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Validating the biosocial model of borderline personality disorder: Findings from a longitudinal study

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Abstract

This longitudinal study aimed to validate the biosocial theory of borderline personality disorder (BPD) by examining the transactional relationship between individual vulnerabilities and parental invalidation, and their links to BPD symptoms. We recruited a sample of 332 adolescents (mean age = 14.18 years; 58.3% female) residing in Singapore and administered self-report measures across three time-points (six months apart). Results from our path analytic model indicated that parental invalidation, impulsivity, and emotional vulnerability exhibited unique predictive associations with emotion dysregulation six months later. There was also a reciprocal prospective relationship between emotion regulation difficulties and BPD symptoms. Using random-intercepts cross-lagged panel models, we found partial evidence for a within-individual reciprocal relationship between parental invalidation and emotional vulnerability, and a unidirectional relationship of within-individual changes in impulsivity positively predicting changes in parental invalidation six months later. Overall, the study provided partial empirical support for the biosocial model in a Singaporean context.

Keywords: biosocial model; borderline personality disorder; parental invalidation; emotional vulnerability; impulsivity

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Borderline personality disorder (BPD) is a severe mental health condition characterized by dysregulation in emotional, behavioral, interpersonal, and self-identity domains (American Psychiatric Association, 2013). Symptoms of BPD include affective instability, inappropriate intense anger, repeated self-harm or suicidal behaviors, and a pattern of unstable and intense interpersonal relationships (American Psychiatric Association, 2013). One of the most influential etiological models of BPD is the biosocial model (Linehan, 1993). The key thesis in the biosocial model is that an individual's emotional vulnerability transacts with an invalidating environment over time, increasing experienced invalidation and worsening emotional vulnerability, thereby contributing to emotion dysregulation and BPD symptoms (Fruzzetti et al., 2005; Linehan, 1993). Recent studies identified impulsivity as an additional plausible early vulnerability factor for the development of BPD (Belsky et al., 2012; Crowell et al., 2009; Stepp et al., 2012). Notably, both emotional vulnerability and impulsivity may be biological in origin, and are conditioned and shaped by environmental factors over time.

Several studies found support for the biosocial model by demonstrating the role of emotional vulnerability (Reeves et al., 2010) and parental invalidation in predicting emotion dysregulation or BPD symptoms (Gill et al., 2018; Gill & Warburton, 2014). However, despite the inclusion of impulsivity as a vulnerability factor in the extended biosocial model (Crowell et al., 2009), no studies have examined if impulsivity has unique predictive associations with emotion dysregulation and BPD symptoms, over and above emotional vulnerability and parental invalidation. Furthermore, even though there is a strong emphasis on the transactional relationship between parental invalidation and individual vulnerability factors in the biosocial model, no study has investigated the *reciprocal* nature of the relationship between parental invalidation and child vulnerability factors. The current study aimed to bridge these gaps in the literature by examining (a) whether impulsivity uniquely predict future emotion dysregulation and BPD symptoms, in addition to emotional vulnerability and parental invalidation, and (b) the transactional relationship between parental invalidation, impulsivity and emotional vulnerability.

The biosocial model

Linehan (1993) postulated that emotional vulnerability and an invalidating childhood environment influence each other reciprocally to contribute to the development of emotion dysregulation and BPD symptoms. Emotional vulnerability refers to an individual's predisposition to experience negative affect, and is characterized by heightened emotional reactivity and sensitivity, and a slow return to baseline. On the other hand, an invalidating environment refers to an environment in which an individual's communicated experiences and needs are persistently punished, trivialized, disregarded, or dismissed.

Empirically, studies investigating emotional responding patterns in BPD have yielded mixed findings. Specifically, even though individuals with BPD may report experiencing greater affect

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sensitivity and intensity, evidence from psychophysiological studies do not support a hypothesis of heightened physiological responsivity (Rosenthal et al., 2008) – suggesting possible differential or discordant responses by the various emotional processing systems (e.g., physiological, cognitive) to a stimulus. On the other hand, findings from research examining the associations between parental invalidation and BPD symptoms have provided a more consistent picture, with a recent meta-analysis demonstrating that parental invalidation exhibited small to moderate positive associations with BPD symptoms (Lee et al., 2022).

When examined concurrently, emotional vulnerability and parental invalidation have independently predicted emotion dysregulation or BPD symptoms in non-clinical (Gill & Warburton, 2014) and clinical (Gill et al., 2018) samples. Although the studies demonstrated that emotional vulnerability and parental invalidation were uniquely associated with BPD, the cross-sectional designs limit the interpretation of causal and directional effects.

Meanwhile, longitudinal studies investigating the development of BPD symptoms have focused on examining the roles of child temperament and negative parenting behaviors (such as overprotection or negative expressed emotions) as etiological factors (Arens et al., 2011; Belsky et al., 2012). Specifically, the interaction of negative maternal parenting and high harm avoidance (e.g., a temperament dimension of being shy, pessimistic) (Arens et al., 2011), and physical abuse alone (Belsky et al., 2012) have been found to predict future BPD symptoms. Taken together, both negative parenting behaviors and difficult child temperament have been identified as crucial factors implicated in the development of BPD, thereby providing preliminary support for the biosocial model. Importantly, Belsky and colleagues (2012) demonstrated that individuals who experienced extreme invalidation (i.e., physical abuse) developed greater BPD characteristics compared to those who were not abused. Nonetheless, both negative parenting and aspects of child temperament such as high harm avoidance are broad constructs, which may include features beyond parental invalidation and emotional vulnerability. For instance, negative parenting could include overprotection (e.g., excessive parental interference or involvement in things that a child does, which may or may not be perceived as invalidating), while high harm avoidance includes features such as being easily tired (Cloninger et al., 1993). Furthermore, physical abuse is an extreme form of invalidation, whereas Linehan's (1993) definition of parental invalidation also includes non-abusive forms of parental practices. Therefore, to validate the biosocial model more comprehensively, there is a need to examine whether emotional vulnerability and parental invalidation would predict BPD symptoms longitudinally.

