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A Systematic Review of Risk Factors Prospectively Associated with Borderline Personality Disorder: Taking Stock and Moving Forward

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Abstract

There is an urgent need to identify signs that harbinger onset of borderline personality disorder (BPD). Advancement in this area is required to refine developmental theories, discover etiological mechanisms, improve early detection, and achieve our ultimate goal of prevention. Though many studies have supported a wide range of factors that increase subsequent risk for BPD, this literature has yet to be critically evaluated, and there are no comprehensive reviews that examine and integrate these findings. To address this limitation, we conducted a systematic review to summarize and synthesize the current literature. Electronic databases were systematically searched for prospective, longitudinal studies that examined risk factors of subsequent BPD outcomes (features, symptoms, diagnosis) resulting in a total of 39 studies, reflecting 24 unique samples. Though increased risk for BPD was reliably attributed to multiple factors within social, family, maltreatment, and child domains, the most striking limitation of this research is its lack of disorder-specific findings Additional limitations, including notable heterogeneity in sampling methodology, symptom assessment methodology, and developmental timing of assessments, are discussed in terms of how close are we to pinpointing *who* is most at risk and *why* in an attempt to provide a roadmap for future research.

Keywords

systematic review; borderline personality disorder; risk factors; development

Borderline personality disorder (BPD) is a serious mental illness that typically emerges during adolescence or young adulthood and is characterized by multiple debilitating symptoms, including emotional dysregulation, tumultuous interpersonal relationships, and impulsive behaviors (Chanen, 2015; Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004). The disorder is associated with a high mortality rate: up to 10% of patients commit suicide (Paris & Zweig-Frank, 2001; Zanarini, Frankenburg, Hennen, & Silk, 2003). Furthermore, severe

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psychosocial impairment can linger for decades after remission (Bagge et al., 2004; Paris & Zweig-Frank, 2001; Skodol et al., 2005). The level of impairment associated with BPD and its public health significance are reflected in its prevalence across adult and adolescent clinical settings, with approximately 10–20% of outpatients and up to 50% of inpatients meeting criteria (Glenn & Klonsky, 2013; Korzekwa, Dell, Links, Thabane, & Webb, 2008; Widiger & Weissman, 1991). These devastating consequences speak to the urgent need to identify signs that harbinger onset of the acute, fully developed illness. Successfully identifying such markers would hasten the refinement of developmental theories, accelerate the discovery of etiological mechanisms, enable early detection and diagnosis, and may provide fruitful targets for early intervention. Advancements in these areas are required to achieve our ultimate goal: deflecting the course of personality development away from BPD outcomes.

Along these lines, there has burgeoning interest in early risk factors for the disorder throughout the past decade (Kongerslev & Chanen, 2015). Much of this work supports a wide range of factors that increase subsequent risk for BPD, including broad social indices, family influences, exposure to maltreatment and trauma, and various child characteristics; however, these findings have yet to be thoroughly evaluated. So while this research may signal progress toward our ultimate goal, a comprehensive examination of the existing literature is critical for integrating our current knowledge, identifying remaining gaps, and providing a roadmap for future research. Therefore, we performed a systematic review of all longitudinal, prospective studies that examined risk factors for the development of BPD. Our goals were two-fold. First, to summarize and synthesize results across identified studies, including detailing risk factors that are consistently supported and discussing strengths and limitations of the existing literature. Second, we sought to determine if prevailing evidence regarding BPD risk factors facilitates sharpening developmental theories, explaining etiology, and identifying those in need of early intervention. In sum, how close are we to pinpointing *who* is most at risk and *why*?

Method

We obtained all peer-reviewed, English-language studies published through September 2015 from (a) PubMed, CINAHL, PsycINFO, and ISI Web of Science databases using the following search algorithm: (*borderline personality* AND [*longitudinal* OR *follow-up** OR *prospect**] AND [*precursor** OR *risk factor** OR *prodrom** OR *antecedent** OR *predict**] AND [*diagnosis* OR *development*]); (b) hand searches of reference lists in identified studies and relevant review papers. Eligibility status for all retrieved articles was determined in two stages. First, all studies were screened based upon title and abstract using EPPI-Reviewer software (Thomas, Brunton, & Graziosi, 2010). Next, studies passing the initial title and abstract screen were reviewed based upon the full manuscript. Articles selected for final analysis met the following criteria: (1) prospective, longitudinal studies of any follow-up duration with at least 2 assessment points (2) outcome included BPD features, symptoms, or diagnosis and (3) the predictor (i.e., putative risk factor) was measured prior to the outcome assessment of BPD. Additionally, studies were excluded if they met the following criteria at either stage: (1) tested an intervention, as its impact on the risk factor and BPD outcome could not be determined; or (2) focused solely on heritability or genetic factors, as these are

fixed markers and not sensitive to time of assessment. The three authors performed independent evaluations based on PRISMA-P (Shamseer et al., 2015) guidelines, with methods and inclusion criteria specified in advance and documented in a protocol. Any disagreements between reviewers were resolved by consensus.

We identified a total of 39 studies, resulting from 24 unique samples that met the aforementioned criteria (Figure 1). Participants totaled 43,681 (range = 56 – 6050), the majority of which were female (54%) and Caucasian (69%).[1] Only eight of the 39 included studies utilized clinical samples (23%) and the vast majority used community samples (n = 31, 73%), of which 13 (42%) were 'high-risk' as defined by selection or oversampling based on a putative specific risk factor (i.e., poverty). Across all included studies, risk factors were assessed, on average, at 13 years (SD = 5.9 years; range = birth – 29 years). Outcome assessments occurred between ages 12 and 43 (M = 20 years; SD = 5.4 years), with the majority of studies examining symptoms (n = 29, 74%), eight studies (21%) examining diagnosis, and one study examining features (Belsky et al., 2012). The time between the risk factor assessment and the follow-up BPD assessment ranged from 1 to 28 years (M = 10.5 years, SD = 7 years). Retention of original sample was acceptable (M = 76%, SD = 16%); however, rates varied substantially (39% – 97%).[2]

For the purpose of the current systematic review, studies were categorized based on type of risk examined: (1) broader social factors (e.g., poverty, stressful life events); (2) family factors, including parent psychopathology, parenting behavior/style, and family climate/parent-child relationship; (3) maltreatment and other trauma exposures (e.g., physical abuse, sexual abuse, neglect); and (4) child factors, including cognitive ability, attachment to caregiver, temperament and personality, and psychopathology. Results are discussed within each domain; studies including more than one risk factor appear multiple times. Analyses conducted in multiple steps are discussed in terms of findings from final analyses. Significant variability in study characteristics precluded statistical evaluation of predictive power and effect sizes.

Results

Broader Social Risk Factors

We identified nine studies that examined one or more indices of broader social risk (Table 1). Four studies assessed low socioeconomic status (SES) and results consistently supported a prospective relationship with later BPD outcomes. Crawford and colleagues (2009) found family SES at age 5 to predict BPD symptoms more than 20 years later. Relatedly, Cohen and colleagues (2008) demonstrated a protracted and stable relationship between family SES and BPD symptom trajectories across adolescence and adulthood. Finally, Stepp and colleagues (2014a; 2014b) found receipt of public assistance to predict BPD symptoms across adolescence.

[1]Notably, the number of participants was reduced to 18,238 when examining only the unique samples (n=24); the majority remained female (51%) and Caucasian (69%).

[2]Clinical samples had lower retention rates compared to community samples, (clinical: M = 62%, SD = 13.5%; community: M = 79%, SD = 19.2%; $F(1, 33) = 6.8$, $p = 0.008$); other characteristics did not significantly vary by sample type. Retention rate was not significantly correlated with any other sample characteristic.

Results from an additional four studies demonstrated a fairly consistent link between life stress and later BPD. Stressful life events (i.e., various psychosocial stressors) as measured in infancy, childhood, and adolescence predicted BPD symptoms in adulthood (Carlson, Egeland, & Sroufe, 2009; Cohen et al., 2008). Additionally, chronic (but not acute) family and school stressors in adolescence were linked to BPD symptoms in adulthood (Conway, Hammen, & Brennan, 2015). In contrast, Greenfield and colleagues (2015) did not find an effect of stressful life events measured at age 15 on the likelihood of BPD diagnosis approximately 3 years later. Stark differences in sampling strategies may explain these disparate findings.[3]

Four studies examining the association between family adversity and BPD symptoms produced mixed results, which may be attributable to variability in measurement of family adversity. Positive findings were evident in two community studies examining family adversity (broadly defined) during pregnancy (Winsper, Zanarini, & Wolke, 2012) and across childhood and adolescence (Stepp, Scott, Jones, Whalen, & Hipwell, 2015). Two additional community samples examined more specific indices, namely family disruption (Carlson et al., 2009) and marital conflict (Crawford et al., 2009), no significant prospective associations were found.

