Psychological Medicine (2015), **45**, 2237–2251. © Cambridge University Press 2015
doi:10.1017/S0033291715000318

REVIEW ARTICLE

Clinical and psychosocial outcomes of borderline personality disorder in childhood and adolescence: a systematic review

C. Winsper[1]*, S. Marwaha[1], S. T. Lereya[2], A. Thompson[1], J. Eyden[2] and S. P. Singh[1]

[1] *Division of Mental Health and Wellbeing, Warwick Medical School, University of Warwick, Coventry CV4 7AL, UK*
[2] *Department of Psychology, University of Warwick, Coventry CV4 7AL, UK*

Background. While there is a growing body of research on borderline personality disorder (BPD) in children and adolescents, controversy remains regarding the validity and diagnosis of the disorder prior to adulthood.

Method. MEDLINE, EMBASE, Psych INFO and PubMed databases were systematically searched for articles pertaining to the clinical and psychosocial outcomes (i.e. predictive validity) of BPD first diagnosed in childhood or adolescence (i.e. prior to 19 years of age). All primary empirical studies were included in the review. A narrative synthesis of the data was completed.

Results. A total of 8200 abstracts were screened. Out of 214 full-text articles, 18 satisfied the predetermined inclusion criteria. Quality assessment indicated that most studies had high risk of bias in at least one study domain. Consistent with the adult literature, the diagnostic stability of BPD prior to the age of 19 years was low to moderate, and mean-level and rank-order stability, moderate to high. Individuals with BPD symptoms in childhood or adolescence had significant social, educational, work and financial impairment in later life.

Conclusions. Studies indicate that borderline pathology prior to the age of 19 years is predictive of long-term deficits in functioning, and that a considerable proportion of individuals continue to manifest borderline symptoms up to 20 years later. These findings provide some support for the clinical utility of the BPD phenotype in younger populations, and suggest that an early intervention approach may be warranted. Further prospective studies are needed to delineate risk (and protective) factors pertinent to the chronicity of BPD across the lifespan.

Received 27 June 2014; Revised 7 November 2014; Accepted 29 January 2015; First published online 24 March 2015

Key words: Adolescence, borderline personality disorder, childhood, predictive validity, systematic reviews, youth.

Introduction

Borderline personality disorder (BPD) is a serious mental illness characterized by behavioural and emotional dysregulation, marked impairment in psychosocial functioning and high risk of mortality (Black *et al.* 2004; Leichsenring *et al.* 2011). BPD is associated with a range of long-term negative sequelae, including relationship dysfunction (Daley *et al.* 2000), unemployment (Skodol *et al.* 2002), high levels of treatment utilization (Bender *et al.* 2001) and imprisonment (Black *et al.* 2007). Consequently, BPD can have a devastating impact on individuals, their families, and health and social services.

BPD diagnosis in childhood and adolescence (i.e. prior to the age of 19 years) remains controversial (Chanen & McCutcheon, 2008; Miller *et al.* 2008). Recent reports indicate that clinicians are reluctant to diagnose BPD in younger individuals (Griffiths, 2011; Laurenssen *et al.* 2013). Nevertheless, BPD is unlikely to appear *de novo* in early adulthood, but may be considered as the continuation of precursor symptoms that first emerge during childhood or early adolescence (Crowell *et al.* 2009; Winsper *et al.* 2012). Importantly, the early identification of BPD symptoms may help shed light on aetiological processes (Crowell *et al.* 2009), inform early intervention programmes (Chanen *et al.* 2008*b*) and ensure that young people with personality pathology receive appropriate treatment (Paris, 2013).

Predictive validity reflects the degree to which BPD in childhood or adolescence transitions into adult BPD, and is prognostic of future impairment (Van Os *et al.* 2009). Ascertaining the predictive validity of BPD by considering both diagnostic and psychosocial outcomes is important in view of concerns regarding the lack of diagnostic stability during this developmental phase (Meijer *et al.* 1998). Furthermore, identifying influences on the stability of BPD across early development may help highlight important risk and protective factors, deepening our understanding of the continuity and discontinuity of BPD trajectories across the lifespan.

* Address for correspondence: C. Winsper, Ph.D., Division of Mental Health and Wellbeing, Warwick Medical School, University of Warwick, Coventry CV4 7AL, UK
(Email: catherine.winsper@warwick.ac.uk)

Previous narrative reviews have examined aspects of the predictive validity of adolescent BPD as part of a broader evaluation of the construct. Bondurant *et al.* (2004) reported that the diagnostic stability of adolescent BPD was relatively low, though very few studies were identified (Bernstein *et al.* 1993; Garnet *et al.* 1994; Mattanah *et al.* 1995). Miller *et al.* (2008) reported low to moderate diagnostic and dimensional stability, though again only a limited number of studies were available (Bernstein *et al.* 1993; Garnet *et al.* 1994; Meijer *et al.* 1998; Grilo *et al.* 2001; Chanen *et al.* 2004). Chanen *et al.* (2008*b*) considered BPD in youth (i.e. aged 15–24 years) and reported that mean-level BPD traits were moderately stable, though the authors highlighted the lack of BPD specific data. They also presented evidence from a small number of studies indicating that young people with BPD may experience poorer outcomes (i.e. increased risk of Axis I disorders, social impairment).

Since these reviews, a number of empirical studies have been published. As far as we are aware, however, there are no extant reviews examining this topic using systematic review procedures. Due to the contentious nature of BPD diagnosis in younger individuals, systematic reviews are now required to provide rigorous evidence to inform clinical policy and practice (Hammersley, 2001). The main aim of the current review was to examine the predictive validity of BPD in childhood and adolescence. There were four research questions:

(1) Is BPD diagnosis stable in this age group?
(2) Do BPD symptoms demonstrate mean-level stability in this age group (i.e. do individual BPD scores remain stable over time)?
(3) Do BPD symptoms demonstrate rank-order stability in this age group (i.e. do individuals retain their relative placement in the group)?
(4) Does BPD pathology in this age group predict subsequent problems in diverse spheres of functioning?

Method

Prior to formulating the protocol, C.W. and J.E. conducted a pilot search to ensure that a systematic review in the area had not been published (Sayers, 2007). We used PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines (Moher *et al.* 2009) as a framework for the review.

Search strategy

We searched MEDLINE, EMBASE, Psych INFO and PubMed databases to identify studies reporting on BPD in children and adolescents published between 1980 and January 2014. We chose 1980 as the earliest date for inclusion to parallel when BPD was first conceptualized in the Diagnostic and Statistical Manual (APA, 1980). The search terms (borderline* OR 'emotionally unstable personality disorder' OR BPD) AND (adolescen* OR child* OR young* OR youth* OR teen* OR student*) were entered. Reference lists of included studies were inspected for relevant titles. We also examined reference lists of relevant narrative reviews as a cross-check (Bondurant *et al.* 2004; Chanen & McCutcheon, 2008; Miller *et al.* 2008).

Inclusion and exclusion criteria

Inclusion criteria were:

(1) Primary research published in a peer-reviewed journal;
(2) Participants were under 19 years of age at index assessment. If all participants were at the extreme end of the age range, i.e. 18 years old, the study was excluded. Studies encompassing an age range predominantly comprising under-18-year-olds were included, e.g. 9–19 years (19 years was the maximum);
(3) The study was published in English;
(4) There was information on clinical or psychosocial outcomes;
(5) Studies with any assessment of BPD were included (we placed no restrictions regarding the methods used to diagnose BPD as we anticipated a paucity of available studies).

Studies were excluded if:

(1) BPD was not the exclusive focus of the study (e.g. associations pertained to all cluster B personality disorders);
(2) The sample was primarily defined in terms of another psychopathology (e.g. all participants were self-harmers, only some of whom had BPD);
(3) They were treatment trials.

