**The Clinical and Psychosocial Outcomes of Borderline Personality Disorder (BPD) in Childhood and Adolescence: A Systematic Review**

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**Background**

While there is a growing body of research regarding 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 8,200 abstracts were screened. Out of 214 full text articles, 18 satisfied the predetermined inclusion criteria. Quality assessment indicated that most studies were high risk of bias in at least one study domain. Consistent with the adult literature, the diagnostic stability of BPD prior to age 19 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 age 19 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.

**Keywords:** Borderline Personality Disorder; predictive validity; childhood; adolescence; systematic review

Borderline Personality Disorder (BPD) is a serious mental illness characterised by behavioural and emotional dysregulation, marked impairment in psychosocial functioning and high risk of mortality ([Black *et al.*, 2004](#_ENREF_9), [Leichsenring *et al.*, 2011](#_ENREF_38)). BPD is associated with a range of long-term negative sequelae, including relationship dysfunction ([Daley *et al.*, 2000](#_ENREF_25)), unemployment ([Skodol *et al.*, 2002](#_ENREF_57)), high levels of treatment utilisation ([Bender *et al.*, 2001](#_ENREF_6)) and imprisonment ([Black *et al.*, 2007](#_ENREF_10)). 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 age 19) remains controversial ([Chanen and McCutcheon, 2008](#_ENREF_18), [Miller *et al.*, 2008](#_ENREF_44)). Recent reports indicate that clinicians are reluctant to diagnose BPD in younger individuals ([Griffiths, 2011](#_ENREF_29), [Laurenssen *et al.*, 2013](#_ENREF_37)). 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](#_ENREF_24)). Importantly, the identification of BPD symptoms prior to adulthood may help shed light on aetiological processes ([Crowell *et al.*, 2009](#_ENREF_24)), inform early intervention programs ([Chanen *et al.*, 2008b](#_ENREF_16)), and ensure that younger people with personality pathology receive appropriate treatment ([Paris, 2013](#_ENREF_48)).

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](#_ENREF_59)). Ascertaining the predictive validity of BPD by considering both diagnostic and psychosocial outcomes is important in view of concerns regarding the lack of stability of the diagnosis during this developmental phase ([Meijer *et al.*, 1998](#_ENREF_43)). Furthermore, identifying influences on the stability of BPD across early development may help highlight important risk and protective factors, furnishing our understanding of the continuity and *discontinuity* of BPD trajectories across the lifespan.

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)](#_ENREF_11) reported that the diagnostic stability of adolescent BPD was relatively low, though very few studies were identified ([Bernstein *et al.*, 1993](#_ENREF_7), [Garnet *et al.*, 1994](#_ENREF_28), [Mattanah *et al.*, 1995](#_ENREF_42)). [Miller *et al.* (2008)](#_ENREF_44) reported low to moderate diagnostic and dimensional stability, though again only a limited number of studies were available ([Bernstein *et al.*, 1993](#_ENREF_7), [Chanen *et al.*, 2004](#_ENREF_15), [Garnet *et al.*, 1994](#_ENREF_28), [Grilo *et al.*, 2001](#_ENREF_30), [Meijer *et al.*, 1998](#_ENREF_43)). [Chanen *et al.* (2008b)](#_ENREF_16) considered BPD in youth (i.e., 15-24 years) and found that mean level BPD traits were moderately stable, though the authors highlighted the lack of BPD specific data. Chanen and colleagues also presented limited evidence suggesting poorer outcome (i.e., increased risk of Axis I disorders, social impairment) in young people with borderline pathology.

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](#_ENREF_31)). 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 individuals 18 years or younger?

2) Do BPD symptoms demonstrate mean-level stability in individuals 18 years or younger (i.e., do individual BPD scores remain stable over time)?

3) Do BPD symptoms demonstrate rank order stability in individuals 18 years or younger (i.e., do individuals retain their relative placement in the group)?

4) Does BPD pathology in childhood and adolescence predict problems in diverse spheres of functioning in later life?

**Method**

Prior to formulating the protocol, C.W. and J.E conducted a pilot search to ensure that a systematic review pertaining to the research question had not been published. We searched the Cochrane Database of Systematic Reviews (CDSR); the Centre for Reviews and Dissemination (CRD); and www.pubmed.gov ([Sayers, 2007](#_ENREF_54)). We used PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines ([Moher *et al.*, 2009](#_ENREF_46)) 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 conceptualised in the Diagnostic and Statistical Manual ([APA., 1980](#_ENREF_3)). 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 the reference lists of relevant narrative reviews as a cross check ([Bondurant *et al.*, 2004](#_ENREF_11), [Chanen and McCutcheon, 2008](#_ENREF_18), [Miller *et al.*, 2008](#_ENREF_44)).

***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 to 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 borderline personality symptoms/disorder);

3) They were treatment trials.

***Screening procedure***

After removal of duplicates, 8,200 abstracts were retrieved. If a title appeared potentially eligible but no abstract was available, the full-text article was retrieved. C.W and T.L independently scanned 100% of the abstracts to identify relevant 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 of BPD. It also included a quality assessment tool based on Cochrane Collaboration guidelines ([Higgins and Altman, 2008](#_ENREF_32)). 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 accessor of outcome variables); and reporting bias (any indication of selective reporting).

