=.186$ , one-sided).

### ***3.5 Are High Scores on the Emergent PD Profile Associated with Lower Rates of Recovery from Depression Post-Treatment?***

A Chi Square analysis was conducted using the PD profile to test for association with likelihood of recovery from depression at the 86-week follow up. There was no significant association between high scores on the PD profile (i.e. those scoring in the top quartile) and recovery from depression:  $\chi^2 (1) = 0.77$ ,  $p=.228$ , one-sided.

68.2% of subjects in the top quartile group (high PD) and 63% of the rest (low PD) had recovered by the 86-week follow-up, in so far as their MFQ scores were below the threshold of 27.

### ***3.6 Does the Emergent PD Profile Impact Depression Outcomes Differently Depending on Severity of Depression at the Outset?***

Baseline MFQ scores were used to categorise all subjects into low, moderate and high baseline MFQ groups relative to the whole sample. Groups were created by ranking all cases according to MFQ baseline score, then re-coding into three discrete categories based on these rankings (i.e. top third of rankings constituted the ‘high’ MFQ group). The MFQ ranges for the groups were: 52-65 (high), 43-51 (moderate) and 13-42 (low). It should be noted that these thresholds are not representative of what would count as clinically low versus high scores in the broader adolescent population (noting that 27 is typically considered a threshold indicative of clinical depression); they are reflective of the upper and lower ends of the score distributions in this particular sample.

Multiple regression analyses were conducted separately for these groups to investigate whether emergent PD profile scores may have predicted outcomes differently for subjects who presented with particularly severe depression (i.e. the highest MFQ scores) at baseline. Summary statistics for these analyses are presented in table 9. For these analyses, the independent variables were PD profile score, age, sex and SSRI prescription (baseline MFQ was not included as a covariate, given that this variable was already used to select cases for the analysis); the outcome variable was MFQ score at 86-week follow up. 116 subjects (of the 342 with available follow-up MFQ data) comprised the lowest MFQ group, while 113 subjects were in the moderate and high baseline MFQ groups respectively.

For the ‘high’ MFQ group, the model was marginally significant at  $p < .05$  level ( $R^2 = .10$ ,  $F(4, 105) = 2.77$ ,  $p = .031$ ) but PD profile scores did not predict MFQ at follow-up, ( $\beta = -.15$ ,  $p = .115$ ). The only variable that significantly predicted follow-up MFQ score was SSRI prescription prior to trial entry ( $\beta = -.20$ ,  $p = .032$ ). Subjects who had been prescribed an SSRI

on average had lower MFQ scores at follow-up ( $M=22$ ,  $SD=17$ ) than subjects who had not had an SSRI prescribed ( $M=31$ ,  $SD=18$ ) ( $t(108) = 2.22$ ,  $p=.030$ ).

For the ‘moderate’ baseline MFQ group, the model was not significant overall when the PD profile score were used as predictor ( $R^2 = .04$ ,  $F(4, 108) = 1.11$ ,  $p = .355$ ), with none of the independent variables significantly predicting MFQ scores at follow-up.

For the ‘low’ baseline MFQ group, again the model was not significant overall when the PD profile was used as one of the predictors, but age ( $\beta=-.19$ ,  $p=.046$ ) and gender ( $\beta=.22$ ,  $p=.021$ ) remained the only statistically significant predictors. Results indicated that older subjects had lower MFQ scores at follow-up than younger subjects ( $r=-.16$ ,  $p=.04$ ), although the effect size was small. Females had higher average MFQ scores at follow-up ( $M=20$ ,  $SD=13$ ) than males ( $M=15$ ,  $SD=11$ ) by an average of 5 points.

### ***3.7 Does the Emergent PD Profile Impact Depression Outcomes Differently Depending on Treatment Modality?***

Although the PD profile did not significantly predict depression treatment outcomes overall, the final part of the analysis explored whether there may have been potentially differential and/or interaction effects across the treatment modalities of CBT and STPP.

Hierarchical regression using enter method was conducted to test for this, including predictor variables of baseline MFQ score, age, sex and SSRI prescription (Model 1), CBT/STPP treatment group (Model 2) and interaction between PD Profile and CBT/STPP (Model 3). A summary of results from the analysis is shown in table 10. Each model significantly predicted follow-up MFQ scores:  $R^2 = .10$ ,  $F(4, 333) = 8.91$ ,  $p < .001$  (Model 1);  $R^2 = .10$ ,  $F(6, 331) = 6.02$ ,  $p < .001$  (Model 2);  $R^2 = .10$ ,  $F(8, 329) = 4.57$ ,  $p < .001$ , however the effect was slight with minimal differences between each. While baseline MFQ

score and gender predicted follow-up MFQ (as previously reported), treatment modality did not and there appeared to be no significant interaction between PD profile and treatment type.

## **4.0 DISCUSSION**

### **4.1 *Summary of Hypotheses and Key Findings***

The present study set out to investigate whether emergent personality pathology among adolescents in the IMPACT study might impact upon their depression treatment outcomes. The study had two primary goals: firstly, to develop an emergent-PD ‘profile’ that could be used to measure the extent to which young people scored significantly on pertinent, core dimensions of personality disorder. Secondly, it was hypothesised that adolescents who score highly on the emergent-PD profile will have worse depression treatment outcomes than those with lower scores. Specifically, it was expected that higher scores on the PD profile (thus more significant probable personality pathology) would be associated with greater depression severity post-treatment, even when controlling for initial severity of depression symptoms; with higher likelihood of withdrawal from treatment; and with lower rates of recovery from depression, based on clinical thresholds used in the Moods and Feelings Questionnaire.

The first part of the study, involving the creation of the emergent PD profile, revealed two main constructs: self-harm and negative affectivity (conceptually, suicidality and insecurity relative to relationship changes were also initially indicated but these factors were not retained, as previously discussed). This is broadly consistent with prevalent theoretical and empirical understanding of core dimensions of personality disorder (APA, 2013). The emergent PD profile was then used to test for the key hypotheses.

The results did not, however, support these hypotheses. Higher levels of emergent PD did not appear to predict or moderate either worse depression outcomes, rates of recovery or treatment drop-out among the IMPACT adolescent population when baseline depression

severity was controlled for, and there was no interaction observed with different treatment modality.

Overall, the finding that the majority of young people in the study appeared to have recovered from major depression by the final follow-up time (and that, given the follow-up was at 86 weeks from start of treatment, the recovery effect was maintained) was encouraging. Moreover, this was the case for all adolescents including those scoring highly on the PD profile. However, there still remained around a third of young people in both high and lower-PD groups whose symptoms persisted at clinically significant levels (i.e. above the typical threshold within the MFQ measure). It was beyond the scope of the present study to explore in detail what may account for this, but nevertheless two important considerations arise out of these findings: firstly, what was it about the specialist treatments used in the trial (CBT and STPP) that worked towards improving depression outcomes *regardless* of the personality pathology that young people presented with? The assumption on which the initial hypothesis in this study was predicated – that PD would have an impact on treatment response – was sensible and logical in the context of previous research and theoretical understanding of PD, but it appears not to be true and it is important to explore this. Secondly, why did so many adolescents not recover from depression in the longer-term even after specialist treatment? Initial level of depression will almost certainly account for some of the latter (and, indeed, baseline MFQ score was the only consistent predictor of MFQ scores post-treatment in the present study), but it is likely that other factors are at play given that the actual amount of variance explained by baseline severity (whether in combination with the other predictors or independently) was not conclusive. These questions are addressed accordingly, preceded by reflections on the strengths and limitations of the present study that may have influenced the findings.

#### **4.2 *Methodological Strengths and Limitations***

A notable strength of the present study is that emergent personality pathology was conceptualised and analysed using a dimensional rather than a categorical approach. This is consistent with most recent research which suggests that dimensional models of personality disorder are clinically, empirically and theoretically superior to categorical models. One argument often cited in support of the dimensional approach is the extensive comorbidity between what have traditionally been considered categorically distinct personality disorders, a notion which has propelled a shift in how we conceptualise psychopathology generally, and PD specifically (Carragher et al, 2015). This change has also been acknowledged in the DSM-5 (APA, 2013) whereby a dimensional model of PD is presented for the first time. The significance of this is a recognition that PD traits form a spectrum rather than discrete categories, on which all individuals can find themselves to lesser or greater extent. Those at the extremes of the continuum are most probably to present with personality pathology; in any case, the dimensional perspective allows for the assessment of severity and extent of symptoms rather than merely their presence or absence. Mulder argues that the distribution of personality disorder symptoms among depressed patient groups in the majority of well-designed trials is continuous, and gives no support to the idea that there are distinct and separate personality disorder categories. As such, it is arguably preferable to use dimensional scores of PD features rather than categorical PD markers as predictors of outcome in multivariate analysis, (Mulder, 2002).

Another strength of the present study was that baseline severity of depression was controlled for when assessing the impact of PD profile scores on depression outcomes. This increased the likelihood of identifying variability in outcome being uniquely attributable to PD features, rather than aspects related to the severity or chronicity of depression. The large sample size was also an advantage in that the study was sufficiently powered to detect an

effect that could be clinically and empirically meaningful. However, it could also be argued that the difficulty of measuring treatment response in adolescents who have comorbid depression and elevated PD features remains even if we control for initial severity per se. This could be due to the fluctuating nature of personality status over time compounded by concurrent mood changes (Bateman & Tyrer, 2014) and because there is likely to be at least some overlap between the PD dimension studied (such as negative affectivity, or suicidality in the present study) and depressive symptoms.

There are several limitations to the present study that require attention. Perhaps one of its biggest challenges is that, due to the overall sample size, it was not plausible to conduct a confirmatory factor analysis alongside exploratory factor analysis, to validate the PD Profile structure even though this had been the original statistical plan. The division of the sample into two random groupings left insufficient numbers in each to meet basic assumptions for factor analytical methods. The validation of the PD profile is urgently needed and could be conducted in future research. In light of this, the present study does not claim for the PD profile or the presented findings to apply beyond the studied sample. An attempt was made to consider external validity, by correlating the PD profile with an existing validated measure of BPD (ZAN:BPD), as used in the IMPACT study. However, this analysis indicated that the two measures were not associated, which highlights problems with the validity of the PD profile. However, it should also be noted that the ZAN:BPD itself was questionably reliable in terms of its utility in the IMPACT study (though its validity as a scale per se is well known). This may undermine its usefulness as an external validity benchmark in the present study.

In the process of exploratory factor analysis, items were not excluded even where some had 15% missing data (this is somewhat above what some analysts recommend, citing 5-10% missing data as acceptable). The decision was made to retain these items in the



interest of preserving sufficient scope and relevance of information towards constructing the PD profile. Moreover, although broad recommendations exist, to the author's knowledge there is not a formally agreed cut-off for missing data at item-level (Dong & Peng, 2013) and the 15% threshold in the current study seemed reasonable.

The chosen factor rotation method was orthogonal, although it could be argued that oblique rotation would have been less artificial in assuming that factors are allowed to relate to each other rather than being independent (as is likely the case with most psychological and social constructs). For the same reason, factor scores would ideally be computed using the regression method, allowing for the assumption that these may be correlated to some degree. However, orthogonal rotation and independent factor scores, as used in the present study, were chosen to enable easier interpretation of latent variables and the use of these in subsequent regression analyses.

While missing data analysis was conducted, further in-depth analyses were not pursued to demonstrate the plausibility of data being missing at random (MAR) even though this was presumed due to the design of the study. Fortunately, research has shown that violation of the MAR assumption does not seem to seriously distort parameter estimates (Collins et al, 2001).

Where multiple regression analyses were done to assess if PD profile scores predicted depression treatment outcomes, a potential limitation of the study is that it did not look at subjects' *clinically significant change* in scores at the end of treatment (note that in the original IMPACT study, improvement by five points on the MFQ scale was considered clinically significant). This could have enabled more intricate analysis of how subjects' depression severity changed over the course of treatment and whether personality pathology may have interacted with this. Another limitation, of course, is that the analyses did not ultimately identify good predictors of treatment response, and it is possible that other

predictors (not included in the models in the present study) may have accounted for differences in outcomes either independently or in interaction. Variables such as the actual number of treatment sessions completed, the quality of the therapeutic alliance, socioeconomic factors and specific comorbidities were not assessed but this could be a valuable goal for further research.

Outcomes in the present study were taken at the final follow-up point in the IMPACT study (86 weeks following start of treatment). This time point was specifically included in the original IMPACT protocol as it enabled analysis of longer-term treatment effects and relapse rates (and lack of), which had not been previously done in trials of such a scale. In the present study, outcomes at this point were considered particularly meaningful in that they were more likely to represent stable, sustained effects following treatment. It is possible, however, that had depression treatment outcomes been considered at the earlier follow-up time points (e.g. 36/52 weeks), results may have been slightly different in capturing subjects' more immediate treatment response (i.e. depression symptomatology/severity closer to the end of therapy).

Finally, the analyses in the present study involved predicting mean outcomes overall across the sample. It is possible that analysis of individual subjects' trajectories throughout treatment and beyond could have more sensitivity at picking up PD moderation effects and differential treatment response, even if overall outcomes do not significantly differ between subjects at different points on the emergent-PD continuum. Future research should consider this approach and would therefore significantly expand upon the current study's findings.

#### ***4.3 Why Might Different Treatments for Depression be Effective Regardless of the Personality Pathology Features Young People Present With?***

The finding that adolescents' response to depression treatment in the IMPACT study was not moderated by their personality pathology features, and that there was no interaction with

specific treatment type (CBT/STPP) is in some ways an optimistic outcome. Clinically, it should be reassuring that comorbid PD features among adolescents in CAMHS settings do not have to be seen as an obstacle towards positive treatment response (Mulder, 2002).

Recent research into the mechanisms of effective therapy among depressed individuals with comorbid personality pathology is limited, although there is some evidence based on adult case studies to suggest that it is the common aspects underlying various psychotherapeutic interventions – rather than specific techniques – that are related to more positive outcomes; this includes a high quality working alliance between the client and therapist, a structure and routine inherent to regular therapeutic intervention, as well as clients' motivation and commitment to change (Pereira, 2014). Research in the past has given some indication that patterns of outcome among depressed adults with comorbid personality disorders are worse in most treatment modalities compared to cognitive therapy, although these differences are not notable (Shea et al, 1990; Hardy et al, 1995). In adult populations, a small body of evidence seems partial towards structured psychotherapeutic interventions (such as CBT) as opposed to less structured ones (Mulder, 2002). General conclusions must be tentative given the limited research available, but it seems likely that the impact of personality pathology on depression treatment outcome will be at least partly dependent on how the treatment is conducted, aside from what the treatment protocol actually involves. Few, if any, randomised controlled trials have been conducted to assess the effectiveness of specific treatment modalities among an adolescent population with comorbid major depression and personality pathology. One possibility for the finding that both CBT and STPP treatment was, overall, effective regardless of subjects' PD features is that both treatments were fairly structured within the parameters of the trial. Both involved routine sessions, each treatment was manualised and conducted by highly experienced clinicians who were trained in the treatment protocols. In parallel to therapy sessions, subjects also attended

regular research assessments regardless of which treatment they had been allocated to, which in itself provided additional structure and one to one space for the adolescent. Whether or not these aspects influenced treatment outcomes cannot be established in the present study, but it is one possible explanation to explore further.

Another possibility is that there were simply not enough young people in the present sample with diagnosable PD symptoms in order to manifest the predicted moderation. Had the proportion of adolescents scoring very highly on the PD profile been greater (and, ideally, also on a parallel diagnostic measure such as the ZAN:BPD), it is possible that a more significant impact on treatment outcome would be observed.

#### ***4.4 Recovery from Depression: Possible Mechanisms to Explain the Persistence of Symptomatology Following Treatment***

At post-treatment follow-up, the majority of adolescents in the present study reported depression symptoms below a clinically significant threshold on the MFQ measure; however, at least a third were still above this threshold, regardless of presentation on the PD profile. It is important to understand why a significant proportion of young people do not recover, and to consider whether such insight can help to adapt treatment more effectively in future.

Although it was not the focus of the present study, subjects in the IMPACT trial presented with a variety of potential comorbid symptoms as well as moderate to severe depression – some of this information was captured in the kSADS interview data (not analysed here). Depression severity at the outset was clearly significant within the study population. As previously noted, it is rare for mental disorders to occur discretely and categorically; a more realistic picture is one of significant and overlapping comorbidity of psychiatric difficulties, changing clinical presentation over time and – in some cases – experiences of chronic struggles and distress, impacted by social and environmental

conditions across the lifespan that impede individuals' resilience and ability to get onto a path of recovery.

Recent research has focused on untangling this complex picture by examining the structure of psychiatric disorders much more broadly, identifying a possible underlying propensity for any psychopathology (Lahey et al, 2012; Caspi et al, 2014). Based on data from the Dunedin Multidisciplinary Health and Development Study in New Zealand (a longitudinal cohort study of health and behaviour), Caspi and colleagues identified one general psychopathology dimension based on the adult population, referring to it as the 'p factor', which seemed to explain psychiatric disorders by reflecting their common fundamental aspects. Increased life impairment, negative developmental history and compromised early-life brain function are thought to be associated with higher 'p factor' scores, and the higher a person scores on p, the worse that person fares on indicators relevant to severity and duration of disorder (Caspi et al, 2014). Comparable research has since been conducted by Patalay and colleagues among children and adolescents (Patalay et al, 2015). Similarly, a general psychopathology dimension was identified which significantly predicted future psychopathology and academic functioning. Most recently, Wright and colleagues conducted a longitudinal study to validate the structural model of PD including both general and specific features, and to consider the stability of PD over time with both of these in mind. Their study indicated the importance of shared variance for understanding both the relationship between PD and psychosocial dysfunction as well as its stability (Wright et al, 2016).

The findings from these studies are highly significant: firstly, they provide an empirically, clinically and intuitively appealing perspective on psychopathology given the aforementioned complexity that individuals present with in clinical settings. Secondly, they encourage more comprehensive consideration of individuals' risk and resilience factors for a

range of psychiatric disorders. Thirdly, the existence of an underlying psychopathology dimension would suggest that one cannot assume a specific relationship between an individual's disorder (e.g. depression) and its treatment – presumably either in terms of its effectiveness or lack of – without considering her general psychopathology profile. Finally, and of particular relevance to the present findings, the 'p factor' might ultimately be better than the PD concept at explaining treatment non-response. In this case, one might expect that adolescents' scores on measures tapping into early adversity or broader personality dimensions (such as the NEO:FFI, which was used in the IMPACT trial initially) could show moderation of treatment outcome. Unfortunately, these measures were only completed for a small part of the overall sample.

If the p factor concept is correct, treatments should relate to 'p' as a matter of precedence (Caspi et al, 2014). It is conceivable that many - if not most - of the young people in the IMPACT study might have elevated propensity to psychopathology more generally, given the baseline severity of depression as well as high observed comorbidity overall. Within this context, therapeutic intervention which is relatively structured and targeted specifically at major depression may not be able to account for the complex array of factors and experiences shaping each individual's psychiatric outcomes. As such, there will be a proportion of young people for whom the available treatments in the trial (and in clinical settings generally) will have lower effectiveness in alleviating distress and other symptomatology particularly where there is likely overlap between dimensions of personality pathology and depression.

