Stressful Life Events and Borderline Personality Disorder Traits

An Elastic Net Analysis with a Subsequent Multivariate Co-Twin Control Regression Analysis

Vilde Sofie Arneberg
Vilde Fodstad

Submitted as a Cand.psychol Thesis at The Department of Psychology (30 points)

The Faculty of Social Sciences

UNIVERSITY OF OSLO

Spring 2021

Abstract

Authors: Vilde Sofie Arneberg and Vilde Fodstad

Title: Stressful Life Events and Borderline Personality Disorder Traits

Supervisor: Eivind Ystrøm

Background: Borderline personality disorder is thought to be a serious mental disorder with a series of challenges in everyday life, and is characterized by severe psychosocial impairment and a high mortality rate due to suicide (Lieb et al., 2004). Multiple studies have found a high prevalence of traumatic experiences in childhood among BPD patients, and BPD is the most frequently studied out of the PDs due to the association with early traumatic experiences. We addressed the following problems: One, by applying a regularization regression method for model sparsity, what types of stressful life events are associated with borderline personality disorder traits? Two, to what extent is the association between the combined stressful life events and BPD accounted for by familial genetic and environmental background factors?

Method: In total 2801 twins between the age of 19 and 36 participated, recruited from The Norwegian National Medical Birth Registry. The sample was obtained from the Norwegian Institute of Public Health (NIPH) Twin Panel. The data was first collected from 1992-1998 (Q1-Q2), and later from 1999 (wave 1) to 2011 (wave 2). In wave 1, each twin in a pair was interviewed by a different interviewer. The interview study in wave 1 consisted of two parts: a Norwegian computerized version of the Munich-Composite International Diagnostic Interview (M-CIDI) assessing ICD-10 lifetime diagnoses, and The Norwegian version of the Structured Interview for DSM-IV Personality (SIDP-IV) assessing the DSM-IV Axis II personality disorders. Further, a total of 2284 twins were re-interviewed by telephone between 2010 and 2011 in wave 2, approximately 10 years later. In this wave, the participants were also mailed a questionnaire that consisted of questions about normative personality, maladaptive personality, and substance abuse. In addition, the experience of stressful life events was assessed. An overview of prevalence of BPD traits and experienced SLEs was presented in a descriptive manner. Negative binomial Poisson regression was used to explore which SLEs could predict BPD traits. An Elastic net analysis was then performed to develop

the final logistic regression model for prediction. At last, discordance within twin pairs was examined and a co-twin control was performed to differentiate between environmental and genetic factors in our sample. The data in this study is based on already collected data material from the Axis I / Axis II study.

Conclusion: We found that in childhood, life-threatening experiences and an unpredictable and unsafe early life environment is associated with the development of BPD traits. In adulthood, life-threatening experiences, economic issues, and relationship conflicts seem to have the same association. Our results indicate that these experiences can predict 22% of the development of BPD traits. In the debate about how stressful life events are associated with the development of BPD traits, it does not appear to be a causal factor. Our results point to there being something in the shared family environment that causes both the stressful life events and the development of BPD traits. It is therefore of importance to limit the effect of, or prevent, familial environmental background factors causing both SLEs and BPD traits.

Acknowledgements

This graduate thesis was written as a part of the Graduate Program in Psychology at the University of Oslo.

The field of personality psychology has fascinated us since we enrolled in the Graduate Program, where personality disorders have been of special interest. We therefore consider ourselves fortunate to have met our supervisor Eivind Ystrøm during the course PSYC4500, where his enthusiasm and expertise on this particular field of psychology motivated us to explore this subject.

We would like to express our gratitude to Axis I / Axis II study and Ted Reichborn-Kjennerud for providing us with access to our data sample. A special thanks goes to our supervisor Eivind Ystrøm for his guidance during our work with this graduate thesis. He has motivated us, answered all our questions, suggested topics of examination, and been frequently available for us. For this, we would like to express our deepest appreciation.

We would like to thank Nils Henrik and Ola for being extremely supportive and patient during this time. Writing a graduate thesis during a pandemic has been challenging, but with our supportive network of friends and family we are now proud and relieved to say that this was possible.

Finally, we could not have done this without one another. We thank each other for all the hours with teamwork spent on Zoom with laughter and tears.

Table of contents

Tables

Figures

1.1 Maladaptive personality development

When a child is born into the world, a loving and secure attachment with one's caregiver makes it possible to portray the world as a safe place. When the child signalizes discomfort, fear and uncertainty, the secure caregiver makes sure to soothe the child and help to regulate its emotions. The child's early view of the world as a safe place with loving and predictable relationships is formed, as well as the phenomenon called *personality* starts to develop. *Personality* refers to the enduring patterns of cognition, behavior and emotion that helps us adjust to the environment to secure social survival (Heim & Westen, 2014). This definition defines the dynamic of the ongoing interaction between these patterns, but also the potential for flexibility and variation of responding.

Alas, this dynamic can also occur in a more rigid manner. If the same child is born into the world under different circumstances, where the caregiver or significant others don't respond to its needs, or the child faces other stressful experiences in childhood or adolescence, he/she might portray the world as an unsafe place with unpredictable relationships, fear and uncertainty. This perception of the world might blend into the child's personality development in a way that helps adjust to the unsafe environment and secure survival. The child's personality development is embossed with an unhealthy pattern of emotion, behavior, cognition, and interpersonal relationships, where the flexibility and variation of responding is limited. The personality development is viewed as maladaptive, or *disordered*, where the child, and later the adult, would potentially meet new situations with fear and skepticism, and further view new relationships as unsafe. This, in some cases, will lead to the development of a disordered personality.

A *personality disorde*r is a type of mental disorder in which you have a rigid and unhealthy pattern of thinking, functioning, and behaving (Torgersen, 2000). The APA Diagnostic and Statistical Manual of Mental Disorders (DSM) 5th edition presents ten clinically defined personality disorders with a common, yet different dysfunction within the areas of *cognition, behavior, emotion, and interpersonal relationships*. Common for these PDs is an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the individual's culture, and leads to clinically significant distress or impairment. According to Kendler (2008) (as cited in Bornovalova, Hicks, Iacono & McGue, 2009), it was found that a personality disorder is moderately inherited, and governed by specific aspects of the environment, and are hypothesized to be genetically influenced forms of psychopathology that have their onset in adolescence or early adulthood, and show a pattern of dysfunction throughout the lifespan.

1.1.1 Borderline Personality Disorder

One of these ten clinically defined personality disorders is *borderline personality disorder (BPD),* which is characterized by a pervasive pattern of instability in emotional, behavior, cognition, and interpersonal relationships (Leichsenring, Leibing, Kruse, New & Leweke, 2011). The clinical symptoms and signs normally include 1. *emotional;* with inappropriate, intense anger or difficulty controlling anger, a chronic feelings of emptiness and affective instability due to a marked reactivity of mood, 2. *behavioral;* with recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior, and impulsivity in at least two areas that are potentially self-damaging and do not include suicidal or self-mutilating behavior, 3. *cognition*; with transient, stress-related paranoid ideation or severe dissociative symptoms, and identity disturbance: markedly and persistently unstable self-image or sense of self, and 4. *interpersonal;* with frantic efforts to avoid real or imagined abandonment and has a pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation. This personality disorder is thought to be a serious mental disorder with a series of challenges in everyday life and is characterized by severe psychosocial impairment and a high mortality rate due to suicide (Lieb, Zanarini, Schmahl, Linehan & Bohus, 2004).

Table 1

Diagnostic Criteria for Borderline Personality Disorder (BPD). Defined by The American Psychiatric Association's (APA) Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; APA, 2000) (Sayrs & Whiteside, 2006)

Criteria	Description
Emotional	Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights)
	Chronic feelings of emptiness
	Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days)
Behavioral	Recurrent suicidal behavior, gestures, or threats, or

self-mutilating behavior

Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating) and do not include suicidal or self-mutilating behavior

Cognitive	Transient, stress-related paranoid ideation or severe dissociative symptoms
	Identity disturbance: markedly and persistently unstable self-image or sense of self
Interpersonal	Frantic efforts to avoid real or imagined abandonment that do not include suicidal or self-mutilating.
	A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation

1.2 The Association Between Borderline Personality Disorder and Stressful Life Events

Multiple studies have found a high prevalence of traumatic experiences in childhood among BPD patients, and some studies finding that childhood abuse is nearly a ubiquitous experience within this group of patients (Wolberg, 1952; Golier et al., 2003). BPD is the most frequently studied out of the PDs due to this association with early traumatic experiences. A psychological trauma is a response to an event that the person finds highly stressful (Leonard, 2020). Such stressful and traumatic events can be categorized as what is known to be called *stressful life events* (SLEs), and was defined by Holmes and Rahe (1967) (as cited in Dohrenwend, 1973), as *"any event that evoked or was associated with any coping or adapting behavior in the involved individual"*.

Johnson, Cohen, Brown, Smailes & Bernstein (1999) reported that documented childhood maltreatment was associated with increased risk of BPD symptoms. Further, Afifi and colleagues (2011) examined this association in a community sample aged 20 years and older. They found that childhood adversity was highly prevalent in people with PDs, and most consistently associated with BPD and three other types of PD. The findings support the link between childhood adversity and BPD. This association has been researched additionally, and in a case-control study by Wingenfeld and colleagues (2011), a majority of BPD patients

reported early sexual and physical abuse, and found further that early life stress was considered a risk factor in the development of this personality disorder.

Other researchers such as Liotti, Pasquini, Cirrincione & Italian Group for the Study of Dissociation (2000) also found predictive factors for the development of BPD to be the child's early traumatic experiences, as well as mourning process in the mother within the first two years of a child's life. Furthermore, Johnson and colleagues (2001) reported that children exposed to maternal verbal abuse had an increased risk of developing personality disorders.

Lobbestael, Arntz & Bernstein (2010) examined a sample of both patients and nonpatients, to investigate the relationship between five different types of childhood maltreatment (sexual abuse, physical abuse, emotional abuse, physical neglect and emotional neglect) and the 10 PDs in DSM-IV. They found sexual abuse, emotional abuse, and emotional neglect to be associated with BPD. According to these studies, SLEs experienced during childhood creates significant risk at developing BPD traits.

While multiple studies have found an association between SLEs in childhood and BPD traits, less research is conducted on SLEs in adulthood associated with BPD (Reichborn-Kjennerud et al. 2015). In 2012, McGowan, King, Frankenburg, Fitzmaurice & Zanarini did a 10-year follow-up, and found that BPD patients reported a declining rate of experiences of abuse in adulthood. It's important to note that findings from studies using a clinical sample of BPD patients may not necessarily be generalizable to nonpatients. A recent study by Conway, Boudreaux & Oltmanns (2018) examined the longitudinal effect of stress exposure on BPD in older adults. The participants were a community sample of adults between the ages of 55 and 64, and measures of BPD and stressors were assessed three times over 5 years. The study found that exposure to stressful life events had no notable effect on the subsequent severity of BPD. Taken together, these results indicate a transient effect of SLEs in adulthood on BPD traits.

1.2.1 Researching SLEs

Stressful life events often occur in childhood. Childhood, defined by McLeod and Almazan (2003) (as cited by Sawyer, Azzopardi, Wickremarathne & Patton, 2018), is the *"pre-adult years of life encompassing infancy through adolescence",* where The UN Convention on the Rights of the Child defines a child as an individual aged 0-18 years. Based on these definitions, stressful life events experienced during childhood would be defined as SLEs experienced between 0-18 years of age, and SLEs experienced in adulthood would be SLEs experienced after 18 years of age.

According to the presented definitions of childhood, the aforementioned importance of SLEs during childhood refers to life events experienced before the individual is 18 years old. Some studies have also found that especially SLEs such as sexual and physical abuse during childhood is associated with the development of BPD (Wingenfeld et al., 2011). Based on this information, the experience of physical and sexual abuse during ages 0-18 years might be strongly associated with the development of BPD traits.

Based on a study by Contractor, Weiss, Natesan Batley & Elhai (2020), different types of SLEs could be clustered together attributed to various reasons. For example, physical and sexual assault are perpetrated by another individual, while natural disasters are conceptualized as uncontrollable, hazardous, and threatening natural phenomena with profound impacts on society and functioning. Preliminary evidence has supported the clustering of SLEs across diverse measures, for example interpersonal vs. non-interpersonal SLEs and intentional vs. non-intentional SLEs. Further, Bae, Kim, Koh, Kim & Park (2008) found a six-factor solution for potential clustering of SLEs, including *physical assault, accident/injury, natural disaster, witnessing death, sexual abuse, criminal assault,* and *man-made disaster*.

