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Childhood Trauma and Borderline Personality Disorder Traits: A Discordant Twin Study

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A discordant twin design was utilized to examine the potentially causal effects of childhood trauma (CT; i.e., emotional abuse, physical abuse, sexual abuse, and witnessing violence) on borderline personality disorder traits (BPD traits) in early adulthood. The participants were 2,808 twins between 17 and 23 years from the Oslo University Adolescent and Young Adult Twin Project. BPD traits were assessed by the Structured Interview for *DSM–IV* Personality (SIDP-IV), and CT was assessed using the Childhood Trauma Interview (CTI). BPD traits (h^2 = .50) and CT (h^2 = .33–.69) were both found to be moderately heritable. Small but statistically significant associations between CT and BPD traits were found in the total sample. After controlling for shared environmental and genetic factors in the discordant twin pairs, the analyses showed little to no evidence for causal effects of CT on BPD traits. The results indicated that the associations between CT and BPD traits stem from common genetic influences. These findings are inconsistent with the widely held assumption that CT causes the development of BPD.

General Scientific Summary

This study suggests that exposure to trauma in childhood and/or adolescence does not lead to later development of borderline personality disorder traits. Rather, the association between trauma and borderline personality disorder traits is better accounted for by shared genetic influences.

Keywords: borderline personality disorder, childhood trauma, discordant twin design, genetic, adolescents

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Borderline personality disorder (BPD) is characterized by a pervasive pattern of intense and unstable interpersonal relations, an unstable sense of self, intense and fluctuating emotions, impulsive behaviors and recurrent suicidal behavior (American Psychiatric Association, 2013). Although BPD usually has its onset in adolescence, diagnosing BPD before age 18 has been controversial (Kaess et al., 2014). However, empirical studies have shown that BPD can be diagnosed in early adolescence with similar reliability, validity and stability as BPD in adulthood (Kaess et al., 2014; Miller et al., 2008).

The first studies that investigated environmental factors in the etiology of BPD were published in the 1970s (Zanarini, 2000). Since then, the research literature has been concerned with trauma in childhood and adolescence, especially abuse and neglect, as potential causes of this personality disorder (Ball & Links, 2009; Newnham & Janca, 2014). Several research findings have contributed to the commonly accepted assumption of a causal relationship between childhood trauma (CT; often defined as experiences that occur before age 18) and BPD. First, several studies have reported high prevalence of reported maltreatment in childhood and adolescence among persons with BPD. This is summarized and meta-analyzed for BPD traits in childhood (Ibrahim et al., 2018), adolescence (Winsper et al., 2016) and adulthood (Porter et al., 2020). Second, there seems to be a dose-response relationship between maltreatment and number of BPD symptoms (Charak et al., 2018; Ibrahim et al., 2018; Pietrek et al., 2013; Zanarini et al., 2002).

Many clinicians believe that sexual abuse is the single most important cause for developing BPD. Indeed, a systematic review of studies investigating the association between sexual abuse and BPD from 1997 to 2017 indicated that childhood sexual abuse is an important risk factor for BPD (de Aquino Ferreira et al., 2018). However, several studies have found that other types of maltreatment may be just

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as important or more important risk factors for developing BPD (Charak et al., 2018; Lobbestael et al., 2010; Zanarini et al., 2020).

While the etiology of BPD was long considered to be exclusively environmental, the first larger twin study of BPD, published in 2000, established that it also has a considerable genetic/biological basis (Gunderson et al., 2018; Torgersen et al., 2000). Studies that have measured BPD traits at one measurement occasion have reported heritability estimates between .35 and .42 (Bornovalova et al., 2013; Distel et al., 2008; Kendler et al., 2008; Torgersen et al., 2008). Other studies that have attempted to reduce error variance by using longitudinal designs or combining different measures for assessing BPD have reported heritability estimates around .70, implying that additive genetic factors may explain 70% of the total variation in BPD traits (Bornovalova et al., 2009; Reichborn-Kjennerud et al., 2015; Torgersen et al., 2012). Furthermore, results from an extended twin-family study suggest that dominant genetic effects also contribute to individual differences in BPD traits (Distel et al., 2009). Notably, findings from twin studies consistently indicate that shared environmental factors make little to no contribution to causing individual differences in BPD traits.

Although the heritability of BPD has been found to be high, genetic factors do not fully explain familial variance. As described, several studies have highlighted various types of CT as risk factors for the development of this personality disorder. However, the fundamental problem of causal inference is whether the association between CT and BPD is due to a direct effect of CT on the development of BPD or arises from the influence of confounding variables correlated with both. One way to reduce the problem with confounding variables is to use a discordant twin design. The discordant twin design relies on comparing the individual level association between exposure and outcome (i.e., analyzed in a standard regression model) with the association observed within discordant twin pairs (i.e., only one member of a twin pair has been exposed to the potential risk factor). This within-pair association represents the difference in outcome among twin pairs discordant on exposure and is often used to strengthen the claim of a causal effect of the exposure on the outcome (McGue et al., 2010). Twins reared together share the same family environment, MZ twins are genetically identical while DZ twins share, on average, half of their segregating genes. Thus, the DZ within-pair association completely controls for shared environmental factors and partially for genetic factors while the MZ within-pair association completely controls for both shared environmental and genetic factors. Importantly, since measures of the environment, such as life events, are not entirely environmental in nature but are partly influenced by genetic factors (e.g., Kendler & Baker, 2007), it is important to control for shared genetic factors when examining the relationship between CT and BPD traits.