## 5.0 CONCLUSION

The present study found no significant effect of emergent personality pathology on depression treatment outcomes among adolescents in the IMPACT study, where personality pathology was measured using an emergent PD profile specifically developed for the purposes of the study. Overall, this is an encouraging finding if it suggests that emergent PD features which young people in CAMHS may present with need not be an obstacle towards positive response to depression treatment. There also appeared to be no differences in treatment outcomes (in terms of drop-out, recovery rates and follow-up depression severity) between the two specialised treatment modalities and some of the reasons for this have been considered in this paper.

Due to methodological limitations, the findings presented in this study cannot be extrapolated beyond its sample. However, further research could build upon the results by conducting confirmatory factor analysis in an effort to validate the profile as an emergent PD marker. Further research could also focus on analysing individual trajectories of depressed adolescents with PD features in terms of treatment response and outcome, versus analysing mean outcomes at population-level, as this may yield results that are most sensitive to the potential moderating impact of personality pathology on young people's experiences of depression.

Important theoretical considerations have been presented that may help interpret why a significant proportion of adolescents in the study (regardless of their scores on the PD profile) reported depression symptoms which remained above clinical threshold once treatment had finished; this includes the possibility of an underlying propensity to psychopathology (the 'p' factor), the complexity of which may not be accounted for by psychological treatments that are targeted towards specific disorders at a given time point. Whether this perspective is correct remains to be seen, but it is fertile ground for further

debate about how treatment can be developed in a way that is appropriately trans-diagnostic, pragmatic yet appropriate in the context of complex clinical presentations, and acceptable to young people experiencing substantial levels of distress in their lives.



**Table 1**

**Core Components of Personality Disorder**

<b>Core Element of PD</b>	<b>Description</b>	<b>Relevant Measure Identified</b>	<b>Item Identified</b>	
<b>Negative Affectivity</b>	High neuroticism	Frequent and intense experiences of high levels of negative emotions (e.g., anxiety, depression, guilt/ shame, worry, anger) and their behavioural (e.g., self-harm) and interpersonal (e.g., dependency) manifestations. Emotions that are easily aroused, intense, and/or out of proportion to events and circumstances. Insecurity and worry/fear around separations and rejection; nervousness. Difficulty recovering from mood of hopelessness or feeling miserable; pessimism about the future; pervasive shame and/or guilt; feelings of inferior self-worth; thoughts of suicide and suicidal behaviour.	Depressive Experiences Questionnaire (DEQ)	DEQ 5 – it bothers me that relationships with people change
	Emotional Instability/ Dysregulation			DEQ 12 – never really feel safe in close relationship with a parent or friend
	Depressivity		DEQ 17 – if someone I cared for became angry at me, scared they would leave me	
	Anxiousness		Revised Children’s Manifest Anxiety Scale (RCMAS)	RCMAS 12 (in YPQ). I felt alone even when there were people with me
	Unstable self-esteem			RCMAS 14 (in YPQ). My feelings got hurt easily RCMAS 28. I often worried about something bad happening to me
	Rosenberg Self-Esteem Scale (RSES)	RSES 1 – At times I felt I was no good at all RSES 6 – I certainly felt useless at times RSES 9 – felt that I was a failure		
<b>Detachment</b>	Withdrawal/avoidance of personal and emotional attachments	Avoidance of socioemotional experience, withdrawal from interpersonal interactions and lack of initiation of social contact (including friendships and intimate/sexual relationships); preference for being alone rather than with others. Restricted affective experience and expression, limited capacity to experience pleasure, enjoyment or joy or to engage with life experiences. Expectations of—and sensitivity to—signs of interpersonal ill-intent or harm; doubts about loyalty and fidelity of others; feelings of being mistreated, used, and/or persecuted by others.	Revised Children’s Manifest Anxiety Scale (RCMAS)	RCMAS 27 (in YPQ) A lot of people were against me
	Suspiciousness			
	Restricted affectivity			
	Anhedonia			
	Difficulty with reciprocity/mutuality			
<b>Antagonism</b>	Low agreeableness	Behaviours Checklist (BC)	BC 1 – deliberately broke the rules or disobeyed others (parents. Teachers)	
	Manipulativeness			BC 4 – deliberately hurt or threatened someone (e.g. bullying)
	Callousness			

	Hostility	empathy, including an unawareness of others' needs and feelings and a readiness to use others for the purpose of self-enhancement. Lack of guilt or remorse about the negative or harmful effects of one's actions on others. Use of seduction, charm, glibness, or ingratiation to achieve one's ends and control others. Dishonesty, fabrication, misrepresentation of self; Attention-seeking (and admiration-seeking) behaviour.		BC 6 – deliberately lied or cheated to get what I wanted
<b>Disinhibition</b>	Low conscientiousness	Orientation toward immediate gratification; impulsive/spur of the moment behaviour driven by current thoughts, feelings, and external stimuli, without consideration for past learning or future consequences. Disregard for obligations or commitments, agreements and promises; carelessness with others' property. Self-harming behaviour under emotional distress. Difficulty concentrating and focusing on tasks or maintaining goal-directed behaviour, easily diverted by extraneous stimuli. Engagement in dangerous, risky, and potentially self-damaging activities, unnecessarily and without regard to consequences or one's own limitations; recklessness.	Ruminative Responses Scale (RRS)	RRS 27 – (when I feel depressed) I do something reckless or dangerous RRS 34 – I take my feelings out on somebody else
	Impulsivity		Risk-Taking and Self-Harming Inventory for Adolescents (RTSHIA)	RTSHIA 1 – taken chances when doing something RTSHIA 2 – deliberately cross road dangerously RTSHIA 3 – put self in risky situation (e.g. cheating in class)
	Risk-taking			RTSHIA 4. Have you been suspended or dropped out of school?
	Self-injurious behaviour			RTSHIA 5. Stayed out late at night, without your parents knowing where you were? RTSHIA 6. Have you participated in gang violence, physical fights or held a weapon? RTSHIA 7. Had so much alcohol that you were really drunk?
	Distractibility			RTSHIA 8. Used drugs (such as marijuana, cocaine, LSD, etc)? RTSHIA 9. Smoked tobacco? RTSHIA 10. Intentionally cut your skin? RTSHIA 11. Intentionally burned yourself with a hot object (such as a cigarette)? RTSHIA 12. Intentionally bitten yourself, to the extent that you broke your skin? RTSHIA 13. Intentionally banged your head against something, hit or punched yourself, to the extent that you caused a bruise to appear? RTSHIA 14. Intentionally prevented wounds from healing or picked at areas of your body to point of drawing blood?

RTSHIA 15. Intentionally scraped, scrubbed or scratched your skin to the point of breaking your skin or drawing blood?

RTSHIA 16. Intentionally rubbed a sharp object (such as sandpaper) or dripped anything toxic (such as acid) onto your skin?

RTSHIA 17. Exercised an injured part of your body intending to hurt yourself?

RTSHIA 18. Deliberately broken a bone in your body either by making yourself fall or in another way?

(19 is a descriptive item pertinent to 18)

RTSHIA 20. Intentionally pulled hair out?

RTSHIA 21. Deliberately inhaled something harmful (excluding cigarette smoke or drugs) or swallowed something inedible?

RTSHIA 22. Starved yourself to hurt or punish yourself?

RTSHIA 23. Used laxatives (a drug that makes you go to the toilet) to hurt or punish yourself?

RTSHIA 24. Forced yourself to eat too much to hurt or punish yourself?

RTSHIA 25. Stayed in a friendship or relationship with somebody who repeatedly hurt your feelings on purpose?

RTSHIA 26. Tried to make yourself suffer by thinking horrible things about yourself?

RTSHIA 27. Taken an overdose? (i.e. Taken an excessive amount of medication without having been prescribed this dosage)

RTSHIA 28. Seriously thought about harming a part of your body?

RTSHIA 29. Seriously thought about killing yourself?

RTSHIA 30. Tried to kill yourself?

RTSHIA 31. Intentionally hurt yourself in any of the above mentioned ways to that it led to hospitalisation or injury severe enough to require medical treatment?

RTSHIA 32. Engaged in any other self-destructive behaviours not asked about in this questionnaire?

<b>Psychoticism</b>	<p>Odd/eccentric beliefs  Lack of lucidity  Depersonalization  Derealisation  Confused/unclear self-image  Inaccurate self-appraisal</p>	<p>Exhibiting a range of odd, eccentric, or unusual behaviours and beliefs incongruent with one's culture, both in content (i.e. actual beliefs) and in terms of process (e.g. unusual ways of perceiving things/people, experience of dissociation). Belief that one has unusual abilities, such as mind reading, thought-action fusion. Unusual experiences of reality, including hallucination. Saying unusual or inappropriate things. Odd or unusual thought processes and experiences, including depersonalization, derealisation, and dissociative experiences; thought-control experiences.</p>	No items identified on usable scales	No items identified on usable scales
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Table 1. cont.

**Table 2**

**Initial Screening of Data: Valid and Missing Values, and Central Tendency across 46 Items Based on Entire Sample (n=465)\***

<b>Item</b>	<b>Valid</b>	<b>Missing</b>	<b>Median</b>	<b>Mode</b>	<b>Range</b>	<b>Minimum</b>	<b>Maximum</b>
5. It bothers me that relationships with people change.	386	79	6.00	7	998	1	999
12. I never really feel sage in a close relationship with a parent or a friend.	386	79	4.00	4	998	1	999
17. If someone I cared about became angry with me, I would feel frightened that he or she might leave me.	386	79	5.00	7	998	1	999
27. I do something reckless or dangerous	408	57	2.00	1	998	1	999
34. I take my feelings out on someone else	408	57	2.00	2	998	1	999
1. Taken chances while doing something (e.g. not wearing your helmet and other safety gear)?	449	16	2.00	2	3	0	3
2. Deliberately crossed the road dangerously?	449	16	2.00	2	999	0	999
3. Put yourself in a risky situation (such as a classroom cheating, travelling without a valid ticket, shoplifting etc.) knowing that you may get caught?	449	16	1.00	0	3	0	3
4. Have you been suspended or dropped out of school?	449	16	.00	0	999	0	999
5. Stayed out late at night, without your parents knowing where you were?	449	16	1.00	0	999	0	999
6. Have you participated in gang violence, physical fights or held a weapon?	449	16	.00	0	3	0	3
7. Had so much alcohol that you were really drunk?	449	16	1.00	0	999	0	999
8. Used drugs (such as marijuana, cocaine, LSD, etc.)?	449	16	.00	0	999	0	999
9. Smoked tobacco?	449	16	1.00	0	3	0	3
10. Intentionally cut your skin?	449	16	2.00	0	999	0	999

<b>11. Intentionally burned yourself with a hot object (such as a cigarette)?</b>	449	16	.00	0	999	0	999
<b>12. Intentionally bitten yourself, to the extent that you broke your skin?</b>	449	16	.00	0	999	0	999
<b>13. Intentionally banged your head against something, hit or punched yourself, to the extent that you caused a bruise to appear?</b>	449	16	1.00	0	999	0	999
<b>14. Intentionally prevented wounds from healing or picked at areas of your body to point of drawing blood?</b>	449	16	1.00	0	999	0	999
<b>15. Intentionally scraped, scrubbed or scratched your skin to the point of breaking your skin or drawing blood?</b>	449	16	1.00	0	999	0	999
<b>16. Intentionally rubbed a sharp object (such as sandpaper) or dripped anything toxic (such as acid) onto your skin?</b>	449	16	.00	0	999	0	999
<b>17. Exercised an injured part of your body intending to hurt yourself?</b>	449	16	.00	0	999	0	999
<b>18. Deliberately broken a bone in your body either by making yourself fall or in another way?</b>	449	16	.00	0	999	0	999
<b>20. Intentionally pulled hair out?</b>	449	16	.00	0	999	0	999
<b>21. Deliberately inhaled something harmful (excluding cigarette smoke or drugs) or swallowed something inedible?</b>	449	16	.00	0	999	0	999
<b>22. Starved yourself to hurt or punish yourself?</b>	449	16	.00	0	999	0	999
<b>23. Used laxatives (a drug that makes you go to the toilet) to hurt or punish yourself?</b>	449	16	.00	0	999	0	999
<b>24. Forced yourself to eat too much to hurt or punish yourself?</b>	449	16	.00	0	999	0	999
<b>25. Stayed in a friendship or relationship with somebody who repeatedly hurt your feelings on purpose?</b>	449	16	.00	0	999	0	999
<b>26. Tried to make yourself suffer by thinking horrible things about yourself?</b>	449	16	1.00	0	999	0	999

<b>27. Taken an overdose? (i.e. Taken an excessive amount of medication without having been prescribed this dosage)</b>	449	16	.00	0	999	0	999
<b>28. Seriously thought about harming a part of your body?</b>	449	16	2.00	0	999	0	999
<b>29. Seriously thought about killing yourself?</b>	449	16	2.00	2	999	0	999
<b>30. Tried to kill yourself?</b>	449	16	.00	0	999	0	999
<b>31. Intentionally hurt yourself in any of the above mentioned ways to that it led to hospitalisation or injury severe enough to require medical treatment?</b>	449	16	.00	0	999	0	999
<b>32. Engaged in any other self-destructive behaviours not asked about in this questionnaire?</b>	449	16	.00	0	999	0	999
<b>45. I felt alone even when there were people with me</b>	465	0	2.00	3	999	0	999
<b>47. My feelings got hurt easily</b>	465	0	2.00	3	999	0	999
<b>60. A lot of people were against me</b>	465	0	2.00	2	999	0	999
<b>61. I often worried about something bad happening to me</b>	465	0	2.00	2	999	0	999
<b>73. I deliberately broke the rules or disobeyed people (e.g. parents, teachers or supervisors)</b>	465	0	1.00	1	999	0	999
<b>76. I deliberately hurt or threatened someone (e.g. bullying or fighting)</b>	465	0	.00	0	999	0	999
<b>78. I deliberately lied or cheated to get what I wanted</b>	465	0	.00	0	999	0	999
<b>RSES_1_R</b> At times I felt I was no good at all	461	4	2.0000	3.00	3.00	.00	3.00
<b>RSES_6_R</b> I certainly felt useless at times	458	7	2.0000	2.00	3.00	.00	3.00
<b>RSES_9_R</b> I felt that I was a failure	460	5	2.0000	3.00	3.00	.00	3.00

\*Items indicated in dark grey were subsequently removed (n=6) due to limited variability, where >80% of responses fell within the same value. All items were from the RTSHIA measure.

**Table 3**

**Selected Items for Analysis (n=40)**

<b>Item No.</b>	<b>Original Measure</b>	<b>Scale</b>	<b>Item Label</b>
1.	DEQ	1 = Strongly Disagree 2-6 in between, not labelled 7 = Strongly Agree 999 = Missing	5. It bothers me that relationships with people change.
2.			12. I never really feel safe in a close relationship with a parent or a friend.
3.			17. If someone I cared about became angry with me, I would feel frightened that he or she might leave me.
4.	RTSHIA	0 = Never 1 = Once 2 = More than once 3 = Many times 999 = Missing	1. Taken chances while doing something (e.g. not wearing your helmet and other safety gear)?
5.			2. Deliberately crossed the road dangerously?
6.			3. Put yourself in a risky situation (such as a classroom cheating, travelling without a valid ticket, shoplifting etc.) knowing that you may get caught?
7.			4. Have you been suspended or dropped out of school?
8.			5. Stayed out late at night, without your parents knowing where you were?
9.			7. Had so much alcohol that you were really drunk?
10.			8. Used drugs (such as marijuana, cocaine, LSD, etc.)?
11.			9. Smoked tobacco?
12.			10. Intentionally cut your skin?
13.			11. Intentionally burned yourself with a hot object (such as a cigarette)?
14.			12. Intentionally bitten yourself, to the extent that you broke your skin?
15.			13. Intentionally banged your head against something, hit or punched yourself, to the extent that you caused a bruise to appear?
16.			14. Intentionally prevented wounds from healing or picked at areas of your body to point of drawing blood?
17.			15. Intentionally scraped, scrubbed or scratched your skin to the point of breaking your skin or drawing blood?



18.			16. Intentionally rubbed a sharp object (such as sandpaper) or dripped anything toxic (such as acid) onto your skin?
19.			17. Exercised an injured part of your body intending to hurt yourself?
20.			20. Intentionally pulled hair out?
21.			21. Deliberately inhaled something harmful (excluding cigarette smoke or drugs) or swallowed something inedible?
22.			22. Starved yourself to hurt or punish yourself?
23.			25. Stayed in a friendship or relationship with somebody who repeatedly hurt your feelings on purpose?
24.			26. Tried to make yourself suffer by thinking horrible things about yourself?
25.			27. Taken an overdose? (i.e. Taken an excessive amount of medication without having been prescribed this dosage)
26.			28. Seriously thought about harming a part of your body?
27.			29. Seriously thought about killing yourself?
28.			30. Tried to kill yourself?
29.	<b>RCMAS</b>	0 = Never 1 = Sometimes 2 = Mostly 3 = Always 999 = Missing	45 (12). I felt alone even when there were people with me
30.			47 (14). My feelings got hurt easily
31.			60 (27). A lot of people were against me
32.			61 (28). I often worried about something bad happening to me
33.	<b>RSES</b>	0 = Never 1 = Sometimes 2 = Mostly 3 = Always 999 = Missing	RSES_1_R (At times I thought I was no good at all)
34.			RSES_6_R (I certainly felt useless at times)
35.			RSES_9_R (I felt that I was a failure)
36.	<b>Behaviour Checklist</b>	0 = Never 1 = Sometimes 2 = Mostly 3 = Always 999 = Missing	73 (1). I deliberately broke the rules or disobeyed people (e.g. parents, teachers or supervisors)
37.			76 (4). I deliberately hurt or threatened someone (e.g. bullying or fighting)
38.			78 (6). I deliberately lied or cheated to get what I wanted
39.	<b>RRS</b>	1 = Almost Never 2 = Sometimes 3 = Often	27. I do something reckless or dangerous

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4 = Almost Always  
999 = Missing

40.

