Borderline Personality Disorder Traits in Early Adulthood: Development, Causes and Predictors

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Submitted for the degree of PhD at the
Department of Psychology, Faculty of Social Sciences, University of Oslo

2022

Series of dissertations submitted to the
Faculty of Social Sciences, University of Oslo
No. 932

ISSN 1504-3991

Cover: UiO.
Print production: Graphics Center, University of Oslo.

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Acknowledgements

I would like to express my deepest gratitude for the guidance provided by my supervisors, Nikolai Czajkowski, Svenn Torgersen, and Trine Waaktaar. First, I want to express my sincerest appreciation to Svenn. Your knowledge of psychopathology, personality psychology and genetics has given rise to numerous interesting conversations, which have been of crucial help when writing the papers. Your tremendous interest in the field of psychology has inspired me. Thank you also for all the emotional support you have given throughout this period, and for dedicating so much of your time to this project. I would also like to extend my deepest gratitude to Nikolai. Your expertise in statistics and twin methodology has been essential for carrying out this project. Thank you so much for sharing your knowledge and for all the help with statistical modelling. Last but not least, Trine, thank you so much for providing constructive feedback on the papers in this thesis. I am also very grateful for your valuable guidance in the research process. Your continuous positivity and drive inspire me.

I would also like to express my thankfulness to my fellow PhD student and close friend, Live Skow Hofgaard. Beyond our frequent conversations about behavior genetics, your continuous emotional backing has been extremely valuable. Thank you for sharing frustrations and laughs with me.

Outside the research arena, I would like to thank my family and friends for always cheering for me. Special thanks to my twin sister, Sigrid, and my mother, Unni. Your encouraging words have meant so much to me throughout this process. Finally, my deepest appreciation goes to my boyfriend, Pål-Anders. Thank you for always patiently listening to my lengthy ramblings, for your encouragement and continuous support during this period. Your belief in me has kept my motivation high during this process.

Summary

Borderline personality disorder (BPD) was classified as a diagnosis in the Diagnostic and Statistical Manual of Mental Disorders, Third edition (DSM-III) in 1980. Since then, the causes of BPD have been extensively theorized and studied. For a long time, its etiology was assumed to be exclusively environmental. Especially traumatic experiences in childhood and young age have long been considered important environmental risk factors for BPD. From the early 2000s, genetically informative studies have revealed that also genes are important in the development of BPD. A complication has been that what we have considered and measured as environmental factors, such as life events, are in fact partly influenced by genes. This finding has important implications for our understanding of the causes of BPD, as almost everything we know about its development comes from pure association studies that do not control for the potential confounding effects of shared genetic influences between potential risk factors and BPD.

By using data from a Norwegian population-based twin sample, the overreaching aim of this thesis was to enhance the knowledge about the development of BPD traits in early adulthood. More specifically, we examined whether childhood trauma and reported life events throughout adolescence have direct effects on levels of BPD traits in early adulthood, after controlling for shared environmental and genetic influences. Beyond studying the causes of BPD, we examined whether levels of sense of coherence (SOC) and feelings of loneliness in adolescence predicted levels of BPD traits in early adulthood. Not much is known about possible precursors of BPD in adolescence before personality disorders usually are diagnosed. To enhance the knowledge about the causal architecture behind these possible predictors of BPD traits (i.e., SOC and loneliness), we also examined their heritability and stability, and examined whether life events influenced levels of these characteristics in adolescence.

The findings presented in this thesis suggest that childhood trauma (i.e., emotional abuse, physical abuse, sexual abuse, and witnessing violence) and negative life events in adolescence are associated with BPD traits mainly due to common genetic influences. That is, these measured environments during childhood and adolescence do not seem to have any causal effects on levels of BPD traits in early adulthood. With respect to SOC and loneliness, we found these constructs to be moderately heritable. In addition, both SOC and loneliness showed trait-like stability in adolescence similar to what is found for personality traits in general. Reported life events did not predict levels of SOC or loneliness throughout adolescence. Rather, life events were correlated with SOC and loneliness mainly for genetic reasons. Furthermore, lower levels of SOC and feelings of loneliness already at the age of 12 years were associated with increased levels of BPD traits in early adulthood. The prediction of SOC and loneliness on BPD traits increased in strength later in adolescence with also shorter time before the assessment of BPD traits. Findings from genetically informative analyses showed that the associations were mainly attributable to shared genetic influences. Together, these findings indicate that low levels of SOC and feelings of loneliness may be important indicators of later development of BPD due to their common genetic influences.

List of Papers

Paper I

Skaug, E., Czajkowski, N. O., Waaktaar, T., & Torgersen, S. (2022). Childhood trauma and borderline personality disorder traits: A discordant twin study. *Journal of Psychopathology and Clinical Science*, 131(4), 365-374. https://doi.org/10.1037/abn0000755

Paper II

Skaug, E., Czajkowski, N. O., Waaktaar, T., & Torgersen, S. (2022). The relationship between life events and sense of coherence in adolescence. A longitudinal twin study. *Journal of Research in Personality, 99,* Article 104259. https://doi.org/10.1016/j.jrp.2022.104259

Paper III

Skaug, E., Czajkowski, N. O., Waaktaar, T., & Torgersen, S. (2022). *The longitudinal relationship between life events and loneliness in adolescence: A twin study.* [Manuscript submitted for publication]. Department of Psychology, University of Oslo.

Paper IV

Skaug, E., Czajkowski, N. O., Waaktaar, T., & Torgersen, S. (2022). *The role of sense of coherence and loneliness in borderline personality disorder traits: A longitudinal twin study.* [Manuscript submitted for publication]. Department of Psychology, University of Oslo.

Aims of the Thesis

Borderline personality disorder (BPD) is characterized by intense and unstable emotions, impulsivity, an unstable identity, and problems with interpersonal relationships (American Psychiatric Association, 2013). Identifying environmental factors that influence the development of BPD has long been an important aim in research on this complex and severe personality disorder. Traumatic experiences in childhood have received most empirical attention, and such experiences have been proposed to be important factors in the etiology of BPD (Ball & Links, 2009; Kaess, 2020). In addition, genetically informative studies have shown that BPD has a substantial genetic basis (Gunderson, Herpertz, Skodol, Torgersen, & Zanarini, 2018). Unfortunately, much of what we know about potential environmental risk factors stem from studies that do not control for shared genetic influences between possible risk factors and BPD (Porter et al., 2020; Winsper et al., 2016). This makes it difficult to draw causal conclusions, because measures of the environment are shown to be partly heritable (Kendler & Baker, 2007). Finding genetic influences on measured environments challenges the commonsense assumption that life experiences have unidirectional effects from environment to person, and highlights the importance of controlling for genes as potential confounding variables in the association between environmental exposures and BPD traits. Today, there exist very few genetically informative studies on this research area. Although the risk of BPD is assumed to be multifactorial resulting from a combination of genetic influences and the influence of life experiences, its etiology is still unclear (Bassir Nia et al., 2018; Bohus et al., 2021; Gunderson et al., 2018).

Causal inference is of central importance in order to advance our understanding of the development of BPD and not least to guide prevention efforts. Therefore, it is crucial to examine whether factors that are believed to cause this personality disorder really do increase the risk for developing BPD. Furthermore, numerous studies have shown that BPD usually

has its onset in adolescence (Sharp, Chanen, & Cavelti, 2021). However, there is a lack of robust evidence regarding precursor signs of BPD (Bohus et al., 2021). Identifying warning signals for later development of BPD are crucial to facilitate early detection, prevention, and intervention.

The aim of this thesis was to contribute to a better understanding of the development of BPD by (1) examining its causes and (2) examining whether two central but underexplored features of the disorder, i.e., low sense of coherence (SOC) and feelings of loneliness, already from early adolescence predict BPD traits in the beginning of adulthood.

Regarding the first aim, we examined the causes of BPD traits in early adulthood by applying two different approaches available within the classical twin design: by quantifying the relative contribution of genetic and environmental influences on individual differences in BPD traits, and by including childhood trauma and life events (i.e., measured environments) in the analyses. The first approach, by using a univariate twin model, allows us to partition the total variance in BPD traits into its genetic and environmental variance components. However, a variance decomposition does not inform us about which environmental factors underly individual differences in BPD traits. Therefore, we also examined whether childhood trauma and life events throughout adolescence could explain some of the environmental variance in BPD traits. More specifically, we examined whether these measured environments have direct effects on levels on BPD traits, after controlling for the potential confounding effects of shared environmental and genetic influences.

With respect to the second aim, we examined whether BPD traits in early adulthood could be predicted from levels of SOC and loneliness throughout adolescence. By using a genetically informative sample, we were also able to examine to what degree the predictive ability of SOC and loneliness on BPD traits could be attributed to a causal effect of the

predictors or stem from shared genetic influences. Identity disturbance and problems with interpersonal relationships are core features of BPD (Gunderson et al., 2018). However, not much is known about whether these indicators of BPD can be observed already in early adolescence before a diagnosis of BPD usually is set. Identity disturbance involves problems in understanding oneself, having distrust in one's capacity to manage challenges, and having problems finding meaning in life (Neacsiu, Herr, Fang, Rodriguez, & Rosenthal, 2015). The concept of SOC is thus closely related to identity, as it reflects the degree to which one is perceiving oneself and the world as comprehensible, manageable, and meaningful (Antonovsky, 1990). Furthermore, the consequences of problems with relations to other people may be a perceived feeling of loneliness (Hauschild et al., 2018; C. E. Miller, Townsend, & Grenyer, 2021). Thus, SOC and loneliness may be central features of BPD beyond what we can read directly from the diagnostic criteria. Conceptually, these two constructs are also closely linked to problems related to self and interpersonal functioning, which are considered the core pathology of personality disorders in the alternative model for personality disorders in DSM-5 (American Psychiatric Association, 2013).

Basic Concepts in the Thesis

Sense of Coherence

The theory of SOC was introduced by Aaron Antonovsky in the late 1970's, aiming to explain the relationship between stressors, coping, and health (Antonovsky, 1979). Antonovsky emphasized that all people have to face various stressors in the course of living, and that how we cope with these stressors will directly influence our health. Furthermore, he emphasized that we cannot successfully cope with a stressor or problem unless we (1) understand the character of the problem, (2) believe that we have the recourses to cope with the situation, and (3) find it meaningful to cope with the challenges in question (Antonovsky, 1990). These coping resources were named SOC, reflecting the extent to which one is

perceiving the world as (1) *comprehensible*, (2) *manageable*, and (3) *meaningful* (Antonovsky, 1987). Supporting Antonovsky's assumption of a relationship between SOC and health, numerous studies have found that SOC is associated with both mental health and quality of life. This is summarized in review papers of both adult (Eriksson & Lindström, 2006, 2007) and adolescent (Länsimies, Pietilä, Hietasola-Husu, & Kangasniemi, 2017) samples.