Extension of the biosocial model

The biosocial model has been extended to include impulsivity as an early vulnerability factor implicated in the development of BPD (Crowell et al., 2009). Impulsivity could be operationalized as a predisposition to rapidly engage in unplanned behaviors without consideration for negative consequences (Moeller et al., 2001). Longitudinal studies have demonstrated that impulsivity is an early precursor to BPD symptoms. Belsky and colleagues (2012) followed children from birth till 12 years of age, and found that children who exhibited BPD symptoms also exhibited high levels of impulsivity, more behavioral and emotional problems, and poor cognitive function at five years old. Stepp and colleagues (2012) provided corroborative evidence by examining the prospective relations between severity of attention deficit hyperactivity disorder (ADHD) symptoms (which includes inattention, impulsivity, and hyperactivity), and BPD symptoms. Specifically, amongst girls, ADHD symptoms at age eight, as well as the rate of increase of ADHD symptoms from age 10 to 13, exhibited unique prospective associations with BPD symptoms measured at age 14 (Stepp et al., 2012). Cumulatively, the studies suggest that impulsivity could be observable from an early age, and is implicated in the development of BPD.

Taken together, specific components of the biosocial model have obtained empirical support to varying degrees in different studies. No research however has yet examined the role of each major etiological factor, namely emotional vulnerability, impulsivity, and parental invalidation in predicting the development of emotion dysregulation and BPD symptoms simultaneously in a single study.

Longitudinal associations between individual vulnerabilities and parental invalidation

Apart from identifying critical factors implicated in the development of BPD, the biosocial model emphasizes the *transactional* relationship between an individual's vulnerability factors (emotional vulnerability and impulsivity) and parental invalidation (Crowell et al., 2009; Fruzzetti et al., 2005; Linehan, 1993). Specifically, a child who is emotionally vulnerable may often display strong negative emotions or a high incidence of impulsive behaviors, making it challenging for parents (or caregivers) to attend to their emotional needs (Linehan, 1993). As a result, parents may invalidate the child by dismissing their emotions, attributing the display of negative emotions to undesirable personal characteristics, or minimizing the difficulties that the child is facing, thereby limiting opportunities to learn adaptive emotion regulation or problem-solving skills. Consequentially, the child may engage in increasingly impulsive behaviors, have a lower threshold in reacting negatively, display more intense emotional or behavioral responses (e.g., self-harm behaviors), and/or stay emotionally aroused for a longer duration (Fruzzetti et al., 2005). In response, parents who are increasingly unable to meet the responses and demands of the child may resort to using more invalidation, thereby creating a negative feedback loop between the child's behaviors and the parents' invalidating behaviors.

The potential influence of parental invalidation on child emotional vulnerability and impulsivity has been supported partially by empirical research. Experimental studies demonstrated that individuals who were invalidated, relative to those who were validated, experienced stronger physiological arousal (Shenk & Fruzzetti, 2011), greater negative affect (Greville-Harris et al., 2016; Shenk & Fruzzetti, 2011; Weber & Herr, 2019), and had more difficulty labeling and accepting their emotions (Woodberry et al., 2008). While the studies demonstrate that invalidation indeed result in negative emotional reactions, it remains uncertain if invalidation could lead an individual to experience *greater* reactivity and sensitivity, and a *slower* return to baseline. Among parents and children, parental invalidation was found to be positively associated with (Mahtani et al., 2019; Tan et al., 2014), or predictive of future engagement in (You & Leung, 2012) non-suicidal self-injurious behaviors, which are associated with impulsivity (Hamza et al., 2015).

Separately, the influence of child behaviors on parenting has been documented in the general parenting literature. A review of

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the parenting and child behavior literature provided empirical support for the bidirectional relationships between a child's high tendency to feel frustration or exhibit impulsivity and negative parenting (Kiff et al., 2011). Within the BPD literature, Hallquist and colleagues (2015) found longitudinal reciprocal associations between poor self-control (an aspect of impulsivity) and harsh punishment, such that poor self-control is both preceded by, and increases harsh punishment. Importantly, poor self-control and harsh punishment had both direct and indirect prospective associations with BPD symptoms. While the study demonstrated the bidirectional influence of harsh punishment and poor self-control and the relationship between these constructs and BPD symptoms, it remains to be investigated whether the broader construct of parental invalidation would display a reciprocal relationship with emotional vulnerability and/or impulsivity.

Beyond the above issues, the majority of the literature examining the effect of parenting behaviors on child outcomes (and vice versa), has not differentiated between- versus within-subject effects. Parenting and child behaviors consist of both stable and variable components, with the stable components being relatively time-invariant while the variable components reflect fluctuations within an individual that could occur across time. Therefore, in the study of reciprocal relationships, it is important to differentiate the stable (between-person effect) and the variable (within-person effects) components of the construct to prevent inaccurate conclusions such as the identification of non-existent reciprocal effects, failure to detect effects that are present, or erroneously indicate a negative influence of one variable on the other (Hamaker et al., 2015). In this study, our analyses would specifically assess *within-subject* changes in levels of parental invalidation and individual vulnerabilities across one year by separating between-subject effects from within-subject effects, which would enable a more accurate assessment of the reciprocal relationship between these constructs.

Specific aims and hypotheses

The current study aimed to validate the components and the transactional nature of the biosocial model using a Singaporean community sample. The study utilized a longitudinal design, in which participants were followed up across three six-monthly measurement occasions. Specifically, we examined (a) whether parental invalidation, impulsivity, and emotional vulnerability would each exhibit unique, prospective associations with emotion dysregulation and BPD symptoms (Model 1), (b) whether emotion dysregulation would prospectively predict BPD symptoms (and vice versa) (Model 1), and (c) whether parental invalidation and individual vulnerabilities (emotional vulnerability and impulsivity) would transact across time in a negative feedback loop (Model 2 and Model 3).

We hypothesized that parental invalidation, impulsivity, and emotion vulnerability at baseline (Wave 1) would exhibit unique positive prospective associations with emotion dysregulation and BPD symptoms 6 months later (Wave 2). Consistent with previous research (Stepp et al., 2014), we also hypothesized that emotion dysregulation and BPD symptoms at Wave 2 would positively predict BPD symptoms and emotion dysregulation at Wave 3, respectively. In addition, we hypothesized that parental invalidation and emotional vulnerability/impulsivity would transact across time, such that fluctuations in the level of parental invalidation would positively predict variations in the level of emotional vulnerability/impulsivity at a subsequent time point (and vice versa). Figures 1, 2, and 3 depict Models 1, 2, and 3, respectively.

Method

Participants

The current study utilized a subset of the data contributed by 332 adolescents who participated in a three-wave longitudinal study conducted in Singapore. We have previously addressed a different research question utilizing only baseline data from the same dataset (Lee et al., 2023). All procedures were approved by the National University of Singapore Institutional Review Board. We recruited 333 families (parents and their adolescents) through advertising in online parenting forums or parenting groups on Facebook, or via a survey company. The study's inclusion criteria included the requirement of at least one parent to participate with an adolescent (aged between 12 and 17 years old) from the same family, and proficiency in English. Individuals with developmental disorders or hearing or visual impairment (apart from corrected vision) were excluded from the study. Participants were reimbursed SGD$5 per 30 minutes of research participation.