To date, twin studies examining the effect of CT on BPD are scarce. Existing discordant twin studies provide preliminary evidence that CT does not causally impact BPD traits (Berenz et al., 2013; Bornovalova et al., 2013). The study by Bornovalova, et al. (2013) indicated that the relationship between childhood abuse (emotional, physical and sexual abuse) and BPD traits is likely explained by shared genetic factors. However, Berenz, et al. (2013) did not have sufficient power to analyze discordant MZ and discordant DZ twins separately. Because a comparison of effects within discordant DZ twins relative to discordant MZ twins is necessary to determine to what extent the confounding is due to shared environmental and/or genetic factors, the results could not say anything about the relative confounding effects of shared environmental and genetic factors. Clearly, more genetically informed studies are needed to contribute to more robust conclusions and a better understanding of this research area.

The aim of the present study was to assess whether there is evidence for a causal effect of CT (emotional abuse, physical abuse, sexual abuse and witnessing violence) on levels of BPD traits in early adulthood. To do so, we employed a series of multilevel discordant twin models. We sought to address the limitations of previous twin studies by utilizing a large genetically informed sample, allowing separate analyses of discordant MZ and discordant DZ twins, and to examine the relationship between subtypes of CT in relation to BPD traits.

Method

Sample and Procedure

Data for the study was collected as part of the Oslo University Adolescent and Young Adult Twin Project (Torgersen & Waaktaar, 2019). All twin pairs born in Norway between 1988 and 1994 were invited to participate. The twins were identified through the Norwegian Medical Birth Registry. The present study utilized data from a face-to-face interview with the twins when they were around 19 years of age (M = 19.1, SD = 1.2, range = 17.5 to 23.3). Each twin in a pair was interviewed by different interviewers. The sample consisted of 2,808 twins (58% females), including 1,384 complete twin pairs and 40 incomplete twin pairs. Of the complete pairs, the zygosity distribution was 540 monozygotic (MZ) and 844 dizygotic (DZ) twin pairs. The study was approved by the Norwegian Data Inspectorate and the Regional Committees for Medical and Health Research Ethics. American Psychological Association ethical standards were followed in the conduct of the study. Since the study included entire national birth cohorts, no a priori power analysis was performed.

Zygosity Determination

The zygosity of same-sex twin pairs were partially determined through a 12-item zygosity scale where questions about similarity in appearance, how often the twins have been mixed-up with each other, and whether they believe that they are monozygotic or dizygotic were asked (Torgersen, 1979). To validate the zygosity scale, cheek swabbed DNA was drawn from a subsample of twin pairs. Seventeen genetic markers were tested, with an estimated probability of misclassification less than $p < .0001$. The scores on the zygosity scale were analyzed using discriminant analysis and a cutting point for the discriminant score was established based on the results of the gene testing. Five hundred thirteen of the 1,006 same-sex twin pairs were gene-tested. Those with a discriminant score close to the cutting point were oversampled for DNA tests. It appeared that 14 out of the 513 twin pairs were misclassified according to the discriminant analysis. Correcting for the oversampling, the questionnaire misclassified 2.13% of the same-sex twins. As almost all of the misclassified pairs were gene tested, results showed that .64% of the same-sex twin pairs are expected to be misclassified (.45% when including the whole twin sample).

Measures

Borderline Personality Disorder Traits

The Structured Interview for *DSM–IV* Personality (SIDP-IV; Pfohl et al., 1997) in Norwegian translation was used to assess BPD traits. SIDP-IV has been used in a number of studies in various countries, including Norway (Helgeland et al., 2005; Kendler et al., 2008), and in several studies of personality disorders in adolescence (e.g., Korsgaard et al., 2016; Speranza et al., 2011). The ratings are based on behavior typical for the past five years, and each criterion is scored on a scale ranging from 0 to 3 (0 = *absent*; 1 = *subthreshold*; 2 = *present*; 3 = *strongly present*).

The proportion of individuals meeting full *DSM–IV* criteria for BPD was too low (i.e., 1.3%) to perform reliable analyses with BPD as a categorical variable. To increase statistical power and utilize a larger amount of information, we analyzed the number of endorsed BPD criteria at either the clinical or subclinical level (i.e., score $\geq$1), resulting in possible scores between 0 and 9. Interrater reliability was assessed based on two raters' scoring of 55 audiotaped interviews, of which 53 of the recordings were of satisfactory quality to be scored. The intraclass correlation coefficient (ICC) for the number of endorsed BPD criteria scored $\geq$1 was .77 ($p < .001$).

Exposure to Trauma in Childhood and Adolescence

The Childhood Trauma Interview (CTI; Fink et al., 1995) in Norwegian translation was used to assess exposure to trauma. The CTI is a semistructured interview for the retrospective assessment of six areas of interpersonal trauma occurring during childhood and adolescence, including separations and losses, physical neglect, emotional abuse, physical abuse, sexual abuse and witnessing violence. The CTI has been shown to have high reliability and validity (Fink et al., 1995; Roy & Perry, 2004), and has been used in several studies examining different areas of mental health (Laporte et al., 2011; Simeon et al., 2001; Vrshek-Schallhorn et al., 2014).