With respect to the development of SOC, Antonovsky stated that SOC develops throughout childhood and adolescence, and becomes stabilized by the end of young adulthood (Antonovsky, 1987). He pointed to three types of life experiences that were assumed to promote the development of SOC. First, consistency and structure in the rearing environment were believed to facilitate a perception of the world as comprehensible. Second, a balance between demands in life and available resources was assumed to contribute to the development of the manageability component. Third, active participation in shaping one's life was believed to contribute to viewing the world as meaningful (Antonovsky, 1987).

Beyond these theoretical assumptions regarding the development of SOC, not much is known empirically about which factors are influencing its development (Mittelmark et al., 2017; Rivera, Garcia-Moya, Moreno, & Ramos, 2013). Studies on adolescent populations have found that negative life events both in the family (such as parental divorce, parental illness, and family conflict) and in the school context (such as peer pressure and pressure of school work) are associated with lower levels of SOC (Marsh, Clinkinbeard, Thomas, & Evans, 2007; Moksnes, Rannestad, Byrne, & Espnes, 2011; Natvig, Hanestad, & Samdal, 2006; Ristkari, Sourander, Rønning, Nikolakaros, & Helenius, 2008). Positive events, on the other hand, such as social support and family closeness, have been associated with higher levels of SOC (Marsh et al., 2007; Natvig et al., 2006; Olsson, Hansson, Lundblad, & Cederblad, 2006). However, the cross-sectional nature of existing studies makes conclusions

regarding direction of effect challenging. Furthermore, although Antonovsky argued that a person's SOC is determined solely by experiences in childhood and adolescence, genetically informative studies have demonstrated that SOC also has a genetic basis. To my knowledge, only two studies have investigated the heritability of SOC. In a study by Hansson et al. (2008), the heritability of SOC was estimated to 35%, while Silventoinen et al. (2014) reported a heritability of 45%.

Loneliness

Humans have fundamental needs for social connectedness and belonging, and people may feel lonely when these needs are not being met (Baumeister & Leary, 1995). Definitions of loneliness emphasize that loneliness is a subjective feeling that is different from being physically alone. The most widely cited definition of loneliness was provided by Letitia Anne Peplau and Daniel Perlman. They defined loneliness as a negative emotional response to a perceived discrepancy between the desired and the experienced quantity and/or quality of social relationships (Peplau & Perlman, 1982). More specifically, loneliness is the unpleasant feeling that follows a situation where the quantity of interpersonal relationships is smaller than one desires, or a situation in which the desired quality (e.g., emotional support) of existing relationships is not met. This is distinguishable from objective social isolation. That is, being alone in an objective sense do not necessarily make people lonely, and people may feel lonely even when surrounded by a big social network (de Jong Gierveld, van Tilburd, & Dykstra, 2018).

Whereas there is a consensus in the research literature that loneliness is a subjective experience, a debate exists on whether loneliness should be conceptualized as a unidimensional or multidimensional construct (Yang, 2019). Different conceptualizations of loneliness are reflected in how researchers are measuring loneliness. Most empirical studies measure loneliness as a unidimensional construct. For example, a commonly used measure of

loneliness is to ask the participants to respond to the single item ‘I feel lonely’. However, the use of direct questions to measure loneliness has been criticized because this approach may result in underreporting due to the social stigma attached to loneliness, and because single indicators generally have lower reliability than measures consisting of multiple items (de Jong Gierveld et al., 2018; Yang, 2019). Therefore, multiple-item scales that measure loneliness indirectly (i.e., avoiding the words ‘lonely’ or ‘loneliness’ and rather include words like ‘left out’ or ‘not close to anyone’) are thought to be better measures of loneliness than a single-item measurement. The most widely used loneliness scales in the research literature, which also measure loneliness as a unidimensional construct, are versions of the UCLA (University of California, Los Angeles) Loneliness Scale (Russell, 1996; Russell, Peplau, & Cutrona, 1980; Russell, Peplau, & Ferguson, 1978). This scale was also employed in the present study.

Loneliness has been associated with many negative health outcomes, such as poor physical health, depression, anxiety, personality disorders, low quality of life, suicidal ideation and suicidal behavior (Erzen & Çikrikci, 2018; Hawkley & Cacioppo, 2010; Maes et al., 2019; McClelland, Evans, Nowland, Ferguson, & O'Connor, 2020; Park et al., 2020; Wang, Mann, Lloyd-Evans, Ma, & Johnson, 2018). Studies indicate that the effects of loneliness on health are complex and that it is likely that the effects are bidirectional. That is, loneliness may contribute to poor health, and poor health may contribute to greater feelings of loneliness (de Jong Gierveld et al., 2018; Lim, Eres, & Vasan, 2020; Maes et al., 2019; Park et al., 2020; Wang et al., 2018).

With respect to predictors of loneliness in childhood and adolescence, life events that cause changes in a person’s social network (e.g., ending of a friendship or change of schools) have theoretically been emphasized as potential causes of loneliness (Peplau & Perlman, 1979). In addition, life events may also be related to loneliness more indirectly. For example,

arguments with parents or peers may give rise to feelings of not being emotionally supported and thus to feelings of loneliness. Weeks and Asher (2012) have provided an overview of the empirical knowledge about predictors of loneliness in childhood and adolescence. Their review showed that peer rejection, not having friends, low friendship quality (e.g., not feeling supported), and peer victimization (e.g., being called mean names) have all been associated with loneliness in childhood and adolescence. In line with this, more recent studies have also found that not having close friends, feeling disliked, and being bullied are associated with greater feelings of loneliness in adolescence and young adulthood (Rönkä, Rautio, Koiranen, Sunnari, & Taanila, 2014; von Soest, Luhmann, & Gerstorf, 2020). Beyond these peer-relationship experiences, family related experiences such as multiple residence moves, multiple changes of school, parental divorce (Lasgaard, Armour, Bramsen, & Goossens, 2016) and low levels of parental care (von Soest et al., 2020) have also been associated with loneliness in adolescence. To summarize, associations between a number of different life experiences and loneliness in childhood and adolescence have been demonstrated. However, most studies are cross-sectional. This makes it challenging to draw causal inferences because the direction of effect is difficult to assess.

In addition to these potential risk factors, individual differences in levels of loneliness are also associated with personality characteristics. That is, the lonely person tends to be more introverted and neurotic, and a bit less agreeable and conscientious than the less lonely person (Buecker, Maes, Denissen, & Luhmann, 2020). Individual differences in loneliness are also shown to be substantially influenced by genetic factors, i.e., studies have reported heritability estimates between 37% and 55% (Goossens et al., 2015).

Life Events

Research on the associations between life events and mental health started in the 1960s with the introduction of the Social Readjustment Rating Scale (Holmes & Rahe, 1967).

This scale included a checklist of 43 events, such as ‘death of a close family member’, ‘major change in financial state’, and ‘troubles with the boss’. Since the publication of Holmes and Rahe’s scale, several life events checklists have been developed. A large number of studies have reported associations between stressful life events and mental disorders, with most studies focusing on depression and post-traumatic stress disorder (Cohen, Murphy, Prather, Murphy, & Prather, 2019; Hammen, 2016). Of note, terms like ‘stressful life events’, ‘negative life events’, and ‘adverse life events’ are often used interchangeably.

Most of the empirical knowledge about the associations between stressful life events and health comes from research that have used life events scales that count the number of stressful life events a person has experienced within a defined time period, usually within the past year (Cohen et al., 2019). That is, the occurrence of life events is used as indicators of life stress rather than subjective judgements of how stressful an environmental exposure was for the individual. In addition, the number of life events are usually summed up to an aggregated score, based on the assumption that the effects of additional life events are cumulative. Common to most checklists is that they cover a range of life events assumed to be representative for the sample studied (Monroe, 2008). For example, checklists developed for children and adolescents include events that aim to cover a representative list of possible events that may be experienced in this period of life (K. E. Grant, Compas, Thurm, McMahon, & Gipson, 2004).

Childhood Trauma

The first instruments designed to measure childhood trauma were developed in the 1980’s (Roy & Perry, 2004). Since then, research on the effects of traumatic experiences on mental health outcomes have expanded exponentially. Empirical findings on the consequences of childhood trauma are summed up in numerous meta-analyses and systematic reviews, reporting strong associations between childhood trauma and a broad range of mental

health outcomes, such as mood disorders, anxiety disorders, substance use disorders, psychosis, schizophrenia, and personality disorders (e.g., Carr, Martins, Stingel, Lemgruber, & Juruena, 2013; Kaufman & Torbey, 2019; Li, D'Arcy, & Meng, 2016; Lindert et al., 2013; Nanni, Uher, & Danese, 2012; Nelson, Klumparendt, Doebler, & Ehring, 2017; Norman et al., 2012; Read, van Os, Morrison, & Ross, 2005; C. J. Rogers et al., 2022). Within the trauma research tradition, the main subtypes of trauma studied include experiences with abuse and neglect (i.e., emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect), witnessing violence and separation from caregivers. Sexual abuse is the most frequently studied type of trauma (Carr et al., 2013), and BPD is probably the diagnostic group that has received most empirical attention when it comes to research on the effects of traumatic experiences in childhood (Berenz et al., 2013).

The earliest studies that examined the effects of childhood trauma on mental health outcomes often focused on single forms of trauma, usually sexual abuse or physical abuse (Bernstein et al., 2003). Although it has long been recognized that maltreated children often experience multiple types of maltreatment (Higgins & McCabe, 2001), most studies have continued to focus on one or a few subtypes of traumatic experiences (Charak, Tromp, & Koot, 2018). Importantly, if one is studying a single type of trauma and does not account for the presence of other co-occurring subtypes, this may lead to a biased picture of the effect of the trauma type studied (Herrenkohl & Herrenkohl, 2009). More recently, there has been an increased focus on studying multiple types of trauma simultaneously in order to examine the independent effect of a particular type of trauma, controlled for other co-existing subtypes (e.g., Charak et al., 2018; Lobbestael, Arntz, & Bernstein, 2010). However, studies differ with respect to which subtypes of trauma that show the greatest independent effect on various mental health outcomes. This may be because there is no consensus in the research literature on how to define and classify abuse and neglect. In addition, studies differ with respect to the

covariates that are included, and results may also depend on the type of sample that is being studied (e.g., clinical vs. nonclinical).