Adolescents recruited were between 12 and 17 years old ($M = 14.18$, $SD = 1.63$, 58.3% female). About 68.3% of the adolescents identified as Chinese, while 17.8%, 6.9%, and 6.6% identified as Malay, Indian, or 'Other' respectively. Approximately 89% of the adolescents were from dual-parent families, while the remaining 11% were from single-parent households. Most parents had completed tertiary education (57.8% for mothers, 61.4% for fathers). About 50% of the adolescents came from households with a monthly combined income of more than SGD$6000.

Procedure

Adolescents completed a battery of questionnaires once every six months across a year (Wave 1: March 2019–January 2020; Wave 2: September 2019–July 2020; Wave 3: March 2020–January 2021). A six-month follow-up period was selected for two reasons. First, as both parenting and child behaviors have stable and variable components, a sufficiently long duration is required to allow for changes in the within-subject (variable) component to occur and be observed. Second, from a contextual perspective, adolescents living in Singapore are expected to sit for examinations approximately once every six months, and may experience changes in classroom settings (e.g., being re-assigned to a new class) once a year. Considering the frequency of natural school events that would typically occur for all adolescents, a six-month observation interval would allow the observations of sufficient variations in the constructs of interest

The questionnaires were administered in English, and were completed either via hardcopy or on an online platform hosted on Qualtrics. Demographic information was obtained at Wave 1 (W1) from a parent using a demographic data form. Adolescents completed the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004), the Personality Assessment Inventory – Adolescent, Borderline Features Scale (PAI-A BOR; Morey, 2007), the Emotion Reactivity Scale (ERS; Nock et al., 2008) and the Barrett Impulsiveness Scale (BIS-11; Patton et al., 1995) at all three data collection waves. In addition, the Invalidating Childhood Environment Scale (ICES; Mountford et al., 2007) was administered at W1, while the Current Parental Invalidation Scale (CPIS; Sturrock & Mellor, 2014) was completed at both Wave 2 (W2) and Wave 3 (W3).

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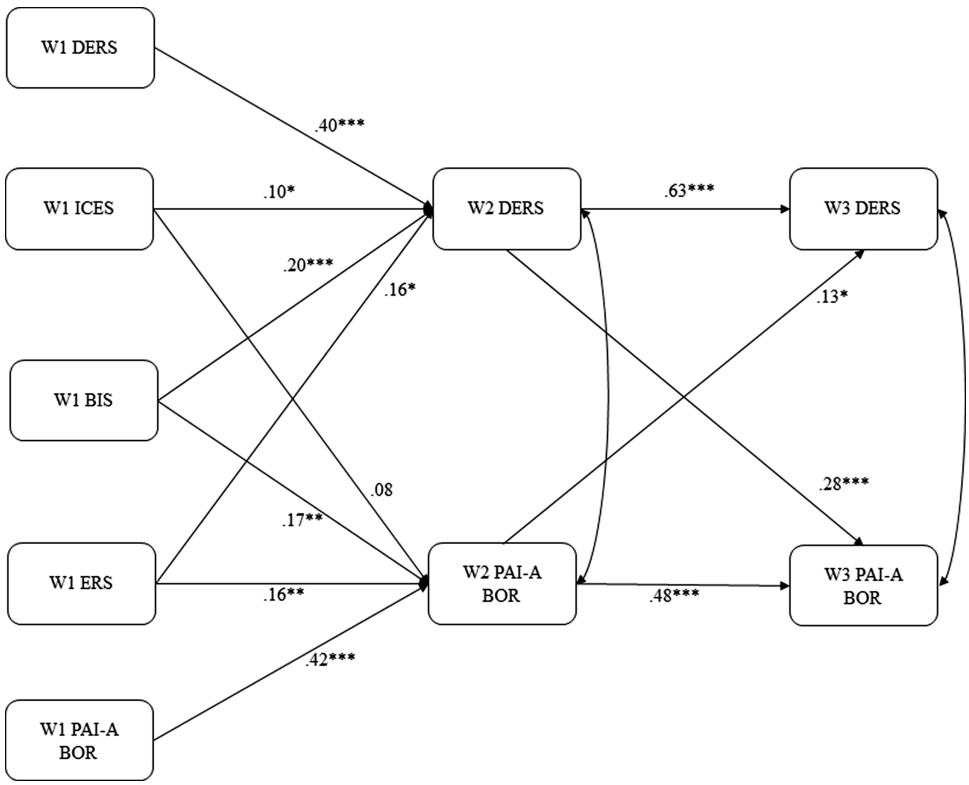

Figure 1. Model 1: longitudinal predictors of emotion dysregulation and symptoms of BPD. The values presented represent the standardized estimates for each path. BIS = Barratt Impulsiveness Scale; CPIS = Current Parental Invalidation Scale; DERS = Difficulties in Emotion Regulation Scale; ERS = Emotion Reactivity Scale; ICES = Invalidating Childhood Environment Scale; PAI-A BOR = Personality Assessment Inventory – Adolescent, Borderline Features Scale; W1 = wave 1; W2 = wave 2; W3 = wave 3. ***p < .001; **p < .01; *p < .05.