34. I take my feelings out on someone else

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**Table 4****Demographic Data and Treatment Allocation**

	Frequency	Percent
<b><u>Sex</u></b>		
Female	335	74.6
Male	114	25.4
Total	449	100.0
<b><u>Ethnicity</u></b>		
Not Known	8	1.8
White British	350	78.0
Irish	3	.7
Any other white background	16	3.6
White and Black Caribbean	8	1.8
White and Black African	8	1.8
White and Asian	7	1.6
Any other mixed background	8	1.8
Indian	1	.2
Pakistani	2	.4
Bangladeshi	3	.7
Any other Asian background	3	.7
Caribbean	8	1.8
African	3	.7
Any other Black background	4	.9
Any other ethnic group	10	2.2
Not stated	7	1.6
Total	449	100.0
<b><u>SSRI prescribed prior to trial entry</u></b>		
No	352	78.4
Yes	88	19.6
Total	440	98.0
System Missing	9	2.0
Total	449	100.0

**Randomisation/Intervention**

BPI	151	33.6
CBT	147	32.7
STPP	151	33.6
Total	449	100.0

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**Table 5****Rotated Factor Loadings (Four Factors)**

<b>Item</b>	<b>1 (negative affectivity)</b>	<b>2 (self-harm)</b>	<b>3 (relationship insecurity)</b>	<b>4 (suicidality)</b>
BC 78. I deliberately lied or cheated to get what I wanted	<b>.817</b>			
RCMAS 45 (12). I felt alone even when there were people with me	<b>.770</b>			
RSES 6. I certainly felt useless at times 2(reversed)	<b>-.727</b>			
RCMAS 47 (14). My feelings got hurt easily	<b>.544</b>			
RCMAS 60 (27). A lot of people were against me	<b>.495</b>			
RTSHIA 22. Starved yourself to hurt or punish yourself?		<b>.855</b>		
RTSHIA 15. Intentionally scraped, scrubbed or scratched your skin to the point of breaking your skin or drawing blood?		<b>.718</b>		
RTSHIA 14. Intentionally prevented wounds from healing or picked at areas of your body to point of drawing blood?		<b>.704</b>		
RTSHIA 21. Deliberately inhaled something harmful (excluding cigarette smoke or drugs) or swallowed something inedible?		<b>.498</b>		
RTSHIA 20. Intentionally pulled hair out?		<b>.446</b>		
RTSHIA 10. Intentionally cut your skin?				
DEQ 5. It bothers me that relationships with people change2			<b>.974</b>	
RTSHIA 29. Seriously thought about killing yourself?				<b>.654</b>
RTSHIA 30. Tried to kill yourself?				<b>.653</b>
<b>Eigenvalues</b>	2.92	2.74	1.58	1.41
<b>Variance Explained (%)</b>	20.90	19.60	11.30	10.04
<b>Cronbach's <math>\alpha</math> (standardised)</b>	.78	.78	N/A	.67

**Table 5a**

**Rotated Factor Loadings (Two Factors)**

<b>Item</b>	<b>1 (negative affectivity)</b>	<b>2 (self-harm)</b>
BC 78. I deliberately lied or cheated to get what I wanted	.937	
RSES 6. I certainly felt useless at times 2(reversed)	-.752	
RCMAS 45 (12). I felt alone even when there were people with me	.635	
RCMAS 47 (14). My feelings got hurt easily	.611	
RCMAS 60 (27). A lot of people were against me		
DEQ 5. It bothers me that relationships with people change2		
RTSHIA 22. Starved yourself to hurt or punish yourself?		.930
RTSHIA 14. Intentionally prevented wounds from healing or picked at areas of your body to point of drawing blood?		.618
RTSHIA 15. Intentionally scraped, scrubbed or scratched your skin to the point of breaking your skin or drawing blood?		.618
RTSHIA 21. Deliberately inhaled something harmful (excluding cigarette smoke or drugs) or swallowed something inedible?		.568
RTSHIA 20. Intentionally pulled hair out?		.463
RTSHIA 30. Tried to kill yourself?		
RTSHIA 29. Seriously thought about killing yourself?		
RTSHIA 10. Intentionally cut your skin?		
<b>Eigenvalues</b>	2.92	2.74
<b>Variance Explained (%)</b>	20.90	19.60
<b>Cronbach's <math>\alpha</math> (standardised)</b>	.80	.78

**Table 6**

***Sum MFQ Scores at Baseline and Follow-Up***

<b>Sum MFQ Score</b>	<b>N</b>	<b>Range</b>	<b>Min</b>	<b>Max</b>	<b>Mean</b>	<b>Std. Deviation</b>	<b>Variance</b>	<b>Skewness</b>	<b>Skewness Std. Error</b>	<b>Kurtosis</b>	<b>Kurtosis Std. Error</b>
<b>Baseline</b>	449	52.00	13.00	65.00	45.83	10.61	112.61	-.57	.12	-.21	.23
<b>T1</b>	301	62.00	2.00	64.00	35.47	13.02	169.56	-.23	.14	-.50	.28
<b>T2</b>	318	60.00	1.00	61.00	33.04	14.06	197.67	-.31	.14	-.58	.27
<b>T3</b>	309	61.00	.00	61.00	27.15	15.76	248.44	.18	.14	-.94	.28
<b>T5</b>	342	63.00	.00	63.00	22.40	15.71	246.85	.67	.13	-.40	.26

**Table 7**

***Linear Model of Predictors of Depression Treatment Outcome (as indicated by follow-up MFQ scores/depression severity)***

	<b>B</b>	<b>SE B</b>	<b>Confidence Interval (95%)</b>	<b>β</b>	<b>t</b>	<b>p</b>
<b>(Constant)</b>	1.32	9.21	-16.79 - 19.43		.14	.886
<b>PD Profile Score – 2</b>	-.65	.64	-1.90 - .60	-.05	-1.02	.307
<b>Age at baseline</b>	-.22	.58	-1.37 - .93	-.02	-.38	.703
<b>Baseline MFQ score</b>	.34	.08	.19 - .49	.23	4.34	.000
<b>Gender Category</b>	5.61	1.89	1.88 – 9.33	.16	2.96	.003
<b>SSRI prescribed prior to trial entry</b>	-3.68	2.09	-7.79 - .44	-.09	-1.76	.080

**Table 8*****Summary of Multiple Regression Analyses for Predictors of Depression Outcome (as indicated by follow-up MFQ scores): By Baseline MFQ Group***

	<b>B</b>	<b>SE B</b>	<b>β</b>	<b>t</b>	<b>p</b>	<b>CI (95%)</b>	
<b>Low MFQ</b>							
(Constant)	33.662	12.480		2.697	.008	8.930	58.395
PD Profile Score - 2 (standardised)	-.052	.762	-.006	-.069	.945	-1.562	1.457
Age at baseline	-1.648	.817	-.187	-2.017	.046	-3.268	-.029
Gender Category	6.069	2.582	.218	2.350	.021	.951	11.186
SSRI prescribed prior to trial entry	-2.807	3.257	-.079	-.862	.391	-9.262	3.649
<b>Moderate MFQ</b>							
(Constant)	10.697	14.389		.743	.459	-17.825	39.219
Age at baseline	.016	.942	.002	.017	.987	-1.852	1.884
Gender Category	5.215	2.968	.171	1.757	.082	-.668	11.099
SSRI prescribed prior to trial entry	.834	3.366	.024	.248	.805	-5.838	7.505
PD Profile Score - 2 (standardised)	-.733	1.014	-.070	-.723	.471	-2.743	1.277
<b>High MFQ</b>							
(Constant)	1.459	19.016		.077	.939	-36.245	39.164
Age at baseline	1.324	1.225	.102	1.081	.282	-1.104	3.753
Gender Category	4.848	4.294	.108	1.129	.262	-3.667	13.362
SSRI prescribed prior to trial entry	-8.774	4.038	-.204	-2.173	.032	-16.781	-.766
PD Profile Score - 2 (standardised)	-2.893	1.821	-.149	-1.589	.115	-6.503	.717



**Table 9**

***Linear Model of Predictors of Depression Treatment Outcome (as indicated by follow-up MFQ scores/depression severity) – Treatment Modality and Interaction Effects***

<b>Model</b>		<b>B</b>	<b>SE B</b>	<b>β</b>	<b>t</b>	<b>p</b>
1	(Constant)	.535	9.176		.058	.954
	Baseline MFQ score	.345	.078	.236	4.454	.000
	Gender Category	5.724	1.891	.161	3.027	.003
	Age at baseline	-.208	.584	-.019	-.356	.722
	SSRI prescribed prior to trial entry	-3.530	2.087	-.089	-1.692	.092
2	(Constant)	1.147	9.239		.124	.901
	Baseline MFQ score	.341	.078	.233	4.382	.000
	Gender Category	5.711	1.895	.161	3.014	.003
	Age at baseline	-.178	.587	-.016	-.303	.762
	SSRI prescribed prior to trial entry	-3.587	2.094	-.090	-1.713	.088
	CBT	-.980	1.993	-.030	-.492	.623
3	STPP	-1.583	2.029	-.048	-.781	.436
	(Constant)	1.760	9.291		.189	.850
	Baseline MFQ score	.336	.079	.229	4.261	.000
	Gender Category	5.626	1.910	.158	2.946	.003
	Age at baseline	-.189	.588	-.017	-.321	.748
	SSRI prescribed prior to trial entry	-3.712	2.105	-.094	-1.763	.079
	CBT	-.924	2.000	-.028	-.462	.644
	STPP	-1.564	2.033	-.047	-.769	.442
	CBTPD	-.482	.868	-.029	-.555	.579
STPPPD	-.542	.976	-.029	-.556	.579	

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## **PART III: CRITICAL APPRAISAL**

Throughout this thesis, I have approached the concept of personality disorder (PD) from two different angles. The literature review focused on a potential antecedent to the development of PD in adolescence and beyond, by looking at the relationship of bullying/victimisation in childhood to emergence of personality pathology. The empirical study concentrated more on its consequences: specifically, on how comorbid PD might impact on outcomes in adolescents with depression. While the two parts are independent, what ultimately unites them is my underlying interest in developmental psychopathology, the development of personality problems among young people, and the validity of assessing these. In the present appraisal, I will discuss firstly how my previous experiences and context have stimulated my curiosity about the development of psychological disorders over the life course, and how I came to conduct the empirical study presented here. Secondly, I will consider some of the assumptions that I had at the outset of this thesis, including my understanding of PD as a concept, and how this has changed over the course of doing my research. I also reflect upon the progress of the research overall, drawing attention to the major challenges I encountered, how I attempted to overcome these and what I have learned through the process. Adopting a critically reflective stance towards a project that has taken a significant amount of time and energy to complete is, admittedly, a somewhat daunting task; however, it is crucial to try to discover the assumptions that frame how we work as clinicians as well as researchers (Brookfield, 1998). The final part of the appraisal aims to summarise some of the key recommendations for future research arising from my work.

### ***Setting the Context: Previous Professional Experiences and their Impact on My Research***

My professional journey within mental health began at the Anna Freud National Centre for Children and Families (previously known as the Anna Freud Centre), where I worked as a research assistant and later regional trial coordinator on the IMPACT study. This, of course,



was the trial on which my current empirical study is based, and it is what inspired my ongoing interest in working with adolescents. Having been involved with the study first-hand from its early stages, I was intrigued by the process of conducting assessments with young people and their families, having the privilege of regular insight into their complex difficulties and life challenges. The process of witnessing significantly distressed teenagers wanting to participate in research which they felt would benefit others in future was a very humbling experience. Equally, the commitment of the young people, their parents, carers and therapists to seek help and treatment was frequently nothing short of inspirational. As the study progressed, I became familiar with the numerous families taking part within my region and grew aware of the many difficulties young people presented with at any one time: not just in terms of major depression, but also other struggles that may have been related to their primary diagnosis, including frequent self-harming, suicidal thoughts, pervasive anxiety and interpersonal struggles. At that time – as a relative novice in the research field – I became interested in the fact that it was extremely rare to see young people with individually discernible mental health problems. In virtually all cases, their stories encompassed not just accounts of ‘depression’ but various degrees of adversity across several domains including home and family life, school life, friendships, and in some cases historical abuse. Simultaneously, I made concerted efforts to look out for signs of resilience. A key lesson from this process was that young people (particularly at the higher end of severity) experience a range of often very complex mental health difficulties, which may be intertwined with each other, and that despite common diagnoses they each also have very individual profiles of risk and resilience. With hindsight, I am convinced that these influence how young people engage with therapeutic intervention in the first place, and how/whether they benefit from it. Moreover, my attention was drawn to the developmental perspective on

psychopathology and how some of these difficulties may evolve over the early part of the life course.

Having had the experience of direct data collection as well as local coordination of the study, I developed good familiarity with the IMPACT trial as well as a strong sense of affiliation with the research team. IMPACT was nearing completion when I began my clinical psychology training, and its timing was impeccable when I first began discussing thesis ideas with my supervisor (himself a principal investigator on the IMPACT study). Working with IMPACT data felt like a natural progression from my previous role and provided scope for a topic that both my supervisor and I had interest in, as well as one that had not yet been tackled by other investigators on the trial: emergent personality pathology among the adolescent population. Moreover, through reflection on my previous clinical experiences with adolescents, I provisionally began to think about hypotheses that could be explored through my thesis. For example, I wondered about those young people who engaged readily with research assessments with myself and attended therapy sessions regularly, versus those who frequently cancelled research appointments and/or missed therapy. I was curious about what might help explain these differences, both with reference to the individuals' inner mental world as well as pragmatic reasons and possibly factors relating to the researcher or therapist themselves. I also thought about the actual interactions that I had with the numerous young people in the IMPACT trial, how they differed in style and in the degree of connectedness and rapport I had felt in the room. I wondered about which factors, aside from depression, might impact on these interactions, and personality characteristics were one of them. All of these considerations contributed to some degree towards further thinking and discussion with my supervisor about emergent personality pathology among this adolescent population.

The correlates of personality pathology among adults have been widely researched, although perhaps the main aspects that have been considered include abuse and childhood maltreatment. In an attempt to conduct an original literature review, we discussed potential relationships that have been less well-studied but for which there could be reasonable, theoretically-based arguments. One such topic that emerged was bullying and peer victimisation. In light of existing models of personality disorder (especially BPD), it seemed conceivable that being a victim of bullying establishes a significantly ‘invalidating context’ and a multitude of ‘high-risk interpersonal transactions’ for the child at a time where a growing proportion of her interactions are within the peer group setting (Linehan, 1993; Crowell et al, 2009). These aspects are thought to contribute to the development of heightened emotional dysregulation over time, which is often seen as a hallmark of personality disorder. The literature review, therefore, focused on associations between childhood bullying and emergent PD in the context of these considerations among others.

### *Assumptions and Perspectives at the Outset*

My knowledge and understanding of personality pathology at the outset of the research was limited, having not previously worked with PD per se in either clinical or research settings. Initial reading around the subject, and the process of developing my research proposal, led me to adopt a relatively categorical, diagnosis-driven perspective on what PD is, its various sub-types and subsequent treatment (e.g. Dialectical Behaviour Therapy, Mentalization-Based Treatment). At this stage, I was predominantly focused on what differentiates alternative forms of PD, rather than what unites them, although I understood that borderline PD is sometimes considered to be the most prototypical variety in relating to symptoms across a whole range of personality disorders. This perspective inevitably shaped how I approached the literature review, for instance when specifying the types of PD that existing

research papers referred to, how they measured symptomatology, and whether they found any significant associations with childhood bullying and those specific disorders. For the empirical paper, meanwhile, my original plan was to test whether PD status predicts depression treatment outcomes by using the Zanarini Rating Scale for Borderline Personality Disorder (ZAN:BPD) (Zanarini et al, 2003). This is a clinician-administered short clinical interview used to assess the severity of BPD, including a diagnostic item at the end (based on number of endorsed items in the measure as ‘definite’ or ‘possible’). I intended to use the diagnostic item to capture PD status and assess whether the presence of PD traits among IMPACT adolescents predicted their depression outcomes in terms of post-treatment severity, as well as treatment non-response/withdrawal. As such, PD status would be conceptualised categorically, rather than using the measure continuously.

It emerged during initial data screening that using the ZAN:BPD measure as above would not be feasible in my study, due to the amount of missing data and concerns regarding the reliability of how the measure was administered in the trial. Subsequently, I considered alternative approaches to capturing PD in the sample and agreed with my supervisor that I could attempt to construct a ‘profile’ of emergent PD, which involved a factor analytical strategy (as described in Part II). This process turned out to be highly educational for me, not least in terms of learning previously unfamiliar statistical techniques. It challenged the assumption that PD necessarily involves distinct sub-categories, encouraged me to think about PD more broadly and to consider not merely its absence or presence, but instead its severity.

Constructing an emergent PD profile (which, for the purposes of the empirical study, was effectively a new measure) required me to firstly consider common latent constructs relevant to PD generally, combine these to create the profile, and then to assess where participants featured on this. In essence, the profile can indeed be understood as a continuum

of severity, whereby all subjects had a score on it but this varied depending on how far they endorsed items that formed each PD construct. All of this was done within the context of exploring recent developments around PD classification in the DSM-5 (APA, 2013) where an alternative model is presented and core, underlying components of all personality pathology are identified. Ultimately, the ‘PD profile’ approach seemed to fit well with current literature, which critiques categorical interpretations of PD in favour of a continuous, dimensional approach. This would suggest that all individuals fall on the continuum *somewhere*, but those at the extremes are likely to present with the most maladaptive functioning and probable PD.

The main hypothesis in the empirical paper hinged on the assumption that emergent PD would predict poorer treatment outcomes among IMPACT adolescents; however, it is useful to unpick this assumption further, since the prediction was itself based on research that would have been guided by its own assumptions. Most previous research which suggested that personality pathology moderates depression outcomes as well as treatment response was also based on categorical conceptualisation of PD; the picture is apparently less clear when PD is considered continuously (Mulder et al, 2002). On reflection, therefore, the original research proposal – including the main hypotheses – were framed by some of the conceptual assumptions presented above and it is helpful to be mindful of this.

### ***Reflections on The Research Process: Main Challenges and Key Learning Points***

The literature review and empirical paper formed essentially two independent pieces of research, and each presented its own challenges. Starting with the literature review, the comparison of fifteen separate studies of varying designs, using different methodologies and measures of bullying as well as PD, was not a straightforward task at the outset. It was necessary to gather the findings in a manner that would make these easier to interpret as well as to draw fair comparisons between them. My attempt to do so involved creating large

matrices/tables with all studies included, identifying the measures used, response scales, dependent/independent variables, as well as considering what was statistically controlled for and possible sources of bias. I also calculated common effect sizes across the study, to be able to compare the significance of their findings. However, fundamentally this review was not a meta-analysis, and it could be argued that such an approach would have yielded empirically more sound conclusions.

The empirical study posed a bigger challenge overall, in the context of several factors. Firstly, I was aware of my personal limits including previous knowledge of and competence in more sophisticated statistical analyses. My approach to this involved nothing extraordinary beside spending significant amounts of time undertaking online statistical tutorials, reading statistical manuals, liaison with my supervisor, university staff and members of the IMPACT research team for advice. Secondly, despite being formerly involved with the IMPACT study, I had not previously had access to the trial data. Therefore, it took significant time to familiarise myself with the dataset, and to sort the data into a format that would be workable for my planned analyses. In itself, this was a highly useful learning exercise. Thirdly, a major challenge emerged upon initial factor analysis, where it transpired that – despite the overall large sample size – it would not be feasible for me to conduct a confirmatory factor analysis (CFA) once the sample was split into two sub-samples and preliminary analyses were run to test for necessary assumptions (CFA is a technique that I had not previously used, but understood would make my project significantly stronger). I did not complete this analysis in the end and instead used the whole sample for exploratory factor analysis. The limitations of doing so have been noted in the discussion (Part II). In brief, the PD profile, as it is, is unvalidated and the findings based on its use must be considered with due caution.