***Data synthesis***

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

**Results**

Of the 8, 200 (data base search = 8, 195; 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 regarding selected articles, 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 information on the outcomes of BPD in childhood or adolescence (**Figure 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 at this stage were: the sample was over 18 years of age; BPD was conflated with another mental disorder; associations with subscales of BPD assessments were reported only; there was no information on clinical or psychosocial outcomes. Studies comprised a mix of clinical and non-clinical populations, and ranged in duration from one to twenty years (see **Table 1).**

**Quality assessment**

Quality assessment indicated that most studies were high risk of bias (systematic error) in one or more 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 over estimation of effects (see discussion for further explication). In general, clinical studies were high risk of bias in more domains than non-clinical studies. All but four studies ([Bernstein *et al.*, 1993](#_ENREF_7), [Chen *et al.*, 2004](#_ENREF_20), [Cohen *et al.*, 2007](#_ENREF_22), [Winograd *et al.*, 2008](#_ENREF_62)) were high risk in terms of sample selection bias. Three studies were high risk in baseline assessment bias ([Biskin *et al.*, 2011](#_ENREF_8), [Meijer *et al.*, 1998](#_ENREF_43), [Stepp *et al.*, 2014](#_ENREF_58)). Nine studies ([Biskin *et al.*, 2011](#_ENREF_8), [Chanen *et al.*, 2004](#_ENREF_15), [Garnet *et al.*, 1994](#_ENREF_28), [Grilo *et al.*, 2001](#_ENREF_30), [Lofgren *et al.*, 1991](#_ENREF_40), [Mattanah *et al.*, 1995](#_ENREF_42), [Meijer *et al.*, 1998](#_ENREF_43), [Wenning, 1990](#_ENREF_61), [Zelkowitz *et al.*, 2007](#_ENREF_66)) were high risk in performance bias. All studies excepting [Bernstein *et al.* (1993)](#_ENREF_7) and [Chanen *et al.* (2004)](#_ENREF_15) were high risk in attrition bias. Two studies were high risk ([Biskin *et al.*, 2011](#_ENREF_8), [Wenning, 1990](#_ENREF_61)) and seven unclear risk ([Bornovalova *et al.*, 2009](#_ENREF_12), [2013](#_ENREF_13), [Cohen *et al.*, 2007](#_ENREF_22), [Crick *et al.*, 2005](#_ENREF_23), [Jovev *et al.*, 2013](#_ENREF_35), [Stepp *et al.*, 2014](#_ENREF_58), [Winograd *et al.*, 2008](#_ENREF_62)) in detection bias. One study was high risk in reporting bias ([Wenning, 1990](#_ENREF_61)).

**The stability of BPD in childhood and adolescence**

***i. Diagnostic stability***

Ten studies examined the stability of BPD diagnosis during a defined follow-up period. Eight utilised clinical populations ([Biskin *et al.*, 2011](#_ENREF_8), [Chanen *et al.*, 2004](#_ENREF_15), [Garnet *et al.*, 1994](#_ENREF_28), [Lofgren *et al.*, 1991](#_ENREF_40), [Mattanah *et al.*, 1995](#_ENREF_42), [Meijer *et al.*, 1998](#_ENREF_43), [Wenning, 1990](#_ENREF_61), [Zelkowitz *et al.*, 2007](#_ENREF_66)) and two non-clinical populations ([Bernstein *et al.*, 1993](#_ENREF_7), [Winograd *et al.*, 2008](#_ENREF_62)). Overall, the level of diagnostic stability across studies (over durations from 2 to 20 years) ranged from 14% to 40%.

*Clinical populations*

[Wenning (1990)](#_ENREF_61) followed up children retrospectively diagnosed with childhood borderline personality over a period of 10 years. Of the original 57 children, 28 (20 ‘angry impulsive;’ 8 ‘borderline psychotic’) were identified for follow-up assessment. Ninety percent of children with ‘angry-impulsive’ borderline disorder, and 75% with ‘borderline psychotic’ disorder received a personality disorder diagnosis at follow-up. Of the ‘angry-impulsive’ types, the most common (75%) diagnosis was BPD and/or antisocial personality disorder.

Over a period of 10-20 years, Lofgren and colleagues (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. Three were diagnosed with BPD (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), likely reflecting the male bias in the sample.

Two studies from the same research group examined the stability of BPD diagnosis in adolescent inpatients from the Yale Psychiatric Institute ([Garnet *et al.*, 1994](#_ENREF_28), [Mattanah *et al.*, 1995](#_ENREF_42)). In the first, 33% of patients with BPD at index assessment met diagnostic criteria for BPD at follow-up 2 years later ([Garnet *et al.*, 1994](#_ENREF_28)). In the second, 23% of individuals with BPD at baseline still met diagnostic criteria at 2-year follow-up ([Mattanah *et al.*, 1995](#_ENREF_42)). In another hospital study, 14% of adolescents diagnosed with BPD at baseline retained diagnosis 3 years later ([Meijer *et al.*, 1998](#_ENREF_43)). A number of the adolescents diagnosed with BPD at index hospitalisation, while no longer carrying a BPD diagnosis, continued to demonstrate sub-clinical levels of disturbance (average Diagnostic Interview for Borderlines [DIB] score of 4.6).

In a study with adolescent outpatients, Chanen et al. (2004) found that BPD diagnosis remained stable over 2 years in 40% of those initially diagnosed. The stability of global personality disorder (i.e., any personality disorder diagnosis) was 74%.