Family Factors

Nineteen studies assessed family factors (Table 2) and seven examined multiple indices.

Parent/Family psychopathology—Ten studies assessed family psychopathology as a risk factor, with seven studies reporting significant positive associations. Six of these studies focused on maternal (or caregiver) psychopathology and found significant associations with offspring BPD. These risk factors included maternal internalizing (Stepp et al., 2014b; Winsper, Wolke, & Lereya, 2015) and externalizing (Conway et al., 2015; Stepp et al., 2014b) disorders as well as maternal BPD (Barnow, Aldinger, Arens, & Ulrich, 2013; Reinelt et al., 2014; Stepp et al., 2014b). Furthermore, Reinelt and colleagues (2014) found an indirect effect of maternal BPD symptoms on offspring BPD symptoms via maladaptive parenting behaviors. Stepp and colleagues (2013) also found that paternal substance use predicted offspring BPD symptoms. Lastly, Belskey and colleagues (2012) found family history of psychiatric hospitalization interacted with maltreatment and maternal negative emotion to predict BPD characteristics.

Three studies examined other maternal characteristics, including maternal ego integration, impulsivity, interpersonal difficulties, and history of serious medical problems (Bezirganian, Cohen, & Brook, 1993; Carlson et al., 2009; Crawford et al., 2009). There were no associations between these maternal factors and later BPD.

Parenting behavior/style—Twelve studies examined risk factors in this subdomain, with the majority focusing on affective parenting dimensions. Results provided consistent

[3]Studies examined community (Carlson et al. 2009; Cohen et al., 2008; Conway et al., 2015) versus clinical samples (i.e., Greenfield et al., 2015). It is also noteworthy that a sizeable portion of the clinical sample already met criteria for BPD at baseline, resulting in a small group of adolescents with emerging BPD between the baseline and follow-up period ($n = 7$; 3.43% of the sample).

evidence for prospective associations between higher BPD symptoms and affective dimensions, such as low warmth, rejection, and low maternal satisfaction with the child (Crawford et al., 2009; Reinelt et al., 2014; Stepp et al., 2014b) as well as hostility and harsh discipline/punishment (Hallquist, Hipwell, & Stepp, 2015; Stepp et al., 2014b; Winsper et al., 2012; Wolke, Schreier, Zanarini, & Winsper, 2012). Moreover, Stepp and colleagues (2014b) found evidence of reciprocal associations between low warmth, harsh punishment, and BPD symptoms across adolescence, such that low warmth and harsh punishment predicted subsequent increases in BPD symptoms; in turn, BPD symptoms predicted subsequent increases in parental levels of low warmth and harsh punishment. While two studies failed to find predictive associations with low affection and harsh discipline (Bezirganian et al., 1993; Johnson, Cohen, Chen, Kasen, & Brook, 2006), it is noteworthy that these were two of the only studies that predicted *onset* of BPD rather than greater mean levels or increases in BPD symptoms.

Those studies examining affective parenting dimensions as they unfold in moment-to-moment observational paradigms found similar results. Specifically, Lyons-Ruth and colleagues (2013) showed disrupted maternal communication at 18 months to predict BPD symptoms at age 18, and Carlson and colleagues (2009) found maternal hostility at 42 months to predict BPD symptoms at age 28. Similarly, Belskey and colleagues (2012) found that maternal expressed negative emotion in middle childhood significantly predicted BPD features at age 12.

Two additional studies examined the influence of behavioral control dimensions of parenting, though findings were somewhat mixed. Bezirganian and colleagues (1993) found a significant interaction between maternal inconsistency and over-involvement at age 14, such that higher levels of both predicted BPD diagnosis at age 16. Conversely, Crawford and colleagues (2009) failed to find an association between inconsistent parenting and BPD symptoms. One final study found a significant association between poor parenting (i.e., both affective and behavioral dimensions) and BPD symptoms in adolescence and adulthood (Cohen et al., 2008).

Family climate and parent-child relationship—Only two of five studies found evidence supporting a prospective link between this sub-domain and subsequent BPD. Stepp and colleagues (2013) found that mother-child discord in adolescence predicted BPD symptoms at age 30 and Hammen and colleagues (2015) found that a broad index of family relationship quality predicted BPD symptoms for those with the OXTR risk genotype. However, the majority of studies did not find support for this risk factor (Bezirganian et al., 1993; Carlson et al., 2009; Greenfield et al., 2015). Overall, these studies used broad observational and self-report measures that may have obfuscated the dynamic, bidirectional nature of parent-child relationships.

Maltreatment and Other Trauma

Fifteen studies examined exposure to maltreatment and other trauma, including forms of child abuse (i.e., physical, sexual, emotional, or verbal abuse) and neglect (i.e., poor parental care, poor supervision, maternal separation); “other” traumatic experiences; and peer

victimization (Table 3). Eight studies found fairly consistent evidence supporting abuse as a risk factor, including associations with physical abuse (Belsky et al., 2012; Bornovalova et al., 2013b; Carlson et al., 2009; Johnson, Cohen, Brown, Smailes, & Bernstein, 1999), verbal abuse (Johnson et al., 2001), emotional abuse (Bornovalova et al., 2013b), and sexual abuse (Bornovalova et al., 2013b; Carlson et al., 2009; Johnson et al., 1999; Stepp et al., 2015). Similarly, evidence consistently supported the link between BPD and neglect, defined broadly (Johnson et al., 1999), or by specific neglectful experiences: early maternal separation (Crawford et al., 2009), inadequate supervision (Johnson, Smailes, Cohen, Brown, & Bernstein, 2000) and poor parental care (Lyons-Ruth et al., 2013). Three of the five studies which examined maltreatment as a composite of types of abuse and neglect also provided support for increased risk for BPD outcomes (Carlson et al., 2009; Crawford et al., 2009; Johnson et al., 1999).

Five studies failed to find an association between abuse or neglect and later BPD: physical and sexual abuse (Thatcher, Cornelius, & Clark, 2005; Wolke et al., 2012), neglect (Carlson et al., 2009), and combined indices of maltreatment (Greenfield et al., 2015; Widom, Czaja, & Paris, 2009). Additionally, two studies that combined indices of maltreatment and other trauma had discrepant findings. Cohen and colleagues (2008) examined “cumulative trauma” (e.g., parent arrest or imprisonment, family suicide, death of a parent), and found a significant association with BPD across adolescence and early adulthood. Conversely, Krabbendam and colleagues (2015) did not find their combined index (i.e., physical or sexual abuse and “other” trauma, e.g., car accident) to predict BPD symptoms in an incarcerated sample of adolescents.

One final study examined exposure to maltreatment and trauma in the form of peer victimization in late childhood (Wolke et al., 2012). Findings demonstrated that both the severity and chronicity of bullying was prospectively associated with BPD symptoms at age 12.

Child Factors

Child factors were examined in 29 studies (Table 4), and eight assessed multiple factors.

Cognitive function—Findings from four studies (three unique samples) supported low IQ, measured from age 5 through adolescence, as a risk factor in adolescence and adulthood (Belsky et al., 2012; Cohen et al., 2008; Winsper et al., 2012; Wolke et al., 2012).

Attachment—Findings were mixed from three studies examining insecure or disorganized attachment. Positive associations were noted when attachment was measured in late childhood and adolescence; disorganized/controlling behavior at age 8 predicted BPD symptoms at age 19 (Lyons-Ruth et al., 2013), and insecure attachment, assessed at age 16 via a questionnaire about peer relationships, predicted BPD across adolescence and adulthood (Crawford et al., 2009). However, attachment disorganization and security, assessed in infancy and toddlerhood, was not associated with BPD symptoms in adulthood (Carlson et al., 2009; Lyons-Ruth et al., 2013).

Temperament/Personality—All 12 studies examining temperament or personality factors demonstrated positive associations with later BPD. Consistent links between negative affectivity (e.g., emotionality, affective instability, angry/tantrums), impulsivity (e.g., low constraint, low self-control, effortful control) and BPD symptoms in adolescence and adulthood were demonstrated (Belsky et al., 2012; Carlson et al., 2009; Crawford et al., 2009; Hallquist et al., 2015; Jovev et al., 2013; Lenzenweger & Desantis Castro, 2005; Stepp et al., 2015; Stepp, Keenan, Hipwell, & Krueger, 2014a; Stepp et al., 2014b; Tragesser et al., 2010; Tragesser, Solhan, Schwartz-Mette, & Trull, 2007). In an examination of more complex processes, Hallquist and colleagues (2015) found poor self-control predicted BPD symptoms via reciprocal effects between poor self-control and parental harsh discipline. Using the same sample, Stepp and colleagues (2015) showed an interaction between negative affectivity and family adversity, such that higher levels of both predicted the highest levels of BPD symptoms across adolescence.

