

Parental Invalidation and Its Associations With Borderline Personality Disorder Symptoms: A Multivariate Meta-Analysis

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Childhood invalidation has been postulated to be implicated in the development of borderline personality disorder (BPD), according to the biosocial model. Despite its significance, most systematic reviews and meta-analyses have focused on examining the associations between extreme forms of invalidation, such as sexual abuse, and BPD. Nonetheless, individuals could experience mild-to-severe levels of parental invalidation, with or without abuse. This study examined the relationship between parental invalidation as an overall construct and BPD symptoms, synthesized across 21 studies (total $N = 7,198$). As most reviewed studies utilized retrospective self-report measures, the effect sizes derived pertained largely to the association between reported childhood parental invalidation and BPD symptoms. We conducted a multivariate meta-analysis to account for the dependence of multiple effect sizes obtained from a single study and to maintain precision in obtained effect sizes. Maternal, paternal, and overall parental invalidation were positively associated with BPD symptoms, with small-to-moderate effect sizes of .26, .23, and .25, respectively. The mean effect size of maternal invalidation was greater than that of paternal invalidation. We also investigated if the parental invalidation–BPD associations were moderated by child gender, age, and culture (degree of individualism). Only *overall* parental invalidation and BPD symptoms association was moderated by child's gender, with larger effect sizes found for samples with a greater proportion of males. Age and culture did not moderate the parental invalidation–BPD association. Risk of publication bias was low. Our findings provide support for the invalidating childhood environment component of the biosocial model.

Keywords: invalidation, borderline personality disorder, biosocial model, parenting, meta-analysis

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Borderline personality disorder (BPD) is a severe and pervasive mental health condition characterized by dysregulation in affective, cognitive, interpersonal, and behavioral domains. BPD affects approximately 1.6% of the general population and 10% and 20% of mental health outpatients and inpatients, respectively (American Psychiatric Association, 2013). One of the most influential models of BPD is the biosocial model, which proposes that BPD develops as a result of an ongoing, reciprocal transactions between an individual's biological vulnerabilities (early impulsivity and emotional vulnerability) and an invalidating childhood environment (Crowell et al., 2009; Linehan, 1993). 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. Impulsivity pertains to inappropriate actions that are initiated quickly without much deliberation. An invalidating environment refers to an environment in which parents (or caregivers) constantly delegitimize a child's expressed emotions by (a) stating that the child's views are inaccurate, (b) misattributing the emotional expressions to undesirable personal characteristics such as oversensitivity, (c) oversimplifying or minimizing difficulties, and (d) discouraging the display of negative affect (Linehan, 1993). Severe forms of invalidation could include sexual and physical abuse. A key feature of invalidation is that it punishes an admissible and reasonable response (Fruzzetti et al., 2005). For example, telling an individual that there is nothing to be angry about when one's wants are denied can be considered an invalidation of emotions. As a whole, an environment can range from being mildly to severely invalidating, with the chronicity and pervasiveness of invalidating responses defining the degree of invalidation (Fruzzetti et al., 2005).

To date, several systematic reviews and meta-analyses have been published on the association between sexual abuse, an extreme form of invalidation, and various mental health outcomes. Results from

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meta-analyses indicated a moderate association ($r = .28$) between sexual abuse and BPD (Fossati et al., 1999), whereas a systematic review concluded that childhood sexual abuse is an important risk factor for the development of BPD (de Aquino Ferreira et al., 2018). Youths with a history of sexual abuse were found to be five times more likely to be diagnosed with BPD than those who were not abused (Winsper et al., 2016). In addition, sexual abuse was associated with an increased risk for a lifetime diagnosis of anxiety, depression, posttraumatic stress disorder, and suicide attempts (Chen et al., 2010). Nonetheless, although childhood sexual abuse has often been reported in inpatients with BPD, with its severity being positively associated with the intensity of BPD symptoms (Zanarini et al., 2002), it is neither a necessary nor sufficient condition for the development of BPD. Furthermore, sexual abuse is an extreme form of invalidation, whereas Linehan's (1993) operationalization of invalidation encompasses both abusive and nonabusive forms of parental practices. A comprehensive examination of the biosocial model would benefit from a quantitative synthesis of the literature specific to an invalidating environment as operationalized by Linehan (1993) and symptoms of BPD. Although the literature has consistently demonstrated a positive association between past parental invalidation and BPD, the effect sizes obtained across the studies varied from small (Keng & Soh, 2018; Sturrock et al., 2009) to medium (DeShong et al., 2015; Robertson et al., 2013). Furthermore, factors contributing to the variance in reported effect sizes have remained largely unexplored. Therefore, this article aimed to fill the gap by conducting a meta-analysis to examine the association between parental invalidation and symptoms of BPD and possible moderators of the association.

Invalidation and the Development of BPD Symptoms

An invalidating environment could contribute to the development of BPD symptoms via several means. First, dismissing or misattributing a child's expressed emotions to undesirable personality traits could contribute to heightened emotion dysregulation (Fruzzetti et al., 2005). Several studies have found that relative to individuals who were validated, those who were invalidated experienced less positive affect, greater worry (Linton et al., 2012), higher negative affect (Greville-Harris et al., 2016; Shenk & Fruzzetti, 2011), and greater physiological arousal (Shenk & Fruzzetti, 2011; Woodberry et al., 2008) and required more time to rate how they felt and how comfortable they were with their emotions (Woodberry et al., 2008). The effect was evident in community adults with BPD features (Woodberry et al., 2008) and college or community participants (Greville-Harris et al., 2016; Linton et al., 2012; Shenk & Fruzzetti, 2011).