Screening procedure

If a title appeared potentially eligible but no abstract was available, the full-text article was retrieved. C.W. and S.T.L. independently scanned 100% of the abstracts to identify articles for full-text retrieval. Full-text articles were read by C.W. to assess for inclusion in the review. S.M. independently reviewed 50% of the full-text articles for inclusion as a reliability check.

Data collection and quality assessment

A data extraction form was developed prior to review. It included author details, country of study, sample characteristics, study design, BPD assessment tool, and information on outcomes. It also included a quality assessment tool based on Cochrane Collaboration

https://doi.org/10.1017/S0033291715000318 Published online by Cambridge University Press

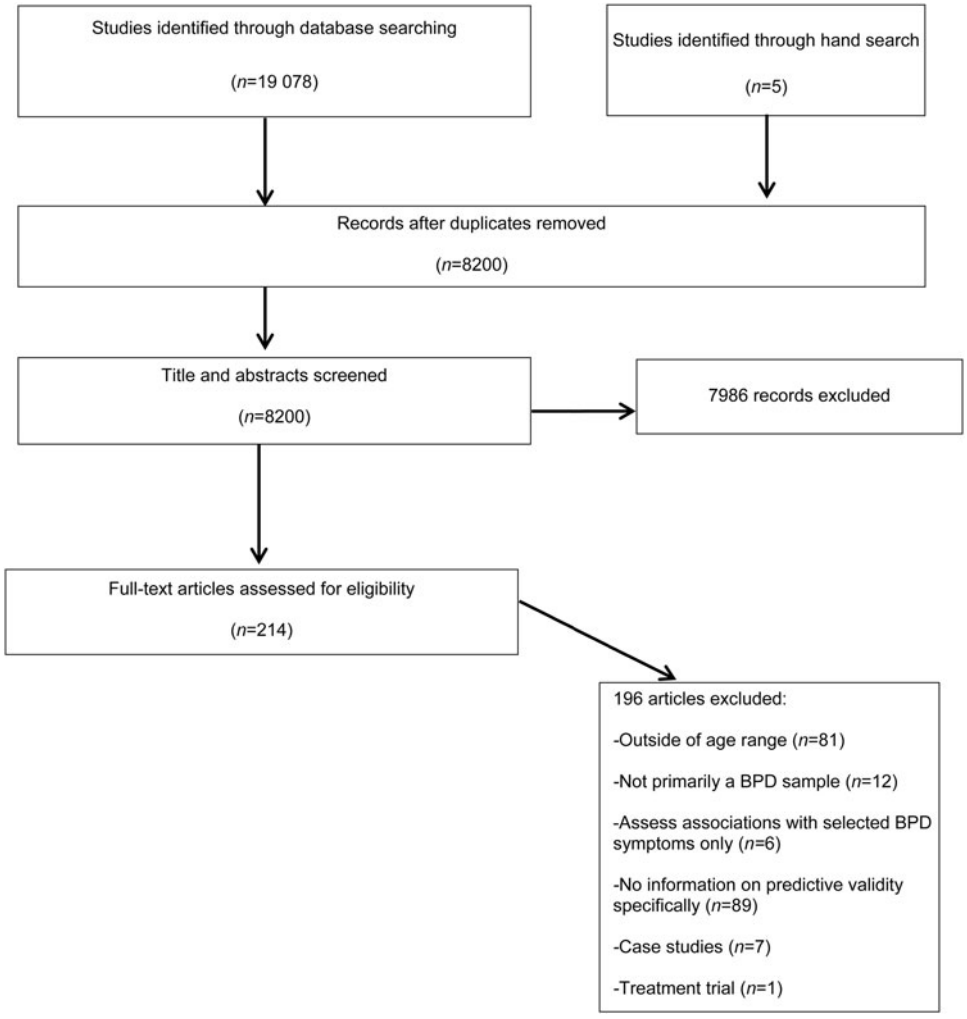

Fig. 1. Flowchart outlining the search and selection strategy. BPD, Borderline personality disorder.

guidelines (Higgins & Altman, 2008). This tool is designed to rate the risk of bias (i.e. systematic error) in each study. We assessed: the quality domains of selection bias (selection of sample, blinding of index assessment); performance bias (events during the study potentially impacting on predictive validity); attrition bias; detection bias (blinding of outcome assessments); and reporting bias (indication of selective reporting).

Data synthesis

Data were not suitable for quantitative synthesis, and thus are qualitatively synthesized within the review.

Results

Of the 8200 (database search = 8195; hand search = 5) abstracts scanned, 214 were selected for full-text retrieval. There was a high level of agreement between raters on articles to be selected for full-text retrieval (>80%). The authors met to discuss discrepancies, which were largely due to uncertainty regarding sample characteristics or age. If there was doubt over whether an abstract should be included for full-text retrieval, the decision was made to include. Of the 214 full-text articles, 18 were identified providing outcome data (Fig. 1). The 50% reliability check indicated a high level of agreement between raters on articles to be included in the review (>80%). The most common reasons for exclusion were: the sample was over 18 years of age; BPD was conflated with another mental disorder; there were no data on clinical or psychosocial outcomes. Studies comprised a mix of clinical and non-clinical populations, and ranged in duration from 1 to 20 years (Table 1).

Quality assessment

Quality assessment indicated that most studies had a high risk of bias (systematic error) in one or more

https://doi.org/10.1017/S0033291715000318 Published online by Cambridge University Press

Table 1. *Details of studies reporting on clinical and psychosocial outcomes of BPD in childhood and adolescence*

First author (year)	Country	Percentage female	Baseline: *n*, age, proportion BPD	Sample frame (control group)	Study design (duration)	BPD assessment (cut-point for diagnosis)	Outcomes
Bernstein (1993)	USA	49.8	733, 9- to 19-year-olds	Community (N.A.)	Prospective (2 years)	Children in the Community-Self Report (1 S.D. > mean/2 S.D. > mean)	Stability of BPD diagnosis
Biskin (2011)	Canada	100	97, mean age = 15.1 years (49 BPD, 48 non-BPD)	Clinical (disruptive behaviour disorders)	Prospective (4 years)	Diagnostic Interview for Borderlines (⩾6)	BPD diagnosis in adulthood; employment; social functioning; treatment utilization
Bornovalova (2009)	USA	100	1118, 14- to 17-year-olds, 339 MZ, 218 DZ twins	Community (N.A.)	Prospective (10 years)	Multidimensional Personality Questionnaire (continuous)	Mean-level and rank-order stability of BPD symptoms
Bornovalova (2013)	USA	100	1280, 14- to 18-year-olds, 390 MZ, 250 DZ twins	Community (N.A.)	Prospective (4 years)	Minnesota Borderline Personality Disorder Scale (continuous)	Substance abuse
Chanen (2004)	Australia	63	101, 15- to 18-year-olds	Clinical (out-patients)	Prospective (2 years)	Structured Clinical Interview-II (>5, continuous)	Categorical and dimensional stability of BPD symptoms
Chen (2004)	USA	52	200, 16-year-olds	Community (N.A.)	Longitudinal (10 years)	Children in the Community-Self Report (continuous)	Partner conflict
Cohen (2007)	USA	50	749, mean age = 13.7 years	Community (N.A.)	Prospective (9 years)	Children in the Community-Self Report (continuous)	Substance use disorder
Crick (2005)	USA	54	400, 9–12 years	Community (N.A.)	Prospective (1 year)	Borderline Personality Features Scale for Children (continuous)	Mean-level stability of BPD symptoms
Garnet (1994)	USA	52	21, 15- to 19-year-olds (all BPD at baseline)	Clinical	Prospective (2 years)	Personality Disorder Examination (five or more symptoms)	Stability of BPD diagnosis
Grilo (2001)	USA	48	60, 15–19 years	Clinical	Prospective (2 years)	Personality Disorder Examination (continuous)	Stability of BPD symptoms
Jovev (2014)	Australia	51	245, 11–13 years	Community (% of large sample from schools)	Prospective (2 years)	Children in the Community-Self Report (continuous)	Stability of BPD symptoms
Lofgren (1991)	USA	26	32, 6- to 10-year-olds (all BPD at baseline)	Clinical (no control group)	Prospective (10–20 years)	'Borderline'[a]criteria delineated in Bemporad *et al.* (1982)	Axis I and Axis II disorders; education; employment
Mattanah (1995)	USA	44	70, 12–18 years (31 BPD at baseline)	Clinical (in-patients)	Prospective (2 years)	Personality Disorder Examination	Stability of BPD disorder and symptoms
Meijer (1998)	Netherlands	50	54, mean age = 15.2 years (18 BPD, 36 non-BPD)	Clinical (in-patients)	Prospective (3.3 years)	Diagnostic Interview for Borderlines (⩾7)	Stability of BPD disorder and symptoms