The non-significant results obtained in the empirical study were a catalyst to help me explore why my hypotheses had not been supported. After all, the findings were not consistent with what was predicted: scores on the PD profile did not predict treatment outcomes. One of the considerations that arose in light of this was that it was not just the PD profile which failed to predict outcomes: amid the covariates included in the analysis, I did not find other substantial predictors either, even where some were statistically significant such as baseline depression severity. This raised an important question about what might be a more reliable predictor (or set of predictors) of depression outcomes, recovery and treatment drop-out: was there a variable that I failed to take into account? Or possibly a combination of several variables that I did not consider where interactions may exist? The original IMPACT study, for instance, found that comorbid behavioural disorder predicted non-response, as indicated by missing primary outcome data (Goodyer et al, 2016). This was not included in the present study as a covariate, since I was not looking at comorbidity at this stage. Yet it is possible that behavioural disorder may predict treatment drop-out and could interact with PD in doing so.

Finding alternative predictors was not the aim of my study, but it is nevertheless important to reflect on the possibility that other predictors may have interacted with PD and influenced adolescents' trajectories through treatment. Given the vast amount of data collected in the IMPACT study, it is possible that I inadvertently omitted information that may have fitted the data better than the variables I tested for. In future, one could look at various aspects such as participants' specific use of health and social care services and help-seeking (as captured by the CA-SUS measure), the extent and type of participants' comorbidity (using data from the kSADS measure), its severity, and the quality of the therapeutic alliance (using data from the Working Alliance Inventory) to consider whether these features might better predict treatment outcomes including recovery from depression

and adherence/engagement with therapy, either individually or in combination with each other and PD. The overall aim of such investigation would be to help clarify which aspects impact significantly upon treatment response for whom. As identified in the IMPACT trial, a challenge for future research is to improve our ability and precision to select the optimum treatment for a given patient with depression (Goodyer et al, 2016).

My empirical paper finished with a discussion of possible mechanisms to explain why a large proportion of adolescents in the trial had not recovered from depression (i.e. below clinical threshold) following treatment. This is directly linked to the challenge noted above, since an understanding of what works for whom also requires some understanding of what underlies the individuals' difficulties. This may also reveal how and why certain therapeutic approaches may not be effective for certain people if they are generalised rather than tailored to the individual.

The mechanism that I focused on and which I think is useful to revisit again here is a proposed underlying propensity for any psychopathology, which Caspi and colleagues have termed the 'p factor' (Caspi et al, 2014). The p factor, they argue, seems to explain various psychiatric disorders by reflecting their common fundamental aspects, with higher scores reflecting more severe and enduring disorders. Further research has recently validated the likelihood of this underlying dimension (e.g. Patalay et al, 2015). Caspi and his team identified a number of things that seem to be associated with higher 'p factor' scores, including individuals' negative developmental experiences, life impairment and deficits in early brain function. In the context of the present study and the finding that no one predictor accounted for substantial variance in depression treatment outcomes, I believe that it was warranted to consider the relevance of the p factor. It is possible that an underlying propensity for psychopathology (itself the product of negative developmental trajectories) may explain why some young people did not significantly improve following treatment, and



why some did not engage with treatment, if the intervention targeted only one prescribed and specific manifestation of their distress (i.e. depression).

Several aspects of the research process have been discussed above, including key limitations of the empirical study and important learning points as the project progressed. This included developing my technical and statistical skills through the process of preparing and conducting the study, as well as increasing my knowledge and broadening my perspective on PD and psychopathology overall. The process of writing both the literature review and empirical study was certainly challenging, intellectually as well as pragmatically, but overall I have found it a highly rewarding and educational experience.

### ***Recommendations for Future Research***

In the context of the present study and the above considerations, several recommendations arise for future research. Firstly, researchers should continue to explore the mechanisms that might be involved in individual responses to depression treatment, including the underlying propensity for psychopathology which may be affected by a constellation of early life experiences and individual risk and resilience factors. Secondly, future research should explore whether and how this propensity can be reliably assessed, since it could be a crucial indicator for planning effective treatment.

With regard to personality pathology more specifically, the validation of a broad, continuous measure of personality disorder (akin to the PD profile here, if not the profile itself) is urgently needed. Further study using such a measure is required to assess whether emergent PD might moderate depression outcomes and treatment response among adolescent populations other than that in the IMPACT study. The PD profile that I developed clearly had several limitations and these could be improved upon, given more time. Firstly, the items that I selected for factor analysis were not independently cross-examined or judged by any other

researcher; therefore, although they are thematically relevant to PD, it would be important for them to undergo some sort of peer review or further assessment to verify their selection. Secondly, as previously noted, CFA should be conducted to explore how accurately these items measure the (four) latent variables and to confirm their structure. Thirdly, the measure would need to be properly validated. To determine the PD profile's construct validity as a priority, future research could examine its association with other measures of personality pathology as well as with PD diagnosis (the ZAN:BPD could be considered among others). One could also explore the profile's association with other dimensional measures of PD drawing on the five-factor model of personality disorder (Trull & Widiger, 2013). It would also be important to evaluate the profile's incremental validity in measuring PD over and above measures of particular aspects such as self-harm and negative affectivity. The measure should also be tested among various samples aside from the adolescent IMPACT population.

The literature review, while not without limitations, suggested that most current research points towards childhood experiences of peer victimisation being associated with greater risk of PD. The most prominent associations appear to be with BPD, although this could be partly because these have been most commonly studied. A number of recommendations arise from this finding. Firstly, schools and other peer settings should make it their priority to tackle victimisation through the use of comprehensive anti-bullying policies, awareness campaigns for children, families and teachers, facilitating a pro-social ethos and ensuring sufficient and accessible channels for reporting bullying incidents. Victims should have easy access to psychological support delivered by staff who are properly trained in counselling, as well as being attuned to signs of emerging mental health difficulties and knowing when to signpost to specialist services. In terms of research recommendations, further prospective studies are urgently needed to help establish the direction and nature of

the relationship between victimisation and PD, and researchers should empirically explore the mechanisms and processes accounting for these associations.

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## **APPENDICES**

## APPENDIX A

### Database Search Strategy (PsychInfo Example)

1. exp Bullying/
2. bullying.mp. [mp=title, abstract, original title, name of substance word, subject heading word, keyword heading word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier]
3. (victimisation or victimization).mp. [mp=title, abstract, original title, name of substance word, subject heading word, keyword heading word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier]
4. exp Victimization/
5. exp Peer Relations/ or exp Victimization/ or exp Bullying/
6. 'peer relations'.mp. [mp=title, abstract, original title, name of substance word, subject heading word, keyword heading word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier]
7. exp Victimization/ or exp School Violence/ or exp Bullying/
8. 'school violence'.mp. [mp=title, abstract, original title, name of substance word, subject heading word, keyword heading word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier]
9. exp Bullying/ or abusive peer relationships.mp.
10. abusive peer relationships.mp.
11. exp Personality Disorders/
12. "personality disorder".mp. [mp=title, abstract, original title, name of substance word, subject heading word, keyword heading word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier]
13. exp Borderline Personality Disorder/ or exp Personality Disorders/ or exp Antisocial Personality Disorder/ or exp Narcissistic Personality Disorder/ or exp Psychopathology/
14. PD.mp.
15. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10
16. 11 or 12 or 13 or 14
17. youth.mp. [mp=title, abstract, original title, name of substance word, subject heading word, keyword heading word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier]
18. childhood.mp. [mp=title, abstract, original title, name of substance word, subject heading word, keyword heading word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier]
19. exp Adolescent Development/ or exp Predelinquent Youth/ or exp Childhood Development/
20. adolescence.mp.
21. adolescen\*.mp. [mp=title, abstract, original title, name of substance word, subject heading word, keyword heading word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier]
22. Predelinquent Youth/ or youth.mp.
23. childhood.mp.
24. school-age.mp.
25. juvenile.mp.
26. child.mp.

27. teenage\*.mp.
28. 17 or 18 or 19 or 20 or 21 or 22 or 23 or 24 or 25 or 26 or 27
29. 15 and 28
30. 16 and 29
31. limit 30 to (english language and humans)

## APPENDIX B

**Table 2**

### *Overview of Study Characteristics*

*Note: N = sample size, IV = Independent Variable, DV = Dependent Variable; PD = Personality Disorder; General bullying/victimisation = experience of bullying assessed as a composite variable, not assessing for victimisation subtypes unless otherwise specified*

Study	Design	N	- Sample characteristics & setting	Types of bullying/victimisation assessed	Source of informant(s) on bullying/victimisation	Types of Personality Disorder assessed	Source of informant(s) on Personality Disorder
1. Sansone et al, 2013	- Non-experimental - Correlational - Cross-sectional - Retrospective	373	- Female Obstetrics/gynaecology outpatients - 18-61 age range - Consecutive outpatients - Convenience sample - Non-random - 71.9% single, low-income - Location: clinic-based, local, U.S	General bullying/victimisation	Self-report only	Borderline personality symptomatology (BPS)	Self-report only
2. Fung and Raine, 2012	- Non-experimental - Correlational - Cross-sectional - Retrospective	3,508	- Schoolchildren (1,966 male, 1,542 female) - 9-15 age range - Population sample, aimed for representativeness in terms of school region, size, socioeconomic diversity and gender - Non-random - Location: 10 x primary and 10 x secondary schools in Hong Kong (drawn from 62 primary schools and 28 secondary schools)	- General bullying/victimisation ('Total peer victimisation') - Specific victimisation Subtypes: • Physical victimization • Social manipulation • Verbal victimization • Attack on property	Self-report only	Schizotypal personality	Self-report only
3. Hengartner et al, 2013	- Non-experimental - Correlational - Cross-sectional - Retrospective	512	- Swiss males and females - 20-41 age range - Stratified PD sample gained from population-based community sample in Zinep epidemiological survey (2012) - Location: canton of Zurich, Switzerland	- General bullying/victimisation, characterised by endorsement of any one or more of the following: • Frequent physical assault at school • Being frequently insulted at school • Being regularly excluded/ignored at school	Self-report only	- Paranoid PD - Schizoid PD - Schizotypal PD - Antisocial PD - Borderline PD - Histrionic PD - Narcissistic PD - Avoidant PD - Dependent PD - Obsessive-compulsive PD	Self-report only



4.	Kawabata et al, 2014	<ul style="list-style-type: none"> <li>- Non-experimental</li> <li>- Correlational</li> <li>- Longitudinal (short-term)</li> <li>- Prospective</li> </ul>	234	<ul style="list-style-type: none"> <li>- Schoolchildren (4<sup>th</sup> and 5<sup>th</sup> grade), 50-50% male/female</li> <li>- 9-11 age range</li> <li>- Community sample</li> <li>- Non-random</li> <li>- Middle-class socioeconomic status (according to teacher ratings)</li> <li>- Location: four schools in large cities in Japan</li> </ul>	<ul style="list-style-type: none"> <li>- Relational victimisation</li> <li>- Physical victimisation</li> </ul>	<ul style="list-style-type: none"> <li>Self-report</li> <li>Teacher-report</li> </ul>	Borderline Personality features (BPF)	Self-report only
5.	Laporte et al, 2012	<ul style="list-style-type: none"> <li>- Non-experimental</li> <li>- Correlational</li> <li>- Cross-sectional</li> <li>- Case-control</li> <li>- Retrospective</li> </ul>	106	<ul style="list-style-type: none"> <li>- Sibling sample: 53 pairs of sisters, one of each pair diagnosed with BPD (DSM-IV)</li> <li>- 18-45 age range</li> <li>- Mean age difference for siblings = 3.17</li> <li>- Clinical sample; referred from psychiatric clinics</li> <li>- Non-random</li> <li>- Location: urban area setting, location unspecified</li> </ul>	Peer victimisation, characterised by endorsement of Emotional and/or physical abuse perpetrated by peers or dating partners	Self-report only	Borderline PD	Self-report only (including clinical interview)
6.	Lereya et al, 2013	<ul style="list-style-type: none"> <li>- Non-experimental</li> <li>- Correlational</li> <li>- Longitudinal (for evaluating main outcome variable of self-harm)</li> <li>- Prospective (for evaluating main outcome variable of self-harm)</li> </ul>	4,810	<ul style="list-style-type: none"> <li>- Schoolchildren</li> <li>- 7-10 years at T1 and 16-17 years at T2</li> <li>- Sample sourced from Avon Longitudinal Study of Parents and Children (ALSPAC) cohort; original birth cohort=13,971 children. Current sample based on children who completed self-harm questionnaire age 16-17 years</li> <li>- Community sample</li> <li>- Non-random</li> <li>- Location: South West England, UK</li> </ul>	General bullying/victimisation, characterised by endorsement of overt bullying (personal belongings taken; threatened or blackmailed; hit or beaten up; tricked in a nasty way; called bad/nasty names) <u>and/or</u> relational bullying (exclusion to upset the child; pressure to do things s/he didn't want to do; lies or nasty things said about others; and games spoiled)	<ul style="list-style-type: none"> <li>- Self-report</li> <li>- Mother-report</li> <li>- Teacher-report</li> </ul>	Borderline PD	Self-report only
7.	Natsuaki et al, 2009	<ul style="list-style-type: none"> <li>- Non-experimental</li> <li>- Correlational</li> <li>- Longitudinal</li> <li>- Prospective</li> </ul>	174	<ul style="list-style-type: none"> <li>- Children who had participated in a week-long summer camp research program at least once between 1993-2002; 69 females, 105 males</li> <li>- 9-12 years at T1, when assessed for victimization and bullying</li> <li>- 15 years at T2, when assessed for PPD [94 maltreated, 80 non-maltreated;</li> <li>- Groups comparable in terms of family socioeconomic status (low) and other demographic characteristics; aimed for ecological validity</li> <li>- Location: local summer camp setting, geographical region not specified</li> </ul>	General bullying/victimisation – defined as ‘submissive, ineffective responses to peer aggression and dominance’	Camp-counsellor report	Paranoid PD	Self-report (in adolescence)

8.	Sansone et al, 2010	Non-experimental Correlational Cross-sectional Retrospective	414	<ul style="list-style-type: none"> <li>- Internal medicine outpatients (130 males, 287 females, 2 undisclosed)</li> <li>- 18-65 age range</li> <li>- Consecutive, convenience sample, non-psychiatric</li> <li>- Non-random</li> <li>- Location: primary care clinic, Midwestern US city</li> </ul>	General bullying/victimisation	Self-report only	Borderline personality	Self-report only
9.	Sourander et al, 2007	Non-experimental Correlational Longitudinal/follow-up Prospective	2540	<ul style="list-style-type: none"> <li>- Male cohort: boys born in 1981</li> <li>- 8 years at T1 (1989); 18-23 at T2</li> <li>- Community sample; current sample taken from Finnish 'From a Boy to a Man' nationwide study (original population sample from which this project arose included 60,007 children); final study sample based on no. Of males for whom complete information on bullying and victimisation was available.</li> <li>- Non-random</li> <li>- Location: national, Finland</li> </ul>	General bullying/victimisation	<ul style="list-style-type: none"> <li>- Self-report</li> <li>- Parent-report</li> <li>- Teacher-report</li> </ul>	Antisocial PD	Finnish military registry information – ICD-10 diagnoses available for all participants at age 18-23
10.	Wolke, et al, 2012	Non-experimental Correlational Longitudinal Prospective	6050	<ul style="list-style-type: none"> <li>- Children, 3112 females, 2938 males</li> <li>- Sample based on The Avon Longitudinal Study of Parents and Children (ALSPAC)</li> <li>- 7.5 years at T1; 11.8 years at T2</li> <li>- Community sample</li> <li>- Non-random</li> <li>- Location: South-west of England, sample/cohort considered broadly representative of children in UK</li> </ul>	<ul style="list-style-type: none"> <li>- General bullying/victimisation (combined victimisation)</li> <li>- Victimization subtypes (for self-report only):</li> <li>- Overt victimisation</li> <li>- Relational victimisation</li> </ul>	<ul style="list-style-type: none"> <li>- Self-report</li> <li>- Mother-report</li> <li>- Teacher-report</li> </ul>	Borderline personality symptoms	Self-report only
11.	Zhu and Chan, 2015	Non-experimental Correlational Cross-sectional Retrospective	3,175	<ul style="list-style-type: none"> <li>- Schoolchildren, 53.2% females, 46.8% males</li> <li>- 15-17 age range</li> <li>- Stratified sample, by geographic and administrative area; three administrative units randomly chosen, then six schools randomly chosen from each administrative unit</li> <li>- Community sample</li> <li>- Location: Xi'an city in Shanxi province in China</li> </ul>	<ul style="list-style-type: none"> <li>- Direct bullying victimisation</li> <li>- Relational bullying victimisation</li> </ul>	Self-report only	<ul style="list-style-type: none"> <li>- Antisocial personality</li> <li>- Borderline personality</li> </ul>	Self-report only
12.	Goodman et al, 2013	Non-experimental Correlational Cross-sectional Retrospective	263	<ul style="list-style-type: none"> <li>- Male sibling pairs; 97 BPD and 166 non-BPD siblings</li> <li>- Sourced via anonymous parent internet survey on National Education Alliance for Borderline Personality Disorder (NEA-BPD) website (US organisation that supports families of BPD patients)</li> <li>- Non-random</li> </ul>	General bullying/victimisation	Parent-report	Borderline PD	Parent-report

				<ul style="list-style-type: none"> <li>- BPD probands mean age: 25.8 ±7.3 years; Non-BPD probands mean age: 25.7±8.2 years</li> <li>- Parent respondent-based, mainly female (93%), mainly white (95%), married (66%), mean parent age = 53.9±7.2 years</li> <li>- Location: web-based, U.S.</li> </ul>				
13.	Goodman et al, 2010	Non-experimental Correlational Cross-sectional Retrospective	321	<ul style="list-style-type: none"> <li>- Female sibling pairs; 234 BPD and 87 non-BPD siblings</li> <li>- Sourced via anonymous parent internet survey on National Education Alliance for Borderline Personality Disorder (NEA-BPD) website (US organisation that supports families of BPD patients)</li> <li>- Non-random</li> <li>- BPD probands mean age = 24.04±8.39 years; Non-BPD probands mean age = 21.59±9.22 years</li> <li>- Parent respondent-based, mainly female (94.8%), white (92.6%), Christian (71.9%), married (63.6%), mean parent age = 50.5±9.2 years</li> <li>- Location: web-based, U.S.</li> </ul>	General bullying/victimisation	Parent-report	Borderline PD	Parent-report
14.	Copeland et al, 2013	Non-experimental Correlational Longitudinal Prospective	1,420	<ul style="list-style-type: none"> <li>- Children to adolescents, 49% female, 51% male, 8% African American, 3% American Indian, 1% Hispanic</li> <li>- 9-13 age range at enrolment in 1993; 26 years at final assessment</li> <li>- Population-based/community sample drawn from Great Smoky Mountain Study of 3 overlapping cohorts of children</li> <li>- Multistage probability sampling</li> <li>- Non-random</li> <li>- Location: sample sourced from 11 counties in Western North Carolina, U.S.</li> </ul>	General bullying/victimisation	Self-report Parent-report	Antisocial PD	Self-report only
15.	Roberts et al, 2008	Non-experimental Correlational Cross-sectional Retrospective	1,396	<ul style="list-style-type: none"> <li>- High-risk male offenders in custody</li> <li>- 18+ years (age range not reported)</li> <li>- Sample obtained from Prison Service Inmate Information System/ Central System Database)</li> <li>- stratified sample obtained, utilised the Offender Group Reconviction Score (OGRS) to oversample low and high scorers</li> <li>- Non-random</li> <li>- Location: sample sourced from 139 prisons in England and Wales</li> </ul>	General bullying/victimisation	Self-report only	<ul style="list-style-type: none"> <li>- Avoidant PD</li> <li>- Dependent PD</li> <li>- Obsessive-compulsive PD</li> <li>- Paranoid PD</li> <li>- Schizotypal PD</li> <li>- Schizoid PD</li> <li>- Histrionic PD</li> <li>- Narcissistic PD</li> <li>- Borderline PD</li> <li>- Antisocial PD</li> </ul>	Self-report only