Second, in an invalidating environment, adaptive attempts at socializing or problem-solving, such as assertive communication, could be discouraged or punished. Meanwhile, inappropriate responses such as self-harm or other impulsive behaviors (e.g., substance use) commonly seen in BPD might be reinforced by the provision of attention and help or elimination of aversive responses (Fruzzetti et al., 2005; Linehan, 1993), further hampering the development of appropriate social and problem-solving skills. Indeed, studies have found positive associations between parental invalidation and nonsuicidal self-injury in Australian (Mahtani et al., 2019) and Singaporean (Tan et al., 2014) samples. In a separate study, perceived family invalidation among Chinese

adolescents was found to predict engagement in nonsuicidal self-injurious behaviors in the following year (You & Leung, 2012).

Potential Moderators of Association Between Parental Invalidation and BPD Symptoms

A review of the literature suggests that the gender of child and parent, the degree of individualism in the participants' culture, and child's age are potential moderators of the parental invalidation-BPD symptoms association.

Gender

According to Bowlby's attachment theory, children form an attachment with their primary caregivers (Bowlby, 1969), with the quality of attachment possibly influencing the development of coping abilities and psychopathology later in life (Zimmer-Gembeck & Skinner, 2016). In many societies, mothers are traditionally the primary caregivers (Cinamon & Rich, 2002) who likely serve as a primary attachment figure for most children (Bowlby, 1969). Therefore, compared with fathers, invalidation experienced from mothers may have a greater impact on children's socioemotional health. In one study, Sturrock and colleagues (2009) found that maternal invalidation, but not paternal invalidation, was positively correlated with symptoms of BPD. The findings were replicated in a Singaporean college sample, with maternal, but not paternal, invalidation remaining as a significant predictor when both maternal invalidation and paternal invalidation were entered into a multiple regression model to predict BPD symptoms (Keng & Soh, 2018). Given these findings, we expect that the association between maternal invalidation and BPD would be stronger than that between paternal invalidation and BPD in the current study.

Apart from the gender of the parent, the child's gender may also moderate the association between parental invalidation and BPD outcomes. Previous research has demonstrated that family invalidation predicted BPD features such as future suicide events (Yen et al., 2015) and suicidal ideation (Selby et al., 2013) for boys but not for girls. In another study, Hay and Ashman (2003) found that relationships with parents had associations with emotional stability only for adolescent boys. In addition, Leong and colleagues (2011) found a positive association between the level of invalidation between couples and depressive symptoms only for husbands but not wives. Taken together, these findings suggest that males may be more reactive to family invalidation and may have fewer resources (such as supportive peer relations) to buffer the impact of family invalidation compared with females. For instance, compared with their female counterparts, males tend to communicate and self-disclose less (Furman & Buhrmester, 1992; Gorrese & Ruggieri, 2012). Therefore, in the current study, we predict that samples with more males (as recipients of invalidating behaviors from parents) would demonstrate stronger associations between parental invalidation and BPD symptoms.

Culture—Degree of Individualism

Culture can be viewed as a set of psychosocial processes, such as beliefs and attitudes, that is shared among a group of people (Tseng, 2001). One dimension of culture is the degree of individualism/collectivism (Hofstede, 2001), with cultures high on individualism

placing more focus on independence, self-expression, and self-reliance and cultures high on collectivism emphasizing interdependence, group harmony, and collective values more. Culture has been proposed as a factor that influences parental emotion socialization (Halberstadt & Lozada, 2011) and the presentation of BPD symptoms (Neacsu et al., 2017). Little research, however, has empirically evaluated the role of cultural dimensions as moderators of the association between parental invalidation and BPD symptoms, with the exception of Keng and Soh (2018), who found that self-construal and conformity moderated the association between invalidation and BPD symptoms. Specifically, maternal invalidation was positively associated with BPD symptoms only for participants with interdependent self-construal and a high level of conformity but not for those with independent self-construal. It is plausible that individuals from collectivistic cultures may be more susceptible to negative effects of invalidation, as they value group identity and harmony more compared with those from individualistic cultures. Therefore, we expect that the association between parental invalidation and BPD symptoms would be stronger for countries that are less individualistic (assessed at the country level).

Age

We also set out to test if child's age would influence the association between parental invalidation and BPD symptoms. The association may be stronger as a child grows older due to the cumulative effects of invalidation. Past empirical studies have demonstrated that parenting behaviors could continue to influence emotion regulation skills in adulthood (Manzeske & Stright, 2009; Tani et al., 2018; Yan et al., 2016), indicating the possibility of a cumulative effect. At the same time, a child may develop other forms of supportive relationships (e.g., peers and romantic partners) that could buffer the effects of past parental invalidation. Given these contrasting possibilities, our meta-analysis would examine, exploratorily, the moderating role of child's age on the association between parental invalidation and BPD symptoms.