Stepp (2014)	USA	100	2212, 14-year-olds	Community (oversampled for low income)	Prospective (4 years)	IPDE-BOR; for details, see Loranger *et al.* (1994)	Mean-level stability of BPD symptoms
Wenning (1990)	USA	47.4	57, 8-year-olds (all BPD at baseline)	Clinical (no control group)	Follow-up study (10 years)	'Borderline'[b] criteria delineated in Vela *et al.* (1983)	Personality disorder diagnosis; psychiatric symptoms
Winograd (2008)	USA	50	748, 9- to 18-year-olds	Community (N.A.)	Prospective (20 years)	Children in the Community-Self Report (continuous)	BPD symptoms/diagnosis; social function; education; employment; service utilization
Zelkowitz (2007)	Canada	19	94, 7- to 12-year-olds (41 BPD, 53 non-BPD)	Clinical (in-patients)	Prospective (5 years)	Diagnostic Interview for Borderlines (⩾7)	BPD diagnosis; psychopathology; functioning

BPD, Borderline personality disorder; N.A., not applicable; MZ, monozygotic; DZ, dizygotic; IPDE-BOR, International Personality Disorders Examination, Borderline Scale.
[a] 'Borderline' diagnosis includes sections on: fluctuation in functioning; anxiety; thought content and processes; relationships with others; lack of control; associated symptoms.
[b] 'Borderline' diagnosis includes sections on: disturbed interpersonal relationships; disturbed sense of reality; excessive intense anxiety; excessive and severe impulsive behaviour; neurotic-like symptoms; uneven distorted development.

domains, i.e. selection, performance, attrition, detection or reporting (Table 2). This suggests that aspects of the study design could have led to an under- or overestimation of effects, rather than implying that the studies are of low quality (for further explanation, see Discussion). In general, clinical studies had a high risk of bias in more domains than non-clinical studies. All but four studies (Bernstein *et al.* 1993; Chen *et al.* 2004; Cohen *et al.* 2007; Winograd *et al.* 2008) were of high risk in sample selection bias. Three studies were of high risk in baseline assessment bias (Meijer *et al.* 1998; Biskin *et al.* 2011; Stepp *et al.* 2014). Nine studies (Wenning, 1990; Lofgren *et al.* 1991; Garnet *et al.* 1994; Mattanah *et al.* 1995; Meijer *et al.* 1998; Grilo *et al.* 2001; Chanen *et al.* 2004; Zelkowitz *et al.* 2007; Biskin *et al.* 2011) were of high risk in performance bias. All studies excepting Bernstein *et al.* (1993) and Chanen *et al.* (2004) were of high risk in attrition bias. Two studies were of high risk (Wenning, 1990; Biskin *et al.* 2011) and seven unclear risk (Crick *et al.* 2005; Cohen *et al.* 2007; Winograd *et al.* 2008; Bornovalova *et al.* 2009, 2013; Jovev *et al.* 2014; Stepp *et al.* 2014) in detection bias. One study was of high risk in reporting bias (Wenning, 1990).

The stability of BPD in childhood and adolescence

Diagnostic stability

Ten studies examined the diagnostic stability of BPD. Eight utilized clinical (Wenning, 1990; Lofgren *et al.* 1991; Garnet *et al.* 1994; Mattanah *et al.* 1995; Meijer *et al.* 1998; Chanen *et al.* 2004; Zelkowitz *et al.* 2007; Biskin *et al.* 2011) and two non-clinical populations (Bernstein *et al.* 1993; Winograd *et al.* 2008). Overall, the diagnostic stability across studies (from 2 to 20 years) ranged from 14 to 40%.

Clinical populations

Wenning (1990) followed up children retrospectively diagnosed with borderline personality over a 10-year period. Of the original 57 children, 28 (20 'angry–impulsive', eight 'borderline psychotic') were identified for follow-up assessment. At follow-up 90% of children with 'angry–impulsive' borderline disorder, and 75% with 'borderline psychotic' disorder received a personality disorder diagnosis. Of the 'angry–impulsive' types, the most common (75%) diagnosis was BPD and/or antisocial personality disorder (ASPD).

Over a period of 10–20 years, Lofgren *et al.* (1991) followed-up 6- to 10-year-olds diagnosed as 'borderline' according to criteria (see Table 1) defined by Bemporad *et al.* (1982). Of an original 32, 19 were located in adolescence or adulthood for repeat assessment. Of these, three were diagnosed with BPD

https://doi.org/10.1017/S0033291715000318 Published online by Cambridge University Press

Table 2. *Quality assessment of studies included in the review*

First author (year)	Selection bias: i. sequence; ii. baseline assessment	Performance bias	Attrition bias	Detection bias	Reporting bias
Bernstein (1993)	i. Part of children in community sample – randomly selected (low risk) ii. Blind assessment by interviewers (low risk)	No indication as community sample (low risk)	94% retained for follow-up assessment (low risk)	Interviewers assessed the children blindly (low risk)	All pre-specified assessments reported (low risk)
Biskin (2011)	i. All patients referred to a treatment programme for adolescent girls with BPD; comparison sample – patients assessed by same clinic during same time period (high risk) ii. No assessment concealment (high risk)	BPD group received specialized treatment could have made an impact on outcome (high risk)	63% of BPD patients retained, 16/48 control patient retained (high risk)	Diagnosing clinician involved in BPD assessments at both time-points (high risk) Other outcomes assessed by same clinician (high risk)	All pre-specified assessments (including non-significant results) reported (low risk)
Bornovalova (2009)	i. Twin sample, thus selection bias (high risk) ii. Assessment concealment unclear though self-report (unclear risk)	No indication as community sample (low risk)	Attrition rates 5–10% for any given assessment (high risk)	Questionnaire but blinding methods not specified (unclear risk)	All pre-specified assessments reported (low risk)
Bornovalova (2013)	i. Twin sample, thus selection bias (high risk) ii. Assessment concealment unclear though self-report (unclear risk)	Some participants received varying levels of treatment could have made an impact on outcome (unclear risk)	Not reported in this study, though reported in previous study with same sample (high risk)	Questionnaire but blinding methods not specified (unclear risk)	All pre-specified assessments reported (low risk)
Chanen (2004)	i. 147 (101 agreed) patients selected from 418 acute referrals (high risk) ii. Assessment concealment (low risk)	Variations in in-patient care across sample could have made an impact on outcome (high risk)	96% retained for follow-up assessment (low risk)	Diagnosing interviewers blind to baseline assessment (low risk)	All pre-specified assessments reported (low risk)
Chen (2004)	i. Part of children in community sample – randomly selected (low risk) ii. Assessment concealment (low risk)	No indication as community sample (low risk)	Follow-up assessments depended on willingness to participate in lengthy interviews (high risk)	Narrative interviews carried out by assessors blind to previous data (low risk)	All pre-specified assessments reported (low risk)
Cohen (2007)	i. Part of children in community sample – randomly selected (low risk) ii. Assessment concealment (low risk)	No indication as community sample (low risk)	85% retained for follow-up assessment (high risk)	Questionnaire but blinding methods not specified (unclear risk)	All pre-specified assessments reported (low risk)