According to Kira (2001), a comprehensive taxonomy of traumas, or SLEs, can give the clinician an organizing pattern of the clients' traumatic exposures. In order to compare our study with other studies in this particular field of science, a clustering of the different SLEs is of importance. This clustering would also be of importance regarding reaching out to clinicians treating clients with BPD symptoms. However, a clustering of SLEs might represent a "one size fits all" aggregation angle regarding prediction of BPD, which would not take into account that each outcome would differ from the next. It is therefore necessary to find a way to predict SLEs regarding BPD in a more optimal way that takes this into account. Further, an Open Science Collaboration (2015) put the question regarding reproducibility on the agenda and expresses concern about the rate and predictors of reproducibility. According to this article, a large portion of replications produced weaker evidence for the original findings. This, the authors say, belies the uncertainty of scientific evidence. Based on this article, it is necessary to explore new ways to predict outcomes within science other than the techniques present today, and to develop a method that could predict SLEs on BPD in a way that would secure reproducibility. This is, according to Maxwell, Lau & Howard (2015) known as the "replication crisis in psychology". When researchers have been interested in the associations between SLEs and BPD, they've usually been investigating one life event at a time. This presents a problem because when

investigating one life event, it may be a different life event that is of importance. Therefore, one should examine multiple life events simultaneously. However, such examination presents the challenge of multiple variables at the same time. An approach to this challenge is the use of a regularization regression method to investigate multiple life events, creating a selection of variables strongly correlated with BPD and regulating non-correlated variables to zero. To secure reproducibility one must have an approach that doesn't test single associations, but compare models representing the data-generating mechanisms in the populations.

1.3 The Nature of The Association Between SLEs and BPD

Several studies confirm the association between stressful life events and BPD (Afifi et al., 2011; Conway et al., 2018; Golier et al., 200; Johnson et al., 1999; Johnson et al., 2001; Liotti et al., 2000; Lobbestael et al., 2010; McGowan et al. 2012; Wingenfeld et al., 2011). In fact, stressful life events like childhood trauma are found to be the most significant environmental risk factor for BPD (Gunderson, Herpertz, Skodol, Torgersen & Zanarini, 2018). However, a proven association does not equal a direct effect of the stressful life events on the development of BPD traits. Finding an association between the exposure (SLEs) and outcome (BPD) is not the same as finding that SLEs cause BPD. An association might be better explained by a third variable or a *confounder*. A confounder is a variable that's a cause of the outcome (BPD) and correlated with, but not affected by, the exposure (SLEs). A second alternative to causal effect is reverse causation where the outcome (BPD) leads to the exposure (SLEs) (McGue, Osler & Christensen, 2010). According to Rutter (2007), although scientists are aware of this, the literature contains multiple studies with correlational evidence implying a causal effect.

It's been proposed that the relationship between SLEs and BPD meets some of the suggested criteria for causality (i.e. Bradford Hill's criteria of causality). In 2009, Ball and Links published a review of the literature on childhood trauma as a causative factor in BPD, and the results support a causal relationship, especially if childhood trauma is considered one variable in a multifactorial etiologic model. More recent studies find support for a dose-response relationship between childhood adversities and BPD. A *dose-response relationship* is present if when the independent variable (here: childhood adversity) increases in duration or degree, the outcome variable (here: BPD) increases as well. When analyzing the response from an epidemiology survey in Switzerland, Hengartner, Ajdacic-Gross, Rodgers, Müller & Rössler (2013) found a strong association between emotional abuse in childhood and BPD, as well as a dose-response relationship impact of childhood adversity on

BPD and the other nine DSM-IV PDs. Ibrahim, Cosgrave & Woolgar (2018) reviewed 10 studies on childhood adversity and the link to BPD features in children and found that the severity of childhood abuse increases the risk of developing borderline features in childhood. Although these findings are thought to support a causal relationship between SLEs and BPD, the association may be better explained by background factors that we are unable to assess to any great extent. These background factors refer to the *effective* environment which is defined by the outcomes they produce. The SLEs are *the measures of* the environment, or "objective" environment, which refers to environmental events as they might be observed by a researcher (Turkheimer & Waldron, 2000).

A causal relationship between SLEs and BPD would mean that the experience of SLEs would alone cause BPD (see Figure 1).

Figure 1

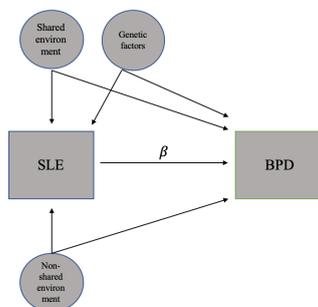*Causal Relationship Between SLEs and BPD*
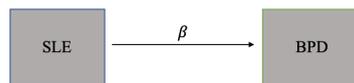
Three background factors, or confounders, might bias the direct effect (β): *genetic factors* (A), *shared environment* (C), and *non-shared environment* (E) (Figure 2). These however does not exclude causality but represents a threat to the validity of the β if not discovered (McGue et al., 2010).

Figure 2

Confounders That Bias the β

These factors can bias the effect alone or together, and according to a review by Carpenter, Tomko, Boomsma & Trull (2012) BPD seems to develop in the context of both biological vulnerabilities and environmental risk factors. Based on this review, impulsivity and emotional sensitivity might be possible key variables in the biological risk, which then interacts with the environment. This is known as the gene-environment correlation model, the *rGE* (Plomin, DeFries & Loehlin, 1977; Scarr & McCartney, 1983). Further, this review supports evidence for the rGE-model for borderline personality traits and a range of adverse life events, indicating that those at risk for BPD are also at increased risk for exposure to environments that may trigger BPD.

This model suggests that genes and environment can be correlated in such a way that genes can influence environmental exposure, and in the review by Carpenter et al. (2012) they found significant rGE for most of the stressful life events, suggesting that genes that are responsible for BPD risk increase risk of exposure to stressful life events. The rGE challenges the causality between SLEs and BPD within the *genetic factor* (A). In this scenario the SLEs serve as a marker of genetic risk for BPD in the family. Further, *shared environment* (C) can be a confounder that affects both the exposure and the outcome. It may be an environment within a family that causes a stressful life event to occur *and* increases the risk of developing BPD traits. Thus, it is not the SLE itself, but the family environment causing the SLEs that also causes BPD traits. In this scenario the SLEs serve as a marker of environmental risk for BPD in the family. As opposed to genetic sequences, if the association is biased by shared environment it is in principle possible to intervene on whatever constitutes the detrimental family environment.

As well as studying both SLE and confounding factors, it is important to study other factors for BPD. These are indexed by additive genetic, shared environmental and non-shared environmental residual factors (see Figure 3).

Figure 3

Complete Model of SLEs on BPD with Possible Confounders and Residual Factors
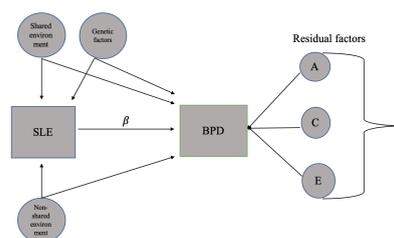
A causal relationship between SLEs and BPD would mean that there is a direct link between the two, and that the β is not biased by one or several confounding variables. If, however, the relationship between SLEs and BPD is caused by a confounding variable, it would mean that this explains both SLEs and BPD. How much remains of shared environmental factors (C) points to how much remains unresolved after accounting for the measures of the environment (SLEs). How much of the total genetic risk (A) in BPD can be explained by SLEs informs us about how much of the genetic risk for BPD can be accounted for by G-E-correlation. Confounding variables would not exclude causality all together, but rather bias our estimates. This is important to clarify when researching the causality between SLEs and BPD.

1.3.1 A Discordant-Twin Design

As in randomized experiments, a proper examination of the mechanisms underlying the relationship between stressful life events and BPD traits requires the participants in a study to differ only in exposure to the life events. Causational reasoning requires a comparison of what happens when an individual experiences the potential risk factor with what would happen if the same individual does not experience it while everything else is equal – this is known as the counterfactual condition (Rutter, 2007). In theory, such a condition eliminates the possibility of confounding variables. A twin design can to some extent mimic such a condition. Twin studies make use of the two existing types of twin pairs, monozygotic (MZ) and dizygotic (DZ). MZ twins share 100% of their genes and DZ share 50% of their genes. Because twins are born into the same family both MZ and DZ twin pairs have the same shared environment (environmental factors that cause twins to become more similar to each other). Specific to each twin is the non-shared environment consisting of environmental factors that causes twins to become less similar to each other. In a classic twin design one can estimate the heritability of the phenotypic trait (e.g. BPD) by comparing the phenotypic similarity of MZ and DZ twins (McGue et al., 2010).

A discordant-twin design can help solve two out of the three aforementioned problems of confounding. The design compares twins who differ in exposure to the risk factor, so that the unexposed twin serves as a control for his/her exposed co-twin (within-pair comparison). A within-pair comparison of discordant DZ pairs allows for complete control of the shared environment, and partial control of genetic factors. A within-pair comparison of discordant monozygotic (MZ) twins makes it possible to control for shared environment and genetic factors and is often used to help investigate causal influence between exposure and outcome

(Slutske et al., 2014). Three possible patterns can be found in a discordant-twin design: no confounding, complete confounding (by shared environment or genetic factors) and partial confounding (McGue et al. 2010). Figure 4 illustrates the three patterns.

Figure 4

Possible Patterns in A Discordant-Twin Design
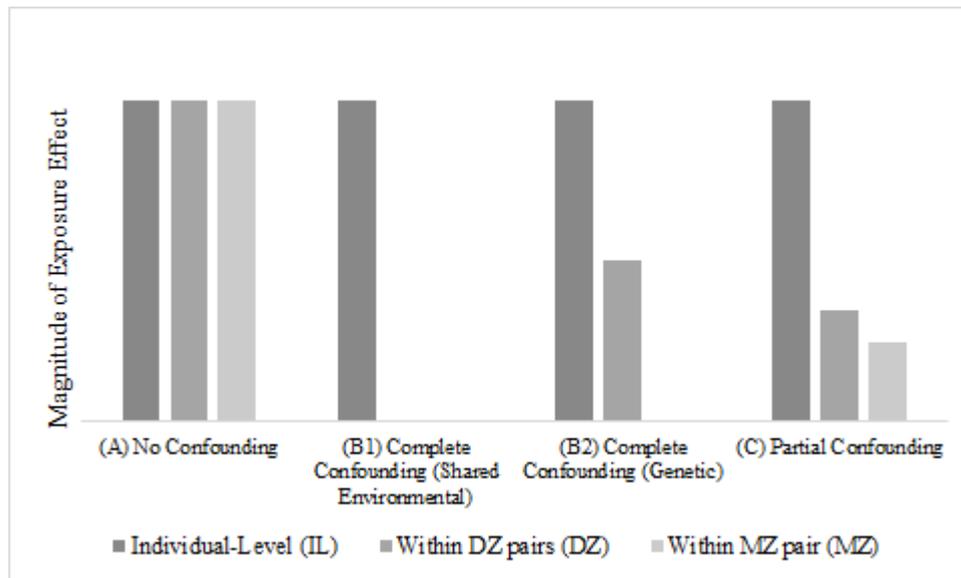
One possible pattern is *no confounding*. If exposure to stressful life events has a causal effect on BPD traits and there is no confounding by shared environment or genes, pattern (A) is expected to be found. The effect of exposure is the same at individual level, within discordant DZ pairs and within discordant MZ pairs. The effect is not reduced when controlling for shared environment and genes. A second pattern is *complete confounding*. Pattern (B1) is expected if the association between SLEs and BPD traits is completely confounded by shared environment. Both DZ and MZ pairs have the same shared environment, and the effect of SLEs on BPD traits is therefore expected to be completely absent in discordant DZ and MZ pairs. Pattern (B2) is expected if the association between SLEs and BPD traits is completely confounded by genetic factors. The effect is reduced in discordant DZ pairs (sharing 50% of genes), and completely absent in discordant MZ pairs (sharing 100% of genes). The third pattern is *partial confounding*. Pattern (C) is expected to be found if the association between SLEs and BPD traits is partially confounded by shared environment and/or genetic factors. The exposure effect is reduced but not completely absent within both discordant DZ and MZ pairs (McGue et al., 2010).