## APPENDIX C

**Table 3**  
**Summary of Measures, Key Findings and Bias Considerations**

Study	Measures of bullying/ Victimization & variable characteristics	Measures of Personality Disorder	Independent Variable(s)	Dependent Variable(s)	Statistical analyses	Statistical control for covariance	Key findings	Effect Size (Pearson's r)	Standardised Effect Size & 95% CI (Cohen's d)	Threats of bias, Methodological limitations, Potential confounding factors
1. Sansone et al, 2013	3 x self-report questions as part of survey: whether bullied (yes/no), for how many years and by how many bullies	Personality Diagnostic Questionnaire (PDQ-4) - borderline personality disorder scale Self-Harm Inventory (SHI) to measure PD symptomatology	Bullying/ Victimization	PD	Point-biserial correlation ( <i>tpb</i> ) (equivalent to Pearson's r, used due to dichotomy of variables)	None reported	<ul style="list-style-type: none"> <li>- Significant relationship between being bullied and both PD measures (PDQ-4: <math>r=.26</math>, <math>p&lt;.001</math>; SHI: <math>r=.30</math>, <math>p&lt;.001</math>)</li> <li>- Significant relationship between no. of years bullied and PDQ-4 (<math>r [106]=.27</math>, <math>p&lt;.01</math>)</li> <li>- Significant relationship between scoring positively on both PD measures and being bullied (<math>X^2 [1, n=373]=22.41</math>, <math>r=.25</math>, <math>p&lt;.001</math>) and longevity of bullying (<i>tpb</i> [106]=.23, <math>p=.017</math>)</li> <li>- No other statistical findings reported</li> </ul>	<ul style="list-style-type: none"> <li>- Positive scoring on PD measure and being bullied: <math>r = .26</math> (PDQ-4) <math>r = .30</math> (SHI)</li> <li>- Duration of bullying and PD (on PDQ-4): <math>r = .27</math></li> <li>- Positive scoring on both PD measures and being bullied: <math>r = .25</math></li> <li>- Positive scoring on both PD measures and Longevity of bullying and PD: <math>r = .23</math></li> </ul>	<ul style="list-style-type: none"> <li>- Positive scoring on PD measure and being bullied: <math>d = .54</math> [0.33, 0.75] (PDQ-4) <math>d = .63</math> [0.42, 0.84] (SHI)</li> <li>- Duration of bullying and PD (on PDQ-4): <math>d = .56</math> [0.35, 0.77]</li> <li>- Positive scoring on both PD measures and being bullied: <math>d = .52</math> [0.31, 0.72]</li> <li>- Positive scoring on both PD measures and Longevity of bullying and PD: <math>d = .47</math> [0.26, 0.68]</li> </ul>	<ul style="list-style-type: none"> <li>- Non-representative sample</li> <li>- Lack of statistical control for potential confounders including socioeconomic status, age, mental health status/diagnoses etc.</li> <li>- Cross-sectional; cannot ascertain direction of correlation; no baseline measures of PD to ascertain change; single-informant, retrospective</li> <li>- Limited measure of bullying: no definition provided, dichotomous response scales; no definitive timescale provided for experience of victimisation ('when you were growing up')</li> </ul>
2. Fung and Raine, 2012	Multidimensional Peer Victimization Scale (MPVS) (16-item self-report scale encompassing experience of physical/verbal victimization, social manipulation and attack on property)	Schizotypal Personality Questionnaire-Child (SPQ-C) (self-report)	Bullying/ Victimization	PD	Correlation (Pearson's r)	None reported	<ul style="list-style-type: none"> <li>- All factors of schizotypy significantly positively associated with all forms of victimization across the whole sample as well as for males/females when assessed separately, invariant of age.</li> </ul> <p>(All sig. correlations as per below reported at <math>p&lt;.001</math>)</p> <ul style="list-style-type: none"> <li>- Across sample, correlation between total victimisation and total schizotypy: <math>r=.39</math></li> </ul>	<ul style="list-style-type: none"> <li>- Relationship between total victimisation and total schizotypy:</li> </ul>	<ul style="list-style-type: none"> <li>- Relationship between total victimisation and total schizotypy:</li> </ul>	<ul style="list-style-type: none"> <li>- Lack of statistical control for potential confounders including socioeconomic status, age, mental health status/diagnoses etc.</li> <li>- Cross-sectional; cannot ascertain direction of correlation; no baseline measures of PD to ascertain change; single-informant, retrospective</li> <li>- no definitive timescale provided for experience of victimisation</li> <li>- Self-report and based on single-respondent (child)</li> </ul>

<p>- <b>Across sample, sig. correlations for total schizotypy with:</b></p> <ul style="list-style-type: none"> <li>• Physical victimisation (PV): <math>r=.26</math></li> <li>• Social manipulation (SM): <math>r=.33</math></li> <li>• Verbal victimisation (VV): <math>r=.35</math></li> <li>• Attack on property (AP): <math>r=.30</math></li> </ul>	<p>Relationship between total schizotypy and:</p> <ul style="list-style-type: none"> <li>- PV: <math>r = .26</math></li> <li>- SM: <math>r = .33</math></li> <li>- VV: <math>r = .35</math></li> <li>- AP: <math>r = .30</math></li> </ul>	<p>Relationship between total schizotypy and:</p> <ul style="list-style-type: none"> <li>- PV: <math>d = .54 [0.47, 0.61]</math></li> <li>- SM: <math>d = .70 [0.63, 0.77]</math></li> <li>- VV: <math>d = .75 [0.68, 0.82]</math></li> <li>- AP: <math>d = .63 [0.56, 0.70]</math></li> </ul>
<p>- <b>Across sample, correlations for total victimisation with:</b></p> <ul style="list-style-type: none"> <li>• SPQ cognitive subscale (SPQCog): <math>r=.35</math></li> <li>• SPQ Interpersonal subscale (SPQInt): <math>r=.30</math></li> <li>• SPQ Disorganised subscale (SPQDis): <math>r=.31</math></li> </ul>	<p>Relationship between total victimisation and:</p> <ul style="list-style-type: none"> <li>- SPQ cognitive subscale(SPQCog): <math>r=.35</math></li> <li>- SPQ Interpersonal subscale (SPQInt): <math>r=.30</math></li> <li>- SPQ Disorganised subscale(SPQDis): <math>r=.31</math></li> </ul>	<p>Relationship between total victimisation and:</p> <ul style="list-style-type: none"> <li>- SPQ cognitive subscale: <math>d = .75 [0.68, 0.82]</math></li> <li>- SPQ Interpersonal subscale: <math>d = .63 [0.56, 0.70]</math></li> <li>- SPQ Disorganised subscale: <math>d = .65 [0.58, 0.72]</math></li> </ul>
<p>- <b>Across sample, sig. correlations reported between:</b></p> <ul style="list-style-type: none"> <li>• PV and SPQCog: <math>r=.24</math>, <math>p&lt;.001</math></li> <li>• PV and SPQInt: <math>r=.20</math></li> <li>• PV and SPQDis: <math>r=.20</math></li> <li>• SM and SPQCog: <math>r=.31</math></li> <li>• SM and SPQInt: <math>r=.25</math></li> <li>• SM and SPQDis: <math>r=.23</math></li> <li>• VV and SPQCog: <math>r=.31</math></li> <li>• VV and SPQInt: <math>r=.27</math></li> <li>• VV and SPQDis: <math>r=.29</math></li> <li>• AP and SPQCog: <math>r=.27</math></li> <li>• AP and SPQInt: <math>r=.22</math></li> <li>• AP and SPQDis: <math>r=.25</math></li> </ul>	<p>- PV and SPQCog: <math>r=.24</math></p> <p>- PV and SPQInt: <math>r=.20</math></p> <p>- PV and SPQDis: <math>r=.20</math></p> <p>- SM and SPQCog: <math>r=.31</math></p> <p>- SM and SPQInt: <math>r=.25</math></p> <p>- SM and SPQDis: <math>r=.23</math></p> <p>- VV and SPQCog: <math>r=.31</math></p> <p>- VV and SPQInt: <math>r=.27</math></p> <p>- VV and SPQDis: <math>r=.29</math></p> <p>- AP and SPQCog: <math>r=.27</math></p> <p>- AP and SPQInt: <math>r=.22</math></p> <p>- AP and SPQDis: <math>r=.25</math></p>	<ul style="list-style-type: none"> <li>- PV and SPQCog: <math>d = .49 [0.43, 0.56]</math></li> <li>- PV and SPQInt: <math>d = .41 [0.34, 0.48]</math></li> <li>- PV and SPQDis: <math>d = .41 [0.34, 0.48]</math></li> <li>- SM and SPQCog: <math>d = .65 [0.58, 0.72]</math></li> <li>- SM and SPQInt: <math>d = .52 [0.45, 0.58]</math></li> <li>- SM and SPQDis: <math>d = .47 [0.26, 0.68]</math></li> <li>- VV and SPQCog: <math>d = .65 [0.58, 0.72]</math></li> <li>- VV and SPQInt: <math>d = .56 [0.49, 0.63]</math></li> <li>- VV and SPQDis: <math>d = .61 [0.54, 0.68]</math></li> <li>- AP and SPQCog: <math>d = .56 [0.49, 0.63]</math></li> <li>- AP and SPQInt: <math>d = .45 [0.38, 0.52]</math></li> <li>- AP and SPQDis: <math>d = .52 [0.45, 0.58]</math></li> </ul>
<p>- 'High' versus 'low' victimization differences found (<math>p&lt;.0001</math>, two-tailed); high victimization scores associated with more than double level in schizotypy compared to 'low victimisation' group</p>		

3. Hengartner et al, 2013	Self-report retrospective questionnaire (author's own) on child adversity; bullying victimization in school assessed categorically (yes/no) based on endorsement of three questions: were you frequently physically assaulted in school/frequently insulted in school/excluded and ignored in school	Self-report, retrospective questionnaire: Assessment of DSM-IV Personality Disorders Questionnaire (ADP-IV) – enables dimensional trait-score and categorical PD diagnosis for each DSM-IV PD	<ul style="list-style-type: none"> <li>- Bullying/ Victimization</li> <li>- Other child adversity factors: <ul style="list-style-type: none"> <li>• Child maltreatment</li> <li>• conduct problems at school</li> <li>• parental separation/ divorce</li> <li>• parents' poverty and parental substance abuse</li> </ul> </li> </ul>	PD	<ul style="list-style-type: none"> <li>- Logistical regression;</li> <li>- bivariate and multivariate correlations</li> <li>- path analysis</li> </ul>	Multiple path analysis to minimise effects of interrelated predictor variables and correlated dependent variables (parents separated/divorced; poverty; conflict with parents; parental substance abuse; conduct problems; emotional abuse; emotional neglect; physical abuse; physical neglect and sexual abuse)	<ul style="list-style-type: none"> <li>- <b>Bullying victimization statistically significantly related to all 10 PDs studied (p&lt;0.05) -correlations reported:</b> <ul style="list-style-type: none"> <li>• Paranoid: r=.180</li> <li>• Schizoid: r=.183</li> <li>• Schizotypal: r=.272</li> <li>• Antisocial: r=.189</li> <li>• Borderline: r=.237</li> <li>• Histrionic: r=.117</li> <li>• Narcissistic: r=.140</li> <li>• Avoidant: r=.236</li> <li>• Dependent: r=.179</li> <li>• Obsessive-compulsive: r=.130</li> </ul> </li> <li>- <b>In path analysis:</b> <ul style="list-style-type: none"> <li>• 'Victim of bullying' was one of the strongest multivariate predictors of nearly all PD trait-scores in the study (p&lt;0.05) except for ASPD (r=.081) and Histrionic PD (r=.019) where associations were statistically not significant.</li> <li>• Sig. associations reported (p&lt;.05) with: <ul style="list-style-type: none"> <li>- Paranoid: r=.08</li> <li>- Schizoid: r=.164</li> <li>- Schizotypal: r=.171</li> <li>- Borderline: r=.105</li> <li>- Narcissistic: r=.090</li> <li>- Avoidant: r=.159</li> <li>- Dependent: r=.142</li> <li>- Obsessive-compulsive: r=.114</li> </ul> </li> </ul> </li> <li>- Authors note that victimisation may therefore represent a covariate of general personality dysfunction rather than specific PD dimensions</li> </ul>	<ul style="list-style-type: none"> <li>- Paranoid: <b>r=.18</b></li> <li>- Schizoid: <b>r=.183</b></li> <li>- Schizotyp.: <b>r=.272</b></li> <li>- Antisocial: <b>r=.189</b></li> <li>- Borderline: <b>r=.237</b></li> <li>- Histrionic: <b>r=.117</b></li> <li>- Narcissist.: <b>r=.14</b></li> <li>- Avoidant: <b>r=.236</b></li> <li>- Depend.: <b>r=.179</b></li> <li>- Obs-comp: <b>r=.130</b></li> <li>- ASPD: <b>r=.081</b></li> <li>- Histrionic PD: <b>r=.019</b></li> <li>- Paranoid: <b>r=.08</b></li> <li>- Schizoid: <b>r=.164</b></li> <li>- Schizotyp: <b>r=.171</b></li> <li>- Borderline: <b>r=.105</b></li> <li>- Narcissist.: <b>r=.090</b></li> <li>- Avoidant: <b>r=.159</b></li> <li>- Depend.: <b>r=.142</b></li> <li>- Obs-comp: <b>r=.114</b></li> </ul>	<ul style="list-style-type: none"> <li>- Paranoid: <b>d = .37 [0.19, 0.54]</b></li> <li>- Schizoid: <b>d = .37 [0.2, 0.55]</b></li> <li>- Schizotyp.: <b>d = .57 [0.38, 0.75]</b></li> <li>- Antisocial: <b>d = .38 [0.21, 0.56]</b></li> <li>- Borderline: <b>d = .49 [0.31, 0.67]</b></li> <li>- Histrionic: <b>d = .24 [0.06, 0.41]</b></li> <li>- Narcissist.: <b>d = .28 [0.11, 0.46]</b></li> <li>- Avoidant: <b>d = .49 [0.31, 0.66]</b></li> <li>- Depend.: <b>d = .36 [0.19, 0.54]</b></li> <li>- Obs-comp: <b>d = .26 [0.09, 0.44]</b></li> <li>- ASPD: <b>d = .16 [-0.01, 0.34]</b></li> <li>- Histrionic: <b>d = .04 [-0.14, 0.21]</b></li> <li>- Paranoid: <b>d = .16 [-0.01, 0.33]</b></li> <li>- Schizoid: <b>d = .33 [0.16, 0.51]</b></li> <li>- Schizotyp: <b>d = .35 [0.17, 0.52]</b></li> <li>- Borderline: <b>d = .21 [0.04, 0.39]</b></li> <li>- Narcissist.: <b>d = .18 [0.01, 0.36]</b></li> <li>- Avoidant: <b>d = .32 [0.15, 0.5]</b></li> <li>- Depend.: <b>d = .29 [0.11, 0.46]</b></li> <li>- Obs-comp: <b>d = .23 [0.05, 0.4]</b></li> </ul>	<ul style="list-style-type: none"> <li>- Stratified sampling method may compromise representativeness of sample compared to general population</li> <li>- Cross-sectional design, cannot ascertain causality on direction of association</li> <li>- Self-report measures: recall bias</li> <li>- Dichotomous measures of victimisation – does not enable exploration of intensity or duration</li> </ul>
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4.	Kawabata et al, 2014	Self- and teacher-rated likert scale surveys (frequency, never>all the time); Peer victimisation encompassed 'relational and physical victimisation', measured using Children's Social Experience Questionnaire-Self-report (CSEQ-S; Crick & Grotpeter, 1996) and teacher-report version (CSEQ-T) (truth, not at all true>always true). Measured at T1 (spring) and T2 (fall semester) – 6-month time interval	Borderline Personality Features Scale for Children (BPFS-C); self-report, truth likert scale. Measured at T1 (spring) and T2 (fall semester) – 6-month time interval	- Bullying/victimisation - Peer exclusivity - Peer aggression - Relational and physical aggression	- PD	- <b>Zero-order correlation (what is this?)</b> - Mixed linear modelling with estimation of maximum likelihood (ML)	Controlled for gender (male/female) and school grade; initial level of BPD	- BPD features at time 1 significantly associated with physical victimisation ( $r=.39$ , $p<.001$ ) and relational victimisation ( $r=.26$ , $p<.01$ )  - BPD features at time 2 significantly associated with physical victimisation ( $r=.34$ , $p<.01$ ) but not relational victimisation ( $r=.18$ , $p<1.0$ )	- BPD1 & physical vict.: <b><math>r = .39</math></b> - BPD1 & relational vict.: <b><math>r = .26</math></b>  - BPD2 & physical vict.: <b><math>r = .34</math></b> - BPD2 & relational vict.: <b><math>r = .18</math></b>	- BPD1 & physical vict.: <b><math>d = .85 [0.57, 1.13]</math></b> - BPD1 & relational vict.: <b><math>d = .54 [0.27, 0.81]</math></b>  - BPD2 & physical vict.: <b><math>d = .72 [0.45, 1.0]</math></b> - BPD2 & relational vict.: <b><math>d = 0.37 [0.1, 0.63]</math></b>	- Non-representative sample; non-random - Short time-span (6 months) for longitudinal observations - Child and teacher informants: no parental recall (parental consent not sought) - BPD measure self-report only (whilst victimisation involved child and teacher reports to form composite scale scores)
5.	Laporte et al, 2012	Childhood Trauma Interview (CTI), looking at frequency, severity and duration of six types of trauma committed by different perpetrators; 'peer victimization' was coded as emotional and physical abuse perpetrated by peers or 'dating partners'	Diagnostic Assessment for Personality Disorders (DIPD-IV) and Diagnostic Interview for Borderline-Revised (DIB-R).	- Bullying/victimisation - Family separation and loss - Intrafamilial childhood abuse/neglect - Extrafamilial childhood abuse/neglect	- PD	- Multiple Conditional Logistic Regression (CLR) - Cochran-Mantel-Haenszel (CMH) tests for association where CLR not feasible due to insufficient discordant sibling pairings	Sibling design: enables some control for variance in family background/socioeconomic context, parenting skills, parental substance abuse between comparison groups	- Physical abuse by peers/victimization associated with greater likelihood of BPD (OR=3.9, 95% CI [1.0, 14.3], $p=.04$ )  - Emotional peer abuse not significantly associated with BPD (OR=0.7, 95% CI [0.2, 2.1], $p=.50$ )  - Sexual peer abuse not significantly associated with BPD (OR=2.8, 95% CI [0.7, 11.6], $p=.10$ )	- Peer Physical abuse and likelihood of BPD: <b><math>r = .35</math></b>  - Peer emotional abuse and likelihood of BPD: <b><math>r = -.10</math></b> - Sexual peer abuse and likelihood of BPD: <b><math>r = .27</math></b>	- Peer Physical abuse and likelihood of BPD: <b><math>d = .75 [-2.96, 4.46]</math></b>  - Peer emotional abuse and likelihood of BPD: <b><math>d = -.19 [-0.33, 0.72]</math></b> - Sexual peer abuse and likelihood of BPD: <b><math>d = .57 [-2.47, 3.61]</math></b>	- Non-representative sample; possible sampling bias in not looking at other siblings; retrospective self-report by both siblings - Did not control for changes in family structure and non-shared environmental factors for sibling pairs over time - Non-BPD sisters showed reported rates of abuse significantly above non-clinical/population norms
6.	Lereya et al, 2013	Child self-report at age 8 and 10, using modified version of Bullying and Friendship Interview Schedule – questions pertaining to overt and/or relational bullying; chronicity also assessed (stable, unstable, none: considered 'stable' if reported at both age 8 and 10). Mother and teacher report gained via 'Strengths and Difficulties Questionnaire' item: "child is picked on or bullied by other children". Mother report	Semi-structured Childhood Interview for DSM-IV Borderline Personality Disorder, UK Version (CI-BPD-UK) based on borderline module of Diagnostic Interview for DSM-IV PD (DIPD-IV). Assessed at 11.7 years.	- Bullying/victimisation - Maladaptive parenting (pre-school period) - Domestic violence - Internalising/externalising behaviour - Depression	- PD - Self-harm	- Binary logistic regression - Multiple mediation analysis - Path analysis - NB: Main outcome variable = self-harm; BPD explored as possible mediator of bullying-SH association and as confounding association with bullying	Multiple mediation analysis and path analysis – controlled for confounding associations between bullying and number of risk exposures and outcomes including: preschool domestic violence, preschool maladaptive parenting, sex, depression, BPD, internalising/externalising behaviour, self-harm (main outcome variable). Logistic regression used to determine selective dropout; no significant differences found	- Being bullied (experiencing stable victimisation) in childhood directly associated with increased risk of BPD in early adolescence (probit regression coefficient=0.585 ( $p<0.05$ )).	-	- Stable victimisation in childhood and risk of BPD in adolescence: <b><math>d = 0.40 [0.30, 0.50]</math></b>	- Correlational, BPD measured at one time-point only, age 11.7 (bullying measured at age 8 and 10) – cannot establish direction of association or causality