Crick (2005)	i. Subsample of ongoing longitudinal study (high risk) ii. Assessment concealment unclear though self-report (unclear risk)	No indication as community sample (low risk)	57% of sample retained (high risk)	Questionnaire but blinding methods not specified (unclear risk)	All pre-specified assessments reported (low risk)
Garnet (1994)	i. Subjects were a small subset of patients from the Yale Psychiatric Institute study (high risk) ii. Assessment concealment not specified (unclear risk)	Sample receiving in-patient treatment (high risk)	Not reported in this study, though small subset of larger outcome study suggests high attrition (high risk)	Diagnosing interviewers blind to baseline assessment (low risk)	All pre-specified assessments reported (low risk)
Grilo (2001)	i. Subjects were a subset of patients from the Yale Psychiatric Institute study (high risk) ii. Interviewers functioned independently to clinical team (low risk)	Sample receiving in-patient treatment (high risk)	36.36% retained for follow-up assessment (high risk)	Diagnosing interviewers blind to baseline assessment (low risk)	All pre-specified assessments reported (low risk)
Jovev (2014)	i. Children overselected for extreme temperament traits (of which only 59% consented to participate) (high risk) ii. Blinding not specified (unclear risk)	No indication as community sample (low risk)	83.67% retained for follow-up assessment (high risk)	Questionnaire but blinding methods not specified (unclear risk)	All pre-specified assessments (including non-significant results) reported (low risk)
Lofgren (1991)	i. Selected from a large sample of hospitalized children – male bias (high risk) ii. Three independent ratings (low risk)	Receiving hospital treatment (high risk)	59.38% retained for follow-up assessment (high risk)	Blind assessment at follow-up (low risk)	All pre-specified assessments reported (low risk)
Mattanah (1995)	i. Hospitalized adolescents from the Yale Psychiatric Institute (high risk) ii. Assessment concealment not specified (unclear risk)	Sample receiving in-patient treatment (high risk)	39.39% retained for follow-up assessment (high risk)	Blind assessment at follow-up (low risk)	All pre-specified assessments reported (low risk)
Meijer (1998)	i. Consecutive admissions to two (long- and short-stay) in-patient facilities (high risk) ii. Author conducted baseline assessments (high risk)	Sample receiving in-patient treatment (high risk)	66.67% retained for follow-up assessment (high risk)	Blind assessment at follow-up (low risk)	All pre-specified assessments reported (low risk)
Stepp (2014)	i. Pittsburgh Girls Study oversampled for low-income neighbourhoods (high risk) ii. Assessment concealment of interviewers unclear (high risk)	No indication as community sample (low risk)	90.25% retained (high risk)	Questionnaire but blinding methods not specified (unclear risk)	All pre-specified assessments reported (low risk)
Wenning (1990)	i. Children selected from a residential treatment service if clinicians believed likely to be borderline (high risk) ii. Blind corroboration of BPD diagnosis (low risk)	Receiving hospital treatment (high risk)	49.12% retained for follow-up assessment (high risk)	Author involved in baseline and follow-up interviews (high risk)	Some assessments not reported on, e.g. social functioning, school performance (high risk)

Table 2 (*cont.*)

First author (year)	Selection bias: i. sequence; ii. baseline assessment	Performance bias	Attrition bias	Detection bias	Reporting bias
Winograd (2008)	i. Part of children in community sample – randomly selected (low risk) ii. Questionnaire assessment but blinding methods unclear (unclear risk)	No indication as community sample (low risk)	85% retained for follow-up assessment (high risk)	Blinding not specified (unclear risk)	All pre-specified assessments (including non-significant reported results) (low risk)
Zelkowitz (2007)	i. Children referred to a day hospital – male bias (high risk) ii. Interviewer blind to other diagnoses (low risk)	Receiving treatment (high risk)	38.30% retained for follow-up (high risk)	Blind assessment at follow-up (low risk)	All pre-specified assessments reported (low risk)

BPD, Borderline personality disorder.

(16%) and 16 (84%) received an Axis II personality disorder diagnosis. Of note, many of the diagnosed personality disorders were male-typical (e.g. antisocial, schizoid), probably reflecting the male bias in the sample.

Two studies from the same research group examined the stability of BPD diagnosis in adolescent in-patients from the Yale Psychiatric Institute (Garnet *et al.* 1994; Mattanah *et al.* 1995). In the first, 33% of patients with BPD at index assessment met diagnostic criteria for BPD 2 years later (Garnet *et al.* 1994). In the second, 23% of individuals with BPD at baseline met diagnostic criteria 2 years later (Mattanah *et al.* 1995). In another hospital study, 14% of adolescents diagnosed with BPD at baseline retained the diagnosis 3 years later (Meijer *et al.* 1998). A number of the adolescents diagnosed with BPD at index hospitalization, while no longer carrying a BPD diagnosis, continued to demonstrate subclinical levels of disturbance.

Chanen *et al.* (2004) found that BPD diagnosis remained stable in 40% of adolescent out-patients diagnosed 2 years previously. The stability of global personality disorder (i.e. any personality disorder diagnosis) was 74%.

Zelkowitz *et al.* (2007) followed 59 adolescents who had received psychiatric day treatment in childhood. Of the children diagnosed with borderline pathology, 14% retained the diagnosis 5 years later. Significant group differences between borderline and non-borderline children were maintained on the interpersonal and cognitive domains of the Diagnostic Interview for Borderlines. Finally, Biskin *et al.* (2011) assessed outcomes of adolescent females who had been referred to a treatment programme for BPD. Of the young women who were diagnosed in adolescence, 35% retained the diagnosis 4 years later.

Non-clinical populations

In the first of two studies with the Children in the Community cohort, Bernstein *et al.* (1993) reported that 29% of adolescents with 'moderate' (dichotomized 1 S.D. above the mean) and 24% of adolescents with 'severe' (dichotomized 2 S.D.s above the mean) BPD symptoms retained the diagnosis after 2 years. In the second study, Winograd *et al.* (2008) reported that extreme BPD symptoms (2 S.D.s above the mean) in adolescence were associated with nine-fold increased risk of BPD 20 years later.

Mean-level stability

Seven studies examined mean-level stability of BPD symptoms. Two utilized clinical (Grilo *et al.* 2001; Chanen *et al.* 2004) and five non-clinical populations (Crick *et al.* 2005; Winograd *et al.* 2008; Bornovalova *et al.* 2009; Jovev *et al.* 2014; Stepp *et al.* 2014).

https://doi.org/10.1017/S0033291715000318 Published online by Cambridge University Press

Mean-level stability ranged from 0.16 to 0.59 over durations of 1–20 years.

Clinical populations

Chanen *et al.* (2004) reported a mean-level stability of intraclass correlation (ICC) = 0.54 in adolescent outpatients. It was indicated by *t* tests that BPD symptoms did not significantly decrease over time ($p = 0.115$). Grilo *et al.* (2001) found that mean-level stability of BPD symptoms in adolescent in-patients was ICC = 0.16. It was indicated by *t* tests that BPD symptom scores significantly decreased over time ($t = 2.23$, $p < 0.05$).

Non-clinical populations

Crick *et al.* (2005) assessed the stability of BPD symptoms over 1 year (autumn year 1; spring year 2; autumn year 2) in a sample of schoolchildren. Correlations between the three time-points ranged from 0.47 to 0.56. In another study with schoolchildren, Jovev *et al.* (2014) found that mean BPD symptoms significantly decreased over 2 years (time 1: mean = 1.67; time 2: mean = 1.30). Low effortful control (i.e. poor self-regulation), however, significantly predicted an increase in BPD symptoms ($\beta = -0.18$, $t = -2.32$, $p = 0.002$).