Study Objectives

The current meta-analysis aimed to clarify the relationship between parental invalidation and BPD and examine moderators of the association. Our moderators of interest were the source of invalidation (maternal vs. paternal), the proportion of males in the sample, degree of individualism (extracted from Hofstede et al., 2010) for the country from which a sample was drawn, and age. We additionally examined if the scale used to measure past parental invalidation would moderate the associations between parental invalidation and BPD symptoms. As the scales included in this study purport to measure parental invalidation according to Linehan's (1993) operationalization, we expect that the types of scales utilized in the reviewed studies would not influence the parental invalidation—BPD association. Our hypotheses are as follows:

Hypothesis 1: Maternal, paternal, and overall parental invalidation would be positively associated with symptoms of BPD.

Hypothesis 2: The effect sizes between parental invalidation and BPD would be stronger for (a) maternal (vs. paternal) invalidation, (b) for samples with more males, and (c) for studies

that recruited samples from countries with lower individualism scores.

Hypothesis 3: We did not a priori hypothesize the moderating role of age given the lack of empirical studies in this area.

Method

Eligibility Criteria

Studies were included if they were written in English language, collected empirical data, included a self-report or observational measure that was in line with Linehan's (1993) operationalization of invalidation, and included a measure on symptoms of BPD. A recent review by Musser and colleagues (2018) identified 47 self-report questionnaires that have been used to measure parental invalidation. Despite the large number of questionnaires used, only two measures, namely, the Invalidating Childhood Environments Scale (ICES; Mountford et al., 2007) and the Coping with Children's Negative Emotions Scale (CCNES; Fabes et al., 1990), were found to closely measure or align with all four components of invalidating environment (inaccuracy, misattribution, discouragement of negative emotions, and oversimplification of problem-solving) as defined by Linehan (1993). Hence, in this review, studies that utilized the ICES (Mountford et al., 2007) and the CCNES (Fabes et al., 1990) were included. Studies that utilized the Socialization of Emotions Scale (SES; Krause et al., 2003), the Recalled Childhood Socialization of Emotion Scale (RCSES; Krause et al., 2003), and the Invalidating Environment—Child Scale (IE-Child; Sauer & Baer, 2010), which were developed based on the CCNES, were also included. We also included studies that utilized the Validating and Invalidating Behaviors Coding Scale (VIBCS; Fruzzetti, 2001), an observational measure that is in line with Linehan's (1993) definition of parental invalidation.

Articles that only measured trauma or abuse were excluded, as our article focused on the association between general parental invalidation (that includes nonabusive forms of invalidation) and symptoms of BPD. Theoretical papers, qualitative research papers, and reported case studies were not included.

Information Sources

Articles were identified via a search of titles and abstracts in the Scopus database. An additional search was conducted via the Proquest Theses and Dissertations database. The search was first conducted in January 2019 and was last conducted in December 2020.

Article Search

Peer-reviewed articles written in English under the subject area of psychology were retrieved. The search terms used were (biosocial model AND borderline personality disorder) OR (biosocial theory AND borderline personality disorder) OR (invalidating environment AND borderline personality disorder) OR (invalid* AND borderline personality disorder) OR (parent* AND borderline personality disorder) OR (communication AND borderline personality disorder). A total of 981 peer-reviewed articles and 199 theses/dissertations were initially retrieved from Scopus and

Proquest Theses and Dissertations databases, respectively. We contributed two additional unpublished data sets.

Study Selection

A two-step screening procedure was carried out. First, screening of the article titles and abstracts for relevance led to the exclusion of 1,029 articles. The full texts of 150 articles were then scrutinized. A further 129 articles were excluded due to the lack of appropriate scales used to measure invalidation, a lack of empirical data (theoretical or review papers), or a failure for contacted study authors to reply with information for required effect sizes. For the current study, 51 effect sizes were extracted from 21 studies with 25 independent samples. Figure 1 depicts the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) flowchart of the article screening process. All studies were cross-sectional, with the exception of the one by Crystal (2017), which adopted a longitudinal design. Only two studies (Gill et al., 2018; Keng et al., 2019) used clinical samples, whereas all other studies recruited college or community participants. Specifically, Gill and colleagues (2018) recruited individuals with BPD traits who were currently engaged in therapy, whereas Keng

and colleagues (2019) recruited psychiatric patients in a hospital setting.