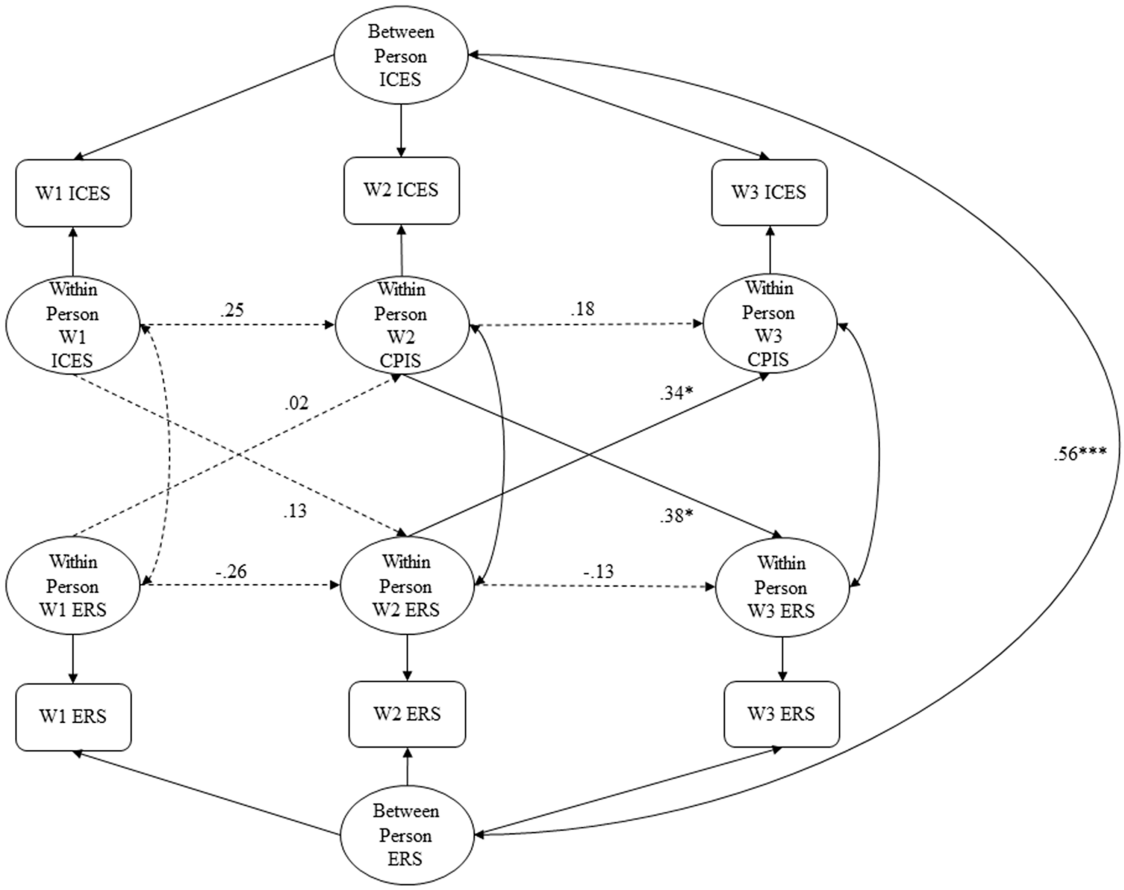

Figure 2. Model 2: longitudinal transactional associations between parental invalidation and emotional vulnerability. The values presented represent the standardized estimates for each path. CPIS = Current Parental Invalidation Scale; ICES = Invalidating Childhood Environment Scale; ERS = Emotion Reactivity Scale; W1 = wave 1; W2 = wave 2; W3 = wave 3. ***p < .001; *p < .05.

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Measures

Demographic data form

The demographic data form requested information regarding the adolescent's gender, age, ethnicity, overall household income, parents' employment status, parents' age, and parents' education level.

Invalidating childhood environment scale (ICES; Mountford et al., 2007)

The ICES is a retrospective self-report measure of parental invalidating behaviors up to age 18 years. The scale consists of 14 items that focus on descriptions of parental invalidating behaviors. Items are rated on a five-point Likert scale, ranging from one (never) to five (all of the time). Adolescents were instructed to respond to all items twice to provide separate ratings for their mothers' and fathers' behaviors up to their current age. Scores of the items were summed to form the maternal and paternal invalidation score. Overall parental invalidation scores were calculated by adding the maternal and paternal invalidation scores. The ICES demonstrated good to excellent internal consistencies in non-clinical samples from the United States and Australia (Robertson et al., 2013; Sturrock et al., 2009) and undergraduate samples from Singapore (Keng & Soh, 2018; Keng & Wong, 2017). The measure demonstrated excellent reliability in our current sample (Wave 1: $\alpha = .91$).

Current parental invalidation scale (CPIS; sturrock & mellor, 2014)

The CPIS (Sturrock & Mellor, 2014) aimed to measure current parental invalidation and was adapted from the ICES by modifying items in the original ICES (Mountford et al., 2007) to present-tense. The adapted measure demonstrated good internal consistencies ($\alpha = .80$–$.83$) in a sample of predominantly Caucasian adults (Sturrock & Mellor, 2014). In our sample, the CPIS demonstrated excellent internal reliability (Wave 2: $\alpha = .93$; Wave 3: $\alpha = .93$).

Emotion reactivity scale (ERS; Nock et al., 2008)

The ERS (Nock et al., 2008) was administered to measure emotional vulnerability. The scale consists of three subscales: sensitivity (e.g., "My feelings get hurt easily."), intensity (e.g., "When I experience emotions, I feel them very strongly/intensely."), and persistence (e.g., "When something happens that upsets me, it's all I can think about for a long time."). The three subscales map onto Linehan's (1993) operationalization of emotional vulnerability, which comprises sensitivity, intensity, and slow return to baseline. Each of the 21 items is rated on a five-point Likert scale, from zero ("Not at all like me") to four ("completely like me"). Item scores were summed, with higher scores indicating greater emotional vulnerability. The ERS has demonstrated excellent internal consistency ($\alpha = .94$) in a sample of adolescents (Nock et al., 2008). The internal reliabilities of the ERS in this sample were excellent (Wave 1: $\alpha = .96$; Wave 2: $\alpha = .97$; Wave 3: $\alpha = .98$).

Barrett impulsiveness scale (BIS-11; Patton et al., 1995)

The BIS (Patton et al., 1995) is a 30-item self-report questionnaire that assesses impulsiveness (e.g., I act "on impulse."). Factor analysis indicated that the scale comprised three second-order factors – attentional impulsiveness, motor impulsiveness, and nonplanning impulsiveness (Patton et al., 1995). A four-point Likert scale is used for rating of items, from 1 (Rarely/Never) to 4 = (Always Always/Always). Scores on the items were summed, with higher scores reflecting greater impulsivity. A systematic review found that the BIS has acceptable to good internal consistencies and criterion-related validity across a range of samples that included undergraduates, psychiatric population, forensic population, adults, and adolescents (Vasconcelos et al., 2012). The BIS demonstrated good internal consistencies across all three waves (Wave 1: $\alpha = .80$; Wave 2: $\alpha = .81$; Wave 3: $\alpha = .84$) in our sample.