Stepp *et al.* (2014) examined the stability of BPD symptoms in a community sample of girls across four time-points (14, 15, 16 and 17 years). All correlations were significant ($p < 0.05$) and were higher between shorter durations: 14–15 years ($r = 0.55$); 15–16 years ($r = 0.59$); 16–17 years ($r = 0.58$); 14–16 years ($r = 0.48$); 15–17 years ($r = 0.52$); and 14–17 years ($r = 0.42$).

In a long-term community study, Winograd *et al.* (2008) found that BPD symptoms at 13.7 and 33.2 years were correlated ($r = 0.39$). The stability of BPD symptoms from mid to early adolescence was $r = 0.516$. BPD symptoms declined on average by $\beta = -0.032$ (S.E. = 0.002) per year, equating to approximately two-thirds of a standard deviation over the 20-year period. In another long-term study, Bornovalova *et al.* (2009) examined mean-level stability of BPD symptoms in a large sample of female twins over multiple assessment points (14, 17, 20 and 24 years). A decline in mean-level BPD traits was observed over the 10-year period from mid adolescence to early adulthood. There was no meaningful change from 14 years (mean = 41.26) to 17 years (mean = 40.86) of age; a moderate change from 14 to 20 years (mean = 37.2) of age; and a large change from 14 to 24 years (mean = 35.19) of age.

Rank-order stability

Two studies assessed the rank-order stability of BPD symptoms (Chanen *et al.* 2004; Bornovalova *et al.* 2009). Chanen *et al.* (2004) reported a rank-order stability of 0.54. Bornovalova *et al.* (2009) reported rank-order stability ranging from 0.53 to 0.73 across four time-points.

Other clinical and psychosocial outcomes of BPD in childhood and adolescence

Education and employment

Clinical populations

During follow-up, Lofgren *et al.* (1991) found that none of the adolescents/adults previously diagnosed as borderline in childhood was self-supporting or living independently. Only 26% were attending school or working (mostly in unskilled or semi-skilled labour). Zelkowitz *et al.* (2007) found that adolescents previously diagnosed with borderline pathology in childhood were significantly more likely than non-borderline psychiatric controls to have changed schools due to behaviour problems. Biskin *et al.* (2011) reported that women previously diagnosed with BPD in adolescence were less likely to be employed (42%) than psychiatric controls (63%). Of note, those who had remitted (20/31) were only slightly more likely to be in employment than those still carrying the BPD diagnosis (45% *v.* 36%).

Non-clinical populations

Winograd *et al.* (2008) found that educational attainment reported at 33 years of age was negatively associated with BPD symptoms in adolescence ($\beta = -0.522$; S.E. = 0.074, $p < 0.01$). Similarly, BPD symptoms in adolescence negatively predicted occupational (ranging from unskilled labour to full professional status) level ($\beta = -0.818$, S.E. = 0.176, $p < 0.01$). Findings remained significant following adjustment for Axis I disorders during early adolescence.

BPD symptoms in early adolescence also significantly predicted reliance on public assistance [odds ratio (OR) 2.90, 95% confidence interval (CI) 1.37–6.16], though the association became non-significant following adjustment for Axis I disorders in adolescence (OR 1.99, 95% CI 0.85–4.69).

Social functioning

Clinical populations

Lofgren *et al.* (1991) found that children diagnosed with borderline pathology had very poor levels of social functioning in adolescence/adulthood. Just one out of 19 subjects had married over the 20-year period. Only 26% reported satisfying relationships with their families, and even fewer (16%), with peers. Of the subjects, 47% described a complete absence of friendships or social life, while 37% reported 'only highly tumultuous relationships'. Zelkowitz *et al.* (2007) reported that

https://doi.org/10.1017/S0033291715000318 Published online by Cambridge University Press

borderline diagnosis 5–7 years previously significantly increased odds of peer problems in adolescence ($\chi^2 = 7.25$, $p < 0.01$). In contrast, Biskin *et al.* (2011) did not find a significant group difference on the Social Adjustment Scale Self Report between those formally diagnosed with BPD *versus* those formerly diagnosed with disruptive behaviour disorder.

Community studies

Chen *et al.* (2004) investigated the association between adolescent BPD and subsequent partner conflict during the transition into adulthood. Narrative descriptions of partner conflict over the previous 10 years were gathered in mid-adulthood. Multilevel growth models indicated that BPD symptoms in adolescence were independently (after controlling for Axis I disorders and other personality disorders) associated with sustained elevations in partner conflict over the 10-year period. Winograd *et al.* (2008) demonstrated that BPD symptoms in mid-adolescence were associated with lower levels of perceived social support over the subsequent 20 years ($\beta = -0.162$, S.E. = 0.026), and this association remained unaltered following adjustment for Axis I disorders. While BPD symptoms in mid-adolescence were also associated with poorer relationship quality, the association did not quite reach statistical significance ($\beta = -0.059$, S.E. = 0.033, $p = 0.075$).

Psychiatric disorders

Clinical populations

Wenning (1990) found that approximately two-thirds of the children who met criteria for BPD at age 8 years had affective conditions at 16–18 years of age. Over half were diagnosed with chronic affective conditions (e.g. cyclothymia, dysthymia), and almost half had experienced recurrent major depression during their post-discharge years. Over one-third had experienced episodes of generalized anxiety disorder, which commonly co-existed or overlapped with depressive episodes. Lofgren *et al.* (1991) reported that 31.6% of borderline children reassessed 10–20 years later were substance abusers. Zelkowitz *et al.* (2007) found that adolescents with borderline pathology in childhood had significantly higher scores on several indices of the Child Behavior Checklist including the withdrawn (64 *v.* 55.8, $p < 0.05$), anxious/depressed (63.5 *v.* 57.3, $p < 0.05$), thought problems (64.3 *v.* 58.6, $p < 0.05$), internalizing (63 *v.* 53.8, $p < 0.05$) and aggression (65.2 *v.* 59, $p < 0.05$) subscales.

Non-clinical populations

Cohen *et al.* (2007) reported that BPD in adolescence was associated with a six-fold increased risk of substance use disorder 9 years later (OR 6.19, 95% CI 1.10–34.92).

Service utilization

Clinical samples

Biskin *et al.* (2011) found that 20-year-olds who had received a BPD diagnosis at age 15 years were more likely to be in current treatment than clinical controls previously diagnosed with disruptive behaviour disorders (BPD = 42%, non-BPD = 19%). Women with persistent BPD (i.e. diagnosis at both 15 and 20 years) were especially likely to be in current treatment (73%, $p < 0.01$). Similarly, Zelkowitz *et al.* (2007) found that adolescents who had been diagnosed with childhood borderline pathology were more likely to have received psychiatric treatment since discharge in comparison with the clinical control group, though this difference did not reach statistical significance (59% *v.* 48%).

Non-clinical samples

Winograd *et al.* (2008) reported an association between BPD symptoms in adolescence and mental health service utilization in adulthood, though this association did not quite reach statistical significance (OR 1.44, 95% CI 0.98–2.11, $p = 0.059$).

Life satisfaction

Winograd *et al.* (2008) found that early adolescent borderline symptoms predicted lower life satisfaction across two decades ($\beta = -0.181$, S.E. = 0.026). The association remained after controlling for Axis I disorders in adolescence.

Discussion

As far as we are aware this is the first systematic review assessing short- and long-term (1–20 years) outcomes of BPD in childhood and adolescence. We investigated the predictive validity of BPD by examining multi-dimensional outcomes of the syndrome. Below we evaluate the findings regarding the stability, and clinical and psychosocial outcomes of BPD in childhood and adolescence.