The section on 'emotional abuse' focuses on experiences of being threatened, humiliated, criticized, shouted at, controlled, ignored or scapegoated. 'Physical abuse' is assessed through questions about experiences of being hit, kicked, thrown into the walls, looked inside a room/closet, choked, cut or burned. The section on 'sexual abuse' includes questions about both contact experiences and noncontact experiences (e.g., sexual threats or watching others engage in sexual activities). The section on 'witnessing violence' includes questions about both domestic and other violence. The section on 'separations and losses' was excluded from the analyses due to inconsistency in rating (based on notes from the interviews). Some interviewers rated 'death of grandparents' consistently as 'separation' while others rated such cases as separation only when the grandparents had been the interviewee's caregivers. In addition, interviewers differed in scoring with regard to 'separation from one of the parents' in case of divorce. Thus, this part of the interview was expected to have high measurement error and consequently low reliability and validity. The section on 'physical neglect' was also excluded from the analyses due to very low prevalence and consequently low statistical power (2.0% of the total sample reported physical neglect and 2.8% was discordant on exposure).

All types of CT included in the analyses (i.e., emotional abuse, physical abuse, sexual abuse, witnessing violence and any CT) were coded dichotomously (0 = *absent*, 1 = *present*).[1] Participants were classified as having experienced 'any CT' if they reported having experienced any of the four CT subtypes included in the analyses. In order to utilize all available information, all traumatic experiences occurring up to the time of the interview were included in the analyses.

Statistical Analyses

Phenotypic Associations

All analyses in the current study were conducted in the statistical package R (R Core Team, 2020). In our initial analyses we examined the phenotypic associations between CT and BPD traits using correlation and linear regression analyses. Biserial correlations were calculated when one variable was dichotomous and the other was continuous. Tetrachoric correlations were calculated when both variables were dichotomous. Biserial and tetrachoric correlations are less prone than Pearson correlations to bias the association between variables when one or both variables are dichotomous (Kirk, 1973; Olsson et al., 1982).

Univariate Twin Models

Before running the discordant twin analyses, univariate twin models were fitted to BPD traits and each CT type to examine the influence of genetic and environmental factors on these phenotypes. These models allow the variance of an observed measure to be partitioned into three separate sources. Additive genetic influences (A; genes that together operate in an additive manner, causing similarity among family members) are inferred when the MZ correlation is greater than the DZ correlation. Nonadditive genetic influences (D; dominance) are inferred when the DZ correlation is less than half of the MZ correlation, whereas shared environmental influences (C; any environmental factors that contribute to similarity among family members) are inferred when the DZ correlation is more than half of the MZ correlation. Of note, A, D and C influences cannot be estimated simultaneously because they are confounded in the classical twin design. Unless the correlation between DZ twins is less than half that of MZ twins, most researchers opt to including A and C parameters in the model. The remaining variance, not accounted for by A + C (or A + D or D + C), is attributed to nonshared environmental influences (E; any factors that contribute to phenotypic dissimilarity between family members, including measurement error).

Using the structural equation modeling R package OpenMx (Neale et al., 2016), univariate twin models were fitted to raw data using full information maximum likelihood. BPD traits were analyzed using standard univariate twin models for continuous data, and each CT was modeled using a threshold model, where a continuous normally

[1] If there is a dose-response relationship between CT and number of BPD symptoms, one should treat the CT types as continuous variables. Surprisingly, severity and duration of trauma showed lower correlations with BPD traits compared to a dichotomous coding of the trauma variables (i.e., on average, the correlations were about 2/3). This is probably due to a large amount of error variance in the scoring of severity and duration compared to a trauma/non-trauma scoring. We therefore chose to study the CT variables dichotomously.

distributed liability is assumed to underlie each of the observed binary variables. The models were fitted with separate means and thresholds for males and females to account for mean-level sex differences in BPD traits and CT. Competing models were compared using Akaike's information criterion (AIC), with lower values indicating better model fit (Akaike, 1987).

Discordant Twin Models

Discordant twin models were fitted to control for potential confounding by shared environmental and additive genetic influences that might contribute to the overall effect of CT on BPD traits in the total study sample. The strength of the association between CT and BPD traits in the total study sample (i.e., individual level) was compared to the effect observed within twin pairs discordant on CT (i.e., twin pairs in which only one of the twins has been exposed to trauma). This within-pair effect gives the expected difference in BPD traits within a twin pair when the twins are discordant on CT exposure. Twins reared together share common environment, MZ twins are genetically identical while DZ twins, on average, share half of their segregating genes. The MZ within-pair effect will therefore completely control for both shared environmental and genetic factors, while the DZ within-pair effect completely controls for shared environmental factors and partially for genetic factors.