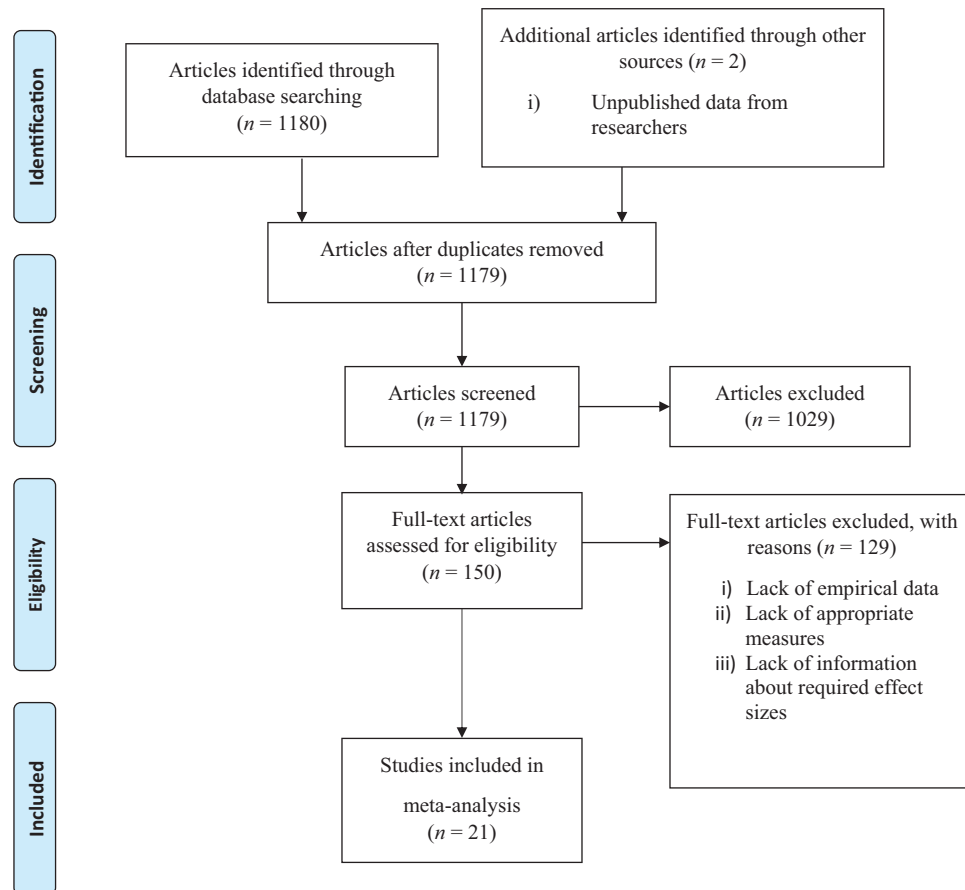
Data Collection Process

All data were coded by the first author. A subset of seven studies (30% of the total included studies) was randomly selected and independently coded by the second author on a standardized spreadsheet. Cohen's κ for categorical variables (e.g., sample type, measure used) were all above 0.99, whereas the intraclass correlation coefficients for continuous variables (e.g., sample sizes, proportion of males in samples, effect sizes) were all 1. Disagreements were resolved through discussion. Attempts were made to contact study authors when required data for computing standardized effect sizes were not available.

Data Items

Following the identification of articles to be included, the Pearson correlation coefficient between the following pairs of variables was extracted whenever available: (a) maternal invalidation and BPD symptoms, (b) paternal invalidation and BPD symptoms, and (c) parental invalidation and BPD symptoms. Note that we did not

Figure 1
PRISMA Flow Diagram



Note. PRISMA = Preferred Reporting Items for Systematic reviews and Meta-Analyses. See the online article for the color version of this figure.

statistically combine the correlation between maternal or paternal invalidation and BPD symptoms to form the correlation between parental invalidation and BPD symptoms if a study did not provide the correlation between parental invalidation and BPD symptoms. As the multivariate meta-analysis method was used to analyze the findings, we were able to obtain multiple effect sizes from a single study. Furthermore, as we were using multivariate meta-analysis, the intercorrelations among the different invalidation variables (e. g., correlation between maternal and paternal invalidation) were also extracted for calculation of dependency of effect sizes.

In addition, data on sample size, scales used to measure parental invalidation and symptoms of BPD, sample type (clinical or non-clinical sample), sample demographics (percentage of males, average age), study type (cross-sectional or longitudinal), and degree of individualism characterizing the country that participants came from (established based on Hofstede et al., 2010) were coded. Due to the very low number of studies that used observational measures ($n = 1$), parent-report parental invalidation measures ($n = 2$), and clinical samples ($n = 2$), we were unable to conduct moderation analysis to investigate possible effects of type of measure, informant type, and clinical status on the association between parental invalidation and BPD. As most effect sizes extracted from the included studies were obtained from self-report retrospective measures of parental invalidation, the references to parental invalidation in our article pertain mainly to reported childhood parental invalidation. We present the identified studies and their associated characteristics in Table S1 in the online supplemental materials.

Summary Measures

In this article, correlation coefficient (r) is the principal summary measure of association between parental invalidation and BPD symptoms. Correlation coefficients extracted from individual studies were first converted to Fisher's Z to be used for the meta-analysis (Hedges & Olkin, 1985). Individual study sample sizes were used to calculate the variance of Z . For purposes of interpretation, the mean effect size and the 95% confidence interval were converted back to r .

Synthesis of Results

Due to the presence of studies providing multiple effect sizes (e. g., correlation coefficients between maternal invalidation and BPD, and between paternal invalidation and BPD), we used multivariate meta-analysis techniques to analyze the data. Multivariate meta-

analysis is the preferred analytic strategy, as it takes into account the relations among the various effect sizes in its estimation procedure (Becker, 2000; Jackson et al., 2012). This is in contrast to running several univariate meta-analyses, which does not consider how the different effect sizes might be associated with each other. Hence, compared with conducting multiple univariate meta-analyses, the estimated mean effect sizes obtained via multivariate meta-analysis would be more accurate and would generally have smaller standard errors (Demidenko, 2013; Riley, 2009).