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

*Non-clinical populations*

In the first of two studies with the Children in the Community (CIC) cohort, [Bernstein *et al.* (1993)](#_ENREF_7) reported that 29% of adolescents with “moderate” (symptom scores dichotomised 1 SD above the mean), and 24% of adolescents with “severe” (symptom scores dichotomised 2 SDs above the mean) BPD symptoms retained the diagnosis after 2 years.

In the second CIC study, [Winograd *et al.* (2008)](#_ENREF_62) reported that extreme BPD symptoms (2 SDs above the mean) in adolescence were associated with an approximately nine-fold increased odds of BPD diagnosis 20 years later.

***ii. Mean level stability***

Seven studies examined the mean level stability of BPD symptoms. Two utilised clinical populations ([Chanen *et al.*, 2004](#_ENREF_15), [Grilo *et al.*, 2001](#_ENREF_30)) and five non-clinical populations ([Bornovalova *et al.*, 2009](#_ENREF_12), [Crick *et al.*, 2005](#_ENREF_23), [Jovev *et al.*, 2013](#_ENREF_35), [Stepp *et al.*, 2014](#_ENREF_58), [Winograd *et al.*, 2008](#_ENREF_62)). Mean level stability ranged from .16 to .59 across studies over durations of 1 to 20 years.

*Clinical populations*

Chanen and colleagues (2004) reported a mean level stability of ICC= .54 in a sample of adolescent outpatients. T-tests indicated that BPD symptoms did not significantly decrease over time (p=.115). [Grilo *et al.* (2001)](#_ENREF_30) found that the mean level stability of BPD symptoms in adolescent inpatients was ICC=.16. T-tests indicated a significant decrease in BPD symptom scores over time (t=2.23, *p*<.05).

*Non-clinical populations*

[Crick *et al.* (2005)](#_ENREF_23) assessed the stability of BPD symptoms over 1 year (autumn year 1; spring year 2; autumn year 2) in a sample of school children. Correlations between the three time-points ranged from .47 to .56. In another short-term study with school children, Jovev et al. (2013) reported that mean BPD symptoms significantly decreased over the 2 year study period (Time 1: M =1.67; Time 2: M =1.30). Low Effortful Control (reflecting poor self-regulation), however, significantly predicted an increase in BPD symptoms over this time period (β= - 0.18, t = -2.32, p=0.002).

 [Stepp *et al.* (2014)](#_ENREF_58) examined the stability of BPD symptoms in a community sample of girls. The correlations between BPD symptom scores across 4 years (at 14, 15, 16 & 17 years) were assessed. All correlations were significant (*p* <.05) and were higher between shorter durations: 14 to 15 (*r* = .55); 15 to 16 (*r*=.59); 16 to 17 (*r*=.58); 14 to 16 (*r* =.48); 15 to 17 (*r*=.52) and 14 to 17 (*r* =.42).

In a long-term community study, Winograd and colleagues (2008) found that BPD symptoms at 13.7 and 33.2 years were correlated (*r*=.39). The stability of BPD symptoms from mid to early adolescence was *r*=.516. Over the two decades of the study, BPD symptoms declined on average by β= -.032 (SE=.002) per year, equating to approximately 2/3 of a SD 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 (at 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 (M=41.26) to 17 (M=40.86) years of age; a moderate change from 14 to 20 (M=37.2) years of age; and a large change from 14 to 24 (M=35.19) years of age.

***iii. Rank order stability***

Only two studies assessed the rank order stability of BPD symptoms ([Bornovalova *et al.*, 2009](#_ENREF_12), [Chanen *et al.*, 2004](#_ENREF_15)). Chanen et al. (2004) reported a rank order stability of .54. Bornovalova et al (2009) reported rank order stability ranging from .53 to .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)](#_ENREF_40) found that none of the adolescents/adults previously diagnosed as borderline in childhood were self-supporting or living independently. Only 26% were attending school or working (mostly in unskilled or semi-skilled labour).[Zelkowitz *et al.* (2007)](#_ENREF_66) found that adolescents previously diagnosed with borderline pathologyin 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 out of the original 31) were only slightly more likely to be in employment than those who still carried the BPD diagnosis (45% versus 36%).

*Non-clinical populations*

[Winograd *et al.* (2008)](#_ENREF_62) found that educational attainment reported at 33 years of age was negatively associated with BPD symptoms in adolescence (β= -.522; SE=.074, *p*<.01). Similarly, BPD symptoms in adolescence negatively predicted occupational (ranging from unskilled labour to full professional status) level (β = -.818; SE=.176, *p*<.01). These findings remained significant following adjustment for Axis I disorders during early adolescence.

BPD symptoms in early adolescence also significantly predicted reliance on public assistance (OR=2.90; 95% CI=1.37, 6.16), though this association became non-significant following adjustment for Axis I disorders in adolescence (OR=1.99; 95% CI=.85, 4.69).

***Social functioning***

*Clinical populations*

[Lofgren *et al.* (1991)](#_ENREF_40) found that children diagnosed with borderline pathology had very poor levels of social functioning in adolescence/adulthood. Only 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. Forty-seven % described a complete absence of friendships or social life, while 37% reported ‘only highly tumultuous relationships.’ [Zelkowitz *et al.* (2007)](#_ENREF_66) reported that borderline diagnosis 5 to 7 years previously significantly increased the odds of peer problems in adolescence (*x* 2 =7.25, *p*<.01). In contrast, Biskin et al. (2011) did not find a significant group difference on the Social Adjustment Scale Self Report (SAS-SR) between those formally diagnosed with BPD versus those formerly diagnosed with disruptive behaviour disorder.