Studies reporting on diagnostic stability

Before summarizing findings and contextualizing within the adult literature, a consideration of methodological limitations and potential impact on stability estimates is warranted.

First, very high rates of attrition were incurred in most studies (Table 2). Attrition analysis has indicated

https://doi.org/10.1017/S0033291715000318 Published online by Cambridge University Press

that retention difficulty may be related to personality pathology at follow-up (Allott *et al.* 2006); thus those most likely to be diagnosed may be lost from the study prior to re-assessment. It is notable that the study with the lowest rate of attrition reported the highest level of diagnostic stability (Chanen *et al.* 2004). Second (and related to attrition), large differences in follow-up period (i.e. 2–20 years) could have partly accounted for variations in diagnostic stability. Generally, shorter studies (e.g. Biskin *et al.* 2011; Chanen *et al.* 2004) yielded higher stability figures. Third, a number of studies were biased in their sampling, utilizing a heavy proportion of male patients (Lofgren *et al.* 1991; Zelkowitz *et al.* 2007). This contradicts the clinical picture in adulthood, in which there are typically a higher proportion of female patients (Skodol & Bender, 2003). Furthermore, gender may influence the trajectory of personality disorder development (Paris, 1997). Of note, Lofgren *et al.* (1991) recruited a predominately (74%) male sample, a large proportion of whom were diagnosed with male preponderance personality disorders at follow-up. Fourth, measurement error could lead to an underestimation of diagnostic stability. Moderate levels of inter-rater and test–retest reliability for categorical diagnosis have been reported (Chanen *et al.* 2004). This problem may be compounded by the quality of assessment tools, and inconsistency in tools between baseline and follow-up (Lofgren *et al.* 1991). While some studies used semi-structured interviews, others relied on screening questionnaires or self-created measures rather than established tools (Bernstein *et al.* 1993). Some studies (Wenning, 1990; Lofgren *et al.* 1991) used broad non-validated criteria for childhood borderline pathology at baseline potentially yielding associations with global personality disorders, rather than BPD specifically. Fifth, many studies recruited hospitalized adolescents (e.g. Meijer *et al.* 1998) or out-patients receiving specific BPD treatments (Biskin *et al.* 2011), which could have potentially reduced diagnostic stability. Finally, it has been noted that dichotomous (i.e. present/not present) classification of personality disorders may artificially widen the gap between those who are just above threshold and those at subclinical levels (i.e. 4/5 symptoms). Some patients demonstrated subclinical levels of BPD at follow-up, but were no longer diagnosed with BPD (Meijer *et al.* 1998).

Accepting these limitations, studies with child and adolescent populations indicate, at best, moderate levels of diagnostic stability, ranging from 14 to 40%. These figures demonstrate considerable overlap with reported stability for adult BPD populations, ranging from 25 to 67% (Pope *et al.* 1983; Barasch *et al.* 1985; Kullgren *et al.* 1986; Paris *et al.* 1987). Rates of global personality disorder stability appear much higher, ranging from 74 to 86% (Wenning, 1990; Lofgren *et al.* 1991; Chanen *et al.* 2004). This suggests that while lower-order individual differences may change, the broad construct of personality disorder may endure for the majority of individuals (Chanen *et al.* 2004).

Studies reporting on mean-level and rank-order stability

Moderate to high levels (Cohen, 1988) of mean-level stability were observed across studies (correlations ranging from 0.39 to 0.59). These figures are comparable with those reported in both clinical (Ferro *et al.* 1998) and community (Johnson *et al.* 1997; Lenzenweger, 1999) adult studies. A low level of dimensional stability (ICC = 0.16) was reported in one study (Grilo *et al.* 2001). The authors suggest that this may have been due to an effective in-patient treatment programme, though it is perhaps more likely that the low levels of stability were attributable to very high levels of attrition in this study. Common to findings regarding individual personality dimensions (e.g. negative affect), studies suggest that BPD symptom levels may decrease with advancing age (Bernstein *et al.* 1993; Grilo *et al.* 2001; Winograd *et al.* 2008; Bornovalova *et al.* 2009; Jovev *et al.* 2014). This highlights mid to late adolescence as a relatively high-risk period for BPD (Stepp *et al.* 2014), and is congruent with previous research demonstrating that personality disorder traits peak in mid-adolescence then follow a linear decline through early to mid-adulthood (Johnson *et al.* 2000). Rank-order associations demonstrated the highest levels of stability, ranging from 0.53 to 0.73. This is consistent with the normative personality literature, which indicates that mean-level traits change over time, while rank-order stability remains relatively stable (Roberts & DelVecchio, 2000).

Studies reporting on other clinical and psychosocial outcomes of BPD

We also assessed the prognostic implications of borderline pathology in terms of long-term psychosocial functioning. It has been suggested by the World Health Organization that functional status may be a better indicator of healthcare needs than symptoms or diagnoses alone (Reed *et al.* 2005). Furthermore, studies indicate that even when individuals with BPD achieve remission, long-term functioning may continue to be suboptimal (Biskin *et al.* 2011). Collectively the evidence suggests that BPD in childhood and adolescence is predictive of impairment in interpersonal, academic, occupational and financial domains, even when psychiatric co-morbidity is accounted for (Winograd *et al.* 2008).

https://doi.org/10.1017/S0033291715000318 Published online by Cambridge University Press

Limitations

Despite our comprehensive search, we identified relatively few studies pertaining to the predictive validity of BPD in childhood and adolescence. Furthermore, studies varied greatly in duration; thus there were insufficient data to provide a quantitative synthesis of the findings. Most of the identified studies were at high risk of bias across one or more domains, which could have led to an underestimation of stability estimates and degree of functional impairment. In particular, our quality assessment indicated that many studies were at high risk of performance and attrition bias. Attrition bias may be especially salient as reports suggest that personality disorder is associated with follow-up contact difficulty (Allott *et al.* 2006). Considering the importance placed on the predictive validity of psychiatric disorders (Van Os *et al.* 2009), our review highlights the need for more high-quality studies in this area. In particular, future studies should utilize validated assessment tools, conduct frequent and repeated assessment of BPD and concomitant psychopathologies, and examine a wide range of psychosocial outcomes, including risk exposures, e.g. bullying (Wolke *et al.* 2012).

Clinical and research implications

Congruent with the theory of homotypic (i.e. prediction of a disorder by the same disorder) and heterotypic (i.e. prediction of a disorder by a different disorder) continuity (Crowell *et al.* 2009), there are probably three groups of youngsters with borderline pathology. These are: those who maintain the diagnosis; those who remit (though they may relapse again); and those who demonstrate heterotypic continuity, i.e. they remain unwell but mental health problems evolve into a different diagnosis (Mattanah *et al.* 1995). Indeed, we found that children and adolescents who had been diagnosed with borderline pathology were more likely to suffer from subsequent psychopathology including substance abuse problems (Lofgren *et al.* 1991; Cohen *et al.* 2007), affective disorders (Wenning, 1990; Zelkowitz *et al.* 2007) and a range of personality disorders (Wenning, 1990; Lofgren *et al.* 1991). An important area of future research will be a deeper understanding of the determinants of persistence of borderline psychopathology over time (Fossati *et al.* 2013). As has been reported within the adult literature (Zanarini *et al.* 2006), childhood sexual abuse (Biskin *et al.* 2011) and temperament (Jovev *et al.* 2014) may predict the stability of BPD across childhood and adolescence. Expanding our understanding of both high-risk (e.g. temperamental predisposition) and protective (e.g. secure attachment) factors could inform future intervention and prevention programmes, promoting more adaptive pathways. Dimensional measures may be especially useful for this endeavour as they allow for the identification of subclinical levels of BPD, enabling early intervention for high-risk individuals who do not quite meet criteria for BPD diagnosis (Chanen *et al.* 2008*b*).