To address the research objectives, we used both random-effects and mixed-effects models to estimate the summarized weighted effect sizes of the various parental invalidation and BPD symptoms and to examine moderators of the associations. Maximum likelihood estimation was used to obtain the following parameter estimates: (a) summary effect size for each of the invalidation type (maternal, paternal, and parental invalidation), (b) the heterogeneity of effect sizes between studies, and (c) the slope parameters for the moderator analyses. All analyses were conducted using both the *metaSEM* (Cheung, 2015) and *mvmeta* (Gasparrini et al., 2012) packages in R (R Development Core Team, 2018).

Risk of Bias Across Studies

Publication bias was assessed via the visual inspection of the funnel plots for asymmetry of effect sizes, p -curve analysis (Simonsohn et al., 2014; Simmons & Simonsohn, 2017), and Orwin's fail-safe N (Orwin, 1983). The funnel plots for maternal, paternal, and parental effect sizes and details on p -curve analysis are presented in Supplemental Material A and Supplemental Material B, respectively.

Results

Risk of Bias Across Studies: Publication Bias

Table 1 presents the results of the publication bias analyses. Based on the statistics, publication bias and p -hacking were not evident, and so, the evidential value of the included studies was not compromised.

Synthesis of Results: Main Analyses

To obtain the mean effect sizes, two random-effects meta-analytic models were conducted. The first was a multivariate model that examined two dependent effect sizes—(a) correlation between

Table 1
Summary of Main Analysis and Publication Bias Analyses

Invalidation	k	N	r	[95% CI]	τ^2	I^2	Publication bias			
							FSN	Z_{half}	Z_{full}	Z_{flat}
Maternal	14	3,963	.26*	[.22, .30]	.004	.51	23	−12.88*	−12.93*	9.23
Paternal	13	3,912	.23*	[.18, .28]	.007	.65	20	−12.15*	−12.32*	8.90
Parental	15	4,438	.25*	[.21, .29]	.003	.42	24	−10.35*	−10.39*	7.19

Note. k = number of studies; N = total number of participants; r = mean effect size in Pearson correlation coefficient; 95% CI = 95% confidence interval of the mean effect size; τ^2 = true between-studies variability; I^2 = ratio of true heterogeneity to total variance observed; FSN = Orwin's fail-safe N ; Z_{half} = Z statistic for the right skewness of the half p -curve; Z_{full} = Z statistic for the right skewness of the full p -curve; Z_{flat} = Z statistic for the test of whether the observed full p -curve is significantly flatter than that of a 33%-power p -curve.

* $p < .001$.

maternal invalidation and BPD and (b) correlation between paternal invalidation and BPD. The second was a univariate model that investigated the effect sizes between parental invalidation and BPD.

For the multivariate model, to minimize type I error, Cheung (2015) advised that all the dependent effect sizes be simultaneously analyzed. Therefore, before estimating the individual mean effect sizes, a comparison between a model that constrained the two dependent mean effect sizes to be zero and a full model without any constraints was made. The constrained model is nested under the full model. This is to test whether the two dependent mean effect sizes are equal to zero. The likelihood ratio statistic was $\Delta\chi^2(df = 2) = 60.09, p < .001$. The null hypothesis that the two effect sizes are zero was then rejected. Because this omnibus multivariate test was significant, the two mean effect sizes were then individually estimated and tested.

The results of the main analysis are shown in Table 1. All the mean effect sizes and their confidence intervals in the metric of Fisher's Z were converted back to Pearson's r for interpretation. The estimated mean effect sizes obtained from the two random-effects models for maternal, paternal, and parental invalidation were .26, .23, and .25, respectively. This is consistent with our hypothesis that maternal, paternal, and overall parental invalidation would be positively associated with symptoms of BPD. Moreover, according to Cohen (1992), the magnitudes of these effects were considered to be small to moderate.

As hypothesized, we expect that the effect sizes would be stronger for maternal as compared with paternal invalidation. From the inspection of the mean effect sizes, the mean effect size of maternal invalidation (.26) is slightly greater than that of paternal invalidation (.23). A further analysis was done to test whether the mean effect sizes of maternal and paternal invalidation were equal. A comparison between a model that was fitted to have the equality constraint on the mean effect sizes and the full model without any constraint was made. The likelihood ratio statistic was $\Delta\chi^2(df = 1) = 7.39, p < .01$. This suggests that the null hypothesis of equal mean population effect sizes between maternal and paternal invalidation was rejected. The mean effect size of maternal invalidation was statistically significantly greater than that of paternal invalidation, supporting our hypothesis that effect sizes for maternal invalidation are stronger than paternal invalidation.