A few twin studies have examined the association between stressful life events and

BPD traits, and the support for a direct effect between the two. Berenz and colleagues (2013) examined whether childhood trauma has a causal effect on PD traits or whether the association can be better explained by common familial factors. Using a discordant-twin design, data from 2780 twins from the Norwegian Twin Registry were analyzed. The results showed that childhood trauma was significantly associated with BPD traits in the general sample, and the association remained significant after accounting for common familial factors in the discordant twin sample. However, the magnitude of the effects was half of that observed in the general sample. This indicates that a considerable portion of the association is better accounted for by familial factors than by a causal effect of childhood trauma. Due to low statistical power the MZ and DZ pairs could not be examined separately, and the results cannot point to whether genetic factors or shared environment were responsible for observed indirect, or mediated, effects (Berenz et al., 2013).

Bornovalova and colleagues (2013) examined the mechanisms underlying the relationship between childhood abuse and BPD traits in adulthood. The sample consisted of 1382 twin pairs participating in the Minnesota Twin Family Study, and MZ and DZ pairs were separated for the discordant-twin design. Their results indicated little to no evidence for a causal effect, and they found the association between childhood abuse and BPD traits can be better explained by common genetic influences (Bornovalova et al., 2013).

Research on the effect of SLEs experience in adulthood on BPD traits using a discordant-twin design, is a limited field. Gunderson (2011) presents that BPD is usually diagnosed in young adulthood, but signs of it often become evident in adolescence. Shiner (2009) further presents research that implies that personality pathology does exist prior to adulthood and predicts adult functioning. According to this study it has become increasingly clear that personality pathology does occur in youths, and the pathways leading to adult PD sometimes begin in childhood. There is especially convincing evidence for the childhood and adolescent manifestations of personality disorders, particularly in youths. Based on this information it is thought that SLEs experienced in adulthood are of less importance in the development of BPD compared to SLEs experienced in childhood.

1.4 Problems to be Addressed

Borderline personality disorder is thought to be a serious mental disorder with a series of challenges in everyday life and is characterized by severe psychosocial impairment and a high mortality rate due to suicide (Lieb et al., 2004). Therefore, it is important to help explain and further investigate if stressful life events are causally related to the development of

borderline personality disorders, and further clarify to what degree genetics and environmental factors play a role. The results can be of great use for public health-related work, specifically mental health work preventing violence, traumatic stress, and other life stressors, and it certainly can be relevant for therapeutic work with people with personality disorders.

Using a longitudinal twin study, we will address the following problems: *One, by applying a regularization regression method for model sparsity, what types of stressful life events are associated with borderline personality disorder traits? Two, to what extent is the association between the combined stressful life events and BPD accounted for by familial genetic and environmental background factors?*

2. Method

2.1 Sample

Our sample is based on already collected data material from the Axis I / Axis II study, that was obtained from the Norwegian Institute of Public Health (NIPH) Twin Panel (Nilsen et al., 2013). The participants were recruited from the Norwegian National Medical Birth Registry, established in 1967. The Registry receives notification of all live births in Norway (Irgens, 2000).

The first wave of data collection on this sample began in 1992, when twins born between 1967 and 1974 were contacted to complete a mail-out questionnaire (Q1). 5864 twins responded to Q1. In 1998 the same sample was contacted again to complete a second questionnaire (Q2). At this time the sample also included twins born in 1975 through 1979. 8045 twins responded to Q2. When asked if they could be contacted again for new studies after the second questionnaire, 6442 twins agreed (Kendler et al., 2017).

2.1.1 Wave 1

Wave 1 includes data from Q2, and interview data collected between 1999 and 2004. For the interview study initiated in 1999, approximately 44% of the twins participated. This included 68 twin pairs which, due to technical problems, had not completed the Q2, but were still recruited. The interviews took place between 1999 and 2004 and were mostly conducted face-to-face. In total 2801 twins between the age of 19 and 36 were interviewed (Tambs et al., 2009). Each twin in a pair was interviewed by a different interviewer, with the majority of interviewers being senior clinical psychology graduate students at the end of their six-year

training course. The other interviewers were experienced psychiatric nurses and two medical students (Berenz et al., 2013).

Zygosity was originally obtained with a combination of questionnaire data, blood samples and microsatellite markers, and in the interview sample it is mainly based on DNA data with an accuracy of 99.1% (Tambs et al., 2009). The sample consists of 1347 monozygotic (MZ) twins and 1454 dizygotic (DZ) twins. All participants gave their written informed consent to participate in the study. Approval was sought and received from the Regional Committee for Medical and Health Research Ethics (#2010/767) (Kendler et al., 2008).

The interview study consisted of two parts: a Norwegian computerized version of the Munich-Composite International Diagnostic Interview (M-CIDI) assessing ICD-10 lifetime diagnoses, and The Norwegian version of the Structured Interview for DSM-IV Personality (SIDP-IV) assessing the DSM-IV Axis II personality disorders. It is the second part to which this thesis will pay its attention. The SIDP-IV interview was conducted after the M-CIDI interview in order to distinguish long-term behavior from temporary states following Axis I disorders (Kendler et al., 2008). The SIDP-IV is a semi-structured interview assessing all DSM-IV Axis II personality disorders, with the criteria scored on a 4-point scale from 0 to 3 (in our analyses the following 4-point scale was used: *not present* (0), *sub-threshold* (0.5), *present* (1) and *strongly present* (1.5)) (Pfohl, Blum & Zimmerman, 1997; Tambs et al., 2009). The interview assesses behaviors, cognitions and feelings that predominated for most of the past 5 years, as it considers it to be representative for long-term personality (Kendler et al., 2008). Questions are organized by themes such as *interests*, *activities,* and *interpersonal relationships* in order to reduce the impression of the interview being designed to assess personality pathology (Jane, Pagan, Turkheimer, Fiedler & Oltmanns, 2006).

2.1.2 Wave 2

The second wave of data on this sample was drawn from a questionnaire completed by participants in 2011, also known as *Wave 2*. A total of 2284 twins were re-interviewed by telephone between 2010 and 2011, approximately 10 years later. A total of 2299 twins received a questionnaire by mail, and written informed consent was obtained from all participants. The questionnaire consisted of questions about normative personality, maladaptive personality, and substance abuse. In addition, the experience of stressful life events was assessed. SLEs were measured by presenting 18 different possible traumatic events as described in the article by Rosenström et al. (2019), and the participants were asked

to report their age at the time the SLEs occurred if they reported *"yes"*. Examples of the questions presented in the SLEs section of the questionnaire are as following: *"Have you ever been threatened with a weapon, held captured or been kidnapped?"*, *"Have you ever experienced the sudden or early death of someone close to you?"*, or *"Were you physically abused as a child?"* (Rosenström et al., 2019). A complete overview of the 18 assessed SLEs is presented in Table 3.

2.2 Statistical Analyses

The statistical analyses were carried out in Stata MP 16. An overview of prevalence is presented in a descriptive manner. Negative binomial regression for overdispersed Poisson distributions was used to explore which SLEs could predict borderline personality disorder traits. An Elastic net analysis was then performed to develop the final logistic regression model for prediction, and a default set-up for Elastic net in Stata MP 16 was followed. At last, discordance within twin pairs was examined, and a co-twin control was further performed to differentiate between environmental and genetic factors in our sample.

2.2.1 Descriptive Statistics

Prevalence is a measure of the frequency of a phenomenon in a population at a particular point in time. To estimate this, the number of participants with the BPD symptoms and/or experienced SLEs, are divided by the total number of participants in the sample. In our study, we investigate the prevalence of experienced BPD symptoms, and experienced SLEs in childhood (up to 18 years) and adulthood (after 18 years). To present an overview of experienced BPD symptoms, and experienced SLEs, three prevalence tables are being presented below (see Table 4, 5 and 10). The prevalence is reported in percentage.

2.2.2 Poisson Regression Analyses

A negative binomial Poisson regression analysis is a type of generalized linear model in which the dependent variable is a count of the number of times an event occurs, in this case number of BPD symptoms. The negative binomial model is chosen when one assumes that the events (i.e. criteria) are dependent, which leads to an overdispersion where the variance is larger than the mean. To assess the types of stressful life events that are associated with borderline personality disorder traits, a negative binomial Poisson regression analysis was performed, with BPD traits as a dependent variable and the documented SLEs in childhood and adulthood as independent variables. Standard errors were corrected for dependence in the

data due to twin pairing using a sandwich estimator. This analysis was performed to explore relative risk of having BPD traits if experiencing each SLE controlled for each other, as well as relative risk at having BPD traits if experiencing any SLE in childhood or adulthood. The sample was divided into two variables, where one is a clustered variable with all SLEs experienced during childhood and the other during adulthood. Five clusters of SLEs were also investigated using this method.

2.2.3 Elastic Net Regression

Elastic net is a linear model-based methodology that shrinks regression coefficients toward zero, and automatically selects predictors with nonzero coefficients. When the number of predictors is large compared to the sample size, traditional variable selection methodologies may have poor prediction performance for external datasets by overfitting random error or noise, and it has been criticized that the goodness of fit, significance, and degrees of freedom do not reflect the reality. To overcome this problem, regularization and shrinkage methods for regression have been developed. Elastic net is a regularization method for regression and classification models which compromises the Least Absolute Shrinkage and Selection Operator (LASSO) penalty (L1) and the Ridge penalty (L2) (Clausen et al., 2019).

Regularization adds a hyperparameter lambda to regression models. Instead of minimizing the residual sum of squares, regularized models flexibly give penalty to parameters insufficiently reducing residual variance. Elastic net is a method combining the penalties of the L1 and L2 norm of Ridge and least absolute shrinkage and selection operator (LASSO) regression, the two most widely used regularization techniques for logistic regression. Overcoming limitations of the two methods respectively, Elastic net shows advantages for variable selection as well as handling of highly correlated variables and has been demonstrated to fit large numbers of predictors (Drukker & Liu, 2019). The Elastic net method tests complete models, but at the cost of sampling distributions for individual parameters. In sum, Elastic net serves as an approach for selecting the most parsimonious model best fitted to the data in the face of a large number of highly correlated factors for an outcome.

The Elastic net method was used to create a predictive "poly event risk score" for SLEs on BPD traits. The method creates a selection of variables strongly correlated with BPD traits, and further regulates non-correlated variables to zero. The chosen model was the one

with the lowest cross-validation error. This is the model best fitted to the data. It is assumed that this is the model with the highest chance of reproducibility in other samples.

2.2.4 Co-Twin Control Analysis

Next, a discordant-twin design was applied in a co-twin control analysis. This analysis allows adjustment for familial confounders on the observed association between SLEs and BPD traits. Members of a twin pair share either 100% (MZ) or on average 50% (DZ) of their segregating genes (van Dongen, Slagboom, Draisma, Martin & Boomsma, 2012). In addition, environmental factors may either have contributed to similarity (shared environmental effects) or difference (non-shared environmental effects) between members of a pair. By assessing effects within twin pairs, the relationship between SLEs and BPD traits is investigated while controlling for possible effects of shared environmental factors and either all (MZ) or half (DZ) of the effect of genetic factors.

Table 2

An Overview of Stages in The Co-Twin Control

Stages	Description	Parameters of interest	Explanation
1	Investigating the effect of SLEs on Bw*	Bw	Concordance & discordance within MZ and DZ twin pairs
2	2a & 2b	Bw & Bb**	Biometric Poisson regression: Investigating difference in relative risk within & between MZ & DZ pairs
	2a. Multilevel Poisson model on poly event risk score (baseline)		Including the fixed effects *sex* & *wave*. Random effects include the participants' unique serial number
	2b. Multilevel Poisson model on poly event risk score (ACE***)		Building on the baseline model. It includes fixed effects of Bw & Bb in MZ & DZ pairs, and random effects for A, C & E
3	Comparing 2a & 2b	A, C, E	Investigating the influence of A, C & E within the poly event risk score

Note. * Bb = between pairs. ** Bw = within pairs. *** ACE = (A) genetic factors, (C) shared environment, and (E) non-shared environment.