*Community studies*

[Chen *et al.* (2004)](#_ENREF_20) investigated the association between adolescent BPD and subsequent partner conflict during the transition into adulthood. Narrative descriptions of partner conflict were gathered in mid-adulthood, and referred to the previous 10 years. Using multilevel growth models, the authors demonstrated 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)](#_ENREF_62) demonstrated that BPD symptoms in mid-adolescence were associated with lower levels of perceived social support over the subsequent 20 years (β= -.162; SE= .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 (β= -.059; SE= .033; P=.075).

***Psychiatric disorders***

*Clinical populations*

[Wenning (1990)](#_ENREF_61) found that approximately two-thirds of the children who met criteria for BPD at age 8 had affective conditions at 16 to 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 generalised anxiety disorder, which commonly co-existed or overlapped with depressive episodes. [Lofgren *et al.* (1991)](#_ENREF_40) reported that 31.6% of borderline children reassessed 10 to 20 years later were substance abusers. [Zelkowitz *et al.* (2007)](#_ENREF_66) found that adolescents with borderline pathology in childhood had significantly higher scores on several indices of the Child Behavior Checklist including the withdrawn (64 vs. 55.8, *p*<.05), anxious/depressed (63.5 vs. 57.3, *p*<.05), thought problems (64.3 vs. 58.6, *p*<.05), internalising (63 vs. 53.8, *p*<.05), and aggression (65.2 vs. 59, *p*<.05) subscales.

*Non-clinical populations*

[Cohen *et al.* (2007)](#_ENREF_22) reported that BPD in adolescence was associated with a six-fold increased odds of Substance Use Disorder 9 years later (OR= 6.19; 95% CI=1.10, 34.92).

***Service utilisation***

*Clinical samples*

Biskin et al. (2011) found that 20 year olds who had received a BPD diagnosis at age 15 were more likely to be in current treatment than clinical controls previously diagnosed with disruptive behaviour disorders (BPD=42%; non-BPD=19%). The subset of women with persistent BPD (i.e., diagnosis at both 15 and 20 years) were especially likely to be in current treatment (73%; *p*<.01). Similarly, [Zelkowitz *et al.* (2007)](#_ENREF_66) found that adolescents who had been diagnosed with childhood borderline pathology were more likely to have received psychiatric treatment since discharge in comparison to the clinical control group, though this difference did not reach statistical significance (59% versus 48%).

*Non-clinical samples*

[Winograd *et al.* (2008)](#_ENREF_62) reported an association between BPD symptoms in adolescence and mental health service utilisation 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 (β = -.181; SE=.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 to 20 years) outcomes of BPD in childhood and adolescence. We investigated the predictive validity of BPD by examining the 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 summarising the findings and contextualising 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 (see **Table 2** for details). Attrition analysis has indicated that retention difficulty may be related to personality pathology at follow-up ([Allott *et al.*, 2006](#_ENREF_1)), 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](#_ENREF_15)). Second, (and related to attrition) large differences in follow-up period (i.e., 2 to 20 years) could have partly accounted for variations in diagnostic stability. Generally, shorter studies (e.g., Chanen et al., 2004; Biskin et al., 2004) yielded higher stability figures. Third, a number of studies were biased in their sampling, utilising a heavy proportion of male patients ([Lofgren *et al.*, 1991](#_ENREF_40), [Zelkowitz *et al.*, 2007](#_ENREF_66)). This contradicts the clinical picture in adulthood, in which there are typically a higher proportion of female patients ([Skodol and Bender, 2003](#_ENREF_56)). Furthermore, gender may influence the trajectory of personality disorder development ([Paris, 1997](#_ENREF_47)). Of note, Lofgren et al. (1990) recruited a predominately (74%) male sample, a large proportion of whom were diagnosed with male preponderance personality disorders (e.g., antisocial, schizoid) 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](#_ENREF_15)). This problem may be compounded by the quality of assessment tools, and inconsistency in tools between baseline and follow-up ([Lofgren *et al.*, 1991](#_ENREF_40)). 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 ([Lofgren *et al.*, 1991](#_ENREF_40), [Wenning, 1990](#_ENREF_61)) 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 hospitalised adolescents (e.g., Meijer et al., 1998) or outpatients receiving specific BPD treatments ([Biskin *et al.*, 2011](#_ENREF_8)). Thus, sample selection may have reduced symptom levels, and subsequently 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 sub-clinical levels (i.e., 4/5 symptoms). Some patients demonstrated sub-clinical levels of BPD at follow-up, but were no longer diagnosed with BPD ([Meijer *et al.*, 1998](#_ENREF_43)).

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% ([Barasch *et al.*, 1985](#_ENREF_4), [Kullgren *et al.*, 1986](#_ENREF_36), [Paris *et al.*, 1987](#_ENREF_49), [Pope *et al.*, 1983](#_ENREF_50)). Rates of global personality disorder stability appear much higher, ranging from 74% to 86% ([Chanen *et al.*, 2004](#_ENREF_15), [Lofgren *et al.*, 1991](#_ENREF_40), [Wenning, 1990](#_ENREF_61)). 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](#_ENREF_15)).