Sharp and colleagues (2015) found that a higher level of experiential avoidance (e.g., tendency to avoid unpleasant thoughts, emotions) was associated with an increase in BPD features over 1 year. Additionally, Carlson and colleagues (2009) demonstrated that disturbances in self-representation (rated from narrative projective tests) predicted BPD symptoms at age 28.

Psychopathology—Sixteen of the 19 studies examining psychopathology as a predictor of later BPD detected at least one significant prospective relationship. In studies examining internalizing psychopathology (i.e., anxiety, depression, dissociation, suicide), consistent links with subsequent BPD were reported (Belsky et al., 2012; Bornovalova et al., 2013b; Conway et al., 2015; Krabbendam et al., 2015; Ramklint, Knorring, Knorring, & Ekselius, 2003; Sharp et al., 2015; Stepp et al., 2013; Thatcher et al., 2005; Widom et al., 2009). Though three studies did not find any evidence of a prospective association between internalizing disorders and BPD (Burke & Stepp, 2012; Miller et al., 2008; Rey, Morris-Yates, Singh, Andrews, & Stewart, 1995), it is notable that these studies utilized clinical samples that were mostly male.

Studies of externalizing psychopathology (i.e., attention deficit hyperactivity disorder, oppositional defiant disorder, conduct disorder, substance use) produced consistent associations with BPD outcomes (Belsky et al., 2012; Bornovalova et al., 2013b; Bornovalova, Hicks, Iacono, & McGue, 2013a; Burke & Stepp, 2012; Conway et al., 2015; Miller et al., 2008; Ramklint et al., 2003; Rey et al., 1995; Stepp et al., 2013; Stepp, Burke, Hipwell, & Loeber, 2012; Stepp et al., 2014b; Thatcher et al., 2005). The only study that did not detect a prospective relationship between externalizing disorders and BPD was in an incarcerated sample of adolescents, with high rates of behavioral and emotional problems at baseline (Krabbendam et al., 2015).

Two studies that failed to find any relationship between psychopathology and BPD were conducted in clinical samples. Thomsen and Mikkelsen (1993) did not find an association between obsessive-compulsive disorder and BPD diagnosis using an inpatient sample. Similarly, in sample of adolescents recruited from a psychiatric hospital, internalizing and externalizing disorders failed to predict BPD diagnosis (Greenfield et al., 2015). Finally,

Winsper and colleagues (2012) did not find a relationship between DSM-IV Axis I disorders at 8 years and BPD symptoms at age 12. However, using the same community sample, Wolke and colleagues (2012) reported bivariate associations between any childhood disorder and BPD symptoms.

Other infant factors—Carlson and colleagues (2009) examined two additional infant characteristics as BPD risk factors (i.e., infant anomalies at birth and overall "non-optimal" functioning) and neither factor was significantly associated with BPD symptoms in adulthood.

Discussion

Results clearly supported multiple factors across social, family, maltreatment, and child domains that increase risk for subsequent BPD outcomes. The most robust risk indicators were low SES, stressful life events, and family adversity in the social domain; maternal psychopathology and affective parenting dimension (low warmth, hostility, harsh punishment) in the family domain; exposure to physical or sexual abuse or neglect in the maltreatment domain; and low IQ, high levels of negative affectivity and impulsivity, and internalizing and externalizing psychopathology, in the child domain. In and of itself the very existence of research capable of examining the development of BPD signals progress in the field, and the plethora of longitudinal studies conducted in both community (e.g., Children in the Community, CIC; Pittsburgh Girls Study, PGS) and clinical samples is remarkable. Taken together, this body of research has improved our understanding of early risk factors of BPD and further strengthened the construct validity of the disorder in adolescence and adulthood (Chanen, 2015).

At the same time, the most striking limitation of this research is its lack of specificity. Previous research demonstrates a nearly identical risk profile for a broad range of internalizing (Hankin, 2006; Murray, Creswell, & Cooper, 2009; Sander & McCarty, 2005) and externalizing (Deater-Deckard, Dodge, Bates, & Pettit, 1998; Shaw, Owens, Vondra, Keenan, & Winslow, 1996) disorders. Consequently, we can conclude that there is a shared set of risk factors that predict poor mental health outcomes (World Health Organization, 2012) and thus, at some level, these factors operate in a similar fashion across many psychiatric disorders. This highlights the concept of multifinality (Cicchetti & Rogosch, 1996), suggesting that we are missing critical elements required to explain divergent trajectories for unique disorder outcomes. Additionally, there was tremendous heterogeneity across several critical study features, including approaches to sample and assessment methodology, and the developmental timing of these assessments. The current systematic review provides an ideal opportunity to weigh the strengths and limitations inherent across study designs as we develop a roadmap for future research endeavors.

Effects of Sampling Methodology

First and foremost, it is imperative to highlight that while 39 studies met our inclusion criteria, only 24 unique samples are represented. For example, almost half of studies demonstrating a positive link between maltreatment and BPD were conducted in the same sample (i.e., 5 of 11 studies used the CIC cohort: Johnson et al., 1999, 2000, 2001; Cohen et

al., 2008; Crawford et al., 2009). Similarly, a third of those studies reporting a positive association between temperament and later BPD utilized the PGS (Hallquist et al., 2015; Stepp et al., 2014a; 2014b; 2015). While this does not necessarily negate these findings, it highlights a potential bias in our interpretation of the strength and consistency of effects. Moving forward, it is critical that we attend to the reproducibility of findings across *unique* samples as opposed to merely counting the sheer number of studies demonstrating significance.

In addition, the consideration of sample ascertainment methods, with regard to interpretation of the current literature and future study design, is imperative as this provides the foundational parameters for the questions we are able to be address. For example, the current review highlights a pattern whereby studies with clinical samples or matched community controls (CC) appeared less likely to detect significant prospective associations between certain risk factors and BPD. Those studies reporting null findings with regard to child psychopathology utilized samples selected on the presence of clinically significant levels of psychopathology (Thomsen et al., 1993; Greenfield et al., 2015). Similarly, maltreatment and trauma were not supported as risk factors of BPD in studies selecting participants based on exposure to maltreatment or incarceration status (Widom et al., 2009; Krabbendam et al., 2015). This can likely be attributed to the sampling of extreme groups *based on the very risk factor of interest*, which precludes examination of the full range of variability. However, since clinical studies are characterized by high levels of clinical severity and are more likely to capture onset of the fully developed illness, they are ideal to examine *onset*. Moving forward, it is important to consider the strengths and limitations of the study design and critically evaluate its ability to uncover the mechanisms that explain the eventual manifestation of BPD.

Effect of Assessment Methodology

Measurement precision is essential as imprecise measurement leads to faulty or incomplete data that can yield flawed conclusions (Rose & Fischer, 2011). Consider two studies examining seemingly similar constructs, family relations (Greenfield et al., 2015) and family relationship quality (Hammen et al., 2015); note their drastically different operationalization: a single “overall measure of intra-family stresses” versus “a multi-method, multi-informant index…of ongoing marital and parental relationship quality,” respectively. It is highly likely that these studies are suffering from the *jingle fallacy*, as they are utilizing common terms to reference different underlying constructs (Thorndike, 1904). Relatedly, the current literature lacked consistency in the measurement of risk factors, including use of informants (parent, child, official documentation), and assessment method (questionnaire, interview, observation). The notable exception was in the domain of child temperament and was the one area in which all identified studies demonstrated complete consistency in their findings. Finally, there was considerable heterogeneity in the measurement of BPD. Specifically, several studies utilized aggregated post-hoc measures of BPD (i.e., CIC) for which the clinical utility is currently unknown. Moreover, it is imperative that we consider the developmental sensitivity of current measures developed for adults being used during adolescence.

In addition to improving data collection methods, we can also strive to improve our statistical approach to understanding the complex nature of risk processes. Research to date has generally examined risk factors as static, unidirectional influences on subsequent BPD. However, a minority of studies reviewed here sought to uncover more nuanced risk processes and found evidence of moderating (Belskey et al., 2012; Bezirganian et al., 1993; Hammen et al., 2015; Jovev et al., 2013; Stepp et al., 2015) and mediating (Bornovalova et al., 2013b; Hallquist et al., 2015; Reinelt et al., 2014; Winsper et al., 2012) mechanisms. These findings highlight the dynamic, transactional progression of risk across development and caution against the use of statistical approaches that inherently reflect the notion that risk factors exist in a vacuum. Moving forward, we must be cognizant of the balance between the breadth and depth when assessing risk factors. The examination of one risk factor in isolation fails to adequately reflect the innate complexity of the development of BPD. Conversely, the inclusion of too many risk factors may obfuscate key prospective relationships. In sum, examining a parsimonious set of theory-driven risk factors can illuminate processes *by which* and *through which* BPD develops.