Difficulties in emotion regulation scale (DERS; Gratz & Roemer, 2004)

The 36-item DERS (Gratz & Roemer, 2004) measures emotion dysregulation. Participants responded to the items using a five-point Likert scale, ranging from one (almost never) to five (almost always). The items form six subscales, which include non-acceptance of emotional responses, difficulty engaging in goal-directed behaviors, difficulty controlling impulses, lack of emotional awareness, low access to strategies for emotion regulation, and lack of emotional clarity. Item scores were summed to obtain a scale score. Higher scores indicate greater difficulties with emotion regulation. The DERS demonstrated good psychometric properties in a community sample of adolescents, with internal consistencies ranging from good to excellent for the various subscales, and good construct validity (Weinberg & Klonsky, 2009). In our study, the DERS demonstrated excellent internal reliabilities for (Wave 1: $\alpha = .94$; Wave 2: $\alpha = .94$; Wave 3: $\alpha = .95$).

Personality assessment inventory – adolescent, borderline features scale (PAI-A BOR; Morey, 2007)

The PAI-A BOR (Morey, 2007) consists of 20 items that assess BPD features through four subscales: affective instability, negative relationships, self-harm, and identity problems. Each item (e.g., "My mood can shift quite suddenly") is rated on a four-point Likert scale, ranging from 0 (false) to 3 (very true). Scores on the items were summed, with higher scores indicating higher BPD features. The PAI-A-BOR demonstrated good internal consistencies, criterion validity, and convergent validity across a clinical and a forensic adolescent sample (Venta et al., 2018). The PAI-A BOR exhibited very good internal consistencies in this study (Wave 1: $\alpha = .84$; Wave 2: $\alpha = .90$; Wave 3: $\alpha = .90$).

Data analytic strategy

We specified a path analytic model to explore the predictive associations between parental invalidation, impulsivity, and emotional vulnerability at baseline, and emotion dysregulation and BPD symptoms at a subsequent time point while controlling for baseline emotion regulation difficulties and BPD symptoms. We also tested whether there were longitudinal reciprocal associations between difficulties in emotion regulation and BPD symptoms in the model.

Two random-intercepts cross-lagged panel models (RI-CLPM) were specified to explore the transactional relationship (a) between parental invalidation and impulsivity, and (b) between parental invalidation and emotional vulnerability, across time. The RI-CLPM takes a multilevel approach to longitudinal data by allowing within- and between-person effects to be distinguished. Therefore, the RI-CLPM overcomes the limitations of the traditional cross-lagged panel model by controlling for stable, between-subject differences and enabling the observation of how constructs are associated with each other at the intra-individual level (Hamaker

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et al., 2015). As the scores are decomposed into between- and within-person sources, the correlation between the random intercepts would indicate how strongly between-person differences in one variable are associated with between-person differences in another variable. On the other hand, a positive cross-lagged association would indicate that a higher-than-average score on a variable (based on the individual's average score on a variable across the three waves) could predict a greater than expected score on another variable at a subsequent time point. Taken together, the strengths of RI-CLPM make it well-suited for the investigation of the longitudinal transactional relationship between parental invalidation and individual vulnerabilities at the intra-individual level.

Results

Preliminary analyses

All analyses were performed using SPSS Version 25.0 (IBM Corp, 2017) and the Lavaan package (Rosseel, 2012) in *R* (R Development Core Team, 2018). The univariate distributions of most variables were normal (skewness and kurtosis < |1|), except for Wave 2 ICES (kurtosis = 1.09). Analyses via Pearson's correlations revealed that all study variables were positively associated with one another (all *ps* < .001). Descriptive statistics and correlations among study variables are presented in Table 1.

In our study, 213 adolescents (64.5%) completed assessments at all three waves. Eighty-one (24.4%) dropped out of the study after the first wave. There were no differences in demographic variables and Wave 1 variables between adolescents who completed the study and those who dropped out before study completion (*ps* > .05). Item-level missing data was minimal (0.09%), and was substituted with the individual's mean for the construct within the specific timepoint. Analyses based on Little's Missing Completely at Random Test revealed that data from the scored scales were missing completely at random ($p > .05$). This allowed us to use maximum likelihood estimation to account for the missing data in our models. Model fit for all models was examined via the comparative fit index (CFI), the Tucker-Lewis Index (TLI), Root Mean Square Error of Approximate (RMSEA), and the standardized root mean square residual (SRMR). For a good fit, SRMR should be less than .08, and RMSEA should be less than .06 with its 90% confidence interval not greater than .10. In addition, CFI and TLI values should be more than .95 to reflect an excellent fit (Hu & Bentler, 1999).

Path analysis

Model 1 (as depicted by Figure 1) yielded acceptable fit to the data (χ^2 (12) = 54.90, $p < .001$; CFI = .971; TLI = .914; RMSEA = .10, 90% CI [.08, .13], SRMR = .06). Results indicated that W1 impulsivity ($b = 0.48$, $SE = .13$, $p < .001$, $\beta = .20$), parental invalidation ($b = 0.15$, $SE = .07$, $p = .041$, $\beta = .10$), and emotional vulnerability ($b = 0.19$, $SE = .08$, $p = .013$, $\beta = .16$) positively predicted W2 emotion dysregulation above and beyond W1 emotion dysregulation. While controlling for baseline BPD symptoms, W1 impulsivity ($b = 0.20$, $SE = .06$ $p = .001$, $\beta = .17$) and emotional vulnerability ($b = 0.09$, $SE = .03$, $p = .008$, $\beta = .16$) positively predicted BPD symptoms six months later. However, W1 parental invalidation did not predict W2 BPD symptoms ($b = 0.06$, $SE = .04$, $p = .116$, $\beta = .08$). Therefore, the hypothesis that impulsivity, emotional vulnerability, and parental invalidation would each exhibit unique positive associations with changes in emotion dysregulation and BPD symptoms over six months was partially supported.

The autoregressive paths and cross-lagged paths between emotion dysregulation and BPD symptoms were also significant across two measurement occasions (i.e., W2 and W3). Specifically, W2 emotion dysregulation positively predicted W3 BPD symptoms ($b = 0.13$, $SE = .03$, $p < .001$, $\beta = .28$) and W3 emotion dysregulation ($b = 0.63$, $SE = .07$, $p < .001$, $\beta = .63$). Similarly, W2 BPD symptoms exhibited positive associations with W3 difficulties in emotion regulation ($b = 0.29$, $SE = .14$, $p = .036$, $\beta = .13$) and W3 BPD symptoms ($b = 0.46$, $SE = .07$, $p < .001$, $\beta = .48$). Therefore, levels of emotion regulation difficulties at an earlier time point predicted BPD symptoms at a subsequent time point, and the relationship also held true in reverse.

Random-intercepts cross-lagged panel model

We first calculated the intra-class correlations to examine the proportion of variance in the variables that could be accounted for by between and within-person components. Our analyses indicated that 75, 63%, and 73% of the variance in parental invalidation, emotional vulnerability, and impulsivity, respectively, could be explained by stable, between-subjects differences.