Within this framework, a causal effect refers to a direct nonshared environmental effect of trauma on BPD traits (McGue et al., 2010). If CT has a *causal effect* on BPD traits, the effect of CT is expected to be the same at the individual level as within discordant DZ and discordant MZ pairs (i.e., controlling for shared environmental and genetic factors would not reduce the association). If the relationship between CT and BPD traits is noncausal and *completely confounded by shared environmental factors*, the effect of CT on BPD traits is expected to be absent within both discordant DZ and discordant MZ pairs. If the relationship is noncausal and *completely confounded by genetic factors*, the effect of CT on BPD traits is expected to be reduced within discordant DZ pairs relative to the effect at the individual level, while the effect within discordant MZ pairs will be completely absent. In the presence of *partial confounding* by shared environmental and/or genetic factors, the effect of CT on BPD traits within discordant DZ and discordant MZ pairs will be reduced relative to the effect at the individual level, but the effects will not be completely absent.

Linear mixed regression models were fitted using the R package lme4 (Bates et al., 2015). Each twin pair represents one cluster, and individual twins (level 1) are nested within twin pairs (level 2). The model included a random intercept across twin pairs, and the within-pair effect of CT was represented by subtracting the pair mean from each twin's own score (that is, $[x_{ij} - \bar{x}_i]$, where $\bar{x}_i$ represents the mean trauma score in twin pair i, and x_{ij} represents the trauma exposure index for individual j in twin pair i). Separate models were fitted to each CT measure, with BPD traits as the outcome variable in each model. First, we conducted separate analyses on MZ and DZ data. Finally, a model including the interaction of zygosity with the within-pair effect of CT in the pooled sample of twins was conducted to determine to what extent any confounding were due to shared environmental and/or genetic factors.

In order to control for potentially important covariates, we replicated the set of models described above accounting for other psychopathology (i.e., affective disorders, anxiety disorders, alcohol or substance abuse, eating disorders and posttraumatic stress disorder) by including these variables as fixed effects in the models. See Table S1 for correlations between other psychopathology and our study variables. In addition, we fitted the models to a subsample excluding the participants who reported trauma after age 12. These analyses ensured temporal precedence of CT (SIDP-IV uses a five-year retrospective period for BPD symptoms).

Cholesky Decomposition Models

To validate the results from the discordant twin models, we fitted a series of bivariate Cholesky decomposition models (Neale & Maes, 2004) using the structural equation modeling package OpenMx (Neale et al., 2016). A potential direct effect of CT on BPD traits should produce a nonshared environmental correlation between these phenotypes, while confounding of genetic and/or shared environmental factors should produce genetic and shared environmental correlations, respectively.

Results

Descriptive Statistics and Phenotypic Associations

Descriptive statistics and correlations between variables are presented in Table 1. All subtypes of CT showed small to moderate positive associations with BPD traits. When excluding the participants who reported trauma after age 12, the correlations between CT and BPD traits were almost identical as when utilizing information from the full sample (see Table S2). Furthermore, neither BPD traits nor CT was associated with age at interview.

Table 1
Descriptive Statistics and Correlations

Variable[a]	*M*	*SD*	1	2	3	4	5	6
1. Sex	0.58	0.49	—					
2. Emotional abuse	0.31	0.46	.04	—				
3. Physical abuse	0.15	0.36	−.13	.59	—			
4. Sexual abuse	0.05	0.21	.45	.40	.37	—		
5. Witnessing violence	0.12	0.33	−.10	.46	.58	.30	—	
6. Any childhood trauma	0.41	0.49	.02	.81	.80	.68	.76	—
7. BPD traits	1.08	1.60	.14	.37	.35	.44	.26	.35

Note. N = 2,808. Tetrachoric correlations are reported for correlations between dichotomous variables; biserial correlations are reported for correlations between dichotomous variables and BPD traits.
[a] Sex coded 0 = male, 1 = female; childhood trauma (2-6) coded 0 = trauma absent, 1 = trauma present; BPD traits = number of endorsed borderline personality disorder criteria either at the clinical or the subclinical level.

The mean age at first exposure was 8.9 (*SD* = 4.3) for emotional abuse, 10.4 (*SD* = 5.2) for physical abuse, 13.0 (*SD* = 3.7) for sexual abuse and 12.7 (*SD* = 4.8) for witnessing violence. Table 2 displays the number and percentage of discordant and concordant MZ and DZ twin pairs.

Results from linear regression analyses are shown in Table 3. Sexual abuse had the strongest specific association with BPD traits, followed by emotional abuse, physical abuse and witnessing violence. That is, when controlled for other subtypes of CT, each CT type still had a statistically significant, albeit reduced, association with BPD traits. Therefore, the unadjusted estimates were used as a basis for comparison in the discordant twin analyses.

Twin correlations and univariate model estimates from the best fitting twin models are given in Table 4. The twin correlations suggested moderate genetic influence on individual differences in all study variables, and modest influence of shared environmental factors in BPD traits and emotional abuse. According to AIC, the AE model was the best fitting model for all variables (see Table S3 for fit statistics and model estimates from the full ACE models). Both BPD traits and CT were moderately heritable, but somewhat lower additive genetic influence was observed for sexual abuse. Given the pattern of twin correlations for sexual abuse, we also fitted an ADE model for this trauma type. The model parameters were estimated to A = .00, D = .41 and E = .59. The ADE model's AIC (-4577.5) was almost identical as the AE model's AIC (-4577.3), indicating similar model fit. Of note, it is often hard to distinguish between A and D influences in studies of twins reared together (Posthuma & Boomsma, 2000).