The multivariate homogeneity test (Jackson et al., 2012) for maternal and paternal invalidation was significant, $Q(df = 25) = 47.09, p < .01$, suggesting that there was heterogeneity between studies in terms of the magnitude of effect sizes. The univariate homogeneity test for parental invalidation was also significant, $Q(df = 14) = 25.95, p < .05$, suggesting that effect sizes were heterogeneous between studies. The multivariate I^2 for maternal and paternal invalidation was .47. Cheung (2015) recommended that individual I^2 be computed for each outcome because a single index is not exactly useful in quantifying the heterogeneity across different outcomes and studies. Table 1 presents the individual I^2 and the estimated variance components for maternal, paternal, and parental invalidation. I^2 is interpreted as the portion of the total observed variance of the effect sizes that is attributed to the between-study heterogeneity. The degree of heterogeneity in the population effect sizes ranged from moderate to moderately high ($I^2 = .42-.65$).

Moderator Analyses

Due to the heterogeneity of effect sizes for maternal, paternal, and parental invalidation, mixed-effects models were run to see if this heterogeneity could be explained by the proposed moderators. Similar to the main analysis, maternal invalidation and paternal invalidation were analyzed together in a multivariate model, whereas parental invalidation was analyzed separately in a univariate model. The results are presented in Table 2.

The Q_{Residual} statistic is a measure of heterogeneity of effect sizes across studies after accounting for the specified moderator. The null hypothesis assumes homogeneity of effect sizes across studies and that the true effect is identical for all studies. If the Q_{Residual} is not statistically significant, this indicates a substantial amount of variability in the effect sizes could be accounted for by the included moderator. However, if the Q_{Residual} is significant, this implies that even after accounting for the included moderator, there is still a significant amount of variability in the effect sizes that could not be accounted for. This also suggests that other moderators that we did not include in our study could potentially explain such variability. In the current study, only the Q_{Residual} statistic for parental invalidation became nonsignificant when percentage of males in the sample was included as a moderator. This suggests

Table 2
Summary of Moderator Analyses

Predictor	Q_{Residual}	Invalidation	k	b	SE	Z
Percentage male	45.49* ($df = 23$)	Maternal	14	-.001	.001	-1.02
		Paternal	13	-.0003	.002	-0.18
		Parental	15	.003	.001	3.02*
Culture—degree of individualism	46.64* ($df = 23$)	Maternal	14	.0004	.001	0.47
		Paternal	13	.0002	.001	0.21
		Parental	15	-.0002	.001	-0.35
Age	44.13* ($df = 23$)	Maternal	14	-.002	.002	-1.27
		Paternal	13	-.004	.002	-1.65
		Parental	15	-.002	.002	-0.77
Invalidation scales	43.13* ($df = 23$)	Maternal	14	-.027	.017	-1.60
		Paternal	13	-.026	.020	-1.27
		Parental	14	.007	.013	0.53

Note. Q_{Residual} = Cochran Q test for residual heterogeneity; k = number of studies; b = slope estimate; SE = standard error of slope estimate; Z = Z statistic for the significance of slope estimate;

* $p < .05$.

that percentage of males could explain a substantial amount of heterogeneity of the effect sizes relating to parental invalidation. On the other hand, the Q_{Residual} statistics across all invalidation types remained significant after the inclusion of the other moderators, indicating that a large amount of variability in the effect sizes remained after accounting for the effects of the moderators.

Child Gender—Percentage of Males in Samples

We examined if child gender would moderate the association between parental invalidation and BPD symptoms. The mean and median of percentage of males in the samples across studies were 29% and 28%, respectively. The percentages ranged from 0% to 49%. The only slope estimate that emerged significant was for parental invalidation ($b = .003$, $SE = .001$, $p < .01$). The positive slope suggests that as percentage of males in the samples increased, effect sizes of parental invalidation also increased. This predictor was able to explain 22.9% of the between-studies variability for parental invalidation. Therefore, our hypothesis that effect sizes between parental invalidation and BPD symptoms would be stronger for samples with more males was supported.

Culture—Degree of Individualism

To investigate if culture moderated the parental invalidation–BPD symptoms association, we first assigned a score to each study to code for the degree of individualism in the country that the participants were from, with higher scores indicating a higher degree of individualism. The scores for each respective study were extracted from the article by Hofstede et al. (2010), which provided estimates of degree of individualism of countries across the world. The mean and median score in the sample of studies were 77 and 91, respectively. The scores ranged from 20 (Singapore) to 91 (United States). The slopes were not significant for all three outcomes, indicating that the effect sizes did not vary as a function of the degree of individualism. Our hypothesis was therefore not supported.

Age

We examined whether the effect sizes varied as a function of the age of recipients of invalidation. The mean and median of ages (in years) of participants in the samples across studies were 23.68 and 19.93, respectively. The ages ranged from 11.34 to 44.96 years. The slopes were not significant for all three outcomes, indicating that the effect sizes did not vary as a function of the age of the participants in the samples. Therefore, age did not moderate the association between parental invalidation and BPD symptoms.