Effects of Developmental Timing

Though the current review highlighted a fairly similar risk profile regardless of the developmental stage at which risk factors and outcomes were assessed, it may be premature to conclude that this signifies insensitivity to the developmental timing of risk or the manifestation of BPD. An alternative explanation may be rooted in the protracted follow-up periods assessing risk at a single time point, which could obscure sensitive developmental periods. For instance, a null finding at a single time point does not necessarily reflect a lack of association during developmental periods outside the sampling frame. This is highlighted in discrepant findings regarding exposure to maternal hostility in early childhood (Carlson et al., 2009) versus adolescence (Johnson et al., 2006). On the other hand, a positive association does not necessarily signify a developmentally critical window. It is possible that the positive association is driven by (1) persistence or change in the risk factor that was not captured (2) that the onset of the BPD outcome could have occurred earlier. Both of these scenarios signal that we are missing the mark with the latter being arguably more vital to refining developmental theories of BPD.

Along these lines, the *developmental timing of the disorder* must also be considered. If our ultimate goal is to prevent BPD, we must prioritize the identification of risk factors and processes that predict *onset* (either of the disorder or of its symptoms). Given that onset is a distinct phase in the course of the illness and may be distinguished by its unique risk profile, it is crucial to discriminate onset from other phases (i.e., prodromal, maintenance, remission, recurrence). We appear to have completely ignored this developmental dimension and thus, have fallen victim to the *jangle fallacy* (Kelley, 1973): equating all BPD outcomes as synonymous regardless of phase variation. In fact, only one study reviewed here considered phase of the disorder as an outcome (Greenfield et al., 2015). As we shift our focus to capture the onset phase, we must also shift away from examining distal factors indicative of shared risk for general psychopathology and instead identify *precursors* (i.e., unique proximal indicators) that are specific BPD (Eaton, Badawi, & Melton, 1995). While this point is certainly applicable to the onset of both symptoms and disorder, it is most urgent to

examine precursors of the acute, fully developed illness if our ultimate goal is to prevent BPD and deflect trajectories of chronic illness. Therefore, future studies should consider *stage of the disorder* in tandem with *developmental timing*, which requires (1) repeated assessments of BPD during developmentally sensitive windows that encompass periods of peak prevalence (i.e., adolescence; Miller, Muehlenkamp, & Jacobson, 2008); (2) ascertainment of a sample with a heightened proclivity to onset during the sampling frame; and (3) the retrospective identification of a prodromal phase to determine the most robust set of precursors (Eaton et al., 1995).

In sum, the current state of the field provides little in the way of identifying *who* is most at risk and *why* BPD (versus some other psychiatric disorder) develops and many unanswered questions remain. Given the public health significance concerning the identification, prevention, and intervention of BPD, we have only a meager amount of research on its causes, developmental trajectory, and response to treatment. Although empirically supported treatments are available, very few psychiatric clinics provide this care, which could account for poor treatment outcomes and heightened use of emergency and inpatient services. In terms of refining developmental theories, what are the mechanisms that explain the etiology of BPD? If our goal is early detection, how do we select those at high risk for BPD? Once identified, how do we intervene with children and youth before they develop the full-blown disorder? Progress in answering these questions may come from addressing these gaps. We have highlighted several key avenues for future endeavors pursuing these research priorities in the hopes of encouraging research that will hasten the pace of these discoveries.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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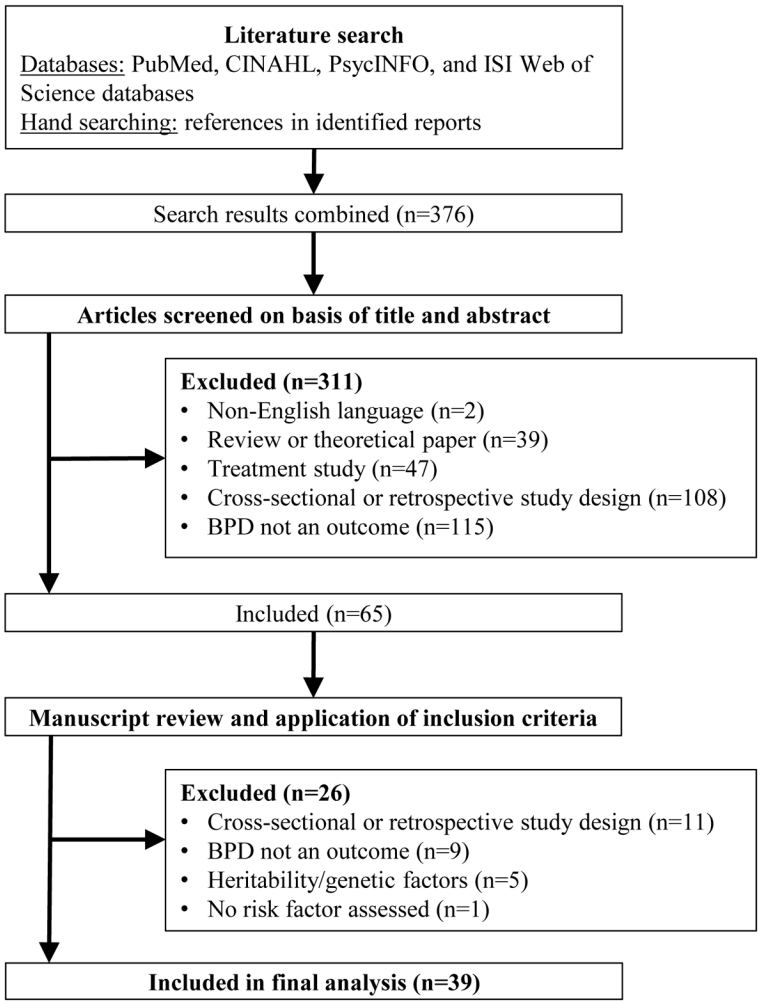

Figure 1.

Study selection flow chart following Preferred Reporting Items for Systematic Reviews and Meta-Analysis Protocols. All articles retrieved from the literature search were first screened on the basis of title and abstract. Those manuscripts deemed eligible from the screening phase were then reviewed using study inclusion criteria. Reasons for study exclusion are listed at each stage. Forty studies were ultimately eligible and included in the final analysis.

Table 1

Summary of Longitudinal Research Investigating Broader Social Risk Factors Predicting Borderline Personality Disorder (BPD)

	Sample			Risk factor		BPD Outcome		
Study	**Characteristics**	**Type (cohort)**	**Follow-up (retention)**	**Construct**	**Age in years**	**Construct (measure)**	**Age in years**	**Summary of results**
Cohen et al. (2008)	n=680; % female NR; 91% Caucasian	Community: Population (CIC)	~20 yrs (70%)	SES	13, 16, 22	BPD sxs (CIC-SR)	13, 16, 22, 33	**Lower SES predicted BPD sxs and effect magnitude remained stable over time.**
				Stressful Life Events	13, 16, 22			**Stressful life events predicted BPD sxs.**
Carlson et al. (2009)	n=162; 49% female; 67% Caucasian	Community: High-Risk	28 yrs (NR)	Life Stress	3–42 mos, 6–11	BPD sxs (SCID-II)	28	**Life stress in infancy/childhood predicted higher BPD sxs.**
				Family Disruption	1–18			NO ASSOCIATION in final analyses. *(Family disruption significant in bivariate analyses)*
Crawford et al. (2009)	n=766; % female NR; 91% Caucasian	Community: Population (CIC)	~20 yrs (78%)	SES	5	BPD sxs (CIC-SR)	13, 16, 22, 33	**Lower SES predicted BPD sxs.**
				Marital Conflict	13			NO ASSOCIATION in analyses.
Winsper et al. (2012)	n=6050; 46% female; 48% Caucasian	Community: Population (ALSPAC)	~12 yrs (43%)	Family Adversity	PREG: 8, 12, 18, 32 wks	BPD sxs (CI-BPD)	12	**Family adversity predicted BPD sxs and was mediated by IQ.**
Stepp et al. (2014a)	n=2282; 100% female; 41% Caucasian	Community: High-Risk (PGS)	14 yrs (93%)	Poverty: Public Assist	6.5	BPD sxs (IPDE-B)	14–19	**Public assistance predicted BPD sxs at age 14.**
Stepp et al. (2014b)	n=2212; 100% female; 41% Caucasian	Community: High-Risk (PGS)	4 yrs (90%)	Poverty: Public Assist	14	BPD sxs (IPDE-B)	14–17	**Public assistance predicted BPD sxs at age 14 and increases in BPD sxs.**
Greenfield et al. (2015)	n=204; 69% female; 70% Caucasian	Clinical: Inpt	4 yrs (71%)	Stressful Life Events	15	BPD dx (Ab-DIB)	18	No ASSOCIATION in analyses.
Stepp et al. (2015)	n=113; 100% female; 33% Caucasian	Community: High-Risk (PGS)	10–13 yrs (97%)	Adversity, Public Assist, Single-Parent	5–16	BPD sxs (IPDE-B)	16–18	**Adversity predicted increases in BPD sxs.**