Borderline Personality Disorder

BPD as a theoretical and clinical psychiatric entity has its origin within psychoanalysis in the 1930s (Stern, 1938), but was not classified as a mental disorder until 1980, in the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III; American Psychiatric Association, 1980), and about 10 years later in the International Classification of Diseases, Tenth Revision (ICD-10; World Health Organization, 1992). BPD is characterized by a pervasive pattern of intense and unstable interpersonal relations, an unstable identity or sense of self, intense and rapidly changing emotions, impulsive behaviors, and self-harming behavior (American Psychiatric Association, 2013). Table 1 presents the DSM-5 criteria for BPD (American Psychiatric Association, 2013). To receive a BPD diagnosis, at least five of nine criteria must be present.

Table 1

DSM-5 Criteria for Borderline Personality Disorder

1	Frantic efforts to avoid real or imagined abandonment [a]
2	A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation
3	Identity disturbance: markedly and persistently unstable self-image or sense of self
4	Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating) [a]
5	Recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior
6	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
7	Chronic feelings of emptiness
8	Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights)
9	Transient, stress-related paranoid ideation or severe dissociative symptoms.

Note. [a] Does not include suicidal or self-mutilating behavior covered in criterion 5.

In DSM-5, personality disorders are diagnosed based on a categorical approach (i.e., one receives the diagnosis or not). The limitations of a categorical classification of personality disorders are acknowledged in the DSM manual itself, and an alternative model for personality disorders is presented in Section III of the DSM-5 manual. This alternative model is based on a more dimensional approach which place personality pathology on a continuum of severity (American Psychiatric Association, 2013). A lengthy discussion regarding the limitations of a categorical approach to personality disorders is beyond the scope of this thesis. However, relevant for this thesis (as we studied BPD dimensionally) are the boundaries between pathology and normality. The categorical approach has been criticized because the boundaries between pathology and normality is determined based on arbitrary thresholds for diagnosis (Morey, Benson, Busch, & Skodol, 2015; Widiger & Trull, 2007). The critics argue that personality disorders are better represented as dimensional constructs. With a dimensional approach, we are not talking about qualitative differences between people in which individuals differ from each other in 'kind' but rather view differences between people as quantitative variations along a continuum representing differences in 'degree'. Supporting this notion, a meta-analysis concluded that psychological differences between people, including differences in psychopathology, reflects quantitative rather than qualitative differences (Haslam, McGrath, Viechtbauer, & Kuppens, 2020). Empirical studies on BPD in particular also suggest that BPD is best conceptualized as a dimensional construct (Edens, Marcus, & Ruiz, 2008; Torgersen et al., 2008; Trull, Distel, & Carpenter, 2010).

Numerous studies have shown that BPD usually has its onset in adolescence (Chanen, 2015; Paris, 2014; Shiner, 2009). This is also emphasized in the definition of personality disorders in DSM-5 (American Psychiatric Association, 2013). However, diagnosing BPD before age 18 has been controversial. A reason for this may be that the BPD diagnosis is

associated with stigmatization, both by the person itself (Rüsch et al., 2006) and by the society at large (Gunderson et al., 2018). Furthermore, there has been a commonly held belief among clinicians that personality is still in flux in adolescence, calling into question the validity of a BPD diagnosis before adulthood (Kaess, Brunner, & Chanen, 2014). However, empirical studies have demonstrated that a diagnosis of BPD in adolescence shows similar reliability, validity, and stability as BPD in adulthood (Chanen, Jovev, McCutcheon, Jackson, & McGorry, 2008; Kaess et al., 2014; A. L. Miller, Muehlenkamp, & Jacobson, 2008; Winsper et al., 2016).

Persons with BPD typically alternate between overinvolvement with and social withdrawal from other people, they may show dramatic shifts in their perception of others, and tend to idealize the persons meeting their needs and to devaluate the same persons when they feel disappointed or overlooked (Gunderson et al., 2018). In addition, they may have difficulties with interpreting other persons' feelings, and tend to perceive them as negatively directed toward themselves as they are hypersensitive to social rejection. Furthermore, an unstable sense of self and difficulties with connecting with their own emotional experiences may lead to a chronic feeling of emptiness. An unstable sense of self may also lead to dramatic shifts in values, in plans for the future, and in sexual identity. Furthermore, intense and fluctuating emotions may be associated with anger and anxiety, especially in social situations, in addition to impulsive behaviors (Gunderson et al., 2018). Self-harm and suicidal behavior are among the most severe symptoms of BPD. A review of studies that have investigated self-harming behavior in persons with BPD reported that the prevalence rate of non-suicidal self-injury was around 17% in adolescent samples of the general population, whereas a prevalence rate around 95% has been reported in adolescent clinical samples (Reichl & Kaess, 2021).

In addition, BPD often co-occurs with other mental disorders, which further complicates the clinical picture, and leads to greater suffering and behavioral difficulties. For instance, studies of both adult (Asherson et al., 2014; Fornaro et al., 2016; B. F. Grant et al., 2008; Pagura et al., 2010; Tate et al., 2022; Tomko, Trull, Wood, & Sher, 2014; Trull et al., 2018) and adolescent (Chanen, Jovev, & Jackson, 2007; Kaess et al., 2013; Loas et al., 2013; Winsper et al., 2016) samples have shown that BPD is associated with high rates of comorbidity with other mental disorders, such as major depressive disorder, bipolar disorders, psychotic disorders, anxiety disorders, post-traumatic stress disorder, substance use disorders, eating disorders, attention deficit hyperactivity disorder, and other personality disorders. Several physical health conditions are also associated with BPD, such as hypertension, cardiovascular disorders, obesity, diabetes, and epilepsy (El-Gabalawy, Katz, & Sareen, 2010; Tate et al., 2022).

BPD is also associated with severe impairments in functioning. Studies of patients in both adult (Skodol et al., 2002) and adolescent (Chanen et al., 2007; Kaess et al., 2013) samples have shown that BPD is associated with severe impairments in functioning across a broad range of domains (e.g., school, work, interpersonal relationships). Studies of the general population have also shown that BPD is associated with severe impairments in functioning, such as poor social functioning (Tomko et al., 2014), poor work performance (Juurlink et al., 2018), disability pensioning (Østby et al., 2014), and impaired quality of life (Cramer, Torgersen, & Kringlen, 2006). Although most persons with BPD will achieve symptomatic remission over time, follow-up studies of adult samples have found that impairments in functioning persist for decades even after symptomatic remission (Alvarez-Tomás et al., 2017; Gunderson et al., 2011; Ng, Bourke, & Grenyer, 2016; Zanarini, Temes, Frankenburg, Reich, & Fitzmaurice, 2018). Also, longitudinal studies on adolescent samples indicate that BPD symptoms in this age period predict long-term impairments in functioning

(Winograd, Cohen, & Chen, 2008; Winsper et al., 2015). Together, these findings highlight the importance of early detection and intervention of this severe and complex disorder.

The first studies examining the etiology of BPD were published in the 1970's (Zanarini, 2000). Inspired by psychodynamic theories, the first studies typically focused on parental separation or loss, and disturbed parental involvement as causal factors. From the late 1980's, empirical studies shifted the focus to examine a range of pathological childhood experiences, especially childhood abuse (e.g., Herman, Perry, & Van Der Kolk, 1989). Since then, researchers have been concerned with childhood trauma (i.e., especially experiences with sexual abuse, emotional abuse, physical abuse, emotional neglect, and physical neglect) as causative factors in the development of BPD (Ball & Links, 2009; Newnham & Janca, 2014). The extensive research on the association between childhood trauma and BPD are summarized in recent systematic reviews and meta-analyses of BPD features in childhood (Ibrahim, Cosgrave, & Woolgar, 2018), BPD in adolescence (Winsper et al., 2016), and BPD in adulthood (Porter et al., 2020), all finding high prevalence of reported childhood trauma among persons with BPD. Studies have also found a dose-response relationship between traumatic experiences in childhood and number of BPD symptoms (Charak et al., 2018; Hengartner, Ajdacic-Gross, Rodgers, Müller, & Rössler, 2013; Pietrek, Elbert, Weierstall, Müller, & Rockstroh, 2013; Zanarini et al., 2002). More specifically, these studies have found that more severe trauma and experiencing multiple types of trauma are associated with number of BPD symptoms. Although sexual abuse is commonly thought to be the most important risk factor in the etiology of BPD, several studies have shown that other types of abuse and neglect may be just as important or even more important for developing BPD (Charak et al., 2018; Johnson, Cohen, Brown, Smailes, & Bernstein, 1999; Lobbestael et al., 2010; Zanarini et al., 2019). Together, all these findings have contributed to the commonly accepted assumption of a causal relationship between childhood trauma and BPD.

The development of BPD was long considered to be exclusively caused by adverse childhood experiences. However, from the early 2000, we have known that BPD also has a substantial genetic basis (Gunderson et al., 2018; Torgersen et al., 2000). Twin studies have demonstrated that the heritability of BPD is high, with genetic influences explaining around 70% of individual differences in BPD traits when BPD is measured longitudinally or with approaches that combine different measurement methods (Bornovalova, Hicks, Iacono, & McGue, 2009; Reichborn-Kjennerud et al., 2015; Torgersen et al., 2012). Such designs aim to reduce measurement error, which would lead to an overestimation of the nonshared environmental influences and consequently to an underestimation of the genetic influences.

Unfortunately, much of what we know about the relationship between childhood trauma and BPD is derived from pure association studies. Commonsense reasoning would suggest that life events, whether stressful life events or childhood trauma, have unidirectional effects from environment to person. However, genetically informative studies have shown that measures of the environment such as life events are partly influenced by genetic factors (Bemmels, Burt, Legrand, Iacono, & McGue, 2008; Kendler & Baker, 2007). Finding genetic influences on environmental measures imply that individuals' genetically influenced behaviors play a role in their choice of environments or elicit certain reactions from the environment. For example, if individuals actively select environments related to their genetic predisposition (e.g., the outgoing person who actively seek new experiences), genetic differences between people would explain some of the variability of environments people finds themselves in, leading to genetic variance in measures of the environment. This is referred to as gene-environment correlations (Plomin, DeFries, & Loehlin, 1977).