*Studies reporting on mean level and rank order stability*

Moderate to high levels ([Cohen, 1988](#_ENREF_21)) of mean level stability were observed across studies (i.e., correlations ranging from .39 to .59). These figures are comparable to those reported in both clinical ([Ferro *et al.*, 1998](#_ENREF_26)) and community ([Johnson *et al.*, 1997](#_ENREF_34), [Lenzenweger, 1999](#_ENREF_39)) adult studies. A low level of dimensional stability (ICC = .16) was reported in one study, ([Grilo *et al.*, 2001](#_ENREF_30)). The authors suggest that this may have been due to an effective inpatient 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](#_ENREF_7), [Bornovalova *et al.*, 2009](#_ENREF_12), [Grilo *et al.*, 2001](#_ENREF_30), [Jovev *et al.*, 2013](#_ENREF_35), [Winograd *et al.*, 2008](#_ENREF_62)). This highlights mid to late adolescence as a relatively high risk period for BPD ([Stepp *et al.*, 2014](#_ENREF_58)), 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](#_ENREF_33)). Rank order associations demonstrated the highest levels of stability, ranging from .53 to .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 and DelVecchio, 2000](#_ENREF_52)).

*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 across domains. It has been suggested by the World Health Organisation (WHO) that functional status may be a better indicator of health care needs than symptoms or diagnoses alone ([Reed *et al.*, 2005](#_ENREF_51)). Furthermore, studies indicate that even when individuals with BPD achieve remission, long-term functioning may continue to be sub-optimal ([Biskin *et al.*, 2011](#_ENREF_8)). Collectively the evidence suggests that BPD in childhood and adolescence is predictive of impairment in interpersonal, academic, occupational and financial domains, even when psychiatric comorbidity is accounted for ([Winograd *et al.*, 2008](#_ENREF_62)).

*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 was 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 bias (e.g., treatment could have reduced stability figures) and attrition bias. Attrition bias may be especially salient as reports suggest that personality disorder pathology is associated with follow-up contact difficulty ([Allott *et al.*, 2006](#_ENREF_1)). Considering the importance placed on the predictive validity of psychiatric disorders ([Van Os *et al.*, 2009](#_ENREF_59)), our review highlights the need for more high quality studies in this area. In particular, future studies should utilise 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](#_ENREF_63)).

*Clinical and research implications*

Congruent with the theory of homotypic (i.e., the prediction of a disorder by the same disorder) and heterotypic (i.e., the prediction of a disorder by another disorder) continuity ([Crowell *et al.*, 2009](#_ENREF_24)), there are likely three groups of young individuals with borderline pathology. Those who maintain the diagnosis; those who remit (though as has been observed with adults, 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](#_ENREF_42)). 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 ([Cohen *et al.*, 2007](#_ENREF_22), [Lofgren *et al.*, 1991](#_ENREF_40)); affective disorders ([Wenning, 1990](#_ENREF_61), [Zelkowitz *et al.*, 2007](#_ENREF_66)) and a range of personality disorders ([Lofgren *et al.*, 1991](#_ENREF_40), [Wenning, 1990](#_ENREF_61)). An important area of future research will be a deeper understanding of the determinants of persistence of borderline psychopathology (and individual symptoms, e.g., affective instability) over time ([Fossati *et al.*, 2013](#_ENREF_27)). As has been reported within the adult literature ([Zanarini *et al.*, 2006](#_ENREF_65)), studies suggest that childhood sexual abuse ([Biskin *et al.*, 2011](#_ENREF_8)) and temperament ([Jovev *et al.*, 2013](#_ENREF_35)) may predict the stability of the disorder across childhood and adolescence. Expanding our understanding of both high risk (e.g., temperamental predisposition) and protective factors (e.g., secure attachment) could inform future intervention and prevention programs to promote more adaptive pathways. Dimensional measures may be especially useful for this endeavour as they allow for the identification of sub-clinical levels of BPD, enabling early intervention for high-risk individuals who do not quite meet criteria for BPD diagnosis ([Chanen *et al.*, 2008b](#_ENREF_16)).

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 likely reflect the developmental stage. As has been observed in the context of Conduct Disorder and subsequent Antisocial Personality Disorder, not all children demonstrating conduct problems will manifest ASPD in adulthood. ([Moffitt and Caspi, 2001](#_ENREF_45)) Nevertheless, the importance of clinically recognising conduct disorder in young people is accepted, and specific diagnostic tools are available ([American Psychiatric Association, 2013](#_ENREF_2), [World Health Organisation, 1992](#_ENREF_64)). Equivalent tools for young people with BPD symptoms are not currently available ([Chanen and Thompson, 2014](#_ENREF_19)).

Considering the high levels of long-term distress and functional impairment observed in children and adolescents with BPD, 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 and Kaess, 2012](#_ENREF_17)). Indeed, recent studies indicate that a range of psychological interventions (i.e., emotion regulation training, cognitive analytic therapy, and mentalisation-based treatment) may be effective in reducing BPD symptoms in adolescents ([Chanen *et al.*, 2008a](#_ENREF_14), [Rossouw and Fonagy, 2012](#_ENREF_53), [Schuppert *et al.*, 2012](#_ENREF_55)).