	Sample			Risk factor		BPD Outcome		
Study	**Characteristics**	**Type (cohort)**	**Follow-up (retention)**	**Construct**	**Age in years**	**Construct (measure)**	**Age in years**	**Summary of results**
Conway et al. (2015)	n=700; 52% female; 92% Caucasian	Community: High Risk (Mater-U)	5 yrs (86%)	Acute Life Stress	15	BPD sxs (SCID-II)	20	NO ASSOCIATION in final analyses. *(Acute stressors significant in bivariate analyses)*
				Chronic Stress	15			**Family & school stressors predicted BPD sxs.** *(All stressors significant in bivariate analyses)*

Note. Ab-DIB=Abbreviated Diagnostic Interview for Borderlines, ALSPAC=Avon Longitudinal Study of Parents and Children, BPD=borderline personality disorder, CI-BPD=Childhood Interview for DSM BPD, CIC=Children in the Community, CIC-SR=CIC Self-Report Scale, dx=diagnosis, Inpt=inpatient; IPDE-B=International Personality Disorders Examination-Borderline Screener, mos=months, Mater-U=Mater University Study, NR=not reported, PGS=Pittsburgh Girls Study, PREG=during pregnancy, SCID-II=Structured Clinical Interview for DSM Axis II Disorders, SES= socioeconomic status, sxs= symptoms, wks=weeks, yrs=years.

Table 2

Summary of Longitudinal Research Investigating Family Risk Factors Predicting Borderline Personality Disorder (BPD)

	Sample			Risk factor		BPD Outcome		
Study	**Characteristics**	**Type (Cohort)**	**Follow-up (Retention)**	**Construct**	**Age in years**	**Construct (Measure)**	**Age in years**	**Summary of results**
Parent/Family Psychopathology								
Bezirganian et al. (1993)	n=776; 48% female; 90% Caucasian	Community: Population	3 yrs (79%)	Maternal Ego Integration; Interpersonal Difficulty; Impulsivity	14	BPD dx (SCID)	16	NO ASSOCIATION in final analyses. (*Maternal ego integration significant in bivariate analyses*)
Carlson et al. (2009)	n=162; 49% female; 67% Caucasian	Community: High-Risk	28 yrs (NR)	Maternal hx of Medical Problems	Prenatal	BPD sxs (SCID-II)	28	NO ASSOCIATION in final analyses. *(Maternal hx of serious medical problems significant in bivariate analyses)*
Crawford et al. (2009)	n=776; % female NR; 91% Caucasian	Community: Population (CIC)	~20 yrs (78%)	Maternal Interpersonal Difficulties	13	BPD sxs (CIC-SR)	13, 16, 22, 33	NO ASSOCIATION in analyses.
Barnow et al. (2013)	n=286; 55% female; % Caucasian NR	Community: Population (Greifswald)	5 yrs (88%)	Maternal BPD & Depression	15	BPD sxs (SCID-II)	20	**Maternal BPD predicted BPD sxs.** *(Maternal BPD and depression significant in bivariate analyses)*
Belskey et al. (2012)	n=1116; 55% female; % Caucasian NR	Community: Population (E-Risk)	7 yrs (96%)	Family hx Psychiatric Hospitalization	12	BPD features (SWAP)	12	***Family hx interacted with maltreatment & maternal negative expressed emotion to predict BPD dimensionally & categorically.***
Stepp et al. (2013)	n=816; 59% female; % Caucasian NR	Community: Population (OADP)	16 yrs (87%)	Maternal/Paternal Depression, Anxiety, BPD, ASPD, Substance Use	24	BPD sxs (IPDE)	30	**Maternal BPD and paternal substance use predicted BPD sxs.** *(Maternal depression & BPD and maternal & paternal anxiety, substance use and ASPD significant in bivariate analyses)*
Reinelt et al. (2014)	n=295; 55% female; % Caucasian NR	Community: Population (Greifswald)	5 yrs (77%)	Maternal BPD sxs	15	BPD sxs (SCID-II)	19	**Maternal BPD sxs predict offspring BPD sxs and this association was mediated by maladaptive parenting style/behavior.**
Stepp et al. (2014b)	n=2212; 100% female; 41% Caucasian	Community: High-Risk (PGS)	4 yrs (90%)	Caregiver ASPD & Depression	11	BPD sxs (IPDE)	14–17	NO ASSOCIATION in final analyses. (*Caregiver ASPD and depression significant in bivariate analyses)*
Conway et al. (2015)	n=700; 52% female; 92% Caucasian	Community: High Risk (Mater-U)	5 yrs (86%)	Maternal Internalizing & Externalizing	15	BPD sxs (SCID-II)	20	**Maternal externalizing dx predicted BPD sxs.**
				Maternal BPD sxs	15			NO ASSOCIATION in analyses.
Winsper et al. (2015)	n=6050; 46% female; 48% Caucasian	Community: Population (ALSPAC)	~12 yrs (43%)	Maternal Alcohol/Tobacco Use, Anxiety & Depression	PREG: 18, 32 wks	BPD sxs (CI-BPD)	12	**Prenatal maternal anxiety and depression at 18wks predicted BPD sxs.** *(All prenatal risks were significant in bivariate analyses)*
Parenting Behavior/Style								
Bezirganian et al, (1993)	n=776; 48% female; 90% Caucasian	Community: Population	3 yrs (79%)	Involvement, Inconsistency, Punishment	14	BPD dx (SCID)	16	**Maternal inconsistency predicted BPD dx and interacted with maternal over-involvement to predict BPD dx.**

	Sample			Risk factor		BPD Outcome		
Study	**Characteristics**	**Type (Cohort)**	**Follow-up (Retention)**	**Construct**	**Age in years**	**Construct (Measure)**	**Age in years**	**Summary of results**
				Involvement & Discipline	14			NO ASSOCIATION in analyses.
Johnson et al. (2006)	n=593; % female NR; 90% Caucasian	Community: Population (CIC)	~28yrs (61%)	Aversive Behaviors & Low Affection	16	BPD dx (DISC; PDQ; SCID-II)	22, 33	NO ASSOCIATION in final analyses. *(Parenting behaviors significant in bivariate analyses)*
Cohen et al. (2008)	n=680; % female NR; 91% Caucasian	Community: Population (CIC)	~20 yrs (70%)	Poor Parenting	13, 16	BPD sxs (NR)	13, 16, 22, 33	**Poor maternal parenting predicted higher BPD sxs.**
Carlson et al. (2009)	n=162; 49% female; 67% Caucasian	Community: High-Risk	28 yrs (NR)	Hostility & Boundary Dissolution	42 mos	BPD sxs (SCID-II)	28	**Maternal hostility in early childhood uniquely predicted BPD sxs.**
Crawford et al. (2009)	n=766; % female NR; 91% Caucasian	Community: Population (CIC)	~20 yrs (78%)	Maternal Child Satisfaction	13	BPD sxs (CIC-SR)	13, 16, 22, 33	**Maternal satisfaction with child predicted BPD sxs.**
				Inconsistency	13			NO ASSOCIATION in final analyses. *(Inconsistency significant in bivariate analyses)*
Belskey et al. (2012)	n=1116; 55% female; % Caucasian NR	Community: Population (E-Risk)	7 yrs (96%)	Negative Emotion	5, 10	BPD features (SWAP)	12	***Maternal negative emotion predicted BPD dimensionally & categorically.***
Winsper et al. (2012)	n=6050; 46% female; 48% Caucasian	Community: Population (ALSPAC)	~12 yrs (43%)	Poor Parenting:	birth-8	BPD sxs (CI-BPD)	12	**Poor parenting predicted BPD sxs.**
Wolke et al. (2012)	n=6050; 46% female; 48% Caucasian	Community: Population (ALSPAC)	~12 yrs (43%)	Hostility, Harsh Discipline	2–7	BPD sxs (CI-BPD)	12	***Maternal hostility and harsh punishment predicted BPD sxs.***
Lynos-Ruth et al. (2013)	n=56; 41% female; 73% Caucasian	Community: High-Risk	21 yrs (74%)	Disrupted Communication	18 mos	BPD sxs (SCID-II)	19	**Communication withdrawal predicted BPD sxs.**
Reinelt et al. (2014)	n=295; 55% female; % Caucasian NR	Community: Population (Greifswald)	5 yrs (77%)	Rejection, Warmth, Overprotection	15	BPD sxs (SCID-II)	19	**Maladaptive parenting (latent factor including both parenting constructs) predicted BPD sxs.**
				Mother-Child Discrepancies	15			
Stepp et al. (2014b)	n=2212; 100% female; 41% Caucasian	Community: High-Risk (PGS)	4 yrs (90%)	Harsh Punishment, Low Warmth	14–17	BPD sxs (IPDE)	14–17	**Significant reciprocal relationships between harsh discipline/low warmth and BPD sxs.**
Hallquist et al. (2015)	n=2228; 100% female; 39% Caucasian	Community: High-Risk (PGS)	4 yrs (90%)	Harsh Discipline	5–14	BPD sxs (IPDE)	14–17	**Harsh discipline predicted BPD sxs at 14.** NO ASSOCIATION with change in BPD sxs.
Family Climate & Parent-Child Relationship								
Bezirganian et al, (1993)	n=776; 48% female; 90% Caucasian	Community: Population	3 yrs (79%)	Parent-Child Closeness	14	BPD dx (SCID)	16	NO ASSOCIATION in analyses.