In stage 1, the discordance of the experience of SLEs within the twin pairs were examined to consider the foundation for a co-twin control method. The design compares twins who differ in exposure to the risk factor and depends on concordance in twin pairs. Concordance and discordance are presented in Table 10.

Introducing stage 2, we tested for overdispersion to allow the use of a Poisson regression. After accounting for random effects, we found no overdispersion on the within-level (i.e. number of BPD criteria after twin pair and time-invariant individual level risk is accounted for). In stage 2a, a multilevel Poisson regression was carried out including BPD traits as the dependent variable and sex and wave as fixed effects. It contained a claim that the sample included would have data (yes/no) on the SLEs in childhood and adulthood. The participants unique serial number was added as a random effect to account for the same person being assessed twice. This is the baseline model. In stage 2b, a second multilevel Poisson regression was carried out building on the baseline model. The fixed effects included within- and between-pair effects of MZ twins, and the additional effect for DZ pairs on the within- and between-pair levels. The additional effects for DZ pairs were included to account for their unique contribution in genetic (1/2 A) and shared environmental factors (C). The additional DZ effects were constrained to be equal. Random effects for A, C and E were included in addition to the twin pairs unique serial number to account for dependence in the data by adjusting for the participants being members of a twin pair. The output was set to display relative risk (RR). This is the ACE model.

In the last stage, the two models were compared in order to examine the influence of A, C and E on the association between SLEs and BPD traits. In line with the principle of parsimony the AIC was considered.

3. Results

3.1 Descriptive Statistics

Table 3

An Overview of The 18 Different SLEs with Description

Type of Stressful Life Event (SLE)	Description
SLE 1	*Have you experienced a life-threatening disease?*
SLE 2	*Have you ever been involved in a life-threatening accident?*
SLE 3	*Have you actively participated in war actions?*
SLE 4	*Have you seen anyone get killed or seriously injured?*
SLE 5	*Have you been threatened with a weapon, held captured or kidnapped?*
SLE 6	*Have you been involved in a fire, flood, or any other kind of natural catastrophe?*
SLE 7	*Have you been raped?*
SLE 8	*Have you been sexually abused in any other way?*
SLE 9	*Except for the questions above, have you been exposed to physical violence?*
SLE 10	*Except for the questions above have you been physically abused as a child?*
SLE 11	*Were you exposed to any other kind of neglect as a child?*
SLE 12	*Did either one of your parents suffer from mental illness and/or alcohol abuse when you were a child?*
SLE 13	*Did your parents divorce or move apart when you were a child?*
SLE 14	*Have you divorced yourself or moved apart from a partner?*
SLE 15	*Have you had long-term economic issues?*
SLE 16	*Have you been unemployed for more than six months?*
SLE 17	*Have you been in a long-term and serious conflict with a relative, close friend, colleague or neighbor?*
SLE 18	*Have you experienced anything else serious that you would like to mention?*

Table 4

Prevalence of Reported SLEs in Childhood and Adulthood in The Total Sample, Including MZ and DZ Twins, Divided by Type of SLE

SLE Childhood Adulthood	All twins (N = 2801) N (%)	MZ twins (N = 1347) N (%)	DZ twins (N = 1454) N (%)
SLE1 *Life-threatening disease*			
Childhood	90 (1.61)	54 (2.00)	36 (1.24)
Adulthood	101 (1.80)	55 (2.04)	46 (1.58)
SLE2 *Involved in a life-threatening* *accident*			
Childhood	94 (1.68)	50 (1.86)	44 (1.51)
Adulthood	161 (2.87)	80 (2.97)	81 (2.79)
SLE3 Actively participated in *war actions*			
Childhood	0	0	0
Adulthood	53 (0.95)	24 (0.89)	29 (1.00)
SLE4 *Seen anyone get killed, or* *seriously injured*			
Childhood	96 (1.71)	44 (1.63)	52 (1.79)
Adulthood	247 (4.41)	124 (4.60)	123 (4.23)
SLE5 *Been threatened with a* *weapon, help captured or* *kidnapped*			
Childhood	56 (1.00)	24 (0.89)	32 (1.10)
Adulthood	203 (3.62)	102 (3.79)	101 (3.47)
SLE6 *Been involved in a fire, flood,* *or any other form of natural* *catastrophe*			
Childhood	76 (1.36)	44 (1.63)	32 (1.10)
Adulthood	140 (2.50)	69 (2.56)	71 (2.44)

SLE7

Been raped

Childhood	90 (1.61)	42 (1.56)	48 (1.65)
Adulthood	79 (1.41)	40 (1.48)	39 (1.34)

SLE8

Been sexually abused in any other way

Childhood	276 (4.93)	136 (5.05)	140 (4.81)
Adulthood	54 (0.96)	29 (1.08)	25 (0.86)

SLE9

Been exposed to physical violence

Childhood	190 (3.39)	92 (3.41)	98 (3.37)
Adulthood	318 (5.68)	178 (6.61)	140 (4.81)

SLE10

Been physically abused as a child

Childhood	138 (2.46)	72 (2.67)	66 (2.27)
Adulthood	1 (0.02)	1 (0.04)	0

SLE11

Experienced serious neglect as a child

Childhood	112 (2.00)	56 (2.08)	56 (1.93)
Adulthood	3 (0.05)	1 (0.04)	2 (0.07)

SLE12

Either one of your parents suffered from any mental illness or alcohol abuse when you were a child

Childhood	526 (9.39)	280 (10.39)	246 (8.46)
Adulthood	21 (0.37)	9 (0.33)	12 (0.41)

SLE13

Your parents got a divorce or moved apart when you were a child

Childhood	762 (13.60)	390 (14.48)	372 (12.79)
Adulthood	74 (1.32)	56 (2.08)	18 (0.62)

SLE14			
Experienced divorce yourself,			
or dissolution of cohabitation			
Childhood	8 (0.14)	2 (0.07)	6 (0.21)
Adulthood	890 (15.89)	468 (17.37)	422 (14.51)
SLE15			
Had any long-term economic			
issues			
Childhood	14 (0.25)	10 (0.37)	4 (0.14)
Adulthood	202 (3.61)	110 (4.08)	92 (3.16)
SLE16			
Been unemployed for more			
than six months			
Childhood	18 (0.32)	10 (0.37)	8 (0.28)
Adulthood	273 (4.87)	135 (5.01)	138 (4.75)
SLE17			
Been in a serious and			
long-term conflict with a			
relative, close friend,			
colleague or neighbor			
Childhood	40 (0.71)	20 (0.74)	20 (0.69)
Adulthood	313 (5.59)	169 (6.27)	144 (4.95)
SLE18			
Been any other serious event			
in your life that you would like			
to mention			
Childhood	142 (2.53)	88 (3.27)	54 (1.86)
Adulthood	374 (6.68)	205 (7.61)	169 (5.81)

The prevalence of reported SLEs in childhood and adulthood in the total sample, including MZ and DZ twins. Of the presented prevalence of SLEs in the total sample, SLE 14 (*Have you divorced yourself or moved apart from a partner?*) during adulthood had the highest percentage, as well as SLE 12 (*Did either one of your parents suffer from mental illness and/or alcohol abuse when you were a child?*) and SLE 13 (*Did your parents divorce or move apart when you were a child?*) during childhood. Further, these were the same reported SLEs with the highest prevalence for both MZ twins and DZ twins.

Table 5

Prevalence of Reported SLEs in Childhood and Adulthood Among Participants with and without BPD Traits

SLE Childhood Adulthood	>0 BPD traits (N = 2151) N (%)	Zero BPD traits (N = 2924) N (%)
SLE1		
Life-threatening disease		
Childhood	40 (1.86)	48 (1.64)
Adulthood	54 (2.5)	46 (1.57)
SLE2		
Involved in a life-threatening accident		
Childhood	44 (2.04)	45 (1.54)
Adulthood	82 (3.81)	76 (2.60)
SLE3 Actively participated in war actions		
Childhood	0	0
Adulthood	23 (1.07)	28 (0.96)
SLE4		
Seen anyone get killed, or seriously injured		
Childhood		
Adulthood	40 (1.86)	54 (1.85)
	107 (4.97)	131 (4.48)
SLE5		
Been threatened with a weapon, help captured or kidnapped		
Childhood	26 (1.21)	29 (0.99)
Adulthood	110 (5.11)	85 (2.91)
SLE6		
Been involved in a fire, flood, or any other form of natural catastrophe		
Childhood	46 (2.14)	27 (0.92)
Adulthood	60 (2.79)	76 (2.60)
SLE7		
Been raped.		
Childhood	55 (2.56)	34 (1.16)

Adulthood	51 (2.37)	23 (0.79)
SLE8		
Been sexually abused in any other way		
Childhood	162 (7.53)	110 (3.76)
Adulthood	32 (1.49)	20 (0.68)
SLE9		
Been exposed to physical violence		
Childhood	120 (5.58)	65 (2.22)
Adulthood	169 (7.86)	136 (4.65)
SLE10		
Been physically abused as a child		
Childhood	90 (4.18)	46 (1.57)
Adulthood	0	0
SLE11		
Experienced serious neglect as a child		
Childhood	79 (3.67)	31 (1.06)
Adulthood	0	0
SLE12		
Either one of your parents suffered from any mental illness or alcohol abuse when you were a child		
Childhood	298 (13.85)	218 (7.46)
Adulthood	6 (0.79)	5 (0.17)
SLE13		
Your parents got a divorce or moved apart when you were a child		
Childhood	362 (16.83)	373 (12.76)
Adulthood	28 (1.30)	42 (1.44)
SLE14		
Experienced divorce yourself, or dissolution of cohabitation		
Childhood	5 (0.23)	3 (0.10)
Adulthood	404 (18.78)	453 (15.49)
SLE15		
Had any long-term economic issues		

Childhood	10 (0.46)	4 (0.14)
Adulthood	118 (5.48)	72 (2.46)
SLE16		
Been unemployed for more than six months		
Childhood		
Adulthood	12 (0.56)	6 (0.21)
	134 (6.23)	126 (4.31)
SLE17		
Been in a serious and long-term conflict with a relative, close friend, colleague or neighbor		
Childhood	27 (1.25)	13 (0.44)
Adulthood	148 (6.88)	148 (5.06)
SLE18		
Been any other serious event in your life that you would like to mention		
Childhood	79 (3.67)	62 (2.12)
Adulthood	154 (7.16)	200 (6.84)

Of the presented prevalence of SLEs among participants with BPD, SLE 12 (*Did either one of your parents suffer from mental illness and/or alcohol abuse when you were a child?*) and SLE 13 (*Did your parents divorce or move apart when you were a child?*) during childhood had the highest reported percentage, as well as SLE 14 (*Have you divorced yourself or moved apart from a partner?*) during adulthood. The same SLEs had the highest percentage in childhood and adulthood among participants without BPD.

3.2 Analyses

3.2.1 Poisson Regression Analyses

To measure which types of stressful life events were associated with borderline personality disorder traits, we carried out several negative binomial Poisson regression analyses.