Reciprocal associations between parental invalidation and emotional vulnerability

Model 2, as depicted by Figure 2, achieved a very good fit to the data (χ^2 (1) = 0.973, $p = .324$; CFI = 1.00; TLI = 1.00; RMSEA = .00, 90% CI [.00, .14], SRMR = .01). At the between-person level, parental invalidation and emotional vulnerability were positively correlated ($r = .56$, $p < .001$). This indicates that adolescents with higher emotional vulnerability across the three waves also reported experiencing greater instances of parental invalidation across the waves.

At the within-subject level, there were positive, cross-lagged associations between W2 parental invalidation and W3 emotional vulnerability ($b = 0.53$, $SE = .24$, $p = .026$, $\beta = .38$), as well as between W2 emotional vulnerability and W3 parental invalidation ($b = 0.31$, $SE = .12$, $p = .011$, $\beta = .34$). This suggests that when an adolescent experienced higher-than personal-average parental invalidation at W2, they would also report higher-than-expected-emotional vulnerability at W3 (and vice versa). On the other hand, the cross-lagged associations between W1 parental invalidation and W2 emotional vulnerability ($b = 0.16$, $SE = .27$, $p = .550$, $\beta = .13$), and between W1 emotional vulnerability and W2 parental invalidation ($b = 0.01$, $SE = .13$, $p = .924$, $\beta = .02$) were not significant.

In addition, all autoregressive paths were not significant ($p > .05$). This implies that there is limited within-subject carry-over effect. In particular, within-subject deviations in levels of reported parental invalidation did not predict deviations in reported parental invalidation at a subsequent time point. The same pattern held true for emotional vulnerability.

Reciprocal associations between parental invalidation and impulsivity

Model 3, as shown in Figure 3, had very good data fit (χ^2 (1) = 2.71, $p = .100$; CFI = 1.00; TLI = .97; RMSEA = .07, 90% CI [.00, .18], SRMR = .02). We observed a positive correlation between the random intercepts, with each intercept representing an individual's

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Table 1. Descriptive statistics and correlations among variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. W1 ICES	–														
2. W2 CPIS	.79	–													
3. W3 CPIS	.72	.79	–												
4. W1 BIS	.34	.35	.34	–											
5. W2 BIS	.33	.40	.41	.71	–										
6. W3 BIS	.25	.29	.40	.69	.79	–									
7. W1 ERS	.39	.41	.39	.40	.44	.38	–								
8. W2 ERS	.43	.56	.54	.32	.53	.41	.61	–							
9. W3 ERS	.37	.46	.47	.37	.42	.44	.65	.66	–						
10. W1 DERS	.40	.43	.41	.55	.54	.48	.69	.53	.58	–					
11. W2 DERS	.40	.52	.51	.50	.65	.57	.57	.74	.59	.66	–				
12. W3 DERS	.37	.44	.55	.51	.57	.62	.53	.62	.74	.67	.73	–			
13. W1 PAI-A-BOR	.41	.47	.45	.53	.49	.42	.65	.56	.60	.67	.59	.56	–		
14. W2 PAI-A BOR	.38	.52	.48	.47	.61	.46	.54	.71	.58	.52	.72	.60	.68	–	
15. W3 PAI-A BOR	.39	.53	.55	.43	.56	.56	.55	.63	.73	.55	.63	.73	.64	.69	–
M	60.62	60.10	58.49	65.60	65.27	64.88	34.82	35.13	32.95	90.69	91.64	90.22	25.68	23.72	22.53
SD	16.58	17.42	17.35	16.58	17.42	17.35	19.63	19.50	21.33	23.16	23.86	24.07	9.69	11.38	11.03
Median	58.00	56.50	56.00	66.00	65.00	66.00	37.00	37.00	35.00	89.00	93.00	95.00	24.00	24.00	21.50
Minimum	28	28	28	35	36	39	0	0	0	40	36	36	6	0	0
Maximum	118	134	117	94	94	92	84	84	84	159	163	152	56	56	55
Skewness	.70	.72	.47	.03	.04	−.02	.05	.05	.10	.25	.10	−.23	.29	.30	.41
Kurtosis	.43	1.09	−.14	.28	.14	−.36	−.69	−.54	−.83	−.06	−.28	−.55	−.31	−.45	−.53

BIS = Barratt Impulsiveness Scale (range: 30–120); CPIS = Current Parental Invalidation Scale (range: 28–140); DERS = Difficulties in Emotion Regulation Scale (range: 36–180); ERS = Emotion Reactivity Scale (range: 0–84); ICES = Invalidating Childhood Environment Scale (range: 28–140); PAI-A BOR = Personality Assessment Inventory – Adolescent, Borderline Features Scale (range: 0–60); W1 = wave 1; W2 = wave 2; W3 = wave 3.
All bivariate correlations were significant ($p < .001$).

average level of impulsivity and parental invalidation respectively ($r = .45$, $p < .001$). Therefore, adolescents who reported having higher impulsivity across the three waves also indicated more instances of parental invalidation across time.

All cross-lagged parameters estimated between W1 and W2 were not significant ($p > .05$). This indicates that at the within-individual level, there was no evidence of reciprocal influences of parental invalidation and impulsivity between W1 and W2. Meanwhile, the cross-lagged association between W2 impulsivity and W3 parental invalidation was significant ($b = 0.40$, $SE = .20$, $p = .050$, $\beta = .24$), even though the cross-lagged association between W2 parental invalidation and W3 impulsivity was not ($b = -0.00$, $SE = .08$, $p = .984$, $\beta = -.00$). Taken together, the relationship between parental invalidation and impulsivity appeared to be unidirectional, such that when individuals exhibited higher impulsivity than usual, they experienced a subsequent increase in parental invalidation. This relationship however did not hold true in reverse.

All autoregressive parameter estimates between W1 and W2 were not significant ($p > .05$), whereas those between W2 and W3 were significant (impulsivity: $b = 0.45$, $SE = .12$, $p < .001$, $\beta = .41$; parental invalidation: $b = 0.31$, $SE = .14$, $p = .032$, $\beta = .30$). Therefore, deviations from an individual's typical level on parental invalidation or impulsivity predicted deviations from the typical level on the same construct between W2 and W3, but not between W1 and W2.