	Sample			Risk factor		BPD Outcome		
Study	**Characteristics**	**Type (Cohort)**	**Follow-up (Retention)**	**Construct**	**Age in years**	**Construct (Measure)**	**Age in years**	**Summary of results**
Carlson et al. (2009)	n=162; 49% female; 67% Caucasian	Community: High-Risk	28 yrs (NR)	Parent-Child Relationship	13	BPD sxs (SCID-II)	28	NO ASSOCIATION in final analyses. *(Parent-child relationship significant in bivariate analyses)*
Stepp et al. (2013)	n=816; 59% female; % Caucasian NR	Community: Population (OADP)	16 yrs (87%)	Cohesion, Discord, Support	17	BPD sxs (IPDE)	30	**Mother-child discord predicted BPD sxs.** (*M-C discord, family cohesion and maternal support significant in bivariate analyses)*
Greenfield et al. (2015)	n=204; 69% female; 70% Caucasian	Clinical: Inpt	4 yrs (71%)	Family Relations	15	BPD dx (Ab-DIB)	18	NO ASSOCIATION in analyses.
Hammen et al. (2015)	n=385; 61% female; 92% Caucasian	Community: High Risk (Mater-U)	5 yrs (47%)	Relationship Quality GxE Interaction (OXTR) × (Relationship Quality)	15	BPD sxs (SCID-II)	20	NO ASSOCIATION with relationship quality. **Relationship quality & OXTR genotype interacted to predict BPD sxs: relationship quality predicted BPD sxs for those with AA/AG genotype, not GG genotype.**

Note. ABC=Antisocial Behavioral Checklist, Ab-DIB=Abbreviated Diagnostic Interview for Borderlines, ALSPAC=Avon Longitudinal Study of Parents and Children, ASPD=antisocial personality disorder, BPD=borderline personality disorder, CI-BPD=Childhood Interview for Diagnostic and Statistical Manual BPD, CIC=Children in the Community, CIC-SR=Children in the Community Self-Report Scale, dx=diagnosis, E-Risk=Environmental Risk Twin Study, FES=Family Environment Scale, GxE=Gene X Environment Interaction, hx=history, Inpt=inpatient, IPDE=International Personality Disorder Examination-Borderline Screener, Mater-U=Mater-University Study, NR=not reported, OADP=Oregon Adolescent Depression Project, OXTR=oxytocin receptor gene, PD=personality disorder, PGS = Pittsburgh Girls Study, PREG=during pregnancy, SCID=Structured Clinical Interview for DSM Axis I Disorders, SCID-II=Structured Clinical Interview for DSM Axis II Disorders, SCID-NP=Structured Clinical Interview for DSM-Nonpatient Version, SCID-Q=Self-Report Personality Disorder Questionnaire, SWAP=The Shelder-Westen Assessment Procedure, sxs=symptoms, wks=weeks, yrs = years.

Table 3

Summary of Longitudinal Research Investigating Maltreatment and Other Trauma Predicting Borderline Personality Disorder (BPD).

	Sample			Risk factor		BPD Outcome		
Study	**Characteristics**	**Type (Cohort)**	**Follow-up (Retention)**	**Construct (Measure)**	**Age in years**	**Construct (Measure)**	**Age in years**	**Summary of results**
Johnson et al. (1999)	n=738; 48% female; 90% Caucasian	Community: Population (CIC)	~17yrs (79%)	Maltreatment: PA, SA, NEG	<10	BPD sxs & dx (CIC-SR)	22	**Maltreatment composite predicted BPD dx and PA, SA and NEG predicted BPD sxs.**
Johnson et al. (2000)	n=738; 48% female; 90% Caucasian	Community: Population (CIC)	~17yrs (79%)	NEG: Emotional, Physical, Supervision	<18	BPD sxs & dx (CIC-SR)	22	**Supervision NEG predicted BPD sxs.** *(All forms of NEG significant in bivariate analyses)*
Johnson et al. (2001)	n=793; 49% female; 90% Caucasian	Community: Population (CIC)	~17yrs (81%)	VA	5, 13 16	BPD sxs & dx (CIC-SR)	22	**VA predicted BPD sxs & BPD dx.**
Thatcher et al. (2005)	n=524; 44% female; 85% Caucasian	Clinical: Inpt, Outpt, Incarcerated (PAARC) & CC	8–12 yrs (NR)	PA or SA	<12	BPD sxs (SCID-II)	18+	NO ASSOCIATION in analyses.
Cohen et al. (2008)	n=680; % female NR; 91% Caucasian	Community: Population (CIC)	~30 yrs (87%)	PA, SA, NEG & Other Trauma	13, 16, 22	BPD sxs (CIC-SR)	13, 16, 22, 33	**Cumulative trauma (maltreatment & other trauma) predicted BPD sxs.**
Carlson et al. (2009)	n=162; 49% female; 67% Caucasian	Community: High-Risk	28 yrs (NR)	Maltreatment: PA, VA, NEG	birth-18 mos	BPD sxs (SCID-II)	28	***Maltreatment predicted BPD sxs.***
				PA, SA or NEG	4 -18			***PA and SA predicted BPD sxs.***
Crawford et al. (2009)	n=766; % female NR; 91% Caucasian	Community: Population (CIC)	~20 yrs (78%)	Maternal Separation	<5	BPD sxs (CIC-SR)	13, 16, 22, 33	**Maternal separation predicted BPD sxs in adolescence and adulthood.**
				Maltreatment: PA, SA, NEG	NR			**Maltreatment predicted BPD sxs.**
Widom et al. (2009)	n=892; 49% female; 90% Caucasian	Community: High-Risk & CC	~ 10 yrs (75%)	Maltreatment: PA, SA, NEG	<11	BPD dx (DIPD-R)	29, 40	NO ASSOCIATION in final analysis. *(Maltreatment significant in bivariate analyses for sxs and dx (OR: 1.65))*

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	Sample			Risk factor		BPD Outcome		
Study	**Characteristics**	**Type (Cohort)**	**Follow-up (Retention)**	**Construct (Measure)**	**Age in years**	**Construct (Measure)**	**Age in years**	**Summary of results**
Belskey et al. (2012)	n=1116; 55% female; % Caucasian NR	Community: Population (E-Risk)	7 yrs (96%)	PA	5, 7, 10	BPD features (SWAP)	12	**PA predicted BPD dimensionally & categorically.**
Wolke et al. (2012)	n=6050; 46% female; 48% Caucasian	Community: Population (ALSPAC)	~12 yrs (43%)	Peer Victimization	8, 10	BPD dx (CI-BPD)	12	**Peer victimization was predictive of BPD dx: chronic victimization robust effects.**
				SA	2–9			NO ASSOCIATION in analyses.
Lynos-Ruth et al. (2013)	n=56; 41% female; 73% Caucasian	Community: High-Risk	21 yrs (74%)	Poor Parental Care	birth - 18 mos	BPD sxs (SCID-II)	19	**Poor parental care predicted BPD sxs.**
Bornovalova et al. (2013b)	n=2764; 55% female; 95% Caucasian	Community: Population (MTS)	7–13 yrs (NR)	PA, SA, or EA	11, 17	BPD sxs (MBPD)	24	***PA, SA, & EA predicted BPD sxs: this was mediated by internalizing & externalizing sxs.***
Greenfield et al. (2015)	n=204; 69% female; 70% Caucasian	Clinical: Inpt	4 yrs (71%)	Maltreatment: PA, SA, NEG	15	BPD dx (Ab-DIB)	18	No ASSOCIATION in final analyses. *(Maltreatment significant in bivariate analysis)*
Krabbendam et al. (2015)	n=184; 100% female; 64% Caucasian	Clinical: Incarcerated	3–6 yrs (55%)	PA, SA & Other Trauma	16	BPD dx (SCID-II)	20	NO ASSOCIATION in analyses.
Stepp et al. (2015)	n=113; 100% female; 33% Caucasian	Community: High-Risk (PGS)	10–13 yrs (97%)	SA	12–16	BPD sxs (IPDE)	16–18	**SA predicted increases in BPD sxs over time.**

Note. Ab-DIB=Abbreviated Diagnostic Interview for Borderlines, ALSPAC=Avon Longitudinal Study of Parents and Children, BPD=borderline personality disorder, CC=community controls, CI-BPD=Childhood Interview for DSM Borderline Personality Disorder, CIC=Children in the Community, DIPD-R=Diagnostic Interview for Personality Disorders- Revised, dx=diagnosis, EA= Emotional abuse, E-Risk=Environmental Risk Twin Study, Inpt=inpatient, IPDE=International Personality Disorders Examination-Borderline Screener, mos=months, MBPD=Minnesota Borderline Personality Disorder Scale, MTS=Minnesota Twin Study, NEG=neglect, NR=not reported, Outpt=Outpatient, PA=physical abuse, PAARC= Pittsburgh Adolescent Research Center, PD=personality disorder, PGS=Pittsburgh Girls Study, SA=sexual abuse, SCID-II= Structured Clinical Interview for DSM-Axis II Disorders, SWAP=The Shelder-Westen Assessment Procedure, sxs=symptoms, VA=verbal abuse, yrs = years.