Finding genetic influences on measured environments opens the possibility that the observed associations between childhood trauma and BPD found in numerous studies, stem from shared genetic influences rather than being a causal effect of the environmental

exposure on BPD. To date, genetically informative studies on the association between childhood trauma and BPD are scarce. However, existing twin studies indicate that childhood trauma does not seem to have causal effects on BPD traits (Berenz et al., 2013; Bornovalova et al., 2013). Bornovalova et al. (2013) found that the observed relationship between childhood abuse (i.e., emotional, physical, and sexual) and BPD traits was accounted for by shared genetic influences. This study used a self-report questionnaire to measure BPD traits, which is generally assumed to have lower validity compared to structured interviews (Vrshek-Schallhorn et al., 2014). Furthermore, the study by Berenz et al. (2013) did not have sufficient power to differentiate between confounding due to genetic and shared environmental factors, nor differentiate between trauma types. Although these studies provide preliminary evidence for a non-causal relationship between childhood trauma and BPD traits, more genetically informative studies are needed to validate the results derived from these studies. Above all, the findings highlight the importance of using genetically informative studies, which are able to separate the environmental effect of an exposure from the potentially confounding effects of shared genetic factors, when looking for environmental factors that contribute to the development of BPD.

Behavior Genetics

Behavior genetics, the study of genetic and environmental influences on individual differences, dates back to the work by Sir Francis Galton in the late 19th century (Neale & Maes, 2004). Galton was the first to use twins in genetic research and his work has given rise to many of the statistical methods that are in use today. In 1918, Ronald Fisher published a paper showing that Mendelian laws of inheritance also applies to human traits that is influenced by a large number of genes (Fisher, 1918). Fisher's work provided the basis for what is now known as the polygenic model, a fundamental principle in behavior genetics

which assumes that individual differences in complex human traits[1] are caused by a large number of genes (Neale & Maes, 2004). However, the view that the environment is the key factor in determining who we are, dominated the field of psychology for decades. From the 1960s, there was a major growth in twin and adoption studies showing that genetic factors contribute substantially to individual differences, including differences in psychopathology (Rutter, Moffitt, & Caspi, 2006). Since then, the field of quantitative behavior genetics has developed exponentially. A variety of different methodological approaches have been developed for quantitative genetic analyses, such as twin studies, family studies, adoption studies, linkage studies, and association studies (Posthuma et al., 2003). This thesis will focus on the classical twin design, which includes monozygotic (MZ) and dizygotic (DZ) twins reared together (Boomsma, Busjahn, & Peltonen, 2002).

Sources of Variance

The goal of quantitative behavior genetics is to understand individual differences in human characteristics. Individual differences in a characteristic of interest may be quantified as variance around the mean in the sample under study. This observed variance is often referred to as the 'phenotypic variance'. Quantitative genetic methods are concerned with understanding differences between people by partitioning this phenotypic variance into proportions due to genetic and environmental influences.

Twin studies typically distinguish between two types of genetic influences: additive and non-additive. Additive genetic influences (A) refer to the effect of multiple genes that together operate in an additive manner. Non-additive genetic influences (D), on the other hand, refer to interactive genetic effects. There are two main types of non-additivity:

[1] Most human traits (e.g., personality traits) are characterized by a continuous distribution and are influenced by multiple genes and environmental influences. Therefore, they are often referred to as quantitative or complex human traits.

dominance (i.e., interaction between alleles at the same locus) and epistasis (i.e., interaction between alleles across different loci). When non-additivity is considered in twin studies, this often refers to dominance because epistatic genetic effects are impossible to identify unless we are studying a trait influenced by a small number of known genes (Neale & Maes, 2004). The importance of genes is often quantified as a proportion relative to all factors influencing individual differences in a trait (i.e., both genetic and environmental). This proportion of the total phenotypic variance accounted for by genetic influences is termed 'heritability'. The heritability of a trait thus quantifies how much of the differences between individuals are accounted for by differences in genes. This is an important point: the heritability of a trait relates to *individual differences* in a particular sample. Thus, heritability does not quantify the extent to which genetic factors influence a trait in a given individual. For example, if the heritability of a trait is 50%, this indicates that 50% of differences between individuals in the trait in the sample under study are due to genetic differences between them. A useful sentence to have in mind when thinking about heritability is that the heritability describes *what is*, not *what could be* (Plomin, 2018). The heritability is like a 'status quo' in a particular sample at a particular time. That is, if you studied a different population or the same population at a different time, the proportion of genetic and environmental influences could be different. The term 'broad-sense heritability' is used when both A and D effects are estimated, whereas the term 'narrow-sense heritability' is used when only A effects are estimated.

Twin studies also separate the environmental influences into two different sources of variance: shared and nonshared. Shared environmental influences (C) refer to any environmental factors contributing to similarity among family members. For example, if the socioeconomic status in a family makes family members similar, this will lead to a C effect in twin studies. Nonshared environmental influences (E), on the other hand, refer to any factors contributing to phenotypic dissimilarity between family members, including measurement

error. Like heritability, the environmental component is also a quantification of the importance of environmental influences in a particular sample at a particular time. For example, in an extreme scenario in which genetic influences explained all variance in a trait, this does not mean that environmental influences *could* not make a difference. Let us imagine that we knew that the only factors contributing to differences in a particular disorder are genes and childhood abuse. For this example, we assume that exposure to childhood abuse is purely environmental in nature and that there is no measurement error in the measured variables. In a sample where none of the participants have experienced childhood abuse, the heritability of the disorder would be 100%. That is, differences in genes would be the only source creating individual differences in the disorder because none of the participants have experienced childhood abuse. In another sample, where some participants have experienced childhood abuse, both genetic and environmental factors (i.e., childhood abuse) would influence individual differences in the disorder.

Decades of research have consistently shown that individual differences in human traits can be attributed to additive genetic and nonshared environmental influences, with negligible influences from the shared environment (Polderman et al., 2015). This is often interpreted as the family environment is without importance in influencing who we are and how we develop. However, a non-existent shared environmental influence does not necessarily mean that experiences objectively shared within a family are without importance, it suggests that growing up in the same family does not make family members similar. As described above, the shared environmental influences refer to experiences making family members similar whereas the nonshared environmental influences refer to any factors making family members dissimilar. Thus, it is important to keep in mind that the environmental variance components refer to the 'effects' of the environment. For example, if environmental

experiences objectively shared by the twins, such as parenting style, do not have equal effects on both twins, they will be included in the estimate of the nonshared environment.

Logic of the Classical Twin Design

Twin studies represent a way of studying sources of variance and covariance without directly measuring specific genes or environments. The classical twin design utilizes the different degree of genetic relationship between MZ and DZ twins reared together to quantify to what extent genetic and environmental influences contribute to the variance within a phenotype or to the covariance between phenotypes. This approach relies on comparing the phenotypic similarity between MZ and DZ twins, usually quantified by comparing the correlation within MZ pairs with the correlation within DZ pairs. Twins reared together share the same family environment, MZ twins are genetically identical whereas DZ twins share, on average, half of their segregating genes. That is, both the A and D effects are correlated at unity among MZ pairs. Within DZ pairs, on the other hand, the A and D effects are expected to correlate at 0.50 and 0.25, respectively. Finally, the C effects are expected to correlate at unity for both zygosity groups.

Any similarity among both MZ and DZ twins must be attributed to the fact that they share genes and/or because they share common environments. Because DZ twins only share half of their segregating genes, the resemblance due to genetic influences will be lower in DZ pairs compared to MZ pairs. Thus, genetic influences are inferred when the MZ correlation is higher than the DZ correlation. If influence of A was the only source to familial resemblance, one would expect that the DZ correlation is half the size of the MZ correlation. If the DZ correlation exceeds half the size of the MZ correlation, influence of C is inferred. However, if the DZ correlation is less than half of the MZ correlation, influence of D is inferred. Of note, D and C effects cannot be estimated simultaneously because they are confounded in the classical twin design (Martin, Eaves, Kearsey, & Davies, 1978). That is, D effects inflates the

MZ correlation relative to the DZ correlation, whereas C effects inflates the DZ correlation relative to the MZ correlation. Although this is a limitation with the classical twin design, a meta-analysis of the heritability of human traits based on fifty years of twin studies suggest that similarity between twins is mainly due to influence of A, indicating that influence of C and D are negligible (Polderman et al., 2015). Finally, any dissimilarity between MZ twins must be due to E influences. For DZ twins, on the other hand, dissimilarity may be due to both E influences and nonshared genetic influences.

Based on the assumptions described above, the MZ and DZ correlations can be used to provide initial estimates of the proportion of *variance* due to genetic and environmental influences. Assuming no D effect, the additive genetic, shared environmental, and nonshared environmental components can be estimated with the following formulas:

$$a^2 = 2[r_{MZ} - r_{DZ}] \quad (1)$$

$$c^2 = 2r_{DZ} - r_{MZ} \quad (2)$$

$$e^2 = 1 - r_{MZ} \quad (3)$$

Where r_{MZ} refers to the MZ correlation, r_{DZ} refers to the DZ correlation, and a^2, c^2 and e^2 refer to additive genetic influences, shared environmental influences and nonshared environmental influences, respectively. As seen in equation 3, the estimate of the nonshared environmental influences includes all variance that does not contribute to similarity between MZ pairs. This estimate will therefore also include measurement error.

The same logic with comparing the correlation within MZ pairs with the correlation within DZ pairs applies when examining sources of *covariance* between traits. In such cases,

we do not consider MZ and DZ correlations for one phenotype, like in the univariate case presented above, but rely on comparing correlations between MZ and DZ twins across different phenotypes (i.e., cross-twin cross-trait correlations). That is, the correlation between trait X in twin 1 and trait Y in twin 2, and vice versa. If the cross-trait correlation is higher among MZ twins compared to DZ twins, genetic influences in the covariance between the traits are inferred. Equation 1-2 can be used to calculate the proportions of covariance between two traits due to additive genetic and shared environmental influences, respectively. However, the formula estimating the influence of the nonshared environment changes in the multivariate case. An estimate of the proportion of covariance due to nonshared environmental influences can be obtained by calculating the difference between the cross-trait correlation (i.e., the phenotypic correlation) and the cross-twin cross-trait correlation among MZ twins.

Whereas the observed twin correlations can be used to calculate initial estimates of genetic and environmental variance (and covariance) components, the formulas presented above do not provide any measure of uncertainty. Today, biometric analyses, implemented as structural equation models, are used for calculation of parameter estimates. However, the pattern of MZ versus DZ correlations are of course the basis for this approach as well. Several biometric models have been developed, each designed to examine different research questions. For example, univariate models and multivariate models such as Cholesky decomposition models and common factor models have been used extensively in twin studies (Røysamb & Tambs, 2016).

Gene-Environment Interactions and Gene-Environment Correlations

In the basic twin model, the phenotypic variance is assumed to equal the sum of the genetic variance plus the environmental variance (i.e., the genetic and environmental influences simply add up, meaning that genes and environment do not *interact* or *correlate*).