Scales Used to Measure Parental Invalidation

We were interested in whether the scales used to measure parental invalidation may influence the effect sizes. There was a total of seven different invalidation scales used by the reviewed studies. These scales included ICES (Mountford et al., 2007), CCNES (Fabes et al., 1990), CCNES—Adolescent Perception Version (Fabes & Eisenberg, 1998), SES (Krause et al., 2003), RCSES (Krause et al., 2003), IE-Child (Sauer & Baer, 2010), and the VIBCS (Fruzzetti, 2001). However, analysis could not be carried out for most of the scales because they were used only in one or two studies. To ensure that the results are stable, we only conducted analyses for scales that were used in at least three

independent samples. As several scales (i.e., CCNES-AP, SES, RCSES, and IE-Child) were developed based on the CCNES (Fabes et al., 1990)¹, we conducted the moderation analyses by comparing ICES (Mountford et al., 2007) against SES, CCNES-AP, RCSES, or IE-Child (Fabes & Eisenberg, 1998; Krause et al., 2003; Sauer & Baer, 2010).

For maternal and paternal invalidation, the number of studies that used ICES (Mountford et al., 2007) and SES/CCNES-AP/RCSES/IE-Child (Fabes & Eisenberg, 1998; Krause et al., 2003; Sauer & Baer, 2010) was nine and five, respectively. For parental invalidation, the number of studies that used ICES (Mountford et al., 2007) and SES/CCNES-AP/RCSES/IE-Child (Fabes & Eisenberg, 1998; Krause et al., 2003; Sauer & Baer, 2010) was six and eight, respectively. The slopes were not significant for all the outcomes, suggesting that the different scales that have been used to measure parental invalidation were likely measuring the same underlying construct.

Discussion

Our main objectives were to investigate the associations between parental invalidation and BPD and its possible moderators (gender, culture, and age). As hypothesized, maternal, paternal, and overall parental invalidation were found to be positively associated with BPD symptoms, with small-to-moderate effect sizes of .26, .23, and .25, respectively. The maternal invalidation–BPD symptoms association was stronger than that for paternal invalidation. Both maternal and paternal invalidation associations with BPD symptoms were not moderated by child's gender, child's age, and degree of individualism. However, the association between overall parental invalidation and BPD symptoms varied as a function of child's gender, with larger effect sizes found for samples with a greater percentage of males. Similarly, the degree of individualism and age did not moderate the association between overall parental invalidation and symptoms of BPD. Our moderator analyses conducted on the self-report measures included in our study indicated that the scales did not influence the effect sizes obtained in the various studies and were likely measuring a similar construct. Our analyses also indicated that there was no evidence of publication bias.

Our findings provide support for the biosocial model that emphasizes the role of an invalidating childhood environment in the development and maintenance of BPD (Linehan, 1993). Similar small-to-medium effect sizes have also been found for the environmental transmission of BPD via poor parenting behaviors in a biological and adoptive study of families conducted by Fatimah and colleagues (2020). The current study also contributes to the growing literature that parenting behaviors, in general, are moderately related to child psychopathology (McLeod, Weisz, et al., 2007; McLeod, Wood, et al., 2007).

¹ The SES scale was first adapted from the CCNES (Fabes et al., 1990) by Krause and colleagues (2003). Further research conducted on the SES (Krause et al., 2003) led to the development of the RCSES (Krause et al., 2003) and IE-Child (Sauer & Baer, 2010). Separately, the CCNES-AP (Fabes & Eisenberg, 1998) is the adolescent self-report version of the CCNES (Fabes et al., 1990) and mirrors the SES (Krause et al., 2003). As the RCSES (Krause et al., 2003) and IE-Child (Sauer & Baer, 2010) were derived from the SES (Krause et al., 2003), they were considered to be similar to SES (Krause et al., 2003) and CCNES-AP (Fabes & Eisenberg, 1998) and were given the same categorical code for purposes of analysis.

We found that maternal, compared with paternal, invalidation had a stronger association with BPD symptoms. Thus, invalidation experienced from mothers, who are potentially the primary attachment figures for many children, may have a greater impact on the child's socioemotional development. Interactions with significant attachment figures aid children in developing an understanding of the self and others and forming expectations about interpersonal interactions (Bowlby, 1973). For instance, children who experienced repetitive invalidation from mothers may expect invalidation from others and perceive innocuous behaviors or words as invalidation. In line with research demonstrating the association between attachment security and both emotion regulation (Abraham & Kerns, 2013; Hershenberg et al., 2011) and interpersonal relationships outcomes (Abraham & Kerns, 2013), such experiences could predispose the child to develop BPD features such as emotion dysregulation and poor interpersonal relationships. Notably, however, there was a significant positive association between paternal invalidation and BPD symptoms, suggesting that invalidation from fathers also plays a role in the development of BPD symptoms in children.

We found that the association between overall parental invalidation and BPD symptoms was stronger in studies with more male participants. Although males are not particularly vulnerable to invalidation from either parent, the overall level of invalidation in the home environment has a greater impact on males than females. Perhaps, compared with girls, boys may self-disclose less (Grabill & Kerns, 2000), thereby limiting their parents' understanding of their emotions and thus have a higher risk of experiencing parental invalidation. In addition, boys may have less peer support to buffer the impact of family invalidation (Furman & Buhrmester, 1992; Gorrese & Ruggieri, 2012). Future studies could investigate if (a) boys have a higher likelihood to perceive responses as invalidating and if (b) the association between parental invalidation and BPD symptoms, as moderated by gender, would further vary as a function of peer relations.