	at 7,8 and 9 years; teacher at 7 and 10 years; Stable/Unstable/None – stable if reported at two or three time points					between subsequently weighted versus unweighted data				
7. Natsuaki et al, 2009	Observation during summer camp (approx.35 hours) and report by summer camp counsellors for each child using Mt. Hope Family Center Bullying-Victim Questionnaire; 10-item survey, 5 x questions each regarding bullying of and by others	OMNI-IV Personality Disorder Inventory conducted in adolescence (mean age – 15); self-report, 210 items; focused on subscale for PPD. Linear t-scores used to categorize subjects into low PPD, moderate PPD and high PPD symptoms as majority of sample did not reach criteria for current clinical diagnosis	- Bullying/victimisation - child maltreatment - externalising problems - social behaviour	- PD	- <b>Multilevel modelling</b>	Controlled for child sex, age and maltreatment status Multiple-informant (mismatched) design lowers risk of shared method variance	- No significant association between PPD in adolescence (mean age=15.3) and victimisation in childhood (between age 9-12). Fixed effects from growth curve models for peer victimisation reported as follows: • High PPD – coefficient=0.12, SE=0.12 • Moderate PPD – coefficient=0.1, SE=0.13 - Average of the moderate and high PPD status (review author’s calculation): beta = 0.11, SE = 0.13	-	- Association between childhood victimisation and adolescent PPD: - <b>d = 0.14 [-0.17, 0.45]</b> (based on combined mod/high PPD status; approximate ES due to insufficient information)	- Non-representative sample, demographical homogeneity – cannot generalise to middle/upper socioeconomic classes - PPD symptoms not measured longitudinally (unlike victimisation experiences) - PPD measurement at symptom level rather than clinical diagnosis – limited clinical implication - Did not measure, or control for, comorbidity
8. Sansone et al, 2010	Self-report yes/no question on survey: “When you were growing up, were you ever a victim of bullying?”; further questions re: duration (in years) and no. of bullies	Personality Diagnostic Questionnaire-4 (PDQ-4) - subscale for borderline personality; Self-Harm Inventory (SHI)	- Bullying/victimisation	- PD - Externalising problems (inc. rage reactions, excessive spending) - Binge eating - Alcohol abuse - Substance abuse - Mental health service utilisation (inc. psychiatric consultation, hospitalisation, counselling, medication)	- Analysis of Variance	Not reported	- Scores on the PDQ-4 significantly higher for those who reported being bullied (M=2.55, SD=2.44) than for those who did not (M=1.41, SD=1.71, F 1,413 = P<.001). Chi square = 18.49  - Scores on the SHI significantly higher for those who reported being bullied (M=2.93, SD=3.84) than for those who did not (M=1.03, SD=2.01, F 1,413=P<.001). Chi square = 30.54	-	- Victimization and scores on PDQ-4 (exceeding cut-off considered indicative of BPD): <b>d = 0.68 [0.36, 0.99]</b>  - Victimization and scores on SHI (exceeding cut-off considered indicative of BPD): <b>d = 0.90 [0.56, 1.24]</b>	- Non-representative sample - Limited measure of bullying experience, including time frame - Focus on symptoms of BPD, not diagnosis - Self-report on both measures, single informant on each variable - Cross-sectional – cannot ascertain direction of association or causality
9. Sourander et al, 2007	“bullying” (being a perpetrator) and victimization (being bullied), and bully-victim assessed at age 8 via child, teacher and parent self-report surveys. 3 alternatives for child: “I bully other children almost every day”, “I bully sometimes” and “usually I do not bully”. Similar alternatives for being victimized by others. Alternatives for	Information gained from military registry when all subjects aged 18 in 1999, then again in 2002 and 2004. Record of all mental health (and general health) diagnoses made by specialized psychiatric services and/or GP, ICD-10. PD grouping considered: antisocial personality disorder	- Bullying/victimisation	- PD - Other adult psychiatric outcomes: • Depression • Anxiety • Psychotic disorders - Substance abuse disorders	- Univariate and multivariate logistic regression	- Parental educational level - Child’s Baseline psychiatric symptoms (as recorded on parent and teacher scales when child aged 8) – including conduct, hyperactivity and emotional difficulties	- Association between frequent <b>victim-only</b> status at age 8 and antisocial personality disorder in early adulthood not statistically significant (OR=1.2, 95% CI [0.4-3.5])  - Statistically significant relationship between <b>bully-victim</b> status at age 8 and antisocial personality disorder in early adulthood (OR=6.8, 95% CI [3.1-15.2], p<.001).	- Association between frequent <b>victim-only</b> status at age 8 and ASPD in early adulthood: <b>r = 0.02 [0.02, 0.05]</b>  - Association between <b>bully-victim</b> status at age 8 and ASPD in early adulthood: <b>r = 0.17 [0.13, 0.21]</b>	- Association between frequent <b>victim-only</b> status at age 8 and ASPD in early adulthood: <b>d = 0.1 [-0.76, 0.96]</b>  - Association between <b>bully-victim</b> status at age 8 and ASPD in early adulthood: <b>d = 1.06 [-2.28, 4.4]</b>	- Sample was male-only; cannot generalise to female in the population - Did not directly assess psychiatric comorbidity in adulthood through structured diagnostic interview; reliance on the military registry being kept up to date - Did not control for potential confounding factors such as maladaptive family environment at baseline or changes in environment or

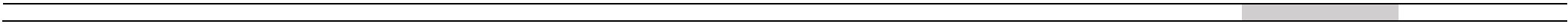


	parent/teacher surveys: “doesn’t apply”, “somewhat applies”, “certainly applies”. Assumed that lack of definition of bullying/victimization given as not described in methodology.						- When adjusted for parental educational level and baseline psychiatric symptoms: OR=3.9, 95% CI [1.4-10.9]	- <b>Adjusted r = 0.12 [0.08, 0.16]</b>	- <b>Adjusted: d = 0.75 [-1.87, 3.37]</b>	socioeconomic status over follow-up period
10. Wolke, et al, 2012	Bullying and Friendship Interview Schedule (child-report) at ages 8 and 10; five items about overt and four items about relational victimization by peers; rated frequency and chronicity of victimization as well as overt/relational or combined. Also used single-item from Strengths and Difficulties Questionnaire (Goodman 1997) for parent and teacher-report of child’s victimization (“picked on or bullied by other children over past 6 months”); parent at child’s age 4, 6.8 and 9; teacher at child’s age 7 and 10).	Childhood Interview for DSM-IV Borderline Personality Disorder: UK edition (CI-BPD-UK), based on Diagnostic Interview for DSM-IV Personality Disorders (Zanarini et al 1996) at 11.8 years of age. BPD not assessed prior to assessment of victimization (no baseline).	- Bullying/victimisation - Sexual abuse - Maladaptive parenting (inc. hitting and hostility) - Multiple family risk factors during pregnancy (inc. social, financial, practical) - Axis I disorders in childhood, including ADHD - Conduct disorder, ODD, depression and anxiety)	- PD	- Logistic regression	Multiple logistic regression analyses with forced entry method used to control for: - Gender - Sexual abuse - Maladaptive parenting (maternal hostility and hitting) - Family adversity/risk factors (social, financial, affective) - Child IQ (measured by WISC-III) Child psychiatric status at 7.5 years (DSM-IV axis I diagnoses made and recorded for conduct, ADHD, ODD, MDD, Anxiety, or ‘no diagnosis’)	<b>Controlling for confounding factors:</b>  - <b>Any victimisation associated with BPD:</b> child report (OR=2.82, 95% CI [2.13-3.72], mother report (OR=2.43, 95% CI [1.86-3.16] and teacher report (OR=1.95, 95% CI [1.34-2.83]) - <b>Chronic victimisation predicts BPD</b> according to child report (OR=5.54, 95% CI [3.86-7.66] and mother report (OR=3.24, 95% CI [2.24-4.68]); teacher-reported chronic victimisation did not significantly predict BPD (OR=1.97, 95% CI [0.67-5.82])  - <b>Combined victimisation</b> (OR=7.10, 95% CI [4.79-10.51] and <b>severity of combined, chronic victimisation</b> (OR=1.59, 95% CI [1.47-1.71] predicted BPD	<b>Any victimisation associated with BPD:</b> - child report: <b>r = 0.15 [0.12, 0.17]</b> - mother report: <b>r = 0.13 [0.1, 0.15]</b> - teacher report: <b>r = 0.1 [0.07, 0.12]</b>  - <b>Chronic victimisation and BPD:</b> - child report: <b>r = 0.26 [0.23, 0.28]</b> - mother report: <b>r = 0.17 [0.14, 0.19]</b> - teacher report: <b>r = 0.1 [0.07, 0.12]</b>  - Combined victimisation and BPD: <b>r = 0.27 [0.25, 0.29]</b>  - Severity of combined, chronic victimisation and BPD: <b>r = 0.07 [0.04, 0.09]</b>	<b>Any victimisation associated with BPD:</b> - child report: <b>d = 0.57 [0.14, 1.01]</b> - mother report: <b>d = 0.49 [0.13, 0.85]</b> - teacher report: <b>d = 0.37 [-0.04, 0.77]</b>  - <b>Chronic victimisation and BPD:</b> - child report: <b>d = 1.01 [-0.02, 2.05]</b> - mother report: <b>d = 0.65 [-0.02, 1.33]</b> - teacher report: <b>d = 0.37 [-1.05, 1.8]</b>  - Combined victimisation and BPD: <b>d = 1.08 [-0.5, 2.66]</b>  - Severity of combined, chronic victimisation and BPD: <b>d = 0.25 [0.25, 0.25]</b>	No measure of BPD prior to bullying assessment – cannot ascertain when symptoms began to manifest - BPD symptoms might be precursors of both victimisation and later BPD
11. Zhu and Chan, 2015	Direct victimization: peer and sibling module of Juvenile Victimization Questionnaire (JVQ;	Personal and Relationships Profile (PRP) (Straus et al 1999) – Borderline Personality Symptoms	- PD - Substance abuse - Self-esteem - PTSD	- Bullying/victimisation	- Structured multiphase logistic regression	- Forward stepwise logistic regression with forced entry method. Aimed to control for:	- Borderline personality trait (BPT) significantly associated with lifetime <i>direct</i> bullying victimisation. BPT –	- N/A – not enough information to calculate r	- N/A – not enough information to calculate d	- Authors suggest correlates ‘lead’ to victimisation but this cannot be established given cross-sectional, correlational design of study

	Finkelhor et al 2005), lifetime and 12-months prevalence. Relational victimization: Relational Aggression Scale (RAS; Crick and Grotpeter 1995). Both measures 5-item, self-report (no measure of chronicity or severity highlighted)	and Antisocial Personality Symptoms subscales used; nine-items each, likert scale of agreement (e.g. "I often lie to get what I want...")	- Attachment - Demographic and socioeconomic status			<ul style="list-style-type: none"> <li>• Gender</li> <li>• Parents' educational level</li> <li>• No. of siblings</li> <li>• Parental marital status</li> <li>• Family socioeconomic status</li> <li>• School district</li> <li>• School type</li> <li>• Smoking</li> <li>• Gambling</li> <li>• Alcohol and substance abuse</li> <li>• Depression</li> <li>• Self-esteem</li> <li>• PTSD</li> </ul> Attachment style	adjustedOR=1.36, 95% CI [1.018, 1.824], p<.05) - BPT significantly associated with lifetime <i>relational</i> victimisation (adjustedOR=1.6, 95% CI [1.178-2.164], p<.01) and with previous-year relational victimisation (OR=1.47, CI not reported, p<.05) - No significant association between BPT and previous-year direct victimisation (OR=1.15, CI not reported) - No significant associations found between Antisocial Personality Trait (APT) and either direct or relational lifetime victimisation (OR=0.98, OR=0.81 respectively, CI not reported) - No significant associations found between APT and either direct or relational previous year victimisation (OR=1.11, OR=1.13 respectively, CI not reported)		- BPT might be consequence of, rather than precursor, of victimisation - Retrospective, child self-report – potential for social desirability response bias - Limited age range in sample means cannot generalise to younger children or young adults	
12. Goodman et al, 2013	No specific measure; Anonymous internet survey included question(s) on child being a victim of bullying, items could be endorsed or not (not clear in paper); terms not defined	Internet-based survey, parent-report. Included BPD diagnostic screen adapted from McLean Screening Instrument for Borderline Personality Disorder (Zanarini et al 2013). Had to score 7+ and have had formal BPD clinical diagnosis to be categorized as 'BPD'	- Bullying/victimisation - Several clinical features across four developmental periods, including: <ul style="list-style-type: none"> <li>• affective lability</li> <li>• sensitivity</li> <li>• developmental delay</li> <li>• separation anxiety</li> <li>• academic difficulties</li> <li>• interpersonal problems</li> <li>• impulsivity</li> <li>• self-harm</li> <li>• self-image concerns</li> <li>• psychotic symptoms</li> <li>• sexual abuse</li> </ul>	- PD	Pearson's Chi-square tests for differences between BPD and non-BPD probands within four reported developmental periods (infancy, toddlerhood, childhood and adolescence) - Hierarchical regression analyses to study interactions between predictors/IVs and outcome/DV (presence or absence of BPD)	Stepwise logistic regression used to control for demographic confounds of age and household income	- Parents reported significantly more bully victimisation for child with BPD compared to non-BPD sibling during childhood period [defined as age 5-13] (OR=4.47, p<.000; CI's not reported) - reported prevalence in BPD probands = 45.36% (n=44), in non-BPD sibling = 15.66% (n=26)	- N/A	- Victimization and BPD: - <b>d = 0.44 [0.19, 0.69]</b>	- Non-representative sample demographically, non-random, internet-based which may involve uptake bias - Did not control for current sibling demographic differences - Did not control for parental psychopathology - Retrospective, parental recall only – potential response bias and/or inaccuracy in recall - Did not distinguish between full, half or step-siblings, whether raised together or apart - Correlational – cannot ascertain causality or direction of association - Not clear how bully victimisation was measured or whether terms defined in the survey
13. Goodman et al, 2010	None specific; Anonymous internet survey included questions on child being	Internet-based survey, parent-report. Included BPD diagnostic screen	- Bullying/victimisation - several clinical features across	- PD	Pearson's Chi-square tests for differences between BPD	Stepwise logistic regression used to control for demographic	- Parents reported significantly more bully victimisation for child with BPD compared to non-BPD sibling during	- Bully victimisation and BPD:	- Bully victimisation and BPD: - <b>d = 0.54 [0.31, 0.77]</b>	- Did not control for parental psychopathology - Retrospective, parental recall only – potential