The presence of interactions or correlations between genes and environment will bias the estimates of genetic and environmental influences. However, gene-environment interactions and gene-environment correlations may be examined in twin studies if the relevant environmental variable contributing to the interaction or the correlation is measured (Evans, Gillespie, & Martin, 2002; Rijsdijk & Sham, 2002).

Gene-environment interactions refer to situations where the effects of environments depend on a person's genotype, or vice versa, situations where genetic influences depend on the environmental context (Briley, Livengood, Derringer, & Kandler, 2018). An interaction between genes and the shared environment will increase the estimate of A (i.e., MZ twins will respond to the shared environment in a similar way because they are genetically identical, whereas DZ twins will respond differently). That is, interaction between genes and the shared environment will inflate the MZ correlation relative to the DZ correlation. An interaction between genes and the nonshared environment, on the other hand, will increase the estimate of E (i.e., the phenotypic similarity between MZ twins, although genetically identical, will be reduced).

Gene-environment correlations refer to situations where a person's genetic predisposition influences which environments the person is exposed to or finds himself/herself in (Briley et al., 2018; Plomin et al., 1977). A correlation between genes and the nonshared environment can either be active or evocative. Active gene-environment correlations refer to situations where an individual actively creates, chose, or select into environments according to his/her genetic predisposition. For example, individuals with a genetic predisposition to high levels of extraversion may seek out social environments. Evocative gene-environment correlations, on the other hand, occur when an individual's genetically influenced characteristics elicit certain reactions from the environment. For example, children with a genetic predisposition to oppositional behavior may elicit negative

reactions from their parents. Both active and evocative gene-environment correlations will increase the estimate of A. In addition to active and evocative gene-environment correlations, gene-environment correlations may also be passive. Passive gene-environment correlations refer to processes where genes and the shared environment are correlated. Such gene-environment correlations occur in situations where parents pass both their genes and correlated environments to their children. For example, parents may provide books and create an intellectually stimulating rearing environment for their child in addition to pass on genes that predispose the child to learning. Passive gene-environment correlations will increase the estimate of C.

Material and Methods

Sample and Procedure

Data for the papers come from the Oslo University Adolescent and Young Adult Twin Project (Torgersen & Waaktaar, 2019; Torgersen & Waaktaar, 2020). All twin pairs born in Norway between 1988 and 1994, identified through the Medical Birth Registry of Norway, were invited to participate. Informed consent was obtained from both the twins and their parents. The data collection started in 2006, when the twins were 12 to 18 years old. Questionnaires were sent to the twins three times, with two years in between measurement waves. The mean ages at Wave-1, Wave-2 and Wave-3 were 15.2 (*SD* = 1.97), 16.9 (*SD* = 1.97) and 19.6 (*SD* = 1.95), respectively. A total of 1,538 twin pairs (56% females) participated in any wave. More specifically, 697 twin pairs (45%) participated in all three waves, 413 twin pairs (27%) participated in two waves, and 428 twin pairs (28%) participated in one wave only. In addition, the twins participated in a face-to-face interview when they were around 19 years (*M* = 19.1, *SD* = 1.2). A total of 1,425 twin pairs were interviewed, of

which 1,210 pairs (85%) had participated in at least one wave. The total twin sample consisted of 658 MZ and 1093 DZ twin pairs.[2]

In order to maximize the number of scales in the questionnaires, and to reduce dropouts and missing data, the complete scales were abbreviated based on results from a pilot study (Torgersen & Waaktaar, 2019). That is, the items in the complete scales with the highest item-to-trait correlations across all age groups and across sex were chosen.

Attrition may potentially lead to biased estimates of genetic and environmental parameters in twin studies (Heath, Madden, & Martin, 1998). Dropout in longitudinal studies are inevitable, and twin studies are particularly vulnerable because data from both twins in a pair is essential to perform analyses. In the present study, the percentage of complete pairs was close to hundred (i.e., when one twin participated, both twins almost always participated). Female gender, Big Five conscientiousness and openness, good school habits, and resilience predicted staying in the project (Torgersen & Waaktaar, 2019). That is, these factors were positively related to both frequency of participation in waves and participation in the interview. To analyze if attrition influenced the heritability estimates in the present sample, MZ and DZ correlations for the Big Five scales were calculated separately for those who participated in one wave, two waves, and three waves, respectively (Torgersen & Waaktaar, 2019). The correlations were similar in magnitude. MZ and DZ correlations for the Big Five scales were also calculated for those who participated in the interview and compared to the correlations for those who declined to participate in the interview. These correlations were also similar in magnitude, suggesting that attrition is not expected to influence the heritability estimates.

[2] The total number of twin pairs sum up to 1,751 because zygosity information was missing from two twin pairs.

Determination of Zygosity

Valid determination of zygosity is extremely important in twin studies. For example, if MZ twins are incorrectly classified as DZ, this may lead to an overestimation of the phenotypic similarity of DZ twins and hence to an overestimation of the shared environmental influences. Determination of zygosity in the present sample was based on a combination of results from a zygosity scale and results from gene testing. A zygosity scale (Torgersen, 1979) was included in the questionnaires at each wave. To validate the zygosity scale, cheek swabbed DNA was drawn from a subsample of same-sex twin pairs. Seventeen gene markers were analyzed, with a likelihood of misclassification of $p < 0.0001$. The scores on the zygosity scale were analyzed using discriminant analysis and a cutting point for the discriminant score was established based on the results of the gene testing. Those with a discriminant score close to the cutting point were oversampled for DNA tests (i.e., a total of 513 of the 1,006 same-sex twin pairs were gene-tested). It appeared that 14 out of the 513 twin pairs were misclassified according to the discriminant analysis. Correcting for the oversampling, the zygosity scale misclassified 2.13% of the same-sex twins. As almost all the misclassified pairs were gene tested, results showed that only 0.64% of the same-sex twin pairs are expected to be misclassified (0.45% when including the whole twin sample).

Measures

Data for the papers in this thesis come from both the self-report questionnaires and the interview. From the interview, we used data on BPD traits and retrospective reports of childhood trauma. Data from the questionnaires include scales measuring SOC, loneliness, and life events.

The Sense of Coherence Scale

SOC was measured by a set of five items from the Sense of Coherence 13-item scale (SOC-13; Antonovsky, 1987). The scale included the following questions: “Do you have the

feeling that you are being treated unfairly?", "Do you have the feeling that you are in an unfamiliar situation and don't know what to do?", "Do you have very mixed-up feeling and ideas?", "Does it happen that you have feelings inside you would rather not feel?" and "How often do you have the feeling that there's little meaning in the things you do in your daily life?". Reponses were given on a 7-point Likert scale ranging from 1 (*very often*) to 7 (*rarely/never*). Average scores were computed with higher scores indicating stronger SOC. The SOC-13 scale has been shown to have good internal consistency, with Cronbach's alpha ranging from 0.70 to 0.92 across studies (Eriksson & Lindström, 2005). The Cronbach's alpha of the SOC scale used in the present study ranged from 0.82 to 0.83 across the study waves, supporting the reliability of the abbreviated 5-item scale.

The R-UCLA Loneliness Scale

The R-UCLA Loneliness Scale consists of 20 indirect measures of loneliness (Russell et al., 1980). The scale used in the present study included the 4-item survey version: "I feel in tune with the people around me", "I can find companionship when I want it", "No one really knows me well", and "People are around me but not with me". Due to good psychometric characteristics, these items were recommended by Russell et al. (1980) when an abbreviated version is needed. In addition, a direct measure of loneliness was included in the scale ("I feel lonely"), resulting in a 5-item scale to measure loneliness. Responses were given on a 5-point Likert scale ranging from 0 (*not typical*) to 4 (*very typical*). Positively worded items were reverse-coded, and average scores were computed with higher scores indicating higher levels of loneliness. Supporting the reliability of our 5-item scale, the internal consistency of the loneliness-scale was $\alpha = 0.77$ at Wave-1, $\alpha = 0.81$ at Wave-2, and $\alpha = 0.84$ at Wave-3.

The Life Events Questionnaire for Adolescents

To measure a spectrum of positive and negative life events in adolescence, the participants were asked if they had experienced any of a set of 38 life events the past year (0

= *no*; 1 = *yes*). Twenty-nine of the events came from the Life Event Questionnaire for Adolescents (LEQ-A; Masten, Neemann, & Andenas, 1994) and nine events were added after the pilot study (Torgersen & Waaktaar, 2019). The set of life events were divided into clusters, based on their independence (i.e., dependent or independent of a person's behavior) and desirability (i.e., positive or negative). Life events from the LEQ-A were assigned to clusters according to Masten et al. (1994), and the additional events were classified based on the authors' evaluation. For example, "I had many arguments with my parents" was classified as negative dependent, "I lost a pet" as negative independent and "I got a new friend" as positive dependent. Positive independent events were not represented in the set of life events, probably due to their rareness or because positive life events are often considered dependent on a person's behavior (Kandler, Bleidorn, Riemann, Angleitner, & Spinath, 2012). Sum scores of the respective life events clusters were used when analyzing the data, with possible values ranging from 0–14 (negative dependent), 0–19 (negative independent) and 0–5 (positive dependent). All items in the life events scale are provided in Table A1.

The Childhood Trauma Interview

The Childhood Trauma Interview (CTI; Fink, Bernstein, Handelsman, Foote, & Lovejoy, 1995) in Norwegian translation was used to assess exposure to trauma. The CTI is a semistructured interview for the retrospective assessment of six areas of interpersonal trauma occurring during childhood and adolescence, including separations and losses, physical neglect, emotional abuse, physical abuse, sexual abuse and witnessing violence. The CTI has been shown to have high reliability and validity (Fink et al., 1995; Roy & Perry, 2004), and has been used in several studies examining different areas of mental health (Laporte, Paris, Guttman, & Russell, 2011; Simeon, Guralnik, Schmeidler, Sirof, & Knutelska, 2001; Vrshek-Schallhorn et al., 2014). The section on 'separation and losses' examines disturbances in attachment, including separation from caregivers, death of caregivers, or placement in

institutions or foster care. The 'physical neglect' section includes questions about lack of supervision and deprivation of food, clothing, and medical care. The section on 'emotional abuse' focuses on experiences of being threatened, humiliated, criticized, shouted at, controlled, ignored, or scapegoated. 'Physical abuse' is assessed through questions about experiences of being hit, kicked, thrown into the walls, looked inside a room/closet, choked, cut, or burned. The section on 'sexual abuse' includes questions about both contact experiences and noncontact experiences (e.g., sexual threats or watching others engage in sexual activities). Finally, the section on 'witnessing violence' includes questions about both domestic and other violence.