Discordant Twin Models

Unstandardized regression coefficients for the effects of CT on BPD traits in the full and discordant twin samples are presented in Figure 1 as a set of bar charts. For all subtypes of CT, the difference between the MZ within-pair effect and the individual level effect were statistically significant (i.e., nonoverlapping 95% confidence intervals). More specifically, the MZ within-pair effect of *emotional*, *physical* and *sexual abuse* on BPD traits were reduced by 70–80% relative to the effect at the individual level, while the MZ within-pair effect of *witnessing violence* was completely absent. Furthermore, all MZ within-pair effects, except from the MZ within-pair effect of emotional abuse (p = .031), failed to reach statistical significance. These results indicate that shared environmental and/or genetic factors are confounding the association between the measured CT subtypes and BPD traits.

A comparison of effects within discordant DZ and discordant MZ pairs help determine to what extent the confounding is due to shared environmental and/or genetic factors. In the models testing the within-pair effects of *emotional abuse*, *sexual abuse* and *any CT,* the interaction effect of zygosity was statistically significant (i.e., the difference between the MZ within-pair effect and the DZ within-pair effect was statistically significant), indicating confounding of genetic factors in the association between these particular types of CT and BPD traits. The same pattern was observed for *physical abuse* and *witnessing violence.* However, while the DZ within-pair effect of *physical abuse* was nearly twice as large in magnitude compared to the MZ within-pair effect, the interaction effect of zygosity did not reach statistical significance (p = .191). For *witnessing violence,* neither the MZ within-pair effect nor the DZ within-pair effect were statistically significant. Thus, although the patterns resemble genetic confounding, confounding by shared environmental factors cannot be ruled out for these trauma types.

When including potentially important covariates (i.e., affective disorders, anxiety disorders, alcohol or substance abuse, eating disorders and posttraumatic stress disorder) in the models, the pattern of estimated regression coefficients were similar as the patterns shown in Figure 1. In addition, the MZ within-pair effects were even lower, which supports the results described above that CT does not seem to have a direct effect on BPD traits. To ensure temporal precedence of CT, we also fitted the models to a subsample excluding the participants who reported trauma after age 12. Results from these analyses also showed similar patterns as displayed in Figure 1. Consequently, the interpretation of results was unchanged by these additional follow-up analyses (see Figure S1 and Figure S2 for estimated regression coefficients).

Finally, we examined the associations between CT and four symptom phenotypes of BPD (Gunderson et al., 2018) as they may show differential associations with CT. Polychoric correlation analyses showed that the strongest association was observed between any CT and the behavioral dysregulation phenotype (r = .38), followed by the affective and/or emotional dysregulation

Table 2
Number and Percentage of Twin Pairs Discordant and Concordant on Childhood Trauma

	Concordant twin pairs with trauma		Concordant twin pairs without trauma		Discordant twin pairs	
Variable	*n*	(%)	*n*	(%)	*n*	(%)
MZ twin pairs (*n* = 540)						
Emotional abuse	102	(18.9)	298	(55.2)	140	(25.9)
Physical abuse	47	(8.7)	409	(75.7)	84	(15.6)
Sexual abuse	6	(1.1)	496	(91.9)	38	(7.0)
Witnessing violence	28	(5.2)	421	(78.0)	91	(16.9)
Any childhood trauma	144	(26.7)	238	(44.1)	158	(29.3)
DZ twin pairs (*n* = 844)						
Emotional abuse	121	(14.3)	442	(52.4)	281	(33.3)
Physical abuse	36	(4.3)	638	(75.6)	170	(20.1)
Sexual abuse	1	(0.1)	765	(90.6)	78	(9.2)
Witnessing violence	18	(2.1)	673	(79.7)	153	(18.1)
Any childhood trauma	179	(21.2)	340	(40.3)	325	(38.5)

Note. MZ = monozygotic; DZ = dizygotic.

Table 3
Regression Models of the Effects of Childhood Trauma on Borderline Personality Disorder Traits

	Unadjusted coefficients		Adjusted for other trauma[b]	
Effect[a]	Unstandardized	Y-standardized[c]	Unstandardized	Y-standardized[c]
Emotional abuse	0.96 [0.84, 1.09]	0.60 [0.53, 0.68]	0.70 [0.57, 0.83]	0.44 [0.36, 0.52]
Physical abuse	1.02 [0.86, 1.18]	0.64 [0.54, 0.74]	0.53 [0.36, 0.71]	0.33 [0.23, 0.44]
Sexual abuse	1.55 [1.27, 1.82]	0.97 [0.80, 1.14]	1.14 [0.88, 1.41]	0.71 [0.55, 0.88]
Witnessing violence	0.78 [0.61, 0.96]	0.49 [0.38, 0.60]	0.29 [0.11, 0.47]	0.18 [0.07, 0.29]
Any childhood trauma	0.90 [0.78, 1.01]	0.56 [0.49, 0.63]		

Note. 95% CI in brackets.
[a] 0 = trauma absent, 1 = trauma present. [b] Each trauma type is adjusted for the other trauma types. [c] The borderline personality disorder outcome was standardized.

phenotype (r = .35), the cognitive and/or self-disturbance phenotype (r = .34) and the interpersonal instability phenotype (r = .27). Overall, results from discordant twin analyses indicated genetic confounding in the relationship between CT and symptom phenotypes of BPD (see Figure S3).