Discussion

The current study aimed to (a) validate key components of the biosocial model, and (b) examine the reciprocal relationship between individual vulnerabilities and parental invalidation (Crowell et al., 2009; Linehan, 1993). Using a sample of community adolescents based in Singapore, we demonstrated that both impulsivity and emotional vulnerability positively predicted residual changes in emotion dysregulation and BPD symptoms six months later. We also found that W1 parental invalidation positively predicted W2 emotion dysregulation, though not W2 BPD symptoms. In addition, levels of emotion regulation difficulties at an earlier time point predicted BPD symptoms at a subsequent time point, and the relationship was also true in reverse. At the within-individual level, we found partial evidence for the reciprocal relationship between parental invalidation and emotional vulnerability, and a unidirectional relationship between impulsivity and parental invalidation six months later.

Our findings provide support for the roles of vulnerability factors and parental invalidation in the development of emotion dysregulation and BPD symptoms (Crowell et al., 2009; Linehan, 1993). We also extended the literature by demonstrating the unique predictive associations between impulsivity and emotion dysregulation or BPD symptoms six months later, over and above parental invalidation and emotional vulnerability. This finding provides additional support for the inclusion of impulsivity as a

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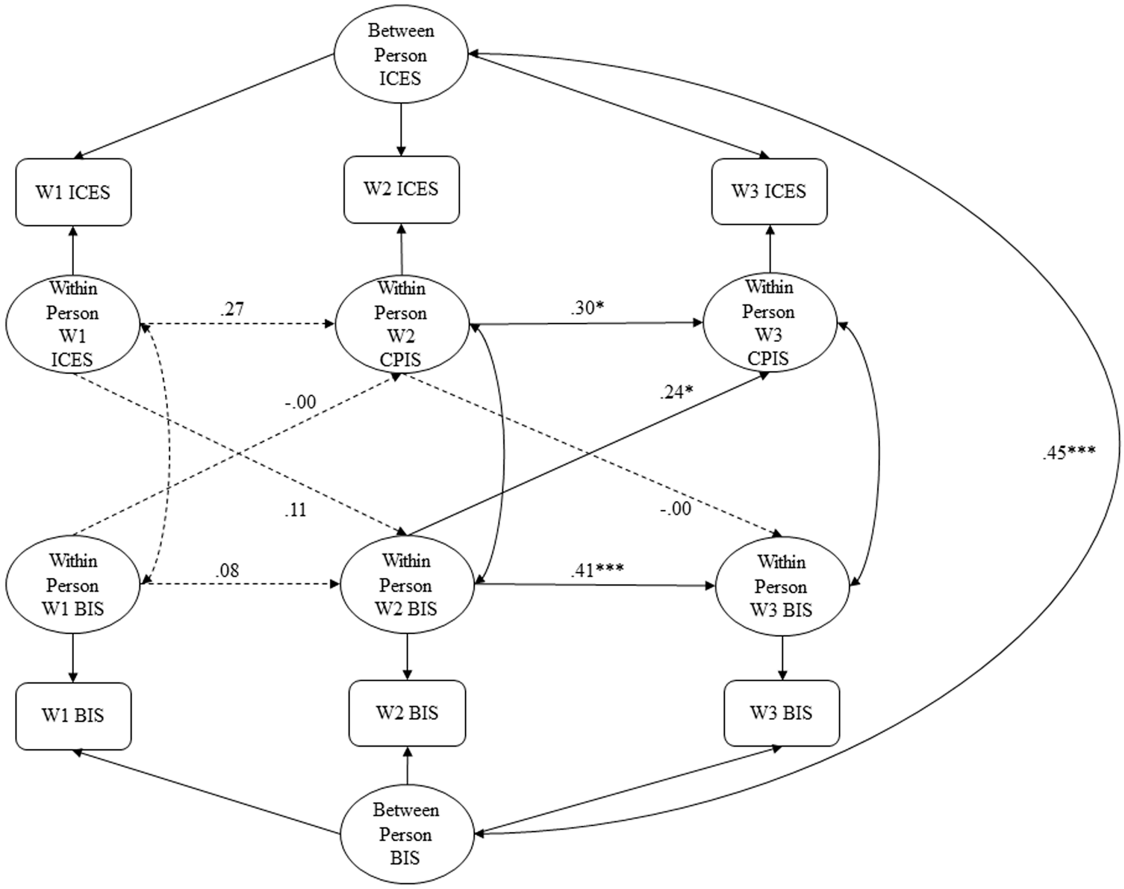

Figure 3. Model 3: longitudinal transactional associations between parental invalidation and impulsivity. The values presented represent the standardized estimates for each path. BIS = Barratt Impulsiveness Scale; CPIS = Current Parental Invalidation Scale; ICES = Invalidating Childhood Environment Scale; ERS = Emotion Reactivity Scale; W1 = wave 1; W2 = wave 2; W3 = wave 3. ***p < .001; *p < .05.

separate vulnerability factor in the biosocial model (Crowell et al., 2009). Our study also found that parental invalidation at W1 predicted W2 emotion dysregulation, a finding consistent with predictions of the biosocial model (Linehan, 1993). However, W1 parental invalidation did not predict BPD symptoms at W2 after controlling for baseline BPD symptoms. It is plausible that the effects of parental invalidation on BPD symptoms may only emerge over a longer time window or in periods when the family experiences elevated stress. Our finding also raises the possibility that there may be other factors that function independently of, or in relation with, parental invalidation that may impact the development of future BPD symptoms. Some possible factors include experiences of peer invalidation or bullying (Selby et al., 2013).

Linehan (1993) conceptualized emotion dysregulation as the core difficulty in BPD. Specifically, BPD symptoms are viewed as consequences of significant emotion regulation difficulties which could then contribute to greater emotion dysregulation. Our study found support for Linehan's (1993) conceptualization by demonstrating a negative feedback loop between emotion dysregulation and BPD symptoms. In particular, emotion dysregulation positively predicted BPD symptoms six months later, and vice versa. Our results corroborate those of Stepp and colleagues (2014), who found that baseline BPD symptoms severity predicted increases in emotion dysregulation over time, which in turn predicted greater BPD symptoms a year later. Difficulties in emotion regulation, which include lack of emotional clarity, problems with impulse control, difficulty engaging in goal-directed behaviors, and/or having poor emotion regulation strategies (Gratz & Roemer, 2004), could contribute to greater instability in the affective (Stepp et al., 2014), behavioral (Selby & Joiner, 2013), and interpersonal domains (Euler et al., 2021; Herr et al., 2013) of BPD. On the other hand, BPD symptoms, such as unstable interpersonal relationships with others and impulsive self-damaging behaviors, may, in turn, contribute to greater emotion dysregulation. Therefore, emotion dysregulation could contribute to, and serve as a consequence of, functioning deficits in the affective, behavioral, and interpersonal domains which manifest as BPD symptoms. Taken together, our study highlights the importance of targeting both deficits in emotion regulation and functioning difficulties (e.g., deficits in interpersonal effectiveness) in various domains during therapy.