The section on 'separations and losses' was excluded from the analyses due to inconsistency in rating. That is, some interviewers rated 'death of grandparents' consistently as separation while others rated such cases as separation only when the grandparents had been the interviewee's caregivers. In addition, interviewers differed in scoring with regards to separation from one of the parents in case of divorce. Thus, this part of the interview was expected to have high measurement error and consequently low reliability and validity. The section on 'physical neglect' was also excluded from the analyses due to very low prevalence and consequently low statistical power (2.0% of the total sample reported physical neglect and 2.8% was discordant on exposure). All types of CT included in the analyses (i.e., emotional abuse, physical abuse, sexual abuse, witnessing violence and any CT) were coded dichotomously (0 = *absent*, 1 = *present*). Participants were classified as having experienced 'any CT' if they reported having experienced any of the four CT subtypes included in the analyses.

The Structured Interview for DSM-IV Personality

The Structured Interview for DSM-IV Personality (SIDP-IV; Pfohl, Blum, & Zimmerman, 1997), in Norwegian translation (Helgeland, Kjelsberg, & Torgersen, 2005),

was used to assess BPD. Although this interview is based on DSM-IV, the criteria for personality disorders in DSM-IV and DSM-5 are identical. SIDP-IV has been used in a number of studies in many countries, including Norway (Kendler et al., 2008). Each criterion is rated on a four-point scale (0 = *absent*, 1 = *subthreshold*, 2 = *present*, and 3 = *strongly present*), and the ratings are based on behavior typical for the past five years. To be diagnosed with BPD, a person must meet at least five of nine criteria. In the present sample, too few participants met the criteria for a BPD diagnosis (i.e., 1.3%) to perform reliable analyses with BPD as a categorically defined diagnosis. We therefore studied BPD dimensionally by calculating the number of endorsed BPD criteria at either the clinical or subclinical level (i.e., score ≥ 1), resulting in possible scores between 0 and 9. Interrater reliability of the dimensional measure of BPD was assessed based on two raters' scoring of 55 audiotaped interviews, of which 53 of the recordings were of satisfactory quality to be scored. The intraclass correlation coefficient for the dimensional measure of BPD (hereafter referred to as BPD traits) was 0.77 ($p < 0.001$).

Statistical Analyses

All statistical analyses were conducted in the statistical package R (R Core Team, 2021). Biometric analyses, implemented as structural equation models, were utilized in all papers, using the R package OpenMx (Neale et al., 2016). Building on model-fitting techniques, structural equation modeling allows us to estimate confidence intervals of parameter estimates and to evaluate the fit of competing models (e.g., determine whether the influence of a parameter is significant). Model fit indices are based on comparing the observed variance/covariance matrix with the expected variance/covariance matrix obtained from the fitted model. When comparing nested models (i.e., a model is nested under another model if it can be obtained from the model with more parameters by fixing one or more parameters to zero), the relative fit of a reduced model may be tested against a model with

more parameters by using the chi square (χ^2) difference test (Kline, 2015). For example, for comparison of a univariate ACE, AE, and CE model. A non-significant χ^2 difference indicates that the restricted model does not lead to significant loss of fit. Thus, the restricted model is preferred. Another widely used fit statistic is the Akaike's information criterion (AIC; Akaike, 1987). Lower values indicate better fit, and the model with the lowest value is selected. The AIC considers both fit and parsimony (i.e., it contains penalties for model complexity), and can be used to compare both nested and non-nested models. The idea of parsimony is based on the principle of selecting the model with a minimum number of parameters needed to explain the data well. Including more parameters in a model usually leads to better model fit. However, overly complex models may result in overfitting, with the consequence that the model does not fit well in other datasets, and in turn limiting generalizability of the findings. Thus, the AIC seeks to find a balance between model fit and parsimony.

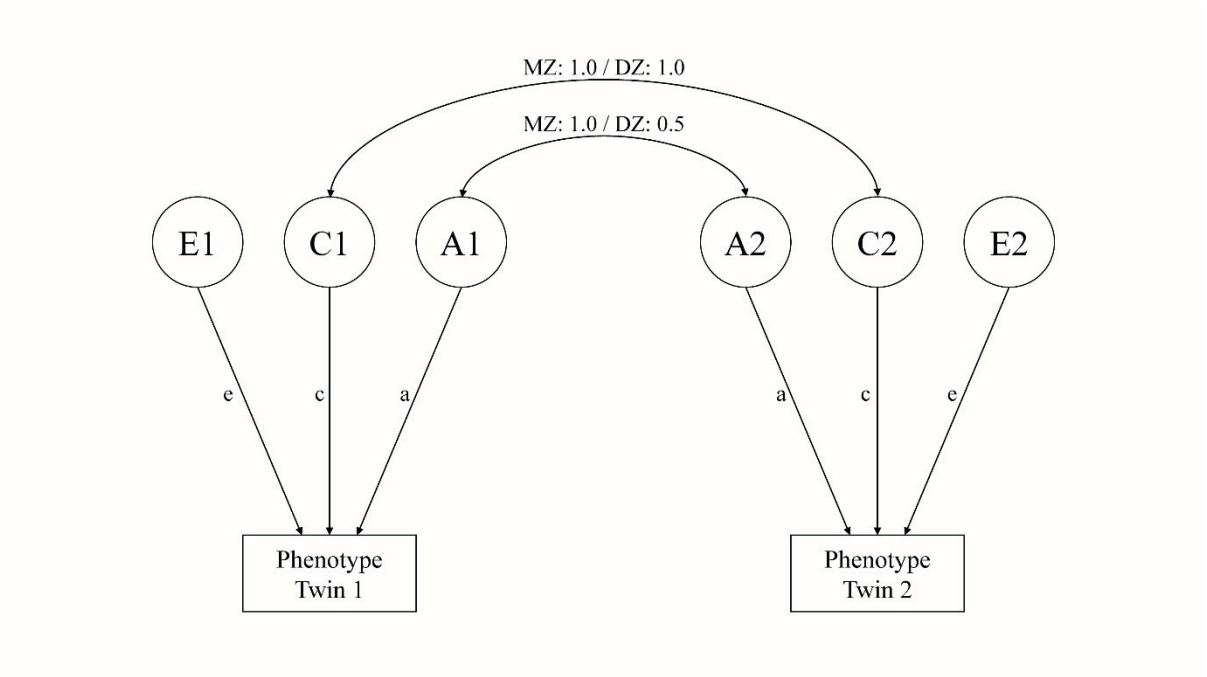
Univariate Twin Models

Univariate twin models seek to explain why people differ in a certain phenotype by partitioning the total variance in the measured variable into genetic and environmental variance components (i.e., the genetic and environmental influences are expressed as proportions of the total variance). Thus, univariate models are used to estimate how important genes and environment are for the observed individual differences in the phenotype of interest. As we have seen, the proportion of the total variance due to genetic influences is termed the heritability of the trait. The ACE model, which is the most widely used model, partitions the variance of a phenotype into additive genetic (A), shared environmental (C) and nonshared environmental (E) components. Figure 1 displays how A, C, and E components influence a phenotype in a pair of twins. After fitting an ACE model, restricted AE and CE models can be fitted. Relative model fit indices can then be obtained in order to choose the

model which best fits the data. Of note, influence of E is always estimated because this component also includes measurement error.

Figure 1

The Univariate ACE Twin Model



Note. MZ = monozygotic; DZ = dizygotic; A; additive genetic influences; C = shared environmental influences; E = nonshared environmental influences.

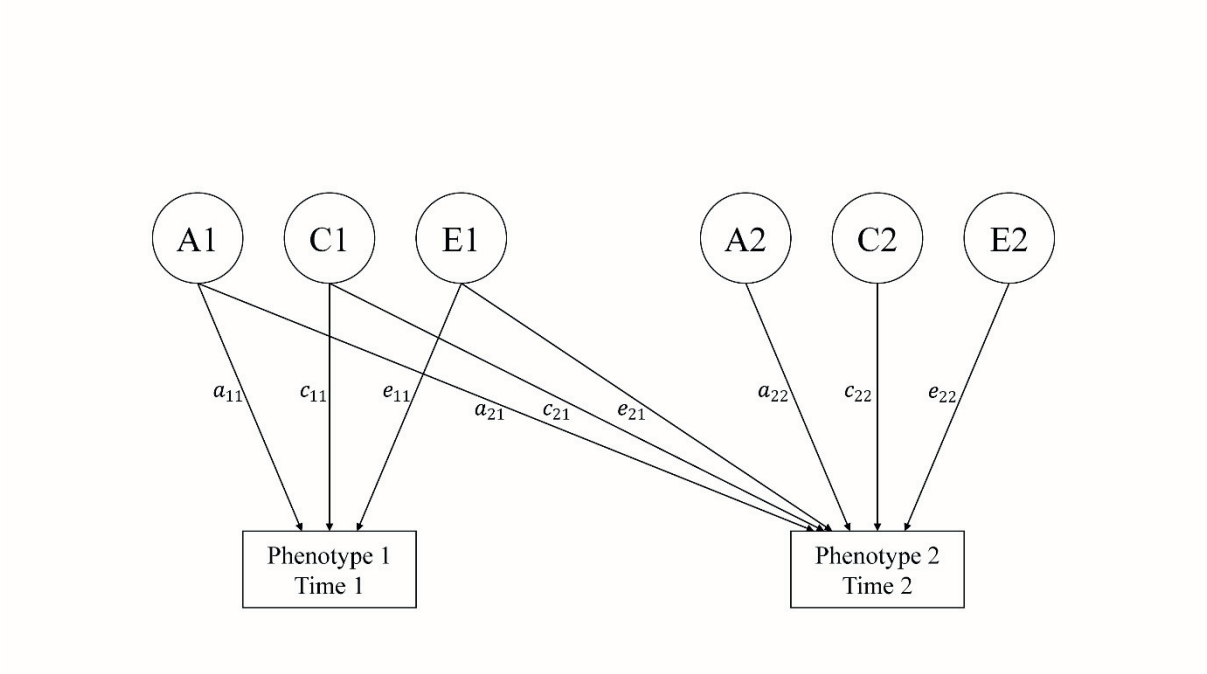
Cholesky Decomposition Models

Whereas univariate twin models are used to estimate genetic and environmental sources of *variance* in a phenotype of interest, multivariate models are used to also estimate sources of *covariance* between phenotypes. One commonly used multivariate model is the Cholesky decomposition model. This model represent a robust method to estimate genetic and environmental sources of variance and covariance, with few theoretical assumptions

(Loehlin, 1996; Neale & Maes, 2004). The Cholesky decomposition specifies as many latent factors as there are variables in the model. The first factor loads on all variables, the second loads on all variables except the first and so on. Figure 2 displays a bivariate Cholesky decomposition model. For simplicity, the model is shown for one twin only.