Table 6

An Overview of Relative Risk of Having BPD Traits if Experienced SLE 1-18

BPD traits	RR	95% CI
SLE1		
Life-threatening disease		
Childhood	1.32	[.92, 1.89]
Adulthood	1.61	[1.20, 2.15]
SLE2		
Involved in a life-threatening accident		
Childhood	1.63	[1.08, 2.46]
Adulthood	1.18	[.93, 1.49]
SLE3		
Actively participated in war actions		
Childhood	1 (omitted)	
Adulthood	.91	[.60, 1.49]
SLE4		
Seen anyone get killed, or seriously injured		
Childhood		
Adulthood	1.38	[.95, 1.99]
	1.04	[.82, 1.32]
SLE5		
Been threatened with a weapon, help		
captured or kidnapped		
Childhood	1.52	[1.04, 2.23]
Adulthood	1.56	[1.24, 1.95]
SLE6		
Been involved in a fire, flood, or any other		
form of natural catastrophe		
Childhood	1.67	[1.13, 2.46]
Adulthood	1.29	[.87, 1.89]
SLE7		
Been raped		
Childhood	2.30	[1.69, 3.13]
Adulthood	2.71	[2.04, 3.59]

SLE8
Been sexually abused in any other way

Childhood	1.84	[1.52, 2.22]
Adulthood	2.64	[1.77, 3.94]

SLE9
Been exposed to physical violence

Childhood	2.10	[1.66, 2.67]
Adulthood	1.92	[1.60, 2.30]

SLE10
Been physically abused as a child

Childhood	2.60	[2.09, 3.22]
Adulthood	1 (omitted)	

SLE11
Experienced serious neglect as a child

Childhood		
Adulthood	3.28	[2.60, 4.14]
	1 (omitted)	

SLE12
Either one of your parents suffered from
any mental illness or alcohol abuse when
you were a child

Childhood	1.91	[1.61, 2.26]
Adulthood	1.17	[.57, 2.40]

SLE13
Your parents got a divorce or moved apart
when you were a child

Childhood	1.48	[1.27, 1.72]
Adulthood	1.35	[.69, 2.62]

SLE14
Experienced divorce yourself, or
dissolution of cohabitation

Childhood	2.89	[1.20, 6.95]
Adulthood	1.26	[1.12, 1.43]
SLE15		
Had any long-term economic issues		
Childhood	1.76	[.94, 3.28]
Adulthood	2.20	[1.84, 2.64]
SLE16		
Been unemployed for more than six months		
Childhood		
Adulthood	2.19	[.94, 5.11]
	1.54	[1.26, 1.87]
SLE17		
Been in a serious and long-term conflict		
with a relative, close friend, colleague or		
neighbor		
Childhood		
Adulthood	2.80	[1.93, 4.05]
	1.69	[1.36, 2.10]
SLE18		
Been any other serious event in your life		
that you would like to mention		
Childhood	2.05	[1.53, 2.74]
Adulthood	1.31	[1.09, 1.59]

When assessing the SLEs individually, results showed that experiencing SLE 7 (*Have you been raped?*), SLE 10 (*Except for the questions above have you been physically abused as a child?*), SLE 11 (*Were you exposed to any other kind of neglect as a child?*, SLE 14 (*Have you divorced yourself or moved apart from a partner?*), and SLE 17 (*Have you been in a long-term and serious conflict with a relative, close friend, colleague or neighbor?*) during childhood, and SLE 8 (*Have you been sexually abused in any other way?*) and SLE 15 (*Have you had long-term economic issues?*) during adulthood enhances the relative risk of having BPD traits. SLE 11 in childhood (*RR = 3.28*), and SLE 8 in adulthood (*RR = 2.64*) gave the highest relative risk of having BPD reported in our sample.

Table 7

An Overview of Relative Risk of Having BPD Traits if Experienced SLEs in Childhood or Adulthood

BPD traits	RR	95% CI
SLEs in *Childhood*	1.38	[1.35, 1.42]
SLEs in *Adulthood*	1.24	[1.18, 1.31]

When assessing SLEs in childhood and adulthood separately, results showed that SLEs during childhood have the highest relative risk of having BPD *(RR = 1.38)* in our sample, compared to SLEs during adulthood *(RR = 1.24)*.

Table 8

An Overview of Relative Risk at Having BPD Traits if Experienced Any of the 5 Different Clusters of SLEs

BPD traits	RR	95% CI
Life-threatening experiences	1.24	[1.15, 1.33]
Sexual and/or physical abuse	1.73	[1.70, 1.75]
SLEs in childhood	1.53	[1.42, 1.64]
SLEs in relationships	1.39	[1.26, 1.53]
Economic issues	1.60	[1.45, 1.76]

When assessing SLEs clustered in categories, results showed that the cluster *"sexual and physical abuse" (RR = 1.73)* gives the highest relative risk of having BPD in our sample.

3.2.2 Elastic Net Regression

Aiming for a predictive model for BPD traits, we carried out an Elastic net regression analysis. The Elastic net revealed 27 SLE variables uniquely related to borderline personality disorder traits (Table 9). Three different alpha (α) values were tested ($\alpha = 1$, $\alpha = 0.75$ and $\alpha = 0.5$), and alpha = 0.5 was chosen to specify Elastic net instead of LASSO ($\alpha = 1$) or Ridge regression ($\alpha = 0$), using 10-fold cross-validation. This α was chosen due to the optimal λ

value, which in this case was $\lambda = 0.02$. We proceeded to carry out a Poisson regression with the selected 27 variables, and the result is shown in Table 9.

Figure 5

Cross-Validation Plot
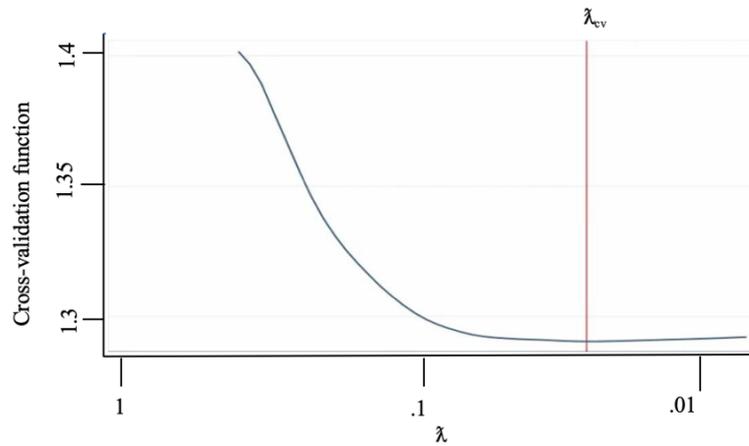
Note. α_{cv} = Cross-validation minimum alpha. $\alpha = .5$. λ_{cv} = Cross-validation minimum lambda. $\lambda = .022$, # Coefficients = 27.

The cross-validation plot from the Elastic net analysis is shown in Figure 5. It has an x-axis with the λ values and a y-axis with the cross-validation function. λ_{cv} marks where on the curve we found the model best fitted to the data.

Table 9

Best Fitting Elastic Net Regression

SLE	RR
SLE4 Childhood	
Have you seen anyone get killed or seriously injured?	*1.17*
SLE5 Childhood	
Have you been threatened with a weapon, held captured or	
kidnapped?	*.73*
SLE6 Childhood	
Have you been involved in a fire, flood, or any other kind of	
natural catastrophe?	*1.19*

SLE7 Childhood

Have you been raped?   *1.46*

SLE8 Childhood

Have you been sexually abused in any other way?   *1.15*

SLE9 Childhood

Except for the questions above, have you been exposed to physical

violence?   *1.40*

SLE10 Childhood

Except for the questions above have you been physically abused as

a child?   *1.19*

SLE11 Childhood

Were you exposed to any other kind of neglect as a child?   *1.88*

SLE12 Childhood

Did either one of your parents suffer from mental illness and/or

alcohol abuse when you were a child?   *1.44*

SLE13 Childhood

Did your parents divorce or move apart when you were a child?   *1.15*

SLE15 Childhood

Have you had long-term economic issues?   *1.24*

SLE17 Childhood

Have you been in a long-term and serious conflict with a relative,

close friend, colleague or neighbor?   *1.12*

SLE18 Childhood

Have you experienced anything else serious that you would like to

mention?   *1.33*

SLE1 Adulthood

Have you experienced a life-threatening disease?   *1.23*

SLE2 Adulthood

Have you ever been involved in a life-threatening accident?   *1.17*

SLE5 Adulthood	
Have you been threatened with a weapon, held captured or	
kidnapped?	*1.21*
SLE7 Adulthood	
Have you been raped?	*1.59*
SLE8 Adulthood	
Have you been sexually abused in any other way?	*1.08*
SLE9 Adulthood	
Except for the questions above, have you been exposed to physical	
violence?	*1.56*
SLE13 Adulthood	
Did your parents divorce or move apart when you were a child?	*.69*
SLE14 Adulthood	
Have you divorced yourself or moved apart from a partner?	*1.11*
SLE15 Adulthood	
Have you had long-term economic issues?	*1.19*
SLE16 Adulthood	
Have you been unemployed for more than six months?	*1.23*
SLE17 Adulthood	
Have you been in a long-term and serious conflict with a relative,	
close friend, colleague or neighbor?	*1.32*
SLE18 Adulthood	
Have you experienced anything else serious that you would like to	
mention?	*1.18*
Wave	*.65*
Sex	*1.05*

We established a predictive score; a poly event risk score for BPD traits. Predicted number of BPD criteria ranged from .32 to 7.64. The risk score was further correlated with

the initial BPD symptom score measured at a single timepoint, $r = .33$. The predicted number of events had a variance of .13, a mean of .65 and a median of .61 events.

3.2.3 Co-Twin Control Analysis

Table 10

Concordance and Discordance. C = Twins That Are Concordant (The Same). D = Twins That Are Discordant (Different) Within Their Twin Pairs

SLE	MZ pairs W1	MZ pairs W2	DZ pairs W1	DZ pairs W2
Childhood	C, D	C, D	C, D	C, D
Adulthood	C	C	C	C
SLE1				
Life-threatening disease				
Childhood				
Experienced	0, 25	0, 12	0, 12	0, 12
Not experienced	488	455	453	453
Adulthood				
Experienced	0, 16	2, 31	0, 8	0, 26
Not experienced	497	480	457	439
SLE2				
Involved in a life-threatening accident				
Childhood				
Experienced	1, 21	1, 21	1, 12	1, 12
Not experienced	494	494	457	457
Adulthood				
Experienced	3, 23	3, 36	1, 21	1, 32
Not experienced	490	477	448	437
SLE3				
Actively participated in war actions				
Childhood				
Experienced				
Not experienced	518	518	471	471
Adulthood				
Experienced	2, 5	4, 5	0, 11	11, 0
Not experienced	511	509	460	460

SLE4

Seen anyone get killed,
or seriously injured

Childhood

Experienced	2, 15	2, 15	0, 20	0, 20
Not experienced	499	499	488	488

Adulthood

Experienced	6, 33	8, 50	0, 35	1, 61
Not experienced	477	458	433	406

SLE5

Been threatened with a
weapon, help captured
or kidnapped

Childhood

Experienced	1, 8	1, 8	2, 9	2, 9
Not experienced	509	509	457	457

Adulthood

Experienced	4, 28	4, 37	2, 26	2, 35
Not experienced	486	477	440	431

SLE6

Been involved in a fire,
flood, or any other form
of natural catastrophe

Childhood

Experienced				
Not experienced	3, 14	3, 14	2, 8	2, 8
	501	501	459	459

Adulthood

Experienced				
Not experienced	0, 23	1, 36	0, 20	1, 34
	496	481	449	434

SLE7

Been raped

Childhood

Experienced	1, 17	1, 17	1, 18	1, 18
Not experienced	498	498	451	451

Adulthood

Experienced	0, 15	0, 17	0, 12	0, 14
Not experienced	501	499	458	456

SLE8
Been sexually abused in
any other way

Childhood

Experienced	11, 41	11, 41	4, 42	4, 42
Not experienced	465	465	425	425

Adulthood

Experienced	0, 12	1, 12	0, 8	0, 9
Not experienced	505	504	463	462

SLE9
Been exposed to
physical violence

Childhood

Experienced	4, 33	4, 33	1, 33	1, 33
Not experienced	474	474	431	431

Adulthood

Experienced	5, 50	6, 80	0, 41	3, 56
Not experienced	456	425	424	406

SLE10
Been physically abused
as a child

Childhood

Experienced	7, 17	7, 17	2, 14	2, 14
Not experienced	492	492	453	453

Adulthood

Experienced		0, 1		
Not experienced	516	515	469	469

SLE11
Experienced serious
neglect as a child

Childhood

Experienced	5, 14	5, 14	3, 13	3, 13
Not experienced	496	496	454	454

Adulthood

Experienced		0, 1		0, 2
Not experienced	515	514	470	468

SLE12
Either one of your

parents suffered from any mental illness or alcohol abuse when you were a child

Childhood

Experienced	38, 45	38, 45	21, 55	21, 55
Not experienced	430	430	394	394

Adulthood

Experienced	0, 2	0, 2	0, 2	0, 9
Not experienced	511	511	460	461

SLE13
Your parents got a divorce or move apart when you were a child

Childhood

Experienced	69, 31	69, 31	67, 13	67, 13
Not experienced	417	417	391	391

Adulthood

Experienced	3, 16	3, 19	1, 4	1, 4
Not experienced	498	495	466	466

SLE14
Experienced divorce yourself, or dissolution of cohabitation

Childhood

Experienced	0, 1	0, 1	0, 2	0, 2
Not experienced	515	515	466	466

Adulthood

Experienced	12, 102	56, 170	7, 75	36, 176
Not experienced	402	290	386	256

SLE15
Had any long-term economic issues

Childhood

Experienced	0, 5	0, 5	0, 1	0, 1
Not experienced	509	509	466	466

Adulthood

Experienced	4, 24	8, 41	1, 21	2, 41
Not experienced	486	465	445	424

SLE16				
Been unemployed for more than six months				
Childhood				
Experienced	0, 5	0, 5	0, 3	0, 3
Not experienced	512	512	461	461
Adulthood				
Experienced	4, 30	12, 55	2, 32	2, 70
Not experienced	483	450	430	392
SLE17				
Been in a serious and long-term conflict with a relative, close friend, colleague or neighbor				
Childhood				
Experienced	1, 7	1, 7	0, 8	0, 8
Not experienced	506	506	459	459
Adulthood				
Experienced	4, 34	20, 69	2, 18	12, 55
Not experienced	476	425	447	400
SLE18				
Been any other serious event in your life that you would like to mention				
Childhood				
Experienced	3, 32	3, 32	0, 18	0, 18
Not experienced	464	464	446	446
Adulthood				
Experienced	0, 46	12,108	1, 31	9, 92
Not experienced	453	379	432	363

Discordance on the experience of SLEs within the twin pairs was present in the sample. This allowed us to carry out a co-twin control analysis.