Our findings of low to moderate stability of BPD diagnosis in childhood and adolescence are congruent with those reported in adult populations. The slightly lower figures probably reflect developmental stage. As has been observed in the context of conduct disorder and subsequent ASPD, not all children demonstrating conduct problems will manifest ASPD in adulthood (Moffitt & Caspi, 2001). Nevertheless, the importance of clinically recognizing conduct disorder in young people is accepted, and specific diagnostic tools are available (World Health Organization, 1992; APA, 2013). Equivalent tools for young people with BPD are not currently available (Chanen & Thompson, 2014). Considering the high levels of long-term distress and functional impairment associated with BPD in childhood and adolescence, a similar recognition of this disorder appears warranted. Early intervention may be indicated, especially if treatments are potentially more benign and effective at the earlier phases of the disorder (Chanen & Kaess, 2012). Indeed, recent studies indicate that a range of psychological interventions (i.e. emotion-regulation training, cognitive analytic therapy and mentalization-based treatment) may be effective in reducing BPD symptoms in adolescents (Chanen *et al.* 2008*a*; Rossouw & Fonagy, 2012; Schuppert *et al.* 2012).

Acknowledgements

C.W. receives funding from the West Midlands Strategic Health Authority and Research and Innovation Division and the Birmingham and Solihull Mental Health Foundation Trust. S.P.S. receives funding from the National Institute for Health Research (NIHR) Collaborations for Leadership in Applied Health Research and Care West Midlands (CLAHRC-WM) initiative.

Declaration of Interest

None.

References

Allott K, Chanen A, Yuen HP (2006). Attrition bias in longitudinal research involving adolescent psychiatric outpatients. *Journal of Nervous and Mental Disease* **194**, 958–961.

https://doi.org/10.1017/S0033291715000318 Published online by Cambridge University Press

APA (1980). *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edn. American Psychiatric Association: Washington, DC.

APA (2013). *Diagnostic and Statistical Manual of Mental Disorders*, 5th edn. American Psychiatric Publishing: Arlington, VA.

Barasch A, Frances A, Hurt S, Clarkin J, Cohen S (1985). Stability and distinctness of borderline personality disorder. *American Journal of Psychiatry* **142**, 1484–1486.

Bemporad JR, Smith HF, Hanson G, Cicchetti D (1982). Borderline syndromes in childhood: criteria for diagnosis. *American Journal of Psychiatry* **139**, 596–602.

Bender DS, Dolan RT, Skodol AE, Sanislow CA, Dyck IR, McGlashan TH, Shea MT, Zanarini MC, Oldham JM, Gunderson JG (2001). Treatment utilization by patients with personality disorders. *American Journal of Psychiatry* **158**, 295–302.

Bernstein DP, Cohen P, Velez CN, Schwab-Stone M, Siever LJ, Shinsato L (1993). Prevalence and stability of the DSM-III-R personality disorders in a community-based survey of adolescents. *American Journal of Psychiatry* **150**, 1237–1243.

Biskin RS, Paris J, Renaud J, Raz A, Zelkowitz P (2011). Outcomes in women diagnosed with borderline personality disorder in adolescence. *Journal of the Canadian Academy of Child and Adolescent Psychiatry* **20**, 168–174.

Black DW, Blum N, Pfohl B, Hale N (2004). Suicidal behavior in borderline personality disorder: prevalence, risk factors, prediction, and prevention. *Journal of Personality Disorders* **18**, 226–239.

Black DW, Gunter T, Allen J, Blum N, Arndt S, Wenman G, Sieleni B (2007). Borderline personality disorder in male and female offenders newly committed to prison. *Comprehensive Psychiatry* **48**, 400–405.

Bondurant H, Greenfield B, Tse SM (2004). Construct validity of the adolescent borderline personality disorder: a review. *Canadian Child and Adolescent Psychiatry Review* **13**, 53–57.

Bornovalova MA, Hicks BM, Iacono WG, McGue M (2009). Stability, change, and heritability of borderline personality disorder traits from adolescence to adulthood: a longitudinal twin study. *Development and Psychopathology* **21**, 1335–1353.

Bornovalova MA, Hicks BM, Iacono WG, McGue M (2013). Longitudinal twin study of borderline personality disorder traits and substance use in adolescence: developmental change, reciprocal effects, and genetic and environmental influences. *Personality Disorders: Theory, Research, and Treatment* **4**, 23–32.

Chanen AM, Jackson HJ, McCutcheon LK, Jovev M, Dudgeon P, Yuen HP, Germano D, Nistico H, McDougall E, Weinstein C (2008*a*). Early intervention for adolescents with borderline personality disorder using cognitive analytic therapy: randomised controlled trial. *British Journal of Psychiatry* **193**, 477–484.

Chanen AM, Jackson HJ, McGorry PD, Allot KA, Clarkson V, Yuen HP (2004). Two-year stability of personality disorder in older adolescent outpatients. *Journal of Personality Disorders* **18**, 526–541.

Chanen AM, Jovev M, McCutcheon LK, Jackson HJ, McGorry PD (2008*b*). Borderline personality disorder in young people and the prospects for prevention and early intervention. *Current Psychiatry Reviews* **4**, 48–57.

Chanen AM, Kaess M (2012). Developmental pathways to borderline personality disorder. *Current Psychiatry Reports* **14**, 45–53.

Chanen AM, McCutcheon LK (2008). Personality disorder in adolescence: the diagnosis that dare not speak its name. *Personality and Mental Health* **2**, 35–41.

Chanen AM, Thompson K (2014). Preventive strategies for borderline personality disorder in adolescents. *Current Treatment Options in Psychiatry* **1**, 358–368.

Chen H, Cohen P, Johnson JG, Kasen S, Sneed JR, Crawford TN (2004). Adolescent personality disorders and conflict with romantic partners during the transition to adulthood. *Journal of Personality Disorders* **18**, 507–525.

Cohen J (1988). *Statistical Power Analysis for the Behavioral Sciences*, 2nd edn. Lawrence Erlbaum: Hillsdale, NJ.

Cohen P, Chen H, Crawford TN, Brook JS, Gordon K (2007). Personality disorders in early adolescence and the development of later substance use disorders in the general population. *Drug and Alcohol Dependence* **88**, S71–S84.

Crick NR, Murray–Close D, Woods K (2005). Borderline personality features in childhood: a short-term longitudinal study. *Development and Psychopathology* **17**, 1051–1070.

Crowell SE, Beauchaine TP, Linehan MM (2009). A biosocial developmental model of borderline personality: elaborating and extending Linehan's theory. *Psychological Bulletin* **135**, 495–510.

Daley SE, Burge D, Hammen C (2000). Borderline personality disorder symptoms as predictors of 4-year romantic relationship dysfunction in young women: addressing issues of specificity. *Journal of Abnormal Psychology* **109**, 451–460.

Ferro T, Klein DN, Schwartz JE, Kasch KL, Leader JB (1998). 30-Month stability of personality disorder diagnoses in depressed outpatients. *American Journal of Psychiatry* **155**, 653–659.

Fossati A, Gratz KL, Maffei C, Borroni S (2013). Emotion dysregulation and impulsivity additively predict borderline personality disorder features in Italian nonclinical adolescents. *Personality and Mental Health* **7**, 320–333.

Garnet KE, Levy KN, Mattanah JJ, Edell WS, McGlashan TH (1994). Borderline personality disorder in adolescents: ubiquitous or specific? *American Journal of Psychiatry* **151**, 1380–1382.

Griffiths M (2011). Validity, utility and acceptability of borderline personality disorder diagnosis in childhood and adolescence: survey of psychiatrists. *Psychiatrist* **35**, 19–22.

Grilo CM, Becker DF, Edell WS, McGlashan TH (2001). Stability and change of DSM-III-R personality disorder dimensions in adolescents followed up 2 years after psychiatric hospitalization. *Comprehensive Psychiatry* **42**, 364–368.