Table 4

Summary of Longitudinal Research Investigating Child Factors Predicting Borderline Personality Disorder (BPD).

	Sample			Risk factor		BPD Outcome		
Study	**Characteristics**	**Type Cohort)**	**Follow-up (Retention)**	**Construct (Measure)**	**Age in years**	**Construct (Measure)**	**Age in years**	**Summary of results**
Cognitive function								
Cohen et al. (2008)	n=680; % female NR; 91% Caucasian	Community: Population (CIC)	~20 yrs (70%)	IQ	13, 16	BPD sxs (CIC-SR)	13, 16, 22, 33	**Lower IQ predicted BPD sxs.**
Belskey et al. (2012)	n=1116; 55% female; %Caucasian NR	Community: Population (E-Risk)	7 yrs (96%)	IQ	5	BPD (SWAP)	12	***Lower IQ predicted higher BPD dimensionally & categorically.***
				Executive Fx	5			NO ASSOCIATION in analyses.
				Theory of Mind	5			***Poor Theory of Mind predicted BPD dimensionally & categorically.***
Winsper et al. (2012)	n=6050; 46% female; 48% Caucasian	Community: Population (ALSPAC)	~12 yrs (43%)	IQ	8	BPD sxs (CI-BPD)	12	**Lower IQ predicted BPD sxs.**
Wolke et al. (2012)	n=6050; 46% female; 48% Caucasian	Community: Population (ALSPAC)	~12 yrs (43%)	IQ	8	BPD dx (CI-BPD)	12	***Lower IQ predicts BPD dx.***
Attachment								
Carlson et al. (2009)	n=162; 49% female; 67% Caucasian	Community: High-Risk	28 yrs (NR)	Attachment	12, 18 mos	BPD sxs (SCID-II)	28	NO ASSOCIATION in final analyses. *(Disorganization significant in bivariate analyses, mediated by self-representation)*
Crawford et al. (2009)	n=766; % female NR; 91% Caucasian	Community: Population (CIC)	~20 yrs (78%)	Attachment	16	BPD sxs (CIC-SR)	13, 16, 22, 33	**Insecure attachment predicted BPD sxs.**
Lynos-Ruth et al. (2013)	n=56; 41% female; 73% Caucasian	Community: High-Risk	21 yrs (74%)	Attachment	18 mos	BPD sxs (SCID-II)	19	NO ASSOCIATION in analyses.
				Disorganized/Controlling	8			**Disorganized/controlling behavior predicted BPD sxs.**
Temperament and Personality								
Lenzenweger et al. (2005)	n=250; 53% female; 72% Caucasian	Community: Population	3 yrs (97%)	Agency, Negative Emotionality, Fear, Affiliation, Constraint	19	BPD sxs (IPDE)	19–22	**Negative emotionality & agency and low fear & constraint predicted BPD sxs (age 19) & increases in BPD sxs.**
						BPD sxs (MCMI-II)	19–22	**Negative emotionality and lower agency constraint predicted BPD sxs (age 19) & lower agency predicted increases in BPD**

	Sample			Risk factor		BPD Outcome		
Study	**Characteristics**	**Type Cohort)**	**Follow-up (Retention)**	**Construct (Measure)**	**Age in years**	**Construct (Measure)**	**Age in years**	**Summary of results**
Tragesser et al. (2007)	n=350; 55% female; 84% Caucasian	Community: High Risk	2 yrs (86%)	Instability, Impulsivity/Self-Harm	18	BPD sxs (PAI-BOR)	20	***Affective instability and impulsivity/self-harm predicted BPD sxs: affective instability most robust predictor.***
Crawford et al. (2009)	n=766; % female NR; 91% Caucasian	Community: Population (CIC)	~20 yrs (78%)	Crying/Demanding; Angry/Tantrum	5	BPD sxs (CIC-SR)	13, 16, 22, 33	**Anger/tantrums predicted BPD sxs.** *(All temperament measures significant in bivariate analyses)*
Carlson et al. (2009)	n=162; 49% female; 67% Caucasian	Community: High-Risk	28 yrs (NR)	Behavior During Feeding	3 mos	BPD sxs (SCID-II)	28	NO ASSOCIATION in final analyses. *(Muscle tone significant in bivariate analyses)*
				Temperament	3, 6, 30 mos			NO ASSOCIATION in final analyses. *(Infant activity at 6 mos & emotionality at 30 mos significant in bivariate analyses)*
				Behavioral, Emotional Attentional & Relational	12			NO ASSOCIATION in final analyses. *(Behavioral and emotional instability and attentional and relational disturbance significant in bivariate analyses)*
				Functioning & Representation	8–12			**Self-representation predicts BPD sxs.**
Crawford et al. (2009)	n=766; % female NR; 91% Caucasian	Community: Population (CIC)	~20 yrs (78%)	Crying/Demanding; Angry/Tantrum	5	BPD sxs (CIC-SR)	13, 16, 22, 33	**Anger/tantrums predicted BPD sxs.** *(All temperament measures significant in bivariate analyses)*
Tragesser et al. (2010)	n=353; 55% female; 84% Caucasian	Community: High Risk	2 yrs (86%)	Negative Affect, Impulsivity	18	BPD sxs (DIB-R)	20	***Negative affect and impulsivity predicted BPD sxs; negative affect was the most robust predictor.***
Belskey et al. (2012)	n=1116; 55% female; % Caucasian NR	Community: Population (E-Risk Twin Study)	7 yrs (96%)	Self-control, Approach, Inhibition	5	BPD (SWAP)	12	***Lower self-control predicted BPD dimensionally & categorically.***
				Impulsivity	5			***Higher impulsivity predicted BPD dimensionally & categorically.***
Jovev et al. (2013)	n=205; 51% female; 96% Caucasian	Community: High-Risk	3 yrs (84%)	Affect, Surgency, Affiliation, Control	13	BPD sxs (CIC-SR)	15	**Effortful control predicted BPD sxs. Associations between affiliation and BPD sxs moderated were by maltreatment.**
Stepp et al. (2014a)	n=2282; 100% female; 41% Caucasian	Community: High-Risk (PGS)	14 yrs (93%)	Emotionality, Activity, Sociability, Shyness	5–8	BPD sxs (IPDE)	14–19	**Temperament predicted BPD sxs at age 14. Higher activity and lower sociability predicted increases in BPD sxs, higher shyness predicted decreases in BPD sxs.**
Stepp et al. (2014b)	n=2212; 100% female; 41% Caucasian	Community: High-Risk (PGS)	4 yrs (90%)	Negative Affectivity; Impulsivity	11, 14	BPD sxs (IPDE)	14–17	**Negative affectivity and impulsivity predicted BPD sxs at age 14**.
Hallquist et al. (2015)	n=2228; 100% female; 60% Caucasian	Community: High Risk (PGS)	4 yrs (90%)	Negative Emotionality	5–8, 11	BPD sxs (IPDE)	14–17	**Negative emotionality & poor self-control predicted BPD sxs at 14 and worsening self-control from predicted increases in BPD sxs.**