**Fig. 1**

**Flowchart outlining the search and selection strategy**

Studies identified through database searching

(*n*=19, 078)

Records after duplicates removed

(*n*= 8, 200)

Title and abstracts screened

(*n*=8, 200)

196 articles excluded:

*-Outside of age range (n=81)*

*-Nor primarily a BPD sample (n=12)*

*-Assess associations with selected BPD symptoms only (n=6)*

*-No information on predictive validity specifically (n=89)*

*-Case studies (n=7)*

*-Treatment trial (n=1)*

Studies identified through hand search

(n=5)

7986 records excluded

Full text articles assessed for eligibility

(*n*=214)

Studies included in review

(*n*=18)

|  |
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| **Table 1. Details of Studies Reporting on Clinical and Psychosocial Outcomes of BPD in Childhood and Adolescence** |
| **First** | **Year** | **Country** | **Percent** | **Baseline** | **Sample frame** | **Study**  | **BPD assessment** | **Outcomes** |
| **Author** |  |  | **female** | **(N, age, % BPD)** | **(control group)** | **Design** | **(cut-point for diagnosis)** |  |
| Bernstein  | 1993 | USA | 49.8% | 733 9-19 year olds  | Community | Prospective | Children in the Community-Self | Stability of BPD diagnosis  |
|   |   |   |   |  | (N/A) |  (2 years) | Report (1 SD>mean/2 SD>mean) |   |
|   |   |   |   |   |   |   |   |  |
| Biskin  | 2011 | Canada | 100% | 97 Mage=15.1 year olds | Clinical (disruptive behaviour | Prospective  | Diagnostic Interview for  | BPD diagnosis in adulthood; employment;  |
|   |   |   |   | (49 BPD; 48 non-BPD)  |  disorders) | (4 years) | Borderlines (≥6)  | social functioning; treatment utilisation |
| Bornovalova  | 2009 | USA | 100% | 1118 14-17 year olds  | Community (N/A)  | Prospective  | Multidimensional Personality  | Mean-level and rank order stability of |
|   |   |   |   | 390 MZ; 250 DZ twins |   | (10 years)  | Questionnaire (continuous) |  BPD symptoms |
| Bornovalova  | 2013 | USA | 100% | 1, 280 14-18 year olds | Community (N/A)  | Prospective  | Minnesota Borderline Personality  | Substance abuse |
|   |   |   |   | 390 MZ; 250 DZ twins |   |  (4 years) | Disorder Scale (continuous) |   |
| Chanen  | 2004 | Australia | 63% | 101 15-18 year olds | Clinical (outpatients)  | Prospective | Structured Clinical Interview -II | Categorical and dimensional stability of  |
|   |   |   |   |   |   |  (2 years) | (>5, continuous) |  BPD symptoms |
| Chen | 2004 | USA | 52% | 200 16 year olds | Community | Longitudinal  | Children in the Community-Self | Partner conflict |
|   |   |   |   |   |   | (10 years) | Report (continuous) |   |
| Cohen | 2007 | USA | 50% | 749 Mage13.7 years | Community | Prospective  | Children in the Community-Self | Substance use disorder |
|   |   |   |   |   |   | (9 years) | Report (continuous) |   |
| Crick | 2005 | USA | 54% | 400 (54% F) 9-12 years | Community (N/A) | Prospective  | Borderline Personality Features | Mean-level stability of BPD symptoms |
|   |   |   |   |   |   | (1 year) | Scale for Children |   |
| Garnet  | 1994 | USA | 52% | 21 15-19 year olds  | Clinical  | Prospective | Personality Disorder  | Stability of BPD diagnosis |
|  |  |  |  | (all BPD at baseline) |  | (2 years) | Examination |  |
| Grilo  | 2001 | USA | 48% | 60 15-19 years | Clinical  | Prospective | Personality Disorder  | Stability of BPD symptoms |
|   |   |   |   |   |   | (2 years) | Examination |   |
| Jovev | 2013 | Australia | 51% | 245 11-13 years | Community (% of large | Prospective | Children in the Community-Self | Stability of BPD symptoms |
|   |   |   |   |   |  sample from schools) | (2 years) | Report (continuous) |   |
| Lofgren  | 1991 | USA | 26% | 32 6-10 year olds | Clinical (no control group) | Prospective | “Borderline” a criteria delineated  | Axis I and Axis II disorders; education;  |
|   |   |   |   | (all BPD at baseline) |   | (10-20 years) |  in [Bemporad *et al.* (1982)](#_ENREF_5) | Employment |
| a “Borderline” diagnosis includes sections on: fluctuation in functioning; anxiety; thought content and processes; relationships to others; lack of control; associated symptoms  |
|  |  |  |  |  |  |  |  |  |
| **Table 1. Details of Studies Reporting on Clinical and Psychosocial Outcomes of BPD in Childhood and Adolescence** |
| **First****Author** | **Year** | **Country** | **Percent****female** | **Baseline****(N, age, % BPD)** | **Sample Frame****(control group)** | **Study****Design** | **BPD assessment****(cut-point for diagnosis)** | **Outcomes** |
| Mattanah  | 1995 | USA | 44% | 70 12-18 years | Clinical - inpatients | Prospective | Personality Disorder  | Stability of BPD disorder and symptoms |
|   |   |   |   |  (31 BPD at baseline) |   | (2 years) | Examination |   |
| Meijer | 1998 | Netherlands | 50% | 54 Mage 15.2 years | Clinical - inpatients | Prospective | Diagnostic Interview for | Stability of BPD disorder and symptoms |
|   |   |   |   |   |   | (3.3 years) | Borderlines (≥7) |   |
| Stepp  | 2014 | USA | 100% | 2, 212 14 year olds | Community - oversampled for  | Prospective  | IPDE-BOR for details, | Mean level BPD symptoms |
|   |   |   |   |   | low income) | (4 years) | see [Loranger *et al.* (1994)](#_ENREF_41) |   |
| Wenning | 1990 | USA | 47.4% | 57 8 year olds  | Clinical | Follow-up study | “Borderline” b  criteria delineated  | PD diagnosis; psychiatric symptoms  |
|  |  |  |  | (all BPD at baseline) | (no control group) | (10 years) | in [Vela *et al.* (1983)](#_ENREF_60) |  |
| Winograd  | 2008 | *USA* | 50% | 748 9-18 year olds  | Community | Prospective | Children in the Community-Self | BPD symptoms/diagnosis; social function;  |
|   |   |   |   |   |   | (20 years) | Report (continuous) | education; employment; service utilisation |
| Zelkowitz  | 2007 | *Canada* | 19% | 94 7 - 12 year olds | Clinical - inpatients | Prospective | Diagnostic Interview for | BPD diagnosis; psychopathology;  |
|   |   |   |   | (41 BPD; 53 non-BPD) |   | (5 years) | Borderlines (≥7) | functioning |
| a IPDE-BOR: International Personality Disorders Examination, Borderline Scale; 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 |