Table 11

Biometric Co-Twin Control. Effect within The Whole Sample, DZ Pairs and MZ Pairs

BPD traits	RR	95% CI
Whole sample	*3.14*	*[2.52, 3.91]*
Within DZ pairs	*1.29*	*[1.06, 1.56]*
Within MZ pairs	*1.33*	*[1.10, 1.60]*

We carried out a multilevel Poisson model and found the effect within MZ pairs to be RR = 1.33 and the effect within DZ pairs to be RR = 1.29. The additional effect of DZ pairs on the between level was RR = 1.03 and equal to no effect. This excluded the possibility for a gene-environment correlation, as the direction of the effect should be negative because the DZ pairs should be less similar if genetic factors play a role. All similarity is assigned to shared environment (C) on the between level.

When comparing the baseline model and the ACE model we found that the AIC decreased from 8976 to 8837. This shows that the ACE model was the better fitted model. The unstandardized coefficient was used as a basis for comparison for the effects of A, C and E. In the baseline model (adjusting for sex and wave) the effects of A = .36, C = .24 and E = .17. In standardized metrics, this corresponds to a heritability (h^2) of 47%, shared environmental effect (c^2) of 31%, and non-shared environmental effect (e^2) of 22%. When including the poly event risk score, the effects in the ACE model was A = .30, C = .14 and E = .16. We found that the shared environmental variance decreased the most with 41.7% (-.1), followed by genetic variance with a decrease of 16.7% (-.06). The non-shared environmental factors did not decrease remarkably (5.9% or -.01). Summing up the explained variance at the three levels, the poly event risk score explained 22% of the total stable, or time-invariant, variance in BPD traits.

4. Discussion

The aim of this study was to investigate what types of stressful life events are associated with borderline personality disorder traits, and to what extent the association is accounted for by familial genetic and environmental background factors. This was done by, respectively, using Elastic net regression and the co-twin control method. Studies have found

an association between SLEs in childhood and BPD traits, and some have suggested experiences of sexual and physical abuse to be of particular importance. We carried out Poisson regression analyses to explore the relative risk of BPD traits if experiencing the assessed SLEs individually, clustered in categories and separated by childhood and adulthood. The Elastic net regression selected which of the SLEs were strongly correlated with BPD traits. While it's been proposed that the relationship between SLEs and BPD traits meets some of the suggested criteria for causality, the problem of confounding might bias the direct effect. Confounding by genetic factors (A), shared environment (C) or non-shared environment (E) represents a threat to the validity of the effect if not discovered. The discordant-twin design made it possible to investigate the association while controlling for shared environment and genetic factors and thereby reduce the problem of confounding.

First, we investigated what types of SLEs are associated with BPD traits. In the presented prevalence of SLEs in the total sample, the following SLEs are of importance: *Have you divorced yourself or moved apart from a partner?* during adulthood had the highest percentage, as well as *Did either one of your parents suffer from mental illness and/or alcohol abuse when you were a child?* and *Did your parents divorce or move apart when you were a child?* during childhood. These were the same reported SLEs with the highest prevalence for both MZ twins and DZ twins, as well as among participants with and without BPD. Previously mentioned studies enhance the experience of early traumatic events, and that childhood adversity was highly prevalent in people with PDs, and most consistently associated with BPD (Afifi et al., 2011; Golier et al., 2003). In our study SLEs experienced in both childhood and adulthood were reported.

Further, the results from our analysis showed that experiencing SLEs such as *Have you been raped?, Have you been physically abused as a child?, Were you exposed to any other kind of neglect as a child?, Have you divorced yourself or moved apart from a partner?,* and *Have you been in a long-term and serious conflict with a relative, close friend, colleague or neighbor?* during childhood enhances the relative risk of having BPD traits, where *Were you exposed to any other kind of neglect as a child?* has the highest relative risk *(RR = 3.28; 95%CI 2.60, 4.14)*. This indicates that if you were exposed to any other kind of neglect as a child, the chance of developing a BPD trait increases with 3.28 times. These results are consistent with previously mentioned studies, indicating that sexual abuse, emotional abuse, and emotional neglect is associated with BPD (Lobbestael et al., 2010; Wingenfeld et al., 2011).

Existing literature indicating a transient effect of SLEs in adulthood on BPD traits is dissimilar to the findings from our study where *Have you been sexually abused in any other way?* and *Have you had long-term economic issues?* during adulthood enhances the relative risk of having BPD traits, with *Have you been sexually abused in any other way? (RR = 2.64; 95%CI 1.77, 3.94)* having the highest relative risk. These results indicate that if you experience sexual abuse during adulthood, the chance of developing a BPD trait increases with 2.64 times. This points to SLEs experienced in adulthood increasing the risk of developing BPD traits. These results are not consistent with existing research finding that SLEs in adulthood had no notable effect on the severity of BPD and BPD patients reporting a declining rate of experiences of abuse in adulthood (Conway et al., 2018; McGowan et al., 2012). When clustering the SLEs and analyzing the relative risk of having BPD traits if experiencing SLEs in childhood or adulthood, our results showed that experiencing SLEs during childhood had a higher relative risk at developing BPD traits than SLEs during adulthood. These findings are consistent with several studies. Liotti and colleagues (2000) found that the child's early traumatic experiences, as well as mourning process in the mother within the first two years of a child's life as predictive factors for BPD. Further, Johnson and colleagues (2001) reported that children exposed to maternal verbal abuse had an increased risk of developing personality disorders. Both Lobbestael and colleagues (2010) and Wingenfeld and colleagues (2011) found that sexual abuse, emotional abuse, and emotional neglect during childhood was associated with BPD. Further, Afifi and colleagues (2011) found that childhood adversity was highly prevalent in people with PDs, and most consistently associated with BPD and three other types of PD. The findings support the association between childhood adversity and BPD. Yet, as previously mentioned, SLEs during adulthood might play a bigger role in this development than first anticipated.

Further, we investigated five clusters of SLEs, finding that the cluster *"sexual and physical abuse" (RR = 1.73; 95%CI 1.70, 1.75)* gave the highest relative risk of having BPD traits out of the five clusters. These results are consistent with previously mentioned studies indicating that sexual abuse is associated with BPD, that a majority of BPD patients reported early sexual and physical abuse, and that early life stress was considered a risk factor in the development of this personality disorder (Lobbestael et al., 2010; Wingenfeld et al., 2011).

Next, to secure replication and further validation, we wanted to develop a model to better predict which SLEs are associated with BPD traits. Our total sample contained 18 different SLEs. These were further divided into two clusters containing all 18 SLEs in *childhood* and in *adulthood*, making a total of 48 SLEs. The regularization regression method

of an Elastic net analysis was applied to develop such a prediction. This analysis created a poly event risk score containing a selection of 27 SLEs strongly correlated with BPD traits, where non-correlated SLEs are put to zero. The risk score was correlated $r = .33$ with BPD traits measured at a single timepoint, indicating that our model is moderately predictive of development of BPD traits. Even though the number of SLEs remaining in our predictive model is still high, several SLEs are put to zero which makes it easier to see a trend of SLEs in childhood and adulthood.

The elected list of SLEs contains 13 SLEs during childhood, and 12 during adulthood as well as the variables *wave* and *sex* (Table 9). The selected SLEs experienced during childhood reflects witnessing and/or experiencing life-threatening experiences such as natural catastrophes, war, and kidnapping. Further, the list reflects experiences of an unsafe and unpredictable childhood environment containing sexual and physical abuse, neglect, violence as well as having a parent suffering from mental illness, having economic issues, and having either one of your parents move away. These findings are consistent with previously mentioned studies underlining the importance of childhood traumas, specifically sexual and physical abuse (Afifi et al., 2011; Golier et al., 200; Johnson et al., 1999; Johnson et al., 2001; Liotti et al., 2000; Lobbestael et al., 2010; Wingenfeld et al., 2011). The SLEs experienced in adulthood reflect life-threatening experiences such as disease, accidents, and abuse, as well as economic issues and abruption of relationships. These findings are in line with our analyses of relative risk, but dissimilar to existing research finding a transient effect of SLEs in adulthood on BPD traits (Conway et al., 2018; McGowan et al., 2012). It's important to note that choosing the best fitting model to the data does not translate to each predictor being individually significant. Each predictor must be included in the model to explain BPD traits in the best possible way, but we cannot say that every SLE can explain BPD traits individually.

We proceeded to investigate to what extent the association between SLEs and BPD traits is accounted for by familial factors by conducting a biometric co-twin control analysis. The results from our analysis do not support a singular causal explanation for the relationship between SLEs and BPD traits as the effect of exposure was reduced when controlling for shared environmental and genetic factors. Only a small part of the association reflects causality, while most of it reflects confounding. Our results are in line with the existing discordant-twin studies examining the SLE-BPD relationship which finds little to no evidence of causal effect of SLEs on BPD traits (Berenz et al., 2013; Bornovalova et al., 2013). However, this is dissimilar to the results from the review by Ball & Links (2009) where they found support for a causal relationship. Also, previous studies have found the experience of

childhood adversity to increase the risk for BPD traits (Hengartner et al., 2013; Ibrahim et al., 2018). Although we did not directly assess the dose-response relationship in this study, findings of this type of relationship are taken to support a causal relationship between SLEs and BPD traits which our study only found to a moderate degree.

When adjusting for the measures of the environment included in the poly event risk score, we found that the shared environmental factors decreased the most. It appears to serve as a confounder between SLEs and BPD traits, meaning it seems to be something in the effective environment that leads to both the experience of SLEs *and* the development of BPD traits (Turkheimer & Waldron, 2000). The pattern of confounding was not similar to complete confounding of shared environment (pattern (B1) in Figure 4). The effect decreased but was not completely absent as one would expect with complete confounding. The effect of exposure was the same in DZ pairs and MZ pairs, supporting a partial confounding (pattern (C) in Figure 4) with a similar decrease in DZ and MZ pairs. Berenz et al. (2013) found that the association was accounted for by familial factors, but low statistical power did not allow them to examine the DZ and MZ pairs separately. The results could therefore not determine whether the effect was due to genetic or shared environmental factors. Our study can serve as an extension to their research by combining MZ and DZ pairs in a single biometric model and aggregating SLE into a poly event risk score, pointing to the association being accounted for by shared environment. The discovery of partial confounding by shared environmental factors is in contrast to the discordant-twin study conducted by Bornovalova et al. (2013). They found no significant shared environmental influences and the association to be better explained by genetic factors. Some shared environmental factors remain unaccounted for by the stressful life events assessed in this study, meaning the influence of the effective environment on BPD traits can't be fully explained with the measures of the environment included in the poly event risk score.