Cholesky Decomposition Models

Table 5 presents genetic and environmental correlations between CT and BPD traits derived from the Cholesky decomposition models. Notably, all shared environmental influences were dropped from the models because this resulted in improvements in model fit, as indicated by the lowest AIC values. There were substantial genetic correlations between CT and BPD traits, while the nonshared environmental correlations were negligible. These results are consistent with the results from the discordant twin models, suggesting confounding by genetic factors in the association between CT and BPD traits.

Discussion

The aim of the study was to examine whether there are causal effects of CT on BPD traits in early adulthood. Although CT is generally thought of as a purely environmental measure, genetically informative studies have found genetic influence on measured environments, such as life events (Kandler et al., 2012; Kendler & Baker, 2007). Results in the present study corroborate prior work mentioned above, finding genetic influence of measured CT (h^2 = .33-.69). BPD traits were also found to be moderately heritable (h^2 = .50). This estimate is in line with results from previous studies that have measured BPD traits at one measurement occasion (Bornovalova et al., 2013; Distel et al., 2008; Kendler et al., 2008; Torgersen et al., 2008).

As to the relationship between CT and BPD traits, we first examined the phenotypic associations in the total study sample. Consistent with previous work (Afifi et al., 2011; Battle et al., 2004; Charak et al., 2018; Lobbestael et al., 2010; Yen et al., 2002; Zanarini et al., 2020), we found associations between CT and BPD traits. Sexual abuse had the strongest independent association with BPD traits, followed by emotional abuse, physical abuse and witnessing violence. However, which subtypes of maltreatment are most prominently and independently associated with BPD differ between studies. Possible reasons for varying research findings may be differences in samples (e.g., clinical vs. nonclinical), definition and classification of abuse/neglect, covariates included and measurement method.

Next, discordant twin models were fitted in order to control for potential confounding of shared environmental and genetic factors in the association between CT and BPD traits. All patterns in Figure 1 resemble a pattern of genetic confounding, indicating that the increased BPD traits associated with CT in the total study sample are not due to a direct effect of CT, but can rather be explained by shared genetic influence. Specifically, in the model testing the within-pair effects of *emotional abuse*, the MZ within-pair effect was statistically significant but modest in magnitude. The DZ within-pair effect was approximately twice as large as the MZ within-pair effect, and this difference in within-pair effects was statistically significant. These results indicate that there might be a small causal effect of emotional abuse on BPD traits, but that a substantial proportion of the observed relationship in the total study sample stems from shared genetic influences. The same pattern was observed for *physical abuse*. In this model, the MZ within-pair effect was statistically nonsignificant, indicating that in discordant

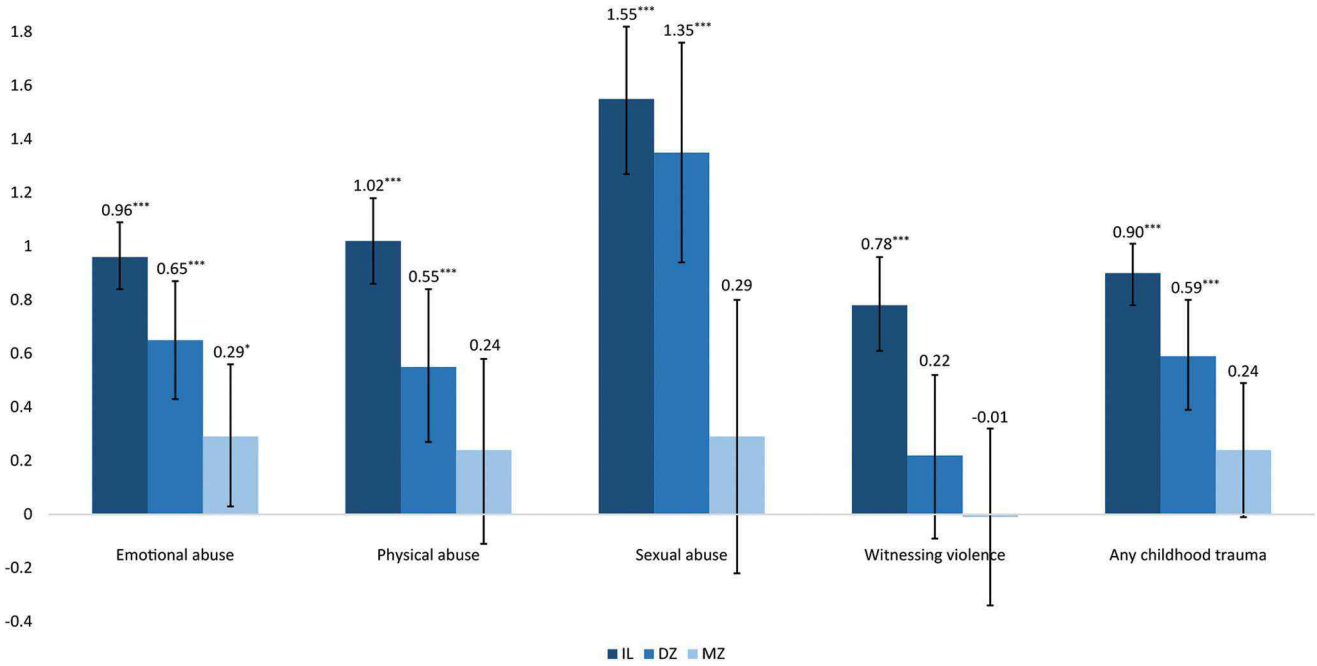
Table 4
Twin Correlations and Univariate Model Estimates From the Best Fitting Twin Models