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| **Table 2. Quality Assessment of Studies included in the Review** |
| **First Author** | **Selection bias**  | **Performance bias** | **Attrition bias** | **Detection bias** | **Reporting bias** |
|  | **(i: sequence; ii: baseline assessment)** |  |  |  |  |
| ***Bernstein 1993*** | **i.** Part of children in community sample - randomly selected | No indication as community sample | 94% retained for follow-up assessment | Interviewers assessed the children | All pre-specified assessments reported |
|  | **(Low Risk)** | **(Low Risk)** | **(Low Risk)** | blindly | **(Low risk)** |
|  | **ii.** Blind assessment by interviewers (**Low Risk)** |  |  | **(Low Risk)** |  |
| ***Biskin 2011*** | **i.** All patients referred to a treatment program for adolescent | BPD group received specialised treatment  | 63% of BPD patients retained  | Diagnosing clinician involved in BPD  | All pre-specified assessments (including |
|  | girls with BPD; Comparison sample - patients assessed by  | could have impacted on outcome | 16/48 control patient retained | assessments at both time-points (**High Risk)** | non-significant results) reported |
|  | same clinic during same time period **(High Risk)** | **(High Risk)** | **(High Risk)** | Other outcomes assessed by same clinician | **(Low risk)** |
|   | **ii.** No assessment concealment **(High Risk)** |   |   | **(High Risk)** |  |
| ***Bornovalova 2009*** | **i.** Twin sample thus selection bias **(High Risk)** | No indication as community sample | Attrition rates 5-10% for any given  | Questionnaire but blinding methods | All pre-specified assessments reported |
|  | **ii.** Assessment concealment unclear though self-report | **(Low Risk)** | assessment **(High Risk)** | not specified **(Unclear Risk)** | **(Low risk)** |
|   |  **(Unclear Risk)** |   |   |   |   |
| ***Bornovalova 2013*** | **i.** Twin sample thus selection bias **(High Risk)** | Some participants received varying levels  | Not reported in this study, though | Questionnaire but blinding methods | All pre-specified assessments reported |
|  | **ii.** Assessment concealment unclear though self-report | of treatment could have impacted on | reported in previous study with same | not specified **(Unclear Risk)** | **(Low risk)** |
|   | **(Unclear Risk)** | outcome **(Low Risk)** | sample **(High Risk)** |   |   |
| ***Chanen 2004*** | **i.** 147 (101 agreed) patients selected from 418 acute referrals | Variations in inpatient care across sample | 96% retained for follow-up assessment | Diagnosing interviewers blind to baseline  | All pre-specified assessments reported |
|  |  **(High Risk)** | could have impacted on outcome  | **(Low Risk)** | assessment **(Low Risk)** | **(Low risk)** |
|   | **ii.** Assessment concealment **(Low Risk)** | **(High Risk)** |   |   |   |
| ***Chen 2004*** | **i.** Part of children in community sample - randomly selected | No indication as community sample | Follow - up assessments depended on  | Narrative interviews carried out by | All pre-specified assessments reported |
|  | **(Low Risk)** | **(Low Risk)** | willingness to participate in lengthy | accessors blind to previous data | **(Low risk)** |
|   | **ii.** Assessment concealment **(Low Risk)** |   | interviews **(High Risk)** | **(Low Risk)** |   |
| ***Cohen 2007*** | **i.** Part of children in community sample - randomly selected | No indication as community sample | 85% retained for follow-up assessment | Questionnaire but blinding methods | All pre-specified assessments reported |
|  | **(Low Risk)** | **(Low Risk)** | **(High Risk)** | not specified **(Unclear Risk)** | **(Low risk)** |
|   | **ii.** Assessment concealment **(Low Risk)** |   |   |   |   |
|  |  |  |  |  |  |
|  |  |  |  |  |  |
| **Table 2. Quality Assessment of Studies included in the Review** |
| **First Author** | **Selection bias** **(i. sequence; ii. baseline assessment)** | **Performance bias** | **Attrition bias** | **Detection bias** | **Reporting bias** |
| ***Crick 2005*** | **i.** Sub-sample of ongoing longitudinal study (High Risk) | No indication as community sample | 57% of sample retained | Questionnaire but blinding methods | All pre-specified assessments reported |
|  | **ii.** Assessment concealment unclear though self-report | **(Low Risk)** | **(High risk)** | not specified **(Unclear Risk)** | **(Low risk)** |
|   | **(Unclear Risk)** |  |  |   |  |
| ***Garnet 1994*** | **i.** Subjects were a small subset of patients from the Yale  | Sample receiving inpatient treatment | Not reported in this study, though small  | Diagnosing interviewers blind to baseline  | All pre-specified assessments reported |