	a victim of bullying, items could be endorsed or not by parent respondent; terms not defined	adapted from McLean Screening Instrument for Borderline Personality Disorder (Zanarini et al 2013). Had to score 7+ and have had formal BPD clinical diagnosis to be categorized as 'BPD'	four developmental periods, including: <ul style="list-style-type: none"> <li>• affective lability</li> <li>• sensitivity</li> <li>• developmental delay</li> <li>• separation anxiety</li> <li>• academic difficulties</li> <li>• interpersonal problems</li> <li>• impulsivity</li> <li>• self-harm</li> <li>• self-image concerns</li> <li>• psychotic symptoms</li> <li>• sexual abuse</li> </ul>	and non-BPD probands within four reported developmental periods (birth, infancy & toddlerhood, childhood and adolescence) <ul style="list-style-type: none"> <li>- Hierarchical regression analyses to study interactions between predictors/IVs and outcome/DV (presence or absence of BPD)</li> </ul>	confounds of age and household income	childhood period [defined as age 5-13] (Chi square = 21.822, p<.000); no OR or CI's reported for victimisation variable <ul style="list-style-type: none"> <li>- Reported prevalence in BPD probands = 32.47% (n=76), in non-BPD sibling = 6.90% (n=6)</li> </ul>	- <b>r = 0.3054 [0.18, 0.42]</b>		response bias and/or inaccuracy in recall <ul style="list-style-type: none"> <li>- No direct questioning about childhood physical or sexual abuse (both found to be predictive of later BPD in previous research as noted in paper)</li> <li>- Did not distinguish between full, half or step-siblings, whether raised together or apart</li> <li>- Correlational – cannot ascertain causality or direction of association</li> <li>- Not clear how bully victimisation was measured aside from yes/no endorsement</li> <li>- Terms not defined in the survey – interpretation of 'bullying victimisation' may not be consistent across respondents</li> </ul>
14. Copeland et al, 2013	Victimisation assessed between the ages of 9 and 16 years, as part of the Child and Adolescent Psychiatric Assessment (completed annual); both the child and the primary caregiver reported on whether the child had been bullied or teased or had bullied others in the 3 months immediately prior to the interview Being bullied or bullying others was counted if reported by either the parent or the child at any childhood or adolescent assessment. If the informant reported that the participant had been bullied or had bullied others, then the informant was asked separately how often the bullying occurred in the prior 3 months in 3 settings: home, school, and community. All participants were categorized as victims only, bullies only, both (bullies/ victims), or neither.	Self-report interviews with the Young Adult Psychiatric Assessment (YAPA).20 The time frame for the YAPA was the 3 months immediately preceding the interview.  DSM-IV diagnoses created via SAS scoring programs which combined data information about onset, duration and intensity of symptoms reported	- Bullying/victimisation <ul style="list-style-type: none"> <li>- Antisocial PD</li> <li>- Other psychiatric outcomes including: <ul style="list-style-type: none"> <li>• Depressive disorders</li> <li>• Anxiety disorders</li> <li>• Generalized anxiety</li> <li>• Panic disorder</li> <li>• Agoraphobia</li> <li>• Alcohol disorders</li> <li>• Marijuana disorder</li> </ul> </li> <li>- Suicidality (inc. suicidal ideation, or a suicide attempt)</li> </ul>	- Weighted logistic regression	Multivariate analyses to control for the effects of childhood and adolescent psychiatric problems (including depressive and anxiety disorders, suicidality, disruptive disorders, substance use) and social/family hardships (including low socioeconomic status, family instability, family dysfunction and maltreatment) Also tested for sex interaction effects	- Being a victim of bullying not significantly associated with antisocial personality disorder (OR=0.3, 95% CI [0.1-1.1], p=.06).  - Being a bully-victim also not significantly associated with ASPD (OR=1.3, 95% CI [0.3-5.3], p=.74)  - <b>Associations remained insignificant when controlled for childhood psychiatric disorders and family hardships:</b> - Victim only: OR=0.3, 95% CI [0.1-1.4], p=.11 - Bully-victim: OR=2.4, 95% CI [0.5-9.3], p=.22)	- Bully victimisation and ASPD: <b>r = -0.1 [-0.15, -0.04]</b>  - Bully-victim and ASPD: <b>r = 0.02 [-0.03, 0.08]</b>  - Adjusted/victimisation and ASPD: <b>r = -0.1 [-0.15, -0.04]</b>  - Adjusted/bully-victim and ASPD: <b>r = 0.07 [0.01, 0.12]</b>	- Bully victimisation and ASPD: <b>d = -0.66 [-0.95, -0.38]</b>  - Bully-victim and ASPD: <b>d = 0.14 [-1.24, 1.52]</b>  - Adjusted/victimisation and ASPD: <b>d = -0.66 [-1.02, -0.3]</b>  - Adjusted/bully-victim and ASPD: <b>d = 0.47 [-1.95, 2.9]</b>	- used overall assessment of bullying, could not distinguish between overt and relational bullying (which may have changed association with outcome, also differentially for males and females)  - Representative of the school settings in this particular area but not U.S. or outside

15. Roberts et al, 2008	Self-report of adverse childhood experiences (before age 16), 'being bullied', dichotomous response Y/N	SCID-II	<ul style="list-style-type: none"> <li>- Bullying/victimisation</li> <li>- Other adverse childhood experiences: <ul style="list-style-type: none"> <li>• family mental health problems</li> <li>• family criminality</li> <li>• family substance abuse problems</li> <li>• parental discord</li> <li>• sexual abuse</li> <li>• neglect</li> <li>• emotional abuse</li> <li>• lack of affection</li> <li>• LAC provision</li> <li>• childhood temperamental features</li> </ul> </li> </ul>	- PD	- Multivariate logistic regression	Adjusted for age, ethnicity, alcohol disorder, drug disorder, psychosis, major depression and comorbid personality disorders	<p><b>- After controlling for age, ethnicity and the Axis I and II disorders, being bullied was significantly associated with greater likelihood of:</b></p> <ul style="list-style-type: none"> <li>- Avoidant PD (OR=1.75, 95% CI [1.13-2.68], p&lt;.01)</li> <li>- Histrionic PD (OR=3.99, 95% CI [1.35-11.76], p&lt;.01),</li> <li>- Borderline PD (OR=1.52, 95% CI [1.05-2.20], p&lt;.05)</li> <li>- Antisocial PD (OR=0.72, 95% CI [0.52-1.00], p&lt;.05)</li> </ul> <p>- No significant association with obsessive-compulsive PD, Paranoid, Schizoid or Narcissistic PD – results not reported by authors</p> <p>- Significant joint effects of childhood adverse environmental factors on PD: <ul style="list-style-type: none"> <li>• Being bullied AND criminality of family members increased likelihood of Avoidant PD (joint effects OR=3.01)</li> <li>• Being bullied AND having criminal influences from peer group <i>reduced</i> likelihood of Avoidant PD (joint effects OR=1.04)</li> <li>• Being bullied AND in local authority care increased likelihood of Borderline PD (joint effects OR=2.17)</li> </ul> </p> <p>- Risk of antisocial PD in adulthood from a) criminality among family members, b) lack of affection from parents, and c) placement in LAC was <i>reduced</i> among prisoners who reported that they had been bullied in childhood (OR = a)1.03, b) not reported, c) 1.50)</p>	<ul style="list-style-type: none"> <li>- Bully victimisation and Avoidant PD: <b>r = 0.09 [0.04, 0.14]</b></li> <li>- Bully victimisation and Histrionic PD: <b>r = 0.08 [0.03, 0.13]</b></li> <li>- Bully victimisation and Borderline PD: <b>r = 0.09 [0.04, 0.14]</b></li> <li>- Bully victimisation and ASPD: <b>r = -0.09 [-0.14, -0.03]</b></li> </ul>	<ul style="list-style-type: none"> <li>- Bully victimisation and Avoidant PD: <b>d = 0.31 [-0.12, 0.74]</b></li> <li>- Bully victimisation and Histrionic PD: <b>d = 0.76 [-2.11, 3.63]</b></li> <li>- Bully victimisation and Borderline PD: <b>d = 0.23 [-0.09, 0.56]</b></li> <li>- Bully victimisation and ASPD: <b>d = -0.18 [-0.18, -0.18]</b></li> </ul>	<ul style="list-style-type: none"> <li>- Not representative of general population and/or clinical population – disproportionate PD among prisoner population (72.9% prevalence of any PD found in present study)</li> <li>- Correlational, cannot determine causal pathways in PD outcome</li> <li>- Cross-sectional nature means cannot distinguish between temperamental features and early adverse experiences; it may be that the former is the result of the latter</li> <li>- Self-report, retrospective; risk of memory suppression/forgetting, recall bias</li> <li>- Dichotomous measure of 'bullying' (and all other adverse childhood experiences)</li> </ul>
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## APPENDIX 1

### DSM-5 Personality Disorder Trait Domains and Facets

(Source: *Diagnostic and Statistical Manual (5<sup>th</sup> Ed)*, American Psychiatric Association, 2013)

DOMAINS (Polar Opposites) and Facets	Definitions
<b>NEGATIVE AFFECTIVITY</b> (vs. Emotional Stability)	Frequent and intense experiences of high levels of a wide range of negative emotions (e.g., anxiety, depression, guilt/ shame, worry, anger) and their behavioral (e.g., self-harm) and interpersonal (e.g., dependency) manifestations.
Emotional lability	Instability of emotional experiences and mood; emotions that are easily aroused, intense, and/or out of proportion to events and circumstances.
Anxiousness	Feelings of nervousness, tenseness, or panic in reaction to diverse situations; frequent worry about the negative effects of past unpleasant experiences and future negative possibilities; feeling fearful and apprehensive about uncertainty; expecting the worst to happen.
Separation insecurity	Fears of being alone due to rejection by—and/or separation from—significant others, based in a lack of confidence in one’s ability to care for oneself, both physically and emotionally.
Submissiveness	Adaptation of one’s behavior to the actual or perceived interests and desires of others even when doing so is antithetical to one’s own interests, needs, or desires.
Hostility	Persistent or frequent angry feelings; anger or irritability in response to minor slights and insults; mean, nasty, or vengeful behavior. <i>See also</i> Antagonism.
Perseveration	Persistence at tasks or in a particular way of doing things long after the behavior has ceased to be functional or effective; continuance of the same behavior despite repeated failures or clear reasons for stopping.
Depressivity	<i>See</i> Detachment.
Suspiciousness	<i>See</i> Detachment.
Restricted affectivity (lack of)	The <i>lack of</i> this facet characterizes <i>low levels</i> of Negative Affectivity. <i>See</i> Detachment for definition of this facet.
<b>DETACHMENT</b> (vs. Extraversion)	Avoidance of socioemotional experience, including both withdrawal from interpersonal interactions (ranging from casual, daily interactions to friendships to intimate relationships) and restricted affective experience and expression, particularly limited hedonic capacity.
Withdrawal	Preference for being alone to being with others; reticence in social situations; avoidance of social contacts and activity; lack of initiation of social contact.
Intimacy avoidance	Avoidance of close or romantic relationships, interpersonal attachments, and intimate sexual relationships.
Anhedonia	Lack of enjoyment from, engagement in, or energy for life’s experiences; deficits in the capacity to feel pleasure and take interest in things.
Depressivity	Feelings of being down, miserable, and/or hopeless; difficulty recovering from such moods; pessimism about the future; pervasive shame and/or guilt; feelings of inferior self-worth; thoughts of suicide and suicidal behavior.
Restricted affectivity	Little reaction to emotionally arousing situations; constricted emotional experience and expression; indifference and aloofness in normatively engaging situations.
Suspiciousness	Expectations of—and sensitivity to—signs of interpersonal ill-intent or harm; doubts about loyalty and fidelity of others; feelings of being mistreated, used, and/or persecuted by others.

DOMAINS (Polar Opposites) and Facets	Definitions
<b>ANTAGONISM (vs. Agreeableness)</b>	Behaviors that put the individual at odds with other people, including an exaggerated sense of self-importance and a concomitant expectation of special treatment, as well as a callous antipathy toward others, encompassing both an unawareness of others' needs and feelings and a readiness to use others in the service of self-enhancement.
Manipulativeness	Use of subterfuge to influence or control others; use of seduction, charm, glibness, or ingratiation to achieve one's ends.
Deceitfulness	Dishonesty and fraudulence; misrepresentation of self; embellishment or fabrication when relating events.
Grandiosity	Believing that one is superior to others and deserves special treatment; self-centeredness; feelings of entitlement; condescension toward others.
Attention seeking	Engaging in behavior designed to attract notice and to make oneself the focus of others' attention and admiration.
Callousness	Lack of concern for the feelings or problems of others; lack of guilt or remorse about the negative or harmful effects of one's actions on others.
Hostility	See Negative Affectivity.
<b>DISINHIBITION (vs. Conscientiousness)</b>	Orientation toward immediate gratification, leading to impulsive behavior driven by current thoughts, feelings, and external stimuli, without regard for past learning or consideration of future consequences.
Irresponsibility	Disregard for—and failure to honor—financial and other obligations or commitments; lack of respect for—and lack of follow-through on—agreements and promises; carelessness with others' property.
Impulsivity	Acting on the spur of the moment in response to immediate stimuli; acting on a momentary basis without a plan or consideration of outcomes; difficulty establishing and following plans; a sense of urgency and self-harming behavior under emotional distress.
Distractibility	Difficulty concentrating and focusing on tasks; attention is easily diverted by extraneous stimuli; difficulty maintaining goal-focused behavior, including both planning and completing tasks.
Risk taking	Engagement in dangerous, risky, and potentially self-damaging activities, unnecessarily and without regard to consequences; lack of concern for one's limitations and denial of the reality of personal danger; reckless pursuit of goals regardless of the level of risk involved.
Rigid perfectionism (lack of)	Rigid insistence on everything being flawless, perfect, and without errors or faults, including one's own and others' performance; sacrificing of timeliness to ensure correctness in every detail; believing that there is only one right way to do things; difficulty changing ideas and/or viewpoint; preoccupation with details, organization, and order. The <i>lack of</i> this facet characterizes <i>low levels</i> of Disinhibition.
<b>PSYCHOTICISM (vs. Lucidity)</b>	Exhibiting a wide range of culturally incongruent odd, eccentric, or unusual behaviors and cognitions, including both process (e.g., perception, dissociation) and content (e.g., beliefs).
Unusual beliefs and experiences	Belief that one has unusual abilities, such as mind reading, telekinesis, thought-action fusion, unusual experiences of reality, including hallucination-like experiences.
Eccentricity	Odd, unusual, or bizarre behavior, appearance, and/or speech; having strange and unpredictable thoughts; saying unusual or inappropriate things.
Cognitive and perceptual dysregulation	Odd or unusual thought processes and experiences, including depersonalization, derealization, and dissociative experiences; mixed sleep-wake state experiences; thought-control experiences.

## APPENDIX 2

### Glossary of Measures Used in the IMPACT Study

(Source: IMPACT Research Team)

- *MFQ: Mood and Feelings Questionnaire*
- *RCMAS: Revised Children's Manifest Anxiety Scale*
- *LOI: Leyton Obsessional Inventory*
- *RSES: Rosenberg's Self-esteem Scale*
- *Behaviours Checklist*
- *RRS: Ruminative Responses Scale*
- *Friendships Questionnaire*
- *K-SADS-PL: Kiddie Schedule for Affective Disorders and Schizophrenia—Present and Lifetime*
- *HoNOSCA: Health of the Nation Outcome Scale for Children and Adolescents*
- *FAD: Family Assessment Device*
- *DSC: Depressed States Checklist*
- *RTSHIA: The Risk-Taking and Self-Harming Inventory for Adolescents*
- *C-SSRS: Classification Suicide Severity Rating Scale*
- *ZAN:BPD: Zanarini Rating Scale for Borderline Personality Disorder*
- *DEQ: Depressive Experiences Questionnaire*
- *APQ: Alabama Parenting Questionnaire*
- *DES-IV: Differential Emotion Scale-IV*
- *NEO-FFI: NEO-Five Factor Inventory*
- *WAI-S: Working Alliance Inventory-Short*
- *CA-SUS: Child and Adolescent Service Use Schedule*
- *SCL:90: Symptom Checklist 90*
- *Life Events Questionnaire*
- *CAM-EE: Cambridge Early Experiences Interview*
- *HCAM: Hampstead Child Adaptation Measure*
- *CGI: Clinical Global Impressions Scale*
- *CDRS: Children's Depression Rating Scale*

Moods and Feelings Questionnaire (MFQ) – part of the young person's questionnaire (YPQ)  
The Moods and Feelings Questionnaire (Angold, Costello, Pickles & Winder, 1987) is a 33-item self-report measure of depressive symptoms. The MFQ consists of a series of descriptive phrases regarding how the subject has been feeling or acting recently. Respondents rate their symptoms over the last 2-week period and items are scored on a 4-point Likert Scale (always=2, mostly=2, sometimes=1, never=0). Higher MFQ scores have been found to predict persistence of major depression (Goodyer et. al. 1997). Work looking at 3 repeat MFQ scores over 12 months, suggests that the MFQ may be detecting trait rather state depressive symptoms. Three trajectories appear to emerge: stable high scorers, stable low scorers and a more volatile mid group for whom the MFQ may be reflecting more immediate response to events.

Revised Children's Manifest Anxiety Scale (RCMAS) –part of the YPQ

The Revised Children's Manifest Anxiety Scale (Reynolds & Richmond, 1978) is a 28-item self-report measure of anxiety symptoms in children. Scoring is on a 4-point Likert Scale (as for the MFQ). Construct validity has been shown by a high correlation between RCMAS



scores and trait scores on the State-Trait Anxiety Inventory for Children, STAIC, (Spielberger 1973, Reynolds, 1980).

Short Leyton Obsessional Inventory (LOI) – part of the YPQ

The Short Leyton Obsessional Inventory (Child Version) is an 11-item screen for current symptoms of obsessive-compulsive disorder (OCD) in children and adolescents (Bamber et al. 2002). It is scored on a 4-point Likert scale (always=3, mostly=2, sometimes=1, never=0).

### **Behaviours Checklist**

The behaviours checklist is an 11-item screen for symptoms of antisocial behaviour which was derived from DSM-III-R criteria for Conduct Disorder. It is scored on a 4-point Likert scale (always=3, mostly=2, sometimes=1, never=0). (Goodyer)

### **RSES: Rosenberg Self Esteem Scale**

The Rosenberg Self-Esteem Scale is a 10-item self-report measure of global self-esteem. It consists of 10 statements related to overall feelings of self-worth or self-acceptance. The items are answered on a four-point scale ranging from strongly agree to strongly disagree.

### **Ruminative Responses Scale (RRS)**

The RRS is a 39-item measure taken from the Nolen-Hoeksema's RDQ. It describes responses to low mood that are self-focused, symptom-focused and focused on the possible consequences and causes of the mood. Four-point Likert scale – almost never =1, sometimes =2, often =3, almost always = 4. Treynor et al (2003) have demonstrated 3 sub-scales: reflection, brooding and depression related items. High ruminative scores have been found to predict an increase in self-report depression scores in young adults (Nolen-Hoeksema, 1992).

### **Friendships Questionnaire**

Eight items on current friendship satisfaction: happy with number of friends, frequency of arrangements, confiding nature of friends, frequency of hurtful teasing and arguments and assesses the respondent's overall friendship satisfaction. (Goodyer). High scores are indicative of greater friendship satisfaction.

### **K-SADS-PL**

Full title: The schedule for affective disorders and schizophrenic disorders for school age children present and lifetime version (Kaufman et al., 1997). The K-SADS-PL is a semi-structured interview measure which generates diagnoses of depressive, anxiety, eating and behaviour disorders according to DSM-IV criteria. It has shown impressive inter rater reliability and construct validity in numerous studies.

### **HoNOSCA**

The Health of the Nation Outcome Scale for Children and Adolescents (HoNOSCA) will be completed (Gowers, Levine, Bailey-Rogers, Shore, & Burhouse, 2002). This instrument measures the outcomes of child and adolescent psychiatric disorders across a range of areas relevant to the *quality* of the child and family's life, including psychiatric symptoms, peer relationships, family functioning and school functioning. HoNOSCA is widely used in the NHS. Indeed, it was used by the Audit Commission in its recent survey of all child mental health services in England. It is of known reliability, sensitive to change, and correlates well with the clinician's judgement of outcome (Gowers et al., 2002). There are 15 items each scored from 0-4 where 0 indicates no problems in that area and 4 indicates very severe problems.

### **McMaster Family Assessment Device (FAD)**

The FAD is used to evaluate families according to the McMaster model of Family Functioning and measure how the family unit works together on essential tasks.