Hammersley M (2001). On 'systematic' reviews of research literatures: a 'narrative' response to Evans & Benefield. *British Educational Research Journal* **27**, 543–554.

Higgins JPT, Altman DG (2008). Assessing risk of bias in included studies. In *Cochrane Handbook for Systematic Reviews of Interventions: Cochrane Book Series* (ed. J. P. T. Higgins and S. Green), pp. 187–241. John Wiley & Sons Ltd: Chichester.

Johnson JG, Cohen P, Kasen S, Skodol AE, Hamagami F, Brook JS (2000). Age-related change in personality disorder trait levels between early adolescence and adulthood: a community-based longitudinal investigation. *Acta Psychiatrica Scandinavica* **102**, 265–275.

Johnson JG, Williams JB, Goetz RR, Rabkin JG, Lipsitz JD, Remien RH (1997). Stability and change in personality disorder symptomatology: findings from a longitudinal study of HIV+ and HIV– men. *Journal of Abnormal Psychology* **106**, 154–158.

Jovev M, Whittle S, Yücel M, Simmons JG, Allen NB, Chanen AM (2014). The relationship between hippocampal asymmetry and temperament in adolescent borderline and antisocial personality pathology. *Development and Psychopathology* **26**, 275–285.

Kullgren G, Renberg E, Jacobsson L (1986). An empirical study of borderline personality disorder and psychiatric suicides. *Journal of Nervous and Mental Disease* **174**, 328–331.

Laurenssen EMP, Hutsebaut J, Feenstra DJ, Van Busschbach JJ, Luyten P (2013). Diagnosis of personality disorders in adolescents: a study among psychologists. *Child and Adolescent Psychiatry and Mental Health* **7**, 3.

Leichsenring F, Leibing E, Kruse J, New AS, Leweke F (2011). Borderline personality disorder. *Lancet* **377**, 74–84.

Lenzenweger MF (1999). Stability and change in personality disorder features: The Longitudinal Study of Personality Disorders. *Archives of General Psychiatry* **56**, 1009–1015.

Lofgren DP, Bemporad J, King J, Lindem K, O'Driscoll G (1991). A prospective follow-up study of so-called borderline children. *American Journal of Psychiatry* **148**, 1541–1547.

Loranger AW, Sartorius N, Andreoli A, Berger P, Buchheim P, Channabasavanna S, Coid B, Dahl A, Diekstra RF, Ferguson B (1994). The International Personality Disorder Examination: The World Health Organization/Alcohol, Drug Abuse, and Mental Health Administration international pilot study of personality disorders. *Archives of General Psychiatry* **51**, 215–224.

Mattanah JJ, Becker DF, Levy KN, Edell WS, McGlashan TH (1995). Diagnostic stability in adolescents followed up 2 years after hospitalization. *American Journal of Psychiatry* **152**, 889–894.

Meijer M, Goedhart AW, Treffers PD (1998). The persistence of borderline personality disorder in adolescence. *Journal of Personality Disorders* **12**, 13–22.

Miller AL, Muehlenkamp JJ, Jacobson CM (2008). Fact or fiction: diagnosing borderline personality disorder in adolescents. *Clinical Psychology Review* **28**, 969–981.

Moffitt TE, Caspi A (2001). Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways among males and females. *Development and Psychopathology* **13**, 355–375.

Moher D, Liberati A, Tetzlaff J, Altman DG (2009). Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *Annals of Internal Medicine* **151**, 264–269.

Paris J (1997). Antisocial and borderline personality disorders: two separate diagnoses or two aspects of the same psychopathology? *Comprehensive Psychiatry* **38**, 237–242.

Paris J (2013). Personality disorders begin in adolescence. *Journal of the Canadian Academy of Child and Adolescent Psychiatry* **22**, 195–196.

Paris J, Brown R, Nowlis D (1987). Long-term follow-up of borderline patients in a general hospital. *Comprehensive Psychiatry* **28**, 530–535.

Pope HG, Jonas JM, Hudson JI, Cohen BM, Gunderson JG (1983). The validity of DSM-III borderline personality disorder: a phenomenologic, family history, treatment response, and long-term follow-up study. *Archives of General Psychiatry* **40**, 23–30.

Reed GM, Lux JB, Bufka LF, Trask C, Peterson DB, Stark S, Threats TT, Jacobson JW, Hawley JA (2005). Operationalizing the international classification of functioning, disability and health in clinical settings. *Rehabilitation Psychology* **50**, 122–131.

Roberts BW, DelVecchio WF (2000). The rank-order consistency of personality traits from childhood to old age: a quantitative review of longitudinal studies. *Psychological Bulletin* **126**, 3–25.

Rossouw TI, Fonagy P (2012). Mentalization-based treatment for self-harm in adolescents: a randomized controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry* **51**, 1304–1313.e3.

Sayers A (2007). Tips and tricks in performing a systematic review. *British Journal of General Practice* **57**, 425.

Schuppert HM, Timmerman ME, Bloo J, van Gemert TG, Wiersema HM, Minderaa RB, Emmelkamp PM, Nauta MH (2012). Emotion regulation training for adolescents with borderline personality disorder traits: a randomized controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry* **51**, 1314–1323.e2.

Skodol AE, Bender DS (2003). Why are women diagnosed borderline more than men? *Psychiatric Quarterly* **74**, 349–360.

Skodol AE, Gunderson JG, McGlashan TH, Dyck IR, Stout RL, Bender DS, Grilo CM, Shea MT, Zanarini MC, Morey LC (2002). Functional impairment in patients with schizotypal, borderline, avoidant, or obsessive–compulsive personality disorder. *American Journal of Psychiatry* **159**, 276–283.

Stepp SD, Whalen DJ, Scott LN, Zalewski M, Loeber R, Hipwell AE (2014). Reciprocal effects of parenting and borderline personality disorder symptoms in adolescent girls. *Development and Psychopathology* **26**, 361–378.

Van Os J, Linscott RJ, Myin-Germeys I, Delespaul P, Krabbendam L (2009). A systematic review and meta-analysis of the psychosis continuum: evidence for a psychosis proneness–persistence–impairment model of psychotic disorder. *Psychological Medicine* **39**, 179–195.

Vela R, Gottlieb E, Gottlieb H (1983). Borderline syndromes in childhood: a critical review. In *The Borderline Child:*

Approaches to Etiology, Diagnosis, and Treatment (ed. K. S. Robson), pp. 31–48. McGraw-Hill: New York.

Wenning K (1990). Borderline children: a closer look at diagnosis and treatment. *American Journal of Orthopsychiatry* **60**, 225–232.

Winograd G, Cohen P, Chen H (2008). Adolescent borderline symptoms in the community: prognosis for functioning over 20 years. *Journal of Child Psychology and Psychiatry* **49**, 933–941.

Winsper C, Zanarini M, Wolke D (2012). Prospective study of family adversity and maladaptive parenting in childhood and borderline personality disorder symptoms in a non-clinical population at 11 years. *Psychological Medicine* **42**, 2405–2420.

Wolke D, Schreier A, Zanarini MC, Winsper C (2012). Bullied by peers in childhood and borderline personality symptoms at 11 years of age: a prospective study. *Journal of Child Psychology and Psychiatry* **53**, 846–855.

World Health Organization (1992). *The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Description and Diagnostic Guidelines*. WHO: Geneva.

Zanarini MC, Frankenburg F, Hennen J, Reich D, Silk K (2006). Prediction of the 10-year course of borderline personality disorder. *American Journal of Psychiatry* **163**, 827–832.

Zelkowitz P, Paris J, Guzder J, Feldman R, Roy C, Rosval L (2007). A five-year follow-up of patients with borderline pathology of childhood. *Journal of Personality Disorders* **21**, 664–674.