	Twin correlations[a]		Univariate model estimates	
Measure	MZ pairs	DZ pairs	A	E
BPD traits	.49 [.42, .55]	.29 [.23, .35]	.50 [.44, .55]	.50 [.45, .56]
Emotional abuse	.61 [.53, .71]	.36 [.25, .45]	.63 [.53, .71]	.37 [.29, .47]
Physical abuse	.69 [.57, .77]	.36 [.23, .47]	.69 [.58, .78]	.31 [.22, .42]
Sexual abuse	.52 [.21, .68]	−.09 [−.24, .24]	.33 [.05, .58]	.67 [.42, .95]
Witnessing violence	.52 [.33, .67]	.21 [.02, .34]	.50 [.35, .63]	.50 [.37, .65]
Any childhood trauma	.59 [.48, .68]	.31 [.23, .39]	.59 [.50, .68]	.41 [.32, .50]

Note. 95% CI in brackets. BPD traits = borderline personality disorder traits; A = additive genes; E = nonshared environment; MZ = monozygotic; DZ = dizygotic.
[a] Pearson correlations are presented for BPD traits, tetrachoric correlations are reported for the remaining five childhood trauma variables.

Figure 1

Average Change in BPD Traits Between CT Exposed and Nonexposed Individuals, With and Without Considering Twin-Pair Membership



Note. Unstandardized regression coefficients B are presented above the bars. CT = childhood trauma; BPD traits = borderline personality disorder traits; IL = individual level (i.e., the average change in BPD traits due to a one-unit change in CT without considering twin-pair membership. Because CT is coded dichotomously, the regression coefficient is interpreted as the average change in BPD traits between CT exposed and nonexposed individuals); MZ = difference in BPD traits within discordant monozygotic twin pairs; DZ = difference in BPD traits within discordant dizygotic twin pairs. The difference between the MZ within-pair effect and the DZ within-pair effect was statistically significant for emotional abuse, sexual abuse and any childhood abuse, i.e., $p = .049$, $p = .003$ and $p = .042$, respectively. * $p < .05$. *** $p < .001$. See the online article for the color version of this figure.

MZ pairs, physical abuse exposed and nonexposed twins did not differ in BPD traits. The DZ within-pair effect, on the other hand, was statistically significant and about twice as large as the MZ within-pair effect. Although this pattern resembles genetic confounding, the observed difference in effects between discordant MZ and discordant DZ twins did not reach statistical significance. Thus, confounding by shared environmental factors cannot be ruled out. However, in the univariate twin models we found no evidence of shared environmental influences accounting for the variance in BPD traits and CT, suggesting that shared environmental factors are unlikely to confound the association between them.

Table 5

Genetic and Environmental Correlations Between Childhood Trauma and Borderline Personality Disorder Traits

Trauma type	Genetic correlation [95% CI]	Nonshared environmental correlation [95% CI]
Emotional abuse	.52 [.42, .62]	.12 [.01, .22]
Physical abuse	.51 [.40, .62]	.07 [−.06, .20]
Sexual abuse	.48 [.26, .85]	.19 [.02, .35]
Witnessing violence	.52 [.37, .65]	−.03 [−.16, .10]
Any childhood trauma	.53 [.43, .63]	.11 [.00, .22]

For the model testing the within-pair effects of *sexual abuse*, the MZ within-pair effect was modest in magnitude and statistically nonsignificant, while the DZ within-pair effect was statistically significant and much greater in magnitude. These results indicate genetic confounding of the relationship between sexual abuse and BPD traits. Although a visual inspection of the estimated regression coefficients for the model testing the within-pair effects of *witnessing violence* resemble genetic confounding, both within-pair effects were small in magnitude and statistically nonsignificant. Of all trauma types, witnessing violence showed the weakest correlation with BPD traits. This may explain the lack of a statistically significant difference between the MZ and DZ within-pair effect. In addition, the effect of witnessing violence in itself may be of less interest, because a large amount of the phenotypic association between witnessing violence and BPD traits could be attributed to the effect of the other trauma types (i.e., when controlled for other trauma types, the effect of witnessing violence dropped considerably). The model testing the within-pair effects of *any CT* makes most sense if the pattern of relationship between all subtypes of CT and BPD traits are pointing in the same direction. As expected from the results above, the analyses indicate genetic confounding of the association between any CT and BPD traits. Results from bivariate twin analyses provided further support for the conclusions derived from the discordant twin analyses, showing substantial genetic

correlations between all types of CT and BPD traits, while the nonshared environmental correlations were negligible.

Genetic confounding can be explained by evocative or active gene-environment correlation (Plomin et al., 1977). From this perspective of behavior genetics, a child inherits the genes and may develop BPD, regardless of whether the child has ever been exposed to CT. In case of an evocative gene-environment correlation, the child's genetically influenced characteristics elicit specific reactions from others. The child's genetic predisposition to, for example, impulsivity or oppositional behavior may elicit reactions from others that increases the likelihood of exposure to adverse life events such as physical aggression from their parents. In case of an active gene-environment correlation, the child actively creates or selects environments that are related to his or her genetic predisposition.