Our study is among the first to examine the within-individual reciprocal relationships between individual vulnerabilities and parental invalidation articulated in the biosocial model (Linehan, 1993). We found partial support for the hypothesized reciprocal relations. Specifically, the cross-lagged associations between individual vulnerabilities and parental invalidation were largely significant between Wave 2 and Wave 3, and not significant between Wave 1 and Wave 2. Therefore, it appears that the reciprocal relations – in particular – that between emotional vulnerability and parental invalidation, could be observed under

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specific circumstances. Notably, the majority of the Wave 2 (September 2019–July 2020) and Wave 3 (March 2020–January 2021) data were collected during the COVID-19 pandemic – a period when many families experienced significant changes in living and work arrangements due to the stay-at-home policies implemented in Singapore. The stay-at-home policies indicated that parents and adolescent spent significantly more time in close proximity, which increased opportunities for interactions to occur. We speculate that the association between parental invalidation and individual vulnerabilities might become more salient during increased interactions and familial stress. Stressful life events could deplete an individual's regulation resources, resulting in less regulation and coping capacities (Stucke & Baumeister, 2006). Indeed, Chung and colleagues (2020) found that among Singaporean parents, the association between the impact of COVID-19 (financial, resource, and psychological) and harsh parenting was mediated by parenting stress. In addition, studies have demonstrated that adolescent's mental health difficulties increased during the pandemic, with positive parental communication and parental conflicts serving as protective and risk factors, respectively (Magson et al., 2021; Panchal et al., 2021). Taken together, the challenges and stress as a result of the pandemic may contribute to a decrease in parents' ability to self-regulate and cope with the demands of parenting. This may result in more frequent invalidating responses to a child's negative emotions or impulsive behaviors, which are also heightened during periods of stress. Invalidating parental responses may then contribute to increases in a child's emotional reactivity (Shenk & Fruzzetti, 2011), thereby, forming a negative feedback loop. Future studies could extend this study by examining potential contextual factors (e.g., low socioeconomic status, increased environmental stress) that may moderate the transactional associations between a child's vulnerabilities and parental invalidation.

The results also showed that while increases in impulsivity temporally contribute to greater parental invalidation, the reverse association did not hold. Several reasons might explain this finding. First, it is important to note that self-report and behavioral lab task measures of impulsivity likely tap onto different aspects of impulsivity, with the former measuring impulsive tendencies, while the latter measuring impulsive states (Cyders & Coskunpinar, 2011, 2012). It is possible that parental invalidation may exert more effects on moment-to-moment impulsive behaviors, which are better captured via behavioral lab tasks (e.g., the GoStop impulsivity paradigm [Dougherty et al., 2005]), rather than the BIS-11 (Patton et al., 1995), which measures overall impulsive tendencies. It is also plausible that the influence of parental invalidation on impulsivity may be more noticeable over a longer term (compared to a short term of six months). For instance, past longitudinal studies examining the effect of parenting behaviors on impulsivity have found that negative parenting behaviors (e.g., strictness / intrusiveness / less clear and consistent discipline) predicted higher impulsivity in children two (Houck & Lecuyer-Maus, 2004) to four years later (Olson et al., 2002).

Our findings suggest that reducing parental invalidation and equipping adolescents with emotion regulation or impulse control skillsets could help mitigate the development of emotion regulation deficits or BPD symptoms. Our study further highlights the possibility that reducing a child's impulsivity or emotion dysregulation tendencies may in turn decrease parental invalidation. These strategies are consistent with approaches employed in existing evidence-based interventions for BPD such as dialectical behavior therapy (DBT) (Linehan, 2014). Empirical studies have demonstrated that dialectical behavior therapy (DBT) adapted for adolescents (Miller et al., 1997, 2007) could be effective in reducing impulsivity and improving emotion regulation (MacPherson et al., 2013; Smith et al., 2019). In addition, parental invalidation could be targeted by equipping parents with validation skills, which is an explicit component within the Family Connections program developed for family members of individuals with BPD (Hoffman et al., 2005).

Our study has several strengths. First, the use of a longitudinal design allowed the investigation of the prospective associations between the components of the biosocial model and BPD symptoms, and the examination of the transactional relationship between individual vulnerabilities and parental invalidation. Second, the recruitment of a community sample of adolescents based in Singapore allowed us to validate the biosocial model in a more collectivistic Asian context. Future research could investigate different ways in which parental invalidation might manifest and influence child behaviors across cultures. The fact that the adolescents recruited in the study were staying with their parents also enables a direct examination of reciprocal influences between emotional vulnerability/impulsivity and parental invalidation. Use of an observational design allows for the examination of BPD symptoms in a naturalistic, community setting, in contrast to a clinical setting where established associations could be a function of clinical interventions.

There are several limitations in our study. All questionnaires utilized in our study are self-report measures, which are subject to social desirability, common method, and/or recall biases. Future studies could consider the use of observational measures or having multiple informants for a construct (e.g., the Urgency-Premeditation-Perseverance-Sensation Seeking-Positive Urgency (UPPS-P) impulsivity scale – parent-report version [Whiteside & Lynam, 2001] to measure child impulsivity). In addition, although the recruitment of an adolescent community sample enabled the examination of BPD in a developmental period in which symptoms of BPD are likely to first manifest (Zanarini et al., 2001), the indicators of impulsivity and emotional vulnerability at this developmental period are likely to have been shaped and potentiated by earlier developmental experiences. Future longitudinal studies could consider recruiting families with young children, and investigate whether the strength of the associations between child vulnerabilities or parental invalidation and BPD symptoms may change over time. This would help to identify the periods in which parental invalidation may exert its strongest influence on individual vulnerabilities and emotion regulation, and inform the development of early intervention programs.

Overall, our study provided empirical support for the biosocial model in a Singaporean context. Our results suggest the possibility that the transactional associations between the vulnerability factors and parental invalidation could be amplified in times of increased familial stress. A further investigation of the contexts in which the transactional relationships are amplified, and possible modifiable factors (e.g., peer relations, early interventions) that might influence the associations between the components of the biosocial model and BPD symptoms would enhance our understanding of the etiology of BPD.

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