We did not find any evidence for genetic confounding, i.e. a gene-environment correlation (rGE), but evidence for genetic effects having indirect effect by mediation through SLEs (Young, Benonisdottir, Przeworski & Kong, 2019). This is dissimilar to findings from the review by Carpenter et al. (2012) which suggests that genes responsible for BPD risk increase the risk of exposure to SLEs. In our study the genetic factors did decrease a small amount when adjusting for the SLEs included in the poly event risk score, supporting that SLEs are linked to some genetic factors for BPD traits. These putative causal effects constitute mediated genetic effects (i.e. SLEs explain genetic variance in BPD through a causal path, since SLEs are influenced by genetic factors). This is in line with the findings of

the SLE-BPD association being explained by genetic factors (Bornovalova et al., 2013). However, the genetic risk for BPD traits seems to be primarily unaccounted for by the stressful life events in the poly event risk score. This means that any genome-wide association study on BPD traits will only to a moderate degree (i.e. our estimate = 16.7%) find alleles giving risk for SLEs, and to a great degree find alleles not having a mediated effect on BPD through SLEs.

4.1 Conclusion

What types of stressful life events are associated with borderline personality disorder traits? In our sample, the experience of SLEs was present in both MZ and DZ twins, as well as in participants with and without BPD traits. With a regularization regression method for model sparsity we learned that life-threatening childhood experiences and an unpredictable and unsafe childhood environment are associated with the development of BPD traits. In adulthood, life-threatening experiences, economic issues, and relationship conflicts seem to have the same association. This indicates that SLEs experienced in adulthood also might affect the development of BPD traits. Our results further indicate that these reported experiences in childhood and adulthood can predict 22% of the development of BPD traits. To what extent are such associations accounted for by familial genetic and environmental background factors? In the debate about *how* stressful life events are associated with the development of BPD traits, it does not appear to be a causal factor. Our results point to there being something in the shared family environment that causes both the stressful life events and the development of BPD traits.

If a child is born into the world not being met on its needs or facing other stressful experiences in childhood or adulthood, he/she might portray the world as an unsafe place with unpredictable relationships, fear, and uncertainty. These stressful experiences seem to indicate a negative family environment, and whatever is in this environment seems to cause the experiences and further affect the development of BPD traits. It is therefore of importance to limit the effect of, or prevent, familial environmental background factors causing both SLEs and BPD traits.

4.2 Strengths and limitations

Our study has several limitations. First, the surveying of traumas was based on retrospective reporting. Although the validity of the reporting probably was enhanced by the use of a semi-structured interview approach, there might still be weaknesses in the reporting

due to limitations in memory, repression, denial or deliberate over- and/or under-reporting. Despite this, there are research results that support the validity of retrospective reporting of serious incidents in childhood (Newbury et al., 2018). Memory for potentially traumatic events appears to be more accurate than memory for non-traumatic events (Lalande & Bonanno, 2011). Psychiatric status also does not appear to be associated with less reliable or valid reporting of early experiences (Brewin, Andrews & Gotlib, 1993). However, measurement errors in the independent variable are always a major problem in regression models. These are problems that affect all types of research on human conditions based on self-reporting and should therefore not put an end to conducting such research.

Second, SLEs experienced in adulthood might be a result of reverse causality, meaning that SLEs in adulthood and BPD are associated, but not as first expected. Instead of SLEs in adulthood causing BPD, it might be the other way around. BPD might cause SLEs in adulthood, where the exposure to BPD causes the risk of experiencing SLEs in adulthood.

Third, we used an approach for selecting the most parsimonious model best fitted to the data at the cost of sampling distributions for individual parameters. A limitation with the Elastic net is that it provides no meaningful way to estimate a confidence interval for the individual predictors included in the model.

Fourth, a discordant twin design checks for all variables shared by the twins (i.e. shared environment and genetic factors) to examine the effect of a non-shared variable, in this case SLEs. Although a discordant twin design eliminates confusion of familial factors, it cannot eliminate confusion of unmeasured unshared variables (McGue et al., 2010). In our study, we did not solve this issue either.

Fifth, although widely used and considered as a valid twin sample, the Norwegian Twin registry contains only Norwegian registered twin pairs. This challenges generalization to other populations. However, Bornovalova et al. (2013) found similar results in a sample of young adult twin pairs from Minnesota, which may indicate that the findings might be generalized to other populations. Either way, it is important to cross validate the findings in this study by replicating the findings in other data sets with samples from other age groups, and with different national backgrounds.

This study also has several strengths. First, the study uses a genetically informative large sample from the Norwegian Twin Registry.

Second, BPD traits in our sample were mapped out by using clinical interviews considered to be representative for long-term personality (Kendler et al., 2008). Further, the participants were interviewed twice, approximately 10 years apart. This takes stability in the

BPD traits into account.

Third, discordant twin studies are a powerful method that makes it possible to separate the effect of an exposure from the influences of a person's genotype and shared family environment. This makes discordant twin designs a good method when shedding light on issues of causality (McGue et al., 2010). The validity of the conclusions drawn from twin studies depends on the fact that twins are representative of the general population. Twins have been 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 individuals in terms of personality (Andrew et al., 2001; Johnson, Krueger, Bouchard & McGue, 2002).

Fourth, when researchers have been interested in the associations between SLEs and BPD, they've usually been investigating one life event at a time. This presents a problem because while investigating one life event, it may be a different life event that is of importance. Therefore, one should examine multiple life events at the same time. By using a regularization regression method for model sparsity, we were able to find the best fitted model to our data set. Due to the replication crisis in psychology, single p-values might not be as informative as first assumed. According to Maxwell et al. (2015), questionable research practices clearly need to be addressed because they produce inflated estimates of effect sizes and render p-values largely uninterpretable. Therefore, when investigating multiple life events and further developing the best fitted model for prediction, this also makes it easier to replicate, and further takes the replication crisis in psychology into account.

References

Afifi, T. O., Mather, A., Boman, J., Fleisher, W., Enns, M. W., Macmillan, H. & Sareen, J. (2011). Childhood adversity and personality disorders: Results from a nationally representative population-based study. *Journal of Psychiatric Research*, *45*(6), 814-822. https://doi.org/10.1016/j.jpsychires.2010.11.008

Andrew, T., Hart, D. J., Snieder, H., de Lange, M., Spector, T. D. & Macgregor, A. J. (2001). Are Twins and Singletons Comparable? A Study of Disease-Related and Lifestyle Characteristics in Adult Women. *Twin Research: the Official Journal of the International Society for Twin Studies, 4*(6), 464-477.

Bae, H., Kim, D., Koh, H., Kim, Y., & Park, J. S. (2008). Psychometric Properties of the Life Events Checklist-Korean Version. *Psychiatry Investigation*, *5*, 163–167. http://dx.doi.org/10.4306/pi.2008.5.3.163

Ball, J. S., & Links, P. S. (2009). Borderline Personality Disorder and Childhood Trauma: Evidence for a Causal Relationship. *Current Psychiatry Reports*, *11*(1), 63-68.

Berenz, E. C., Amstadter, A. B., Aggen, S. H., Knudsen, G. P., Reichborn-Kjennerud, T., Gardner, C. O. & Kendler, K. S. (2013). Childhood Trauma and Personality Disorder Criterion Counts: A Co-twin Control Analysis. *Journal of Abnormal Psychology*, *122*(4), 1070-1076. https://doi.org/10.1037/a0034238

Bornovalova, M. A., Hicks, B. M., Iacono, W. G. & McGue, M. (2009). Stability, Change, and Heritability of Borderline Personality Disorder Traits from Adolescence to Adulthood: A Longitudinal Twin Study. *Development and Psychopathology*, *21*(4), 1335-1353. https://doi.org/10.1017/S0954579409990186

Bornovalova, M. A., Huibregtse, B. M., Hicks, B. M., Keyes, M., McGue, M., & Iacono, W. (2013). Tests of a Direct Effect of Childhood Abuse on Adult Borderline Personality Disorder Traits: A Longitudinal Discordant Twin Design. *Journal of Abnormal Psychology*, *122*(1), 180-194. http://dx.doi.org/10.1037/a0028328

Brewin, C. R., Andrews, B. & Gotlib, I. H. (1993). Psychopathology and Early Experience: A Reappraisal of Retrospective Reports. *Psychological Bulletin, 113*(1), 82-98. https://doi.org/10.1037/0033-2909.113.1.82

Clausen, A. N., Aupperle, R. L., Yeh, H. W., Waller, D., Payne, J., Kuplicki, R., ... & Victor, T. (2019). Machine Learning Analysis of the Relationships Between Gray Matter Volume and Childhood Trauma in a Transdiagnostic Community-Based Sample. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *4*(8), 734-742. https://doi.org/10.1016/j.bpsc.2019.03.001

Contractor, A. A., Weiss, N. H., Natesan Batley, P., & Elhai, J. D. (2020). Clusters of Trauma Types as Measured by the Life Events Checklist for DSM–5. *International Journal of Stress Management*, *27*(4), 380. https://doi.org/10.1037/str0000179

Conway, C. C., Boudreaux, M., & Oltmanns, T. F. (2018). Dynamic Associations Between Borderline Personality Disorder and Stressful Life Events Over Five Years in Older Adults. Personality Disorders: *Theory, Research, and Treatment*, *9*(6), 521. https://doi.org/10.1037/per0000281

Coolidge, F. L., Thede, L. L., & Jang, K. L. (2001). Heritability of Personality Disorders in Childhood: A PreliminaryIinvestigation. *Journal of Personality Disorders*, *15*(1), 33-40. https://doi.org/10.1521/pedi.15.1.33.18645

Distel, M. A., Trull, T. J., Willemsen, G., Vink, J. M., Derom, C. A., Lynskey, M., ... & Boomsma, D. I. (2009). The Five-Factor Model of Personality and Borderline Personality Disorder: A Genetic Analysis of Comorbidity. *Biological psychiatry*, *66*(12), 1131-1138. https://doi.org/10.1016/j.biopsych.2009.07.017

Dohrenwend, B. S. (1973). Life Events as Stressors: A methodological Inquiry. *Journal of Health and Social Behavior*, 167-175. https://doi.org/10.1037/h0035718

Drukker, D., & Liu, D. (2019, September 9th). An Introduction to the Lasso in Stata. *The Stata Blog*. https://blog.stata.com/2019/09/09/an-introduction-to-the-lasso-in-stata/

Fatimah, H., Wiernik, B. M., Gorey, C., McGue, M., Iacono, W. G., & Bornovalova, M. A. (2020). Familial Factors and the Risk of Borderline Personality Pathology: Genetic and Environmental Transmission. *Psychological Medicine*, *50*(8), 1327-1337. https://doi.org/10.1017/ S0033291719001260

Golier, J. A., Yehuda, R., Bierer, L. M., Mitropoulou, V., New, A. S., Schmeidler, J., ... & Siever, L. J. (2003). The Relationship of Borderline Personality Disorder to Posttraumatic Stress Disorder and Traumatic Events. *American Journal of Psychiatry*, *160*(11), 2018-2024. https://doi.org/10.1176/appi.ajp.160.11.2018

Gunderson, J. G. (2009). *Borderline Personality Disorder: A Clinical Guide*. American Psychiatric Pub.