|  |  Psychiatric Institute study **(High Risk)** | **(High Risk)** | sub-set of larger outcome study suggests  | assessment **(Low Risk)** | **(Low risk)** |
|   | **ii.** Assessment concealment not specified (**Unclear Risk)** |   | high attrition **(High Risk)** |   |   |
| ***Grilo 2001*** | **i.** Subjects were a subset of patients from the Yale Psychiatric  | Sample receiving inpatient treatment | 36.36% retained for follow-up  | Diagnosing interviewers blind to baseline  | All pre-specified assessments reported |
|  |  Institute study **(High Risk)** | **(High Risk)** | assessment **(High Risk)** | assessment **(Low Risk)** | **(Low risk)** |
|  | **ii.** Interviewers functioned independently to clinical team |  |  |  |  |
|   | **(Low Risk)** |   |   |   |   |
| ***Jovev 2013*** | **i.** Children over selected for extreme temperament traits (of | No indication as community sample | 83.67% retained for follow-up | Questionnaire but blinding methods | All pre-specified assessments (including |
|  |  which only 59% consented to participate) **(High Risk)** | **(Low Risk)** | assessment **(High Risk)** | not specified **(Unclear Risk)** | non-significant results) reported |
|   | **ii.** Blinding not specified **(Unclear Risk)** |   |   |   | **(Low risk)** |
| ***Lofgren 1991*** | **i.** Selected from a large sample of hospitalised children - male  | Receiving hospital treatment  | 59.38% retained for follow-up  | Blind assessment at follow-up | All pre-specified assessments reported |
|  | bias **(High Risk)** | **(High Risk)** | assessment **(High Risk)** | **(Low Risk)** | **(Low risk)** |
|   | **ii.** Three independent ratings **(Low Risk)** |   |   |   |   |
| ***Mattanah 1995*** | **i.** Hospitalised adolescents from the Yale Psychiatric Institute | Sample receiving inpatient treatment | 39.39% retained for follow-up  | Blind assessment at follow-up | All pre-specified assessments reported |
|  | **(High Risk)** | **(High Risk)** | assessment **(High Risk)** | **(Low Risk)** | **(Low risk)** |
|   | **ii.** Assessment concealment not specified **(Unclear Risk)** |   |   |   |   |
| ***Meijer 1998*** | **i.** Consecutive admissions to two (long and short stay) inpatient | Sample receiving inpatient treatment | 66.67% retained for follow-up assessment | Blind assessment at follow-up | All pre-specified assessments reported |
|  | facilities **(High Risk)** | **(High Risk)** | **(High Risk)** | **(Low Risk)** | **(Low risk)** |
|   | **ii.** Author conducted baseline assessments **(High Risk)** |   |   |   |   |
| ***Stepp 2014*** | **i.** Pittsburgh Girls Study oversampled for low-income | No indication as community sample | 90.25% retained | Questionnaire but blinding methods | All pre-specified assessments reported |
|  |  neighbourhoods **(High Risk)** | **(Low Risk)** | **(High Risk)** | not specified **(Unclear Risk)** | **(Low risk)** |
|   | **ii.** Assessment concealment of interviewers unclear **(High Risk)** |   |   |   |   |
|  |  |  |  |  |  |
| **Table 2. Quality Assessment of Studies included in the Review** |
| **First Author** | **Selection bias** **(i. sequence; ii. baseline assessment)** | **Performance bias** | **Attrition bias** | **Detection bias** | **Reporting bias** |
| ***Wenning 1990*** | **i.** Children selected from a residential treatment service if  | Receiving hospital treatment  | 49.12% retained for follow-up  | Author involved in baseline and follow-up | Some assessments not reported on, e.g.,  |
|  | clinicians believed likely to be borderline **(High Risk)** | **(High Risk)** | assessment **(High Risk)** | interviews **(High Risk)** | social functioning, school performance  |
|   | **ii.** Blind corroboration of BPD diagnosis **(Low Risk)** |   |   |   | **(High Risk)** |
| ***Winograd 2008*** | **i.** Part of children in community sample - randomly selected | No indication as community sample | 85% retained for follow-up assessment | Blinding not specified | All pre-specified assessments  |
|  | **(Low Risk)** | **(Low Risk)** | **(High Risk)** |  **(Unclear Risk)** | (including non-significant reported |
|  | **ii.** Questionnaire assessment but blinding methods unclear |  |  |  | results) **(Low risk)** |
|   | **(Unclear Risk)** |   |   |   |   |
| ***Zelkowitz 2007*** | **i.** Children referred to a day hospital - male bias **(High Risk)** | Receiving treatment  | 38.30% retained for follow-up  | Blind assessment at follow-up | All pre-specified assessments reported |
|   | **ii.** Interviewer blind to other diagnoses **(Low Risk)** | **(High Risk)** | (**High Risk)** | **(Low Risk)** | **(Low risk)** |

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