Figure 2

Bivariate Cholesky Decomposition Model



Note. A and a = latent additive genetic factors and paths; C and c = latent shared environmental factors and paths; E and e = latent nonshared environmental factors and paths.

As displayed in Figure 2, one set of latent factors (A, C, and E) is specified for each of the variables. The first set of latent factors loads on both measured variables, while the second set loads on only the last variable, accounting for the residual variance in the second variable not captured by influences from the first. As in the univariate case, restricted models

(AE and CE) can be compared to the full ACE model to determine which model that best fit the observed data.

In Figure 2, the heritability of Phenotype 1 equals the squared standardized parameter estimate from the latent factor A1 to Phenotype 1 (i.e., a_{11}^2), while the heritability of Phenotype 2 equals the sum of the squared standardized parameter estimate from the latent factor A1 to Phenotype 2 plus the squared standardized parameter estimate from the latent factor A2 to Phenotype 2 (i.e., $a_{21}^2 + a_{22}^2$). Similar reasoning is used when calculating the shared environmental and the nonshared environmental sources of variance. Furthermore, the parameter estimates in Figure 2 can be used to calculate the proportions of the phenotypic correlation between the two phenotypes that are due to genetic and environmental influences, respectively. For example, the proportion of the phenotypic correlation due to genetic influences can be calculated by dividing the genetic influences on the phenotypic correlation by the phenotypic correlation (i.e., $[a_{21} \times a_{11}]/[a_{21} \times a_{11} + c_{21} \times c_{11} + e_{21} \times e_{11}]$).

The Cholesky decomposition model also allows us to estimate genetic and environmental correlations. The genetic correlation describes the extent to which genetic influences on one trait are shared with another trait (Posthuma et al., 2003). A nonzero genetic correlation between two traits implies that both traits are influenced by at least some of the same genetic factors. If the genetic correlation is 1, this implies that the two sets of genetic influences on each trait overlap completely. Of note, a high genetic correlation does not indicate a high phenotypic correlation, nor that most of the phenotypic correlation is due to genetic influences. This is because the genetic contribution to the phenotypic correlation is a function of both the genetic correlation and the heritability of both traits. Thus, if the heritabilities are low, the genetic contribution to the phenotypic correlation will also be low (Posthuma et al., 2003). Similar reasoning applies to the nonshared environmental correlation and to the shared environmental correlation.

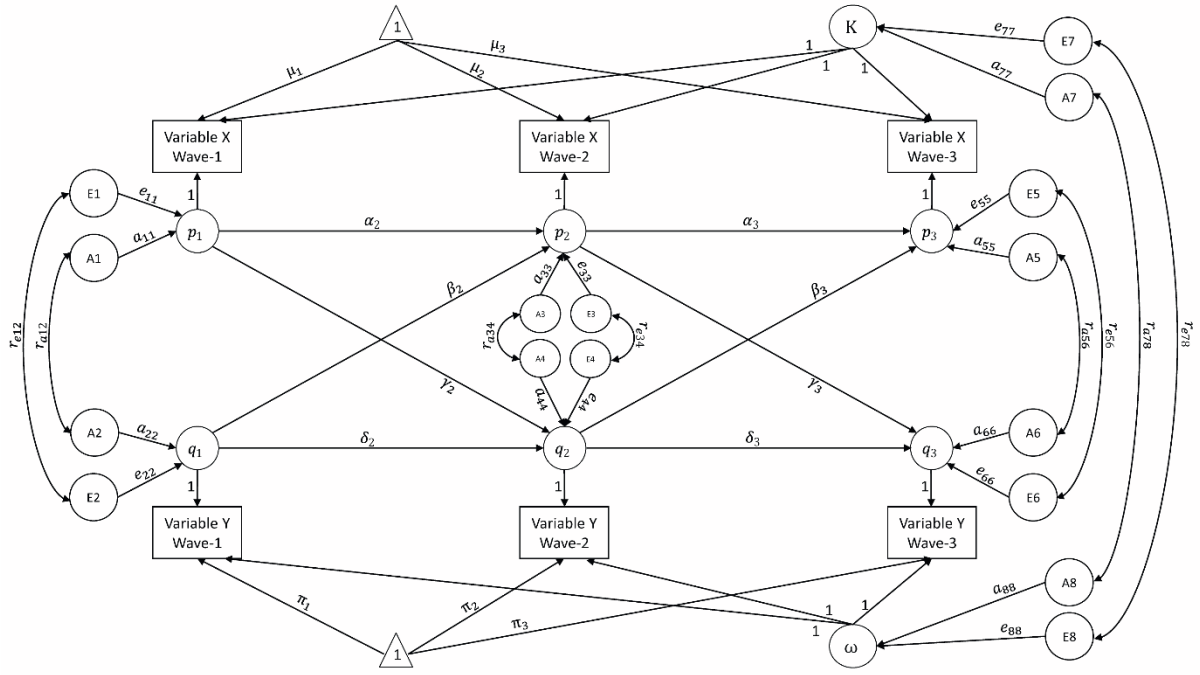
Genetically Informative Random Intercept Cross-Lagged Panel Models

The random intercept cross-lagged panel model (RI-CLPM) was recently presented by Hamaker, Kuiper, and Grasman (2015) as an alternative to the traditional cross-lagged panel model (CLPM). Both these models are concerned with making causal interpretations between variables by estimating cross-lagged effects (i.e., the effect of one construct on another construct measured at a later occasion). In both the RI-CLPM and the CLPM, the cross-lagged parameters are estimated controlling for the previous level (i.e., stability) of the construct being predicted. The difference between these models is that they control for different kinds of stability. The CLPM controls for stability through the inclusion of autoregressive parameters reflecting stability of the rank order of individuals between measurement occasions. This model assumes that all individuals vary over time around the same mean. Thus, if the constructs studied to some extent have a trait-like nature, the autoregressive parameters fail to adequately control for this. As a consequence, the CLPM may produce incorrect estimates of the cross-lagged paths leading to erroneous conclusions regarding the causal effects (Hamaker et al., 2015; Selig & Little, 2012). The RI-CLPM extents the CLPM by including random intercepts that control for trait-like stability. That is, the random intercepts control for stable individual differences in mean levels across the measurement waves (i.e., between-person variance). Thus, the autoregressive and cross-lagged parameters reflect actual within-person processes (i.e., variance due to changes within individuals over time), which are the processes we are interested in when examining potential causal influences of one variable on another.

The RI-CLPM approach can be further extended by partitioning the variances into genetic and environmental components, and by modeling genetic and environmental correlations. Figure 3 displays a figurative representation of a genetically informative RI-CLPM for the relationship between two variables measured at three measurement occasions.

Figure 3

Bivariate Genetically Informative Random Intercept Cross-Lagged Panel Model for Three Waves of Data



Note. For simplicity, the model is shown for one twin only and only additive genetic (A) and nonshared environmental (E) influences are shown. Triangles represent constants for the means, rectangles represent observed variables and circles represent latent variables. μ_t and π_t = temporal grand means; K and ω = random intercept latent factors; p_t and q_t = within-person components; α_t and δ_t = autoregressive parameters; β_t and γ_t = cross-lagged parameters; A and a = latent additive genetic factors and paths; E and e = latent nonshared environmental factors and paths; r_a and r_e = additive genetic correlations and nonshared environmental correlations, respectively.

Because the random intercepts account for stable individual differences in mean levels in the measured constructs across the measurement waves, the variance in the within-person components (p and q) reflect individuals' temporal deviations (or fluctuations) from their

own stable level of the measured variables. The autoregressive parameters are specified between these within-person components. We can think of the autoregressive parameters as 'within-person carry-over effects' (Hamaker et al., 2015). A positive autoregressive parameter indicates that individuals who score higher (or lower) on for example variable X relative to their stable level at one time point, also tend to score higher (or lower) on variable X relative to their stable level at the next measurement occasion. The parameters of main interest in the model, however, are the cross-lagged parameters. Yet, an understanding of the random intercepts and the autoregressive parameters are important in order to understand the interpretation of the cross-lagged parameters. A significant cross-lagged effect indicates that fluctuations in one construct measured at one time point, predicts fluctuations in the other construct measured at a later measurement occasion, after controlling for the within-person carry-over effect in the construct being predicted.

If there are significant cross-lagged effects that are of a size that are practically meaningful, it is useful to examine to what extent these effects can be attributed to genetic and/or environmental influences. Let us consider an example where there is a significant cross-lagged effect from variable X to variable Y. At the extreme, the whole effect could be due to environmental influences. This would indicate that changing the environments that influence variable X are also likely to influence variable Y. For example, if variable Y is some mental health outcome, this indicates that intervening on X could influence the measured mental health outcome. At the other extreme, the cross-lagged effect could be solely attributed to genetic influences. This would indicate that it may be more fruitful to look for other factors than X when searching for factors that influence Y.

In addition to the cross-lagged effects, it is also interesting to calculate the proportion of variance in the measured constructs accounted for by the random intercepts. These proportions inform us about how stable each construct is. Because we have data from twins,

we can also calculate how much of the stability that is due to genetic and environmental factors, respectively. In addition, the genetic and environmental correlations between the random intercepts inform us whether some of the same genetic and environmental factors are influencing the stability in both constructs.

Discordant Twin Analyses

The discordant twin design, or the cotwin control design, is based on the counterfactual model of causality (McGue, Osler, & Christensen, 2010; Rutter, 2007). The counterfactual approach basically asks what the outcome would have been if the exposure did not happen (Hernan, 2004; Rubin, 2001). The fundamental threat to causal inference is to distinguish between whether an observed association between a potential risk factor and an outcome arises because the risk factor causally influences the outcome, or arises because one or more third variables (i.e., confounding variables) are correlated with both the potential risk factor and the outcome. Randomized experiments are often considered the gold standard for causal inference. That is, random assignment to the exposure group and the control group aims to ensure that the control group will provide a valid counterfactual for those in the exposure group (i.e., confounding variables are randomly spread in the groups and the only systematic difference between the groups is the exposure). However, due to the nature of research questions asked in the field of psychology, manipulation of the exposure is often unethical and impossible (Rohrer, 2018). Therefore, we often have to rely on observational data when examining potential causal relations between variables. That being said, randomized experiments are not without limitations, especially in terms of generalization of findings (Ohlsson & Kendler, 2020).