In line with previous studies finding that children exposed to trauma often experience multiple types of trauma (Charak et al., 2018; Herrenkohl & Herrenkohl, 2009; Zanarini et al., 2002), our results showed that the trauma types were interrelated. When studying single types of CT and not controlling for cooccurring subtypes, one may overestimate the negative influence of a particular type of trauma. However, when no causal effects were found for any type of trauma, a control for the cooccurrence of other trauma types should not be necessary. Yet, in addition to our main analyses, we fitted discordant twin models controlled for the effects of cooccurring CT. This did not change the pattern of estimated regression coefficients, nor consequently the conclusions regarding the relationship between trauma exposure and BPD traits.

Our results indicate that CT does not seem to play a causal role in the development of BPD traits. These results confirm findings from the very few twin studies published to date with equivalent CT, personality disorders and methodology (Berenz et al., 2013; Bornovalova et al., 2013). Noteworthy, the results in this study contradict much of the existing research literature regarding the etiology of BPD studied without control for shared environmental and genetic factors. For instance, a systematic review of studies examining the relationship between CT and BPD concluded that CT is an important etiological factor in the development of BPD (Ball & Links, 2009). However, the studies reviewed in that paper were pure correlational studies and did not control for the effects of shared environmental and genetic factors. Importantly, our findings do not justify the infliction of traumatic events, nor that CT does not cause pain and harm in young people's lives. Irrespective of any potential long-term consequences, it should be the ethical imperative for any responsible society to ensure that children's quality of life is good and that they are protected from painful childhood experiences.

Limitations and Strengths

Potential limitations to results should be mentioned. First, the assessment of CT was based on retrospective reporting. Although the validity of the assessment was likely strengthened by the use of a semistructured interview that based the scores on concrete behavior, there may still be bias in the reporting due to memory limitations or deliberate overreporting/underreporting. The risk for potential memory limitations might have been lower if the time delay between CT exposure and reporting were reduced. It is also a question to what degree the measure of CT reflects the perception of recalled memories (e.g., it might be that individuals with BPD symptoms are more likely to recall an experience as abusive/traumatic). However, studies have found that recall bias only explains a small portion of the variance in retrospective reporting of childhood maltreatment (Fergusson et al., 2011) and that retrospective reporting of abuse is highly consistent over time in personality disorder samples (Spinhoven et al., 2012). Of course, a more comprehensive trauma interview would be preferable (e.g., combining interview and questionnaire data or using multiple informants), but this is difficult in large surveys aiming to maximize the number of scales. Second, the lower correlations between BPD traits and continuously scored CT variables compared to a trauma/nontrauma scoring may be due to error variance in the scoring of severity and duration. It could be assumed that dichotomously scored CT variables would be correspondingly unreliable. Supporting its reliability/validity, the dichotomous CT variables showed clear associations with BPD traits, although there is a possibility that the correlations are artificially high. Third, because few participants met the criteria for a BPD diagnosis, we used a dimensional measure of BPD traits that included both clinical and subclinical scores. Even if studies have shown that different symptom levels reflect degrees of severity on a single continuum (Edens et al., 2008; Torgersen et al., 2008), other results may emerge in samples including more individuals with the full range of BPD symptoms. Fourth, although a discordant twin design controls for all variables shared by the twins (i.e., shared environmental and genetic factors), it does not eliminate confounding by unmeasured nonshared environmental variables, that is, environmental factors unique to the individual that influences exposure to CT as well as BPD traits (Ohlsson & Kendler, 2020). However, this limitation will probably only be a problem if we actually had found a direct effect of CT on BPD traits. Fifth, measurement error in the exposure variable will attenuate the within-pair associations to a greater extent than the individual-level associations (Ashenfelter & Krueger, 1994; Griliches, 1979). Consequently, the discordant twin design may fail to find a within-pair effect even when there is in fact a causal effect of exposure on outcome.

Contributing to the present knowledge within the field, this study has several strengths. The discordant twin design eliminates confounding by genetic and shared environmental factors, which makes it a powerful method for examining questions about causality (McGue et al., 2010; Ohlsson & Kendler, 2020). The large sample size provided statistical power to analyze discordant MZ and discordant DZ twins separately, and subtypes of CT in relation to BPD traits. Furthermore, the use of a full cohort, population-based sample strengthens the possibility of generalization of findings. The validity of the conclusions drawn from twin studies depends on twins being representative of the general population. Indeed, twins are shown to be representative of the general population in studies of both mental and somatic health, and twins do not appear to be different from other people with respect to personality (Andrew et al., 2001; Johnson et al., 2002).

Future Research

It is important to cross-validate findings from the present study by replicating the findings in samples from other age groups and other countries. Bornovalova, et al. (2013) found similar results in a sample of young adults from Minnesota, suggesting that findings may generalize to other populations. However, it is possible that BPD symptoms might develop at a later age than assessed in the present study. Future studies should also examine other types of CT and different dimensions of the trauma experience (e.g.,

trauma inside vs. outside the family, age and severity). Importantly, future studies should use methods that are able to separate the effects of an exposure from shared genetic and environmental factors when looking for environmental factors that potentially contribute to the development of BPD.

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