Gunderson, J. G. (2011). Borderline Personality Disorder. *New England Journal of Medicine*, *364*(21), 2037-2042. https://doi.org/10.1056/NEJMcp1007358

Gunderson, J. G., Herpertz, S. C., Skodol, A. E., Torgersen, S. & Zanarini, M. C. (2018). Borderline Personality Disorder. *Nature Reviews Disease Primers, 4*, 1-20. https://doi.org/10.1038/nrdp.2018.29

Hengartner, M., Ajdacic-Gross, V., Rodgers, S., Müller, M., & Rössler, W. (2013). Childhood Adversity in Association with Personality Disorder Dimensions: New Findings in an Old Debate. *European Psychiatry*, *28*(8), 476-482. https://doi.org/10.1016/j.eurpsy.2013.04.004

Heumann, K. & Morey, L. (1990). Reliability of categorical and dimensional judgments of personality disorder. *Am J Psychiatry* 1990; 147:498–500. https://doi.org/10.1176/ajp.147.4.498

Ibrahim, J., Cosgrave, N., & Woolgar, M. (2018). Childhood Maltreatment and its Link to Borderline Personality Disorder Features in Children: A Systematic Review Approach. *Clinical Child Psychology and Psychiatry*, *23*(1), 57-76. https://doi.org/10.1177/1359104517712778

Irgens, L. M. (2000). The Medical Birth Registry of Norway. Epidemiological Research and Surveillance Throughout 30 Years. *Acta Obstetricia et Gynecologica Scandinavica*, *79*(6), 435-439. https://doi.org/10.1034/j.1600-0412.2000.079006435.x

Jane, J. S., Pagan, J. L., Turkheimer, E., Fiedler, E. R., & Oltmanns, T. F. (2006). The Interrater Reliability of the Structured Interview for DSM-IV Personality. *Comprehensive psychiatry*, *47*(5), 368-375. https://doi.org/10.1016/j.comppsych.2006.01.009

Johnson, J. G., Cohen, P., Brown, J., Smailes, E. M. & Bernstein, D. P. (1999). Childhood Maltreatment Increases Risk for Personality Disorders During Early Adulthood. *Archives of General Psychiatry*, *56*(7), 600-606. https://doi.org/10.1001/archpsyc.56.7.600

Johnson, J. G., Cohen, P., Smailes, E. M., Skodol, A. E., Brown, J., & Oldham, J. M. (2001). Childhood Verbal Abuse and Risk for Personality Disorders During Adolescence and Early Adulthood. *Comprehensive Psychiatry*, *42*(1), 16-23. https://doi.org/10.1053/comp.2001.19755

Johnson, W., Krueger, R. F., Bouchard, T. J., & McGue, M. (2002). The Personalities of Twins: Just Ordinary Folks. *Twin Research and Human Genetics*, *5*(2), 125-131. https://doi.org/10.1375/twin.5.2.125

Kendler, K. S., Aggen, S. H., Czajkowski, N., Røysamb, E., Tambs, K., Torgersen, S., ... & Reichborn-Kjennerud, T. (2008). The Structure of Genetic and Environmental Risk Factors for DSM-IV Personality Disorders: A Multivariate Twin Study. *Archives of General Psychiatry*, *65*(12), 1438-1446. https://doi.org/10.1001/archpsyc.65.12.1438

Kendler, K. S., Aggen, S. H., Gillespie, N., Krueger, R. F., Czajkowski, N., Ystrom, E., & Reichborn-Kjennerud, T. (2019). The Structure of Genetic and Environmental Influences on Normative Personality, Abnormal Personality Traits, and Personality Disorder Symptoms. *Psychological Medicine*, *49*(8), 1392. https://doi.org/10.1017/S0033291719000047

Kendler, K. S., Aggen, S. H., Gillespie, N., Neale, M. C., Knudsen, G. P., Krueger, R. F., ... & Reichborn-Kjennerud, T. (2017). The Genetic and Environmental Sources of Resemblance Between Normative Personality and Personality Disorder Traits. *Journal of Personality Disorders*, *31*(2), 193-207. https://doi.org/10.1521/pedi_2016_30_251

Kendler, K. S., Neale, M. C., MacLean, C. J., Heath, A. C., Eaves, L. J., & Kessler, R. C. (1993). Smoking and Major Depression: A Causal Analysis. *Archives of General Psychiatry*, *50*(1), 36-43. https://doi.org/10.1001/archpsyc.1993.01820130038007

Kira, I. A. (2001). Taxonomy of Trauma and Trauma Assessment. *Traumatology*, *7*(2), 73-86.

Lalande, K. M. & Bonanno, G. A. (2011). Retrospective Memory Bias for the Frequency of Potentially Traumatic Events: A Prospective Study. *Psychological Trauma: Theory, Research, Practice, and Policy, 3*(2), 165-170. https://doi.org/10.1037/a0020847

Leichsenring, F., Leibing, E., Kruse, J., New, A. S., & Leweke, F. (2011). Borderline Personality Disorder. *The Lancet*, *377*(9759), 74-84. https://doi.org/10.1016/S0140-6736(10)61422-5

Leonard, J. (2020, June 3rd). What is Trauma? What to Know. *Medical News Today*. https://www.medicalnewstoday.com/articles/trauma

Lieb, K., Zanarini, M. C., Schmahl, C., Linehan, M. M., & Bohus, M. (2004). Borderline Personality Disorder. *The Lancet*, *364*(9432), 453-461. https://doi.org/10.1016/S0140-6736(04)16770-6

Links, P. S., Steiner, M., & Huxley, G. (1988). The Occurrence of Borderline Personality Disorder in the Families of Borderline Patients. *Journal of Personality Disorders*, *2*(1), 14-20. https://doi.org/10.1521/pedi.1988.2.1.14

Liotti, G., Pasquini, P., Cirrincione, R., & Italian Group for the Study of Dissociation. (2000). Predictive Factors for Borderline Personality Disorder: Patients' Early Traumatic Experiences and Losses Suffered by the Attachment Figure. *Acta Psychiatrica Scandinavica*, *102*(4), 282-289. https://doi.org/10.1034/j.1600-0447.2000.102004282.x

Lis, E., Greenfield, B., Henry, M., Guilé, J. M., & Dougherty, G. (2007). Neuroimaging and Genetics of Borderline Personality Disorder: A Review. *Journal of Psychiatry & Neuroscience*, *32*(3), 162.

Lobbestael, J., Arntz, A. & Bernstein, D. P. (2010). Disentangling the Relationship Between Different Types of Childhood Maltreatment and Personality Disorders. *Journal Of Personality Disorders*, *24*(3), 285-295. https://doi.org/10.1521/pedi.2010.24.3.285

MacIntosh, H. B., Godbout, N., & Dubash, N. (2015). Borderline Personality Disorder: Disorder of Trauma or Personality, A Review of the Empirical Literature. *Canadian Psychology/Psychologie Canadienne*, *56*(2), 227. https://doi.org/10.1037/cap0000028

Maxwell, S. E., Lau, M. Y., & Howard, G. S. (2015). Is Psychology Suffering From A Replication Crisis? What Does "Failure to Replicate" Really Mean?. *American Psychologist*, *70*(6), 487. https://doi.org/10.1037/a0039400

McGowan, A., King, H., Frankenburg, F. R., Fitzmaurice, G., & Zanarini, M. C. (2012). The Course of Adult Experiences of Abuse in Patients with Borderline Personality Disorder and Axis II Comparison Subjects: A 10-Year Follow-up Study. *Journal of Personality Disorders*, *26*(2), 192-202. https://doi.org/10.1521/pedi.2012.26.2.192

McGue, M., Osler, M. & Christensen, K. (2010). Causal Inference and Observational Research: The Utility of Twins. *Perspectives on Psychological Science*, *5*(5), 546-556. https://doi.org/10.1177/1745691610383511

McLeod, J. D., & Almazan, E. P. (2003). Connections Between Childhood and Adulthood. In *Handbook of the life course* (pp. 391-411). Springer, Boston, MA.

Newbury, J. B., Arseneault, L., Moffitt, T. E., Caspi, A., Danese, A., Baldwin, J. R. & Fisher, H. L. (2018). Measuring Childhood Maltreatment to Predict Early-Adult Psychopathology: Comparison of Prospective Informant-Reports and Retrospective Self-Reports. *Journal of Psychiatric Research, 96*, 57-64. https://doi.org/10.1016/j.jpsychires.2017.09.020

Nilsen, T. S., Knudsen, G. P., Gervin, K., Brandt, I., Roysamb, E., Tambs, K., Orstavik, R., Lyle, R., Reichborn-Kjennerud, T., Magnus, P., & Harris, J. R. (2013). The Norwegian Twin Registry from A Public Health Perspective: A Research Update. *TwinRes Hum Genet*, *16*(1), 285-295. https://doi.org/10.1017/thg.2012.117

Open Science Collaboration. (2015). Estimating the Reproducibility of Psychological Science. *Science*, *349*(6251). https://doi.org/10.1126/science.aac4716

Pfohl, B., Blum, N., & Zimmerman, M. (1997). *Structured Interview for DSM-IV Personality: Sidp-IV*. American Psychiatric Pub.

Plomin, R., DeFries, J. C., & Loehlin, J. C. (1977). Genotype-Environment Interaction and Correlation in the Analysis of Human Behavior. *Psychological Bulletin*, *84*(2), 309. https://doi.org/10.1037/0033-2909.84.2.309

Reichborn-Kjennerud, T., Czajkowski, N., Ystrøm, E., Ørstavik, R., Aggen, S. H., Tambs, K., ... & Kendler, K. S. (2015). A Longitudinal Twin Study of Borderline and Antisocial Personality Disorder Traits in Early to Middle Adulthood. *Psychological Medicine*, *45*(14), 3121. https://doi.org/10.1017/S0033291715001117

Rosenström, T., Czajkowski, N. O., Ystrom, E., Krueger, R. F., Aggen, S. H., Gillespie, N. A., & Torvik, F. A. (2019). Genetically Informative Mediation Modeling Applied to Stressors and Personality-Disorder Traits in Etiology of Alcohol Use Disorder. *Behavior Genetics*, *49*(1), 11-23. https://doi.org/10.1007/s10519-018-9941-z

Rutter, M. (2007). Proceeding from Observed Correlation to Causal Inference: The Use of Natural Experiments. *Perspectives on Psychological Science*, *2*(4), 377-395. http://dx.doi.org/10.1111/j.1745-6916.2007.00050.x

Sawyer, S. M., Azzopardi, P. S., Wickremarathne, D., & Patton, G. C. (2018). The Age of Adolescence. *The Lancet Child & Adolescent Health*, *2*(3), 223-228. https://doi.org/10.1016/S2352-4642(18)30022-1

Scarr, S., & McCartney, K. (1983). How People Make Their Own Environments: A Theory of Genotype Environment Effects. *Child Development*, *54*(2), 424-435. https://doi.org/10.2307/1129703

Shiner, R. L. (2009). The Development of Personality Disorders: Perspectives From Normal Personality Development in Childhood and Adolescence. *Development and Psychopathology*, *21*(3), 715-734. https://doi.org/10.1017/S0954579409000406

Slutske, W. S., Deutsch, A. R., Richmond-Rakerd, L. S., Chernyavskiy, P., Statham, D. J., & Martin, N. G. (2014). Test of a Potential Causal Influence of Earlier Age of Gambling Initiation on Gambling Involvement and Disorder: A Multilevel Discordant Twin Design. *Psychology of Addictive Behaviors*, *28*(4), 1177. http://doi.org/10.1037/a0035356

Tambs, K., Ronning, T., Prescott, C. A., Kendler, K. S., Reichborn-Kjennerud, T., Torgersen, S., & Harris, J. R. (2009). The Norwegian Institute of Public Health Twin Study of Mental Health: Examining Recruitment and Attrition Bias. *Twin Research and Human Genetics, 12,* 158–168. https://doi.org/10.1375/twin.12.2.158

Tellegen, A., Lykken, D. T., Bouchard, T. J., Wilcox, K. J., Segal, N. L., & Rich, S. (1988). Personality Similarity in Twins Reared Apart and Together. *Journal of Personality and Social Psychology*, *54*(6), 1031. https://doi.org/10.1037/0022-3514.54.6.1031

Torgersen, S. (2000). Genetics of Patients with Borderline Personality Disorder. *Psychiatric Clinics of North America*, *23*(1), 1-9. https://doi.org/10.1016/S0193-953X(05)70139-8

Turkheimer, E., & Waldron, M. (2000). Nonshared Environment: A Theoretical, Methodological, and Quantitative Review. *Psychological Bulletin, 126*(1), 78-108. doi:10.1037/0033-2909.126.1.78. https://doi.org/10.1037/0033-2909.126.1.78

Van Dongen, J., Slagboom, P. E., Draisma, H. H., Martin, N. G., & Boomsma, D. I. (2012). The Continuing Value of Twin Studies in the Omics Era. *Nature Reviews Genetics*, *13*(9), 640-653.

Wingenfeld, K., Schaffrath, C., Rullkoetter, N., Mensebach, C., Schlosser, N., Beblo, T., ... & Meyer, B. (2011). Associations of Childhood Trauma, Trauma in Adulthood and Previous-Year Stress with Psychopathology in Patients with Major Depression and Borderline Personality Aisorder. *Child Abuse & Neglect: The International Journal*, *35*(8), 647-654.

Wolberg, A. R. (1952). The "Borderline" Patient. *American journal of psychotherapy*, *6*(4), 694-710.

Young, A. I., Benonisdottir, S., Przeworski, M., & Kong, A. (2019). Deconstructing the sources of genotype-phenotype associations in humans. *Science*, *365*(6460), 1396-1400. https://doi.org/10.1126/science.aax3710