The degree of individualism failed to moderate the associations between parental invalidation and BPD. Of the reviewed studies, four were studies utilizing Singaporean samples (country-level individualism score: 20); one study was conducted in Argentina (country-level individualism score: 46); and the remaining 16 studies were conducted in the United States, United Kingdom, and Australia (country-level individualism scores of 91, 89, and 90, respectively; Hofstede et al., 2010). Notably, the majority of the studies were conducted in countries with similarly high degrees of individualism. It is plausible that the lack of moderation effect is a result of the limited variance in individualism scores. Alternatively, the use of individualism scores coded at the *country* level may not be a sufficient or accurate reflection of actual cultural values upheld by individual participants. For example, despite the fact that Singapore is considered as a largely collectivistic society, there is likely much variation with regard to the degree of individualism (or collectivism) upheld by individuals. Nonetheless, as most studies were conducted in the United States, United Kingdom, and Australia, the generalizability of our results to parent-child relationships in other cultures is limited. Further attempts at examining the extent to which invalidation and BPD symptom outcomes may vary as a function of cultural variables assessed at the *individual* level, as well as cultural variables that pertain more directly to attitudes or beliefs underlying emotional expression and invalidation, could be beneficial.

The meta-analysis found that the association between parental invalidation and BPD symptoms was not moderated by child's age. The finding suggests that the strength of the association

between parental invalidation and BPD symptoms may vary over time depending on the quality of other forms of relationships. Specifically, the presence of supportive or nonsupportive relationships may, respectively, buffer or exacerbate the effects of parental invalidation. Further investigation of whether the quality of other significant relationships could moderate the association between parental invalidation and BPD symptoms is warranted.

Although our study focused on parental invalidation, it is highly likely for both parental invalidation and *validation* to be present in all families. Despite being related concepts, parental invalidation has been demonstrated to be a construct independent of parental validation. For example, Adrian and colleagues (2018) demonstrated that adolescent self-harm was at its highest frequency when levels of both parental validation and invalidation were high. Therefore, future studies could examine both main and interactive effects of parental invalidation and validation on BPD-related outcomes.

Furthermore, based on the family systems theory, thoughts, behaviors, and emotions experienced in a subsystem (e.g., the parent-child subsystem and the spousal subsystem) can be transferred to another subsystem (Erel & Burman, 1995). Studies have demonstrated that parents could influence each other's parenting behaviors (Ponnet et al., 2013) and parent-child relationships (Li et al., 2018), which in turn influences child emotion regulation (Li et al., 2019). Extending the findings of past research, investigating how parents may influence each other's invalidating behaviors and the child's perception of the overall level of parental invalidation would serve to extend our understanding of how the entire family system could influence the development of BPD symptoms.

Our findings in this review are qualified by several limitations. First, with the exception of the study by McCallum and Goodman (2019) that included an observational rating of parental invalidation, all studies used self-report measures to assess parental invalidation. These measures are subject to biases such as social desirability and recall bias. Furthermore, most studies were cross-sectional, which limits the inference of causality. Even though childhood experiences of invalidation are conceptualized as antecedents of BPD symptoms, current BPD symptoms may influence the perception of past parental invalidation. In addition, the reported effect sizes in these studies were likely inflated estimates due to common time measurement variance. Therefore, future longitudinal studies should measure parental invalidation and BPD symptoms at multiple timepoints and track the reciprocal impact of parental invalidation and BPD symptoms on one another. Studies could also include concurrent methods of assessments, such as semi-structured interviews to measure BPD symptoms and observer-rated tasks (such as the VIBCS; Fruzzetti, 2001) to examine the relative impact of objective and perceived invalidation on BPD symptoms.

Although our findings indicate that parental invalidation is implicated in the development of BPD, it is important to note that none of the primary studies included in the meta-analysis had directly assessed for the occurrence of child sexual abuse or other forms of severe abuses. It is plausible that in some cases, abuse might have occurred alongside nonabusive instances of invalidation for some participants. The effect sizes obtained in our meta-analysis could have been slightly inflated and driven by the associations between plausible abuse and BPD symptoms. Nonetheless, all included studies had used questionnaires that operationalized and measured parental invalidation according to Linehan's (1993), with the range of scores corresponding to mild-to-severe level of invalidation. Therefore, the obtained effect sizes between parental

invalidation and BPD reflect a broad range of parental invalidation (that could overlap with sexual abuse) and BPD symptoms. Future studies could additionally include measures of abuse in addition to general questionnaires on parental invalidation to establish the unique effects of abusive versus nonabusive forms of parental invalidation on BPD symptoms.

Our meta-analysis consolidated the parental invalidation and BPD literature and provided empirical evidence highlighting the importance of an invalidating childhood environment in contributing to the development of BPD symptoms. Our study also supported the invalidating childhood environment component of the biosocial model by only including studies that had operationalized parental invalidation according to Linehan (1993). Our study highlighted the importance of addressing and examining how maternal and paternal invalidation, individually and combined, would impact the child. The establishment of parental invalidation as an important factor associated with BPD symptoms also emphasizes the therapeutic value of explicitly targeting invalidation within the family in therapy settings. An enriched understanding of factors moderating the parental invalidation–BPD association, as well as the effects and mechanisms of invalidation, would allow for the development of targeted interventions to reduce invalidating behaviors and practices within the family environment.

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