Study	Sample: Characteristics	Sample: Type Cohort)	Sample: Follow-up (Retention)	Risk factor: Construct (Measure)	Risk factor: Age in years	BPD Outcome: Construct (Measure)	BPD Outcome: Age in years	Summary of results
				Poor Self-Control	5–8, 10–14			**The effects of poor self-control on age 14 BPD sxs was mediated by reciprocal effects between poor self-control and harsh discipline.**
Sharp et al. (2015)	n=730; 56% female; 30% Caucasian	Community: Population	1 yr (83%)	Experiential Avoidance	16	BPD sxs (BPFS-C)	17	**Experiential avoidance predicted BPD sxs.**
Stepp et al. (2015)	n=113; 100% female; 33% Caucasian	Community: High-Risk (PGS)	10–13 yrs (97%)	Negative Affectivity	16	BPD sxs (IPDE)	16–18	**The effect of negative affectivity on BPD sxs was moderated by family adversity: higher levels of both predicted BPD sxs.**
Psychopathology								
Thomsen et al. (1993)	n=96; 33% female; % Caucasian NR	Clinical: Inpt, Outpt & Controls	16 yrs (85%)	OCD	12	BPD dx (SCID-II)	27	NO ASSOCIATION in analyses.
Rey et al. (1995)	n=250; 44% female; % Caucasian NR	Clinical: Inpt	5–8 yrs (39%)	Internalizing & Externalizing	14	BPD dx (PDE)	20	***Adolescent ADHD (either alone or with CD) predicted BPD dx.***
Ramklint et al. (2003)	n=158; 60% female; % Caucasian NR	Clinical: Inpt	16 yrs (44% RR)	MDD, Disruptive Disorder, Substance Use	14	BPD dx (DIP-Q)	31	**MDD and substance use disorder predicted adult BPD dx.**
Thatcher et al. (2005)	n=524; 44% female; 85% Caucasian	Clinical: Inpt, Outpt, Incarcerated (PAARC)	8–12 yrs (NR)	Alcohol Dependence MDD, CD, ADHD	14–17	BPD sxs (SCID-II)	18+	**MDD and ADHD predicted 'severe' BPD sxs as characterized by latent-class analysis after accounting for covariates.**
Miller et al. (2008)	n=181; 12% female; 23% Caucasian	Clinical: Outpt & CC	10 yrs (NR)	ADHD, Internalizing & Externalizing	9	BPD dx (SCID-II)	18	***Childhood ADHD predicted BPD.*** NO ASSOCIATION between persistent ADHD and BPD.
Belskey et al. (2012)	n=1116; 55% female; % Caucasian NR	Community: Population (E-Risk)	7 yrs (96%)	Internalizing & Externalizing	5	BPD (SWAP)	12	***Internalizing & externalizing sxs predicted higher BPD dimensionally & categorically.***
Burke et al. (2012)	n=142; 0% female; 70% Caucasian	Clinical: Outpt (DTS)	12–18 yrs (80%)	ODD, CD, ADHD, Depression, Anxiety	7–22	BPD sxs (SCID-II)	24	**ODD (behavioral dimension) & ADHD sxs through adolescence predicted BPD sxs.**
				ASPD	18, 19			**ASPD sxs predicted BPD sxs and accounted for association between ADHD and BPD.**
				Alcohol & Drug Use	10–22			NO ASSOCIATION in final analyses. *(Marijuana use significant in bivariate analyses)*
Stepp et al. (2012)	n=1233; 100% female; 42% Caucasian	Community: High-Risk (PGS)	6–9 yrs (94%)	ADHD, ODD	5–13	BPD sxs (IPDE)	14	**ADHD and ODD sxs predicted BPD sxs. Growth in ADHD sxs (10–13yrs) and ODD sxs (8–10yrs) predicted BPD sxs.**

	Sample			Risk factor		BPD Outcome		
Study	**Characteristics**	**Type Cohort)**	**Follow-up (Retention)**	**Construct (Measure)**	**Age in years**	**Construct (Measure)**	**Age in years**	**Summary of results**
Winsper et al. (2012)	n=6050; 46% female; 48% Caucasian	Community: Population (ALSPAC)	~12 yrs (43%)	Axis I dx	8	BPD sxs (CI-BPD)	12	NO ASSOCIATION in final analyses.
Wolke et al. (2012)	n=6050; 46% female; 48% Caucasian	Community: Population (ALSPAC)	~12 yrs (43%)	Axis I dx	8	BPD sxs (CI-BPD)	12	***Any dx predicts BPD sxs.***
Bornovalova et al. (2013a)	n=1280; 100% female; 96% Caucasian	Community: Population (MTS)	3–4 yrs (NR)	Substance Use	14, 18	BPD sxs (MBPD)	14, 18	NO ASSOCIATION in final analyses *(Substance use significant in bivariate analyses)*
Stepp et al. (2013)	n=816; 59% female; % Caucasian NR	Community: Population (OADP)	16 yrs (87%)	Depression, Anxiety, Suicidality, Substance Use, CD/ODD, ADHD	16, 17	BPD sxs (IPDE)	30	**Depression, substance use and suicidality predicted BPD sxs.** *(All dx were significant in bivariate analyses)*
Bornovalova et al. (2013a)	n=2764; 55% female; 95% Caucasian	Community: Population (MTS)	7–13yrs (NR)	Externalizing & Internalizing	11, 17	BPD sxs (MBPD)	24	***Internalizing & externalizing sxs predicted BPD sxs.***
Stepp et al. (2014b)	n=2212; 100% female; 41% Caucasian	Community: High-Risk (PGS)	4 yrs (90%)	CD/ODD sxs	14	BPD sxs (IPDE)	14–17	**CD/ODD sxs predicted BPD sxs at age 14 and increases in BPD sxs.**
Conway et al. (2015)	n=700; 52% female; 92% Caucasian	Community: High Risk (Mater-U)	5 yrs (86%)	Internalizing & Externalizing	15	BPD sxs (SCID-II)	20	**Internalizing dx predicted BPD sxs.** *(Internalizing & externalizing dx significant in bivariate analyses)*
Greenfield et al. (2015)	n=204; 69% female; 70% Caucasian	Clinical: Inpt.	4 yrs (71%)	Depression, Suicide, CD, Alcohol/Drug	15	BPD dx (Ab-DIB)	18	NO ASSOCIATION in analyses.
				Age of Hospitalization & ER Visits	15			**Older admission age for suicidality predicted BPD dx.** *(Admission age and prior hospitalizations significant in bivariate analyses)*
				Global Functioning	15			**Global functioning predicted BPD dx.**
Krabbendam et al. (2015)	n=184; 100% female; 64% Caucasian	Clinical: Incarcerated	3–6 yrs (55%)	PTSS Depression, Dissociation, & Externalizing	16	BPD dx (SCID-II)	20	**Dissociation predicted BPD dx.** *(PTSS, depression & dissociation significant in bivariate analyses)*
Sharp et al. (2015)	n=730; 56% female; 30% Caucasian	Community: Population	1 yr (83%)	Anxiety, Depression sxs	16	BPD sxs (BPFS-C)	17	**Anxiety & depression predicted BPD sxs.**
Other Infant Factors								
Carlson et al. (2009)	n=162; 49% female; 67% Caucasian	Community: High-Risk	28 yrs (NR)	Behavior, Motor Fx	7, 10 days	BPD sxs (SCID-II)	28	NO ASSOCIATION in analyses.
				Infant Anomalies	birth			NO ASSOCIATION in analyses.

Note. Ab-DIB=Abbreviated Diagnostic Interview for Borderline, ADHD=attention deficit hyperactivity disorder, ALSPAC=Avon Longitudinal Study of Parents and Children, APSD=antisocial personality disorder, BPD=borderline personality disorder, BPFS-C=Borderline Personality Disorder Features Scale for Children, CC=community controls, CD=conduct disorder, CI-BPD=Childhood Interview for DSM Borderline Personality Disorder, CIC=Children in the Community, DIB-R=Diagnostic Interview for Borderlines-Revised, DIP-Q=Diagnostic and Statistical Manual-IV and International Classification of Diseases-10 Personality Questionnaire, DIS=Diagnostic Interview Schedule, DISC=Diagnostic Interview Schedule for Children, DTS=Developmental Trends Study, dx=diagnosis, E-Risk=environmental

risk, fx=functioning, HR=hospital record Inpt=inpatient, INT=interview, INT-ASST=interviewer-assisted measure, IPDE=International Personality Disorder Examination, IPDE-BOR=International Personality Disorders Examination-Borderline Screener, IQ=intelligence quotient, MBPD=Minnesota Borderline Personality Disorder Scale, MCMI-II=Millon Clinical Multiaxial Inventory-II, MDD=major depressive disorder, mos=months, MTS=Minnesota Twin Study, NR=not reported, OBS=observation, OCD=obsessive-compulsive disorder, ODD=oppositional defiant disorder, Outpt=outpatient, PAARC=Pittsburgh Adolescent Research Center, PAI-BOR=Personality Assessment Inventory- Borderline Features scale, PDE=Personality Disorder Examination, PGS=Pittsburgh Girls Study, PTSS=post-traumatic stress symptoms, RR=response rate, SCID = Structured Clinical Interview for DSM Axis I Disorders, SCID-II=Structured Clinical Interview for DSM Axis II Disorders, SWAP=Shelder-Westen Assessment Procedure, sxs=symptoms, WM=working memory, yrs=year.