In IMPACT the general functioning sub-scale will be used, which are 12 items rated from strongly agree – strongly disagree, assessing overall health of the family. Higher FAD scores are associated with more unhealthy or pathological family functioning. This is an ideal device not only due to its brevity (about 5 minutes to complete), but also its reputation as a valid and reliable measure in identifying families with clinical needs.

### **Depressed States Checklist (DSC)**

A 28-item measure of cognitive vulnerability to depression. Participants describe how they felt last time their mood ‘went down’ by rating a list of adjectives on a 5-point Likert scale from not at all – extremely. The scale measures affective components of depression and globally negative (self-devaluative) view of the self. The ratio of one to the other has been found to be significantly higher in the previously depressed compared to never depressed, even after controlling for current mood (Teasdale & Cox 2001).

### **RTSHIA: The Risk-Taking and Self-Harming Inventory for Adolescents**

The RTSHIA is based on existing instruments for assessing self-harm and risk taking behaviour, and on clinical descriptions of RT and SH, using items that tap into RT and SH in both direct and indirect ways. There were 13 RT-related items in the original RTSHIA, ranging from mild behaviours such as taking chances while doing one’s hobbies and smoking tobacco, to serious risk-taking such as participating in gang violence and putting oneself at risk of sexual abuse. The 20 SH-related items ranged again from milder behaviours such as picking at wounds and pulling one’s hair out to more serious SH such as taking an overdose and trying to commit suicide. It includes an item developed by Lundh et al. (2007), which enquires about SH leading to hospitalisation or an injury severe enough to require medical treatment. The majority of the SH items are about self-mutilation (e.g. cutting, burning, biting, scratching one’s skin etc), followed by a question about the part(s) of the body that were deliberately injured, if applicable. Three items are about eating disorders (starving oneself, eating too much and using laxatives), two items about self-demeaning behaviour (staying in a friendship or a relationship with somebody who repeatedly hurt one’s feelings and trying to make oneself suffer by thinking horrible things about oneself) and two items are about self-harming ideation, with or without suicidal intent. The items are ordered gradually in terms of severity and expected frequency (from the milder and more frequent to the more serious and rarer). Most items contain the word “intentionally” or end with the phrase “to hurt or punish yourself”. The items are on a 4-point Likert scale, referring to life-long history and respondents can answer each question by selecting “never”, “once”, “more than once”, or “many times”, as employed by Lundh et al.

Vrouva, I., Fonagy, P., Fearon, P. & Rossouw, T. (2009). The risk-taking and self-harming inventory for adolescents (RTSHIA): Development and psychometric investigation in a community and a clinical sample of young people in England.

### **C-SSRS: Classification Suicide Severity Rating Scale**

Systematic administration of a tool designed to track suicidal adverse events across a treatment trial. *Prospective version* of the system developed for the FDA. Way to get better safety monitoring and avoid inconclusive results, this is why the FDA is often recommending C-SSRS in ongoing or future studies. Feasible, low- burden (typical admin time 5 minutes), assesses *both* behavior and ideation, appropriately assesses and tracks suicidal all events. Uniquely address the need for a summary measure of suicidality.

Posner, K., Oquendo, M.A., Gould, M., Stanley, B., Davies, M. (2007). Columbia Classification Algorithm of Suicide Assessment (C-CASA): Classification of Suicidal Events in the FDA's Pediatric Suicidal Risk Analysis of Antidepressants. *American Journal of Psychiatry* 164:7, 1035-1043.

### **ZAN:BPD: Zanarini Rating Scale for Borderline Personality Disorder**

The ZAN-BPD is based on the borderline module of the Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV). The interrater and test-retest reliability of the DSM-III (Zanarini, Frankenburg, Chauncey, & Gunderson, 1987), DSM-III-R (Zanarini & Frankenburg, 2001), and DSM-IV versions of this interview (Zanarini et al., 2000) have all been carefully assessed and found to be in the good to excellent range according to the standards described by Fleiss (1981).

In this system, 0 = no symptoms, 1 = mild symptoms, 2 = moderate symptoms, 3 = serious symptoms, and 4 = severe symptoms. For each anchored-rating point for each criterion, the rating is intended to reflect both frequency and severity of psychopathology. In addition to the criterion-based scales, the interview has four sector scores reflecting the four core areas of borderline psychopathology (Zanarini et al., 1990): affective, cognitive, impulsive, and interpersonal symptoms. There are three affective symptoms in the ZAN-BPD (with a sector score ranging from 0 to 12): inappropriate anger/frequent angry acts, chronic feelings of emptiness, and mood instability. There are two cognitive symptoms (with a sector score ranging from 0 to 8): stress-related paranoia/dissociation and severe identity disturbance. (Identity disturbance was placed in the cognitive realm because it is based on a series of false beliefs, such as that one is good one minute and bad the next.) There are also two impulsive symptoms (with a sector score ranging from 0 to 8): self-mutilative/suicidal efforts and two other forms of impulsivity. Finally, there are two symptoms in the interpersonal realm of BPD (with a sector score ranging from 0 to 8): intense, unstable relationships and frantic efforts of avoid abandonment. The four sector scores sum to provide a total score of borderline psychopathology. This score ranges from 0 to 36.

Zanarini, M. C. (2003). ZANarini Rating Scale For Borderline Personality Disorder (ZAN:BPD): A Continuous Measure of DSM-IV Borderline Psychopathology. *Journal of Personality Disorders*, 17(3), 233-242.

### **DEQ: Depressive Experiences Questionnaire**

Based on a review of the clinical literature, ISO statements were constructed that were not direct symptomatic expressions of depression but rather reflected experiences frequently reported by depressed patients. From this basic list, 66 items were selected by several judges because the items represented a relatively broad range of phenomenological experiences associated with depression without commitment to any particular theoretical formulation. Selected items included such issues as a distorted or depreciated sense of self and others, dependency, helplessness egocentricity, fear of loss, ambivalence, difficulty in dealing with anger, self-blame, guilt, loss of autonomy, and distortions in family relations. Items were presented in both negative and positive directions. Subjects are asked to rate items on a 7-point scale from strongly disagree (1) to strongly agree (7).

Blatt, S. J., D'Afflitti, J. P. and Quinlan, D. M. (1976). Experiences of Depression in Normal Young Adults. *Journal of Abnormal Psychology*, 85, 383-389.

There is an adolescent version of this questionnaire:

S.J. Blatt, S.E. Schaffer, S.A. Bers and D.M. Quinlan. (1990). Adolescent Depressive Experiences Questionnaire. Unpublished research manual, Yale University, New Haven, CT (1990).

S.J. Blatt, C.E. Schaffer, S.A. Bers and D.M. Quinlan, (1992). Psychometric properties of the depressive experiences questionnaire for adolescents, *Journal of Personality Assessment* **59** (1992), pp. 82–98.

There is a shorter 20-item version for adolescents:

Fichman, L., Koestner, R. & Zuroff, D. C. (1994). Depressive styles in adolescents: Assessment, relation to social functioning, and developmental trends. *Journal of Youth and Adolescents*, 23, 315-330.

### **APQ: Alabama Parenting Questionnaire**

There is a parent and child version of this questionnaire. There are 42 questions rated from 1-5 (never, almost never, sometimes, often, always). Some questions ask about both parents whereas others ask about the mother and father separately.

The APQ measures five dimensions of parenting that are relevant to the etiology and treatment of child externalizing problems: (1) positive involvement with children, (2) supervision and monitoring, (3) use of positive discipline techniques, (4) consistency in the use of such discipline and (5) use of corporal punishment. The APQ has good psychometric properties including criterion validity in differentiating clinical and nonclinical groups (Dadds, Maujean, & Fraser, 2003; Frick, Christian, & Wooton, 1999; Shelton et al., 1996). Frick et al. (1999) reported a mean  $r^2$  across its five scales of 0.24 for predicting child symptoms of ODD and CD.

There is also a short 9-item version of this questionnaire.

Frick, P. J. (1991). Alabama parenting questionnaire. University of Alabama: Author.  
Elgar, F. J., Waschbusch, D. A., Dadds, M. R. & Sigvaldason, N. (2007). Development and Validation of a Short Form of the Alabama Parenting Questionnaire  
*Journal of Child and Family Studies*, 16, 243–259.

### **DES-IV: Differential Emotion Scale-IV**

The DES was initially developed by Izard (1972) as a self-report measure of the fundamental emotions: interest, joy, sadness, anger, disgust, contempt, fear, shyness, guilt, and surprise. The original scale was later revised (Izard, Dougherty, Bloxom, & Kotsch, 1974) (DES-HI) and factor analyzed with children as young as 10 by Kotsch, Gerbing, and Schwartz (1982). The DES-IV is identical to the DES-III, except for the addition of two experimental scales: a shame scale and a self-directed hostility scale. The DES-IV instructs children to indicate how often they felt particular emotions during the past week.

There are 36 items. The participants are asked to indicate how often they feel the items in their daily life. It is rated on a 5-point frequency scale (1 = rarely or never, 2 = hardly ever, 3 = sometimes, 4 = often, and 5 = very often).

Blumberg, S. H. & Izard, C. E. (1986). Discriminating Patterns of Emotions in 10- and 11-Year-Old Children's Anxiety and Depression. *Journal of Personality and Social Psychology*, 51, 852-857.

### **NEO-FFI: NEO-Five Factor Inventory**

The NEO-PI is intended to offer both a global portrait of the individual's personality and more detailed information on specific facets of the broad domains.

The NEO-PI was developed over the past 15 years as a measure of the five-factor model: Neuroticism (N) vs. Emotional Stability; Extraversion (E) or Surgency; Openness to Experience (O) or Intellect; Agreeableness (A) vs. Antagonism; and Conscientiousness (C).

The current 181-item version of the inventory has two forms: S for self-reports, and R for observer ratings, with parallel items phrased in first- and third-person. Items are answered along a 5-point Likert scale from strongly disagree to strongly agree, and scales are balanced to control for the effects of acquiescence.

There is a short, 60-item version (the NEO Five Factor Inventory, or NEO-FFI) that gives scores for the five domains only and may be useful when time for assessment is limited. The NEO-FFI scales show correlations of .75 to .89 with the NEO-PI validimax factors. Internal consistency values range from .74 to .89. It contains 70 items and takes approximately 15 minutes to complete.

Costa, P. T., Jr & McCrae, R. R. (1992). Normal Personality Assessment in Clinical Practice: The NEO Personality Inventory, *Psychological Assessment*, 4, 5-13.

Costa, P. T., Jr & McCrae, R. R. (1985). The NEO Personality Inventory manual. Odessa, FL: Psychological Assessment Resources.

### **WAI-S: Working Alliance Inventory-Short form**

The Working Alliance Inventory (Horvath & Greenberg, 1989; WAI) is a 36-item questionnaire that can be administered to both clients and therapists. Each item is responded to using a 7-point format, where 1=never and 7=always. It yields three 12-item, summed subscale scores (Task, Bond, and Goal) as well as one overall score. Horvath and Greenberg (1986) demonstrated adequate reliability for the WAI. Internal consistency estimates of alpha were .93 for the overall client score (with subscale alphas of .85 to .88) and .87 for the overall therapist score (with subscale alphas of .68 to .87). Content validity has been supported through both rational (expert raters agreed that the items reflect the three constructs) and empirical (multitrait-multimethod analyses) methods.

The short version (Tracey & Kokotovic, 1989) has 12 items, with four items per subscale.

Horvath, A. O. & Greenberg, L. S. (1989). Development and validation of the working alliance inventory. *Journal of Counselling Psychology*, 36, 223-233.

Tracey, T. J. & Kokotovic, A. M. (1989). Factor structure of the working alliance inventory. *Journal of Consulting and Clinical Psychology*, 1, 207-210.

### **CA-SUS: Child and Adolescent Service Use Schedule**

A questionnaire used to assess service use adapted specifically for use with this study. This includes information about from the young person (or the parent/carer for young children) about their accommodation, education, use of hospital services, contact with professionals and voluntary organisations, psychotropic medication and any time spent in custody. Parents are asked how they travelled to the treatment, their employment and how much time they took off work.

### **SCL:90: Symptom Checklist 90**

The SCL-90 is intended to measure symptom intensity on nine different subscales. The SCL-90 normally requires between 12 and 20 minutes to complete (Derogatis 2000).

Each item of the questionnaire is rated by the patient on a five-point scale of distress from 0 (none) to 4 (extreme). The SCL-90 consists of nine primary symptom dimensions (somatization, obsessive compulsive, depression, anxiety, hostility, phobic anxiety, paranoid ideation, psychoticism), and three global indices: global severity index, positive symptom distress index, positive symptom total.

The instrument's three global indices of distress are:

Global Severity Index (GSI), Positive Symptom Distress Index (PSDI), Positive Symptom Total (PST)

Derogatis LR, Lipman RS, Covi L (1973): SCL-90: An outpatient psychiatric rating scale—preliminary report. *Psychopharmacol Bull* 9:13–28.

There is also a revised version of this assessment.

Derogatis, L. R. SCL-90-R: Symptom checklist-90-R: administration, scoring & procedures manual. Pearson Assessments

<http://www.pearsonassessments.com/scl90.aspx>

### **Life Events**

The parents of participants are asked to rate 11 major life events during the preceding 12-month period which may have affected their child. These may include changes in school, deaths, household disasters, friendship difficulties and illnesses. Respondents are asked how they felt about the event on a scale of 1= very pleasant/happy, 2= quite pleasant/happy, 3= neither pleasant nor unpleasant, 4= quite unpleasant/sad/painful, 5= very unpleasant/sad/painful. Events which are rated either 4 or 5 and as being upsetting for more than two weeks are then categorised into disappointments, danger to self, a danger to another or as an event involving loss. The latter is inclusive of deaths (human and pets) along with loss of contact with a significant person in the child's life.

### **CAMEEI: The Cambridge Early Experience Interview**

“The Cambridge Early Experience Interview” (Goodyer et al, 2009) is a semi structured interview used to gain insight into important life events and family circumstances during a child's up-bringing. It was developed to be non-judgmental and user friendly and is conducted with the child's primary care-giver. Parents may be asked prior to the interview to fill in a timeline, to help them and the interviewer focus on important events (positive and negative) and their chronology in the child's life. These timelines can be the starting point for

the interview. CAMEEI involves detailing life experiences, the child's age at occurrence, the duration and an interviewer's assessment on their practical impact on the family's everyday life.

Incidences may be coded within time periods, e.g. 1= age 0-5, preschool years; 2= age 5-11, primary years and 3= 11-14, secondary years. Therefore, the CAMEEI offers not only a detailed picture of important events in the child's life and their impact, but also describes social risk from birth through to adolescence. Inter-rater reliability with 48 mothers on a number of core items has been highly satisfactory (kappa ranges from 0.7-0.9). The time taken to complete the CAMEEI varies, but can take from hour to an hour and a half (including the MIDI parent psychiatric assessment).

### **HCAM: Hampstead Child Adaptation Measure**

This is a measure designed to assess the general adjustment of a child. It is a manualisation of the CGAS instrument developed by Shaffer and colleagues (1983). Like the CGAS, it is a 100-point rating scale with descriptions of the overall level of functioning expected within each 10-point interval. Scores above 70 are regarded as falling within the normal range. A score below 30 indicates severe impairment, probably requiring hospitalization. Children rated at 55 or below would be clearly in need of some form of therapeutic help, and often special educational provision.

The manual was influenced primarily by Luborsky's Health Sickness Rating Scale (Luborsky, 1962) and Anna Freud's concept of developmental lines (A Freud, 1963). Raters are required to make judgements on 15 operationalized parameters of the child's adjustment and emotional development relative to his age, physical condition and social circumstances.

Manualisation has considerably improved the reliability of ratings of adaptation (0.85 against 0.75 for the CGAS scale, with this chart material). Change scores (the difference between the start and end of treatment) were highly correlated between raters on both the CGAS and the HCAM but HCAM seems to be a more reliable indicator of change as well as of general level of adaptation.

Target, M., & Fonagy, P. (1992). Raters' manual for the Hampstead Child Adaptation Measure (HACAM), London, UCL.

### **CGI: Clinical Global Impressions Scale**

The CGI is a 3-item observer-rated scale that measures illness severity (CGIS), global improvement or change (CGIC) and therapeutic response. The illness severity and improvement sections of the instrument are used more frequently than the therapeutic response section in both clinical and research settings. The Early Clinical Drug Evaluation Program (ECDEU) version of the CGI is the most widely used format, and asks that the clinician rate the patient relative to their past experience with other patients with the same diagnosis, with or without collateral information. Several alternative versions of the CGI have been developed, however, such as the FDA Clinicians' Interview-Based Impression of Change (CIBIC), which uses only information collected during the interview, not collateral. The CGI has proved to be a robust measure of efficacy in many clinical drug trials, and is easy and quick to administer, provided that the clinician knows the patient well.

The CGI is rated on a 7-point scale, with the severity of illness scale using a range of responses from 1 (normal) through to 7 (amongst the most severely ill patients).

CGI-C scores range from 1 (very much improved) through to 7 (very much worse). Treatment response ratings should take account of both therapeutic efficacy and treatment-related adverse events and range from 0 (marked improvement and no side-effects) and 4 (unchanged or worse and side-effects outweigh the therapeutic effects). Each component of the CGI is rated separately; the instrument does not yield a global score.

Guy W, editor. ECDEU Assessment Manual for Psychopharmacology. 1976. Rockville, MD, U.S. Department of Health, Education, and Welfare

### **CDRS: Children's Depression Rating Scale**

The Children's Depression Rating Scale (CDRS) is a 16-item measure used to determine the severity of depression in children 6-12 years of age. Items are measured on 3-, 4-, 5-, and 6-point scales. The CDRS is derived from the Hamilton Rating Scale for Depression (HAM-D); a score of 15 on the CDRS is equivalent to a score of 0 on the HAM-D. Assessment information is based on parent, child and schoolteacher interviews.

*There is a revised version (CDRS-R) that covers 17 symptom areas of depression and used to diagnose depression and can be repeated to measure response to treatments. CDRS-R total scores range from 17 to 113 and Fourteen of the 17 items are rated on a scale from 1 to 7, with an item score of 3 suggestive of mild, 4 or 5 moderate, and 6 or 7 severe symptoms. The other 3 items are rated on a scale from 1 to 5. Both children and their parents provide input into the first 14 items of the scale. A child's nonverbal behaviour is rated by the observer for items 15 through 17. A CDRS-R  $\geq$  40 suggests the presence of depressive disorder.*

Poznanski, E., Cook, S. & Carroll, B. (1979). A depression rating scale for children. *Pediatrics*, 64, 442-450.

Poznanski EO, Freeman LN, Mokros HB: **Children's Depression Rating Scale-Revised**. *Psychopharmacol Bull* 1985, **21**:979-989.

Poznanski EO, Mokros HB: *Children's Depression Rating Scale, Revised (CDRS-R) Manual*. Los Angeles, Calif: Western Psychological Services; 1995.



### APPENDIX 3

#### SPSS Output: Scree plot (Exploratory Factor Analysis)