The discordant twin design represents a powerful approach for studying questions about causality (McGue et al., 2010). Building on the counterfactual approach, the logic of the discordant twin design can be summarized as using the non-exposed twin as an estimate

of what the exposed twin would have looked like if he or she had not been exposed to the risk factor in question. The non-exposed twin will of course not be a perfect estimate. However, twins reared together share the same family environment and are thus matched on a range of environmental factors (e.g., rearing environment and family-background characteristics), MZ twins are genetically identical while DZ twins, on average, share half of their segregating genes. Thus, when comparing an outcome within discordant MZ twins (i.e., only one of the twins in a pair has been exposed to the potential risk factor), this comparison will completely control for potential confounding of both genetic and shared environmental factors. A comparison within discordant DZ twins will also completely control for shared environmental factors and partly for genetic factors.

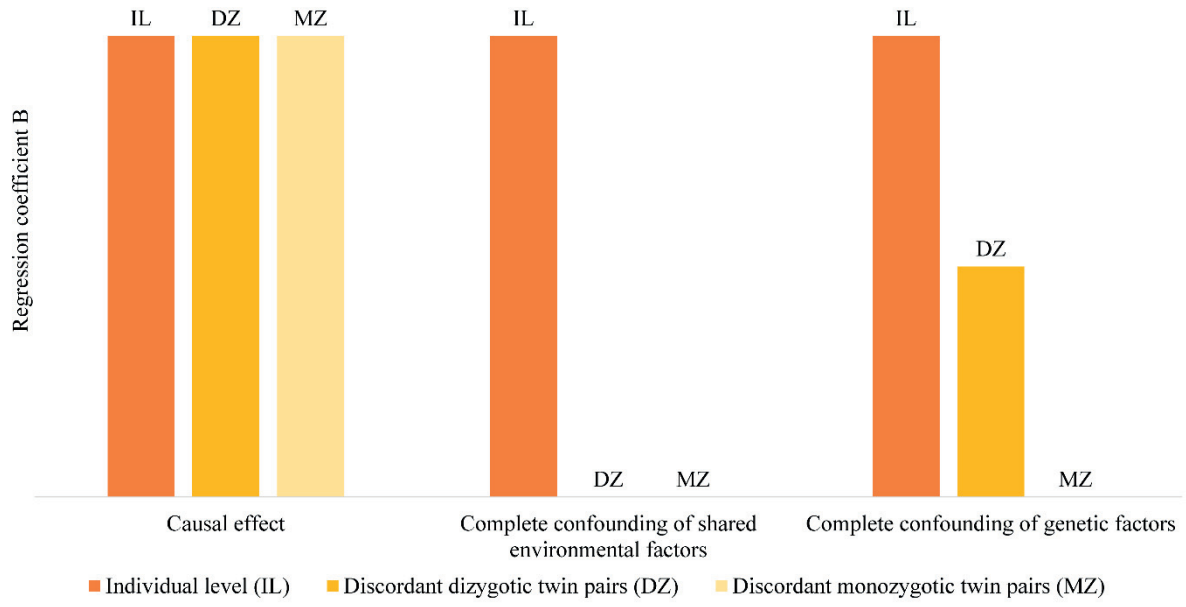
Figure 4 presents three main patterns which can be found in studies with a discordant twin design. If we were interested in whether some exposure has a causal effect on an outcome, we would first estimate the effect of the exposure on the outcome in a standard regression framework without considering twin structure (i.e., referred to as 'individual level' in Figure 4). If the exposure is coded dichotomously (0 = non-exposed, 1 = exposed), the unstandardized regression coefficient at the individual level represents the average difference in the outcome between exposed and non-exposed individuals. This individual level effect can then be compared to the effect observed within discordant twin pairs. This effect represents the average difference in the outcome within twin pairs that are discordant on the exposure. Although MZ twins represent the most stringent control for confounding, the effect within discordant DZ pairs is essential to be able to distinguish between genetic and shared environmental confounding.

If the exposure has a *causal effect* on the outcome, the effect within discordant DZ and discordant MZ pairs are expected to be the same as the effect at the individual level. That is, controlling for shared environmental and genetic factors do not reduce the association

between the exposure and the outcome. If, on the other hand, the association is confounded by shared environmental and/or genetic factors, the effect within discordant DZ and discordant MZ pairs are expected to be lower compared to the effect at the individual level. In a scenario of *complete confounding of shared environmental factors*, the effect of the exposure is expected to be absent within both discordant DZ and discordant MZ pairs. This is because the effect within both discordant DZ and discordant MZ pairs completely control for confounding of shared environmental factors. In a scenario of *complete confounding of genetic factors*, the effect of the exposure is expected to be absent within discordant MZ pairs. Within discordant DZ pairs, on the other hand, the effect is expected to be reduced but not completely absent compared to the effect at the individual level. This is because the effect within discordant DZ pairs only partly controls for genetic factors. These two scenarios of confounding describe complete confounding. If the association between exposure and outcome is *partially confounded* by shared environmental and/or genetic factors, the patterns will be similar as those presented in Figure 4, but the effects within discordant twin pairs will not be completely absent.

Figure 4

Three Possible Patterns of Effects of Predictor on Outcome at the Individual Level Versus Within Discordant Twins



Note. The height of the bars represents the effect of exposure on outcome when measured at the individual level, and within monozygotic and dizygotic twin pairs discordant on exposure. IL = the average difference in outcome due to a one-unit change in the predictor without considering twin-pair membership.; MZ = the average difference in outcome within discordant monozygotic twin pairs; DZ = the average difference in outcome within discordant dizygotic twin pairs.

Discussion of the Results

In this section, I will present and discuss the findings in the four individual papers in this thesis. First, the study aims and the statistical analyses that were used to answer the research questions are presented, followed by a presentation and discussion of the results. Of

note, implications of the research findings will mainly be covered in the concluding section. Paper I and paper IV directly examine the main research objectives of this thesis. However, as will become apparent, findings from paper II and paper III reveal important insights about the nature of SOC, loneliness, and life events that are all relevant for enhancing our understanding about the main aims of this thesis.

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

The aim of paper I, published in the Journal of Psychopathology and Clinical Science (Skaug, Czajkowski, Waaktaar, & Torgersen, 2022a), was to examine the nature of the associations between childhood trauma (i.e., emotional abuse, physical abuse, sexual abuse, and witnessing violence) and BPD traits in early adulthood. More specifically, we used discordant twin analyses to examine whether childhood trauma have direct effects on levels of BPD traits in early adulthood by controlling for the potential confounding effects of shared environmental and genetic factors. In addition, we estimated the relative contribution of genetic and environmental influences in creating individual differences in childhood trauma and BPD traits by using univariate twin analyses.

Numerous studies without control for shared genetic factors have concluded that traumatic experiences in childhood represent important environmental risk factors in the development of BPD (Ball & Links, 2009; Charak et al., 2018; Newnham & Janca, 2014). However, in line with previous work that have found substantial genetic influence on measured environments (e.g., Kendler & Baker, 2007), results from the present study showed that all subtypes of childhood trauma were moderately or highly heritable (i.e., the heritability estimates ranged from 33% to 69% across the trauma types). This highlights the importance of using designs that are able to separate the environmental effect of childhood trauma from the potential confounding effects of genes when examining whether such experiences

actually represent causal factors in the development of BPD traits. The heritability of BPD traits was estimated to 50%, which is similar to estimates reported in previous studies that have measured BPD traits at one measurement occasion (Bornovalova et al., 2013; Distel et al., 2008; Kendler et al., 2008; Torgersen et al., 2008). The remaining variance was attributable to nonshared environmental influences. Indeed, twin studies consistently indicate that shared environmental factors do not create individual differences in levels of BPD traits (Gunderson et al., 2018).

We replicated the well-documented phenotypic associations between childhood trauma and BPD traits. However, results from the discordant twin analyses indicate that the observed associations are not due to causal effects of childhood trauma on BPD traits. Rather, the associations were accounted for by shared genetic influences. This finding implies that childhood trauma and BPD traits are correlated because the genes causing the development of BPD also influence the likelihood of exposure to childhood trauma (i.e., gene-environment correlations). For example, a child's genetic predisposition to impulsivity and defiant behavior may elicit certain reactions from the environment such as physical aggression from others (i.e., evocative gene-environment correlation). Alternatively, the impulsive and oppositional child may select into environments that increases the likelihood of exposure to adverse life events (i.e., active gene-environment correlation). These results corroborate findings from the few studies published to date with analogous childhood trauma, personality disorders and methodology (Berenz et al., 2013; Bornovalova et al., 2013). To validate the results from the discordant twin analyses, we calculated genetic and environmental correlations between BPD traits and each subtype of childhood trauma by fitting a series of bivariate Cholesky decomposition models. Results from these models provided further support for the conclusions derived from the discordant twin analyses. That is, all subtypes of childhood trauma showed substantial genetic correlations with BPD traits, whereas the

environmental correlations were negligible. In sum, the results indicate that childhood trauma does not seem to causally influence levels of BPD traits in early adulthood. Childhood trauma and BPD traits are associated, but these associations are genetically grounded.

Paper II: The Relationship between Life Events and Sense of Coherence in Adolescence. A Longitudinal Twin Study

The main aim of paper II, published in the Journal of Research in Personality (Skaug, Czajkowski, Waaktaar, & Torgersen, 2022b), was to contribute to a better understanding of the causal architecture behind the relationship between life events and SOC. Life experiences in childhood and adolescence have theoretically been assumed to shape the development of SOC (Antonovsky, 1987). However, not much is known empirically about which factors influencing its development. Although cross-sectional studies of adolescent samples have suggested that life events influence levels of SOC (Marsh et al., 2007; Moksnes et al., 2011; Natvig et al., 2006; Olsson et al., 2006; Ristkari et al., 2008), claims about direction of effect is very difficult to assess using cross-sectional studies. In this paper, we examined the longitudinal relationship between three clusters of life events (i.e., negative dependent, negative independent, and positive dependent) and SOC throughout adolescence. Both life events and SOC were measured at three successive time points, with two years in between measurements. More specifically, we examined whether reported life events within the past year predicted subsequent levels of SOC two years later, and vice versa. By using a twin sample, we were also able to examine the nature of the associations. These research questions were studied by using genetically informative RI-CLPMs.

Before we explored the nature of the relationship between life events and SOC, we estimated genetic and environmental sources of variance in the measured constructs. Trivariate Cholesky decomposition models were fitted to data from the three measurement occasions, with separate models for each construct. Person-dependent negative and positive

life events showed substantial heritability, with estimates ranging from 47%–55% (negative dependent) and from 43%–52% (positive dependent) across the study waves. As expected, the heritability of negative life events assumed to be independent of a person's own behavior was lower (i.e., 12%–25%). Not surprisingly, as this cluster of life events included several family-related events (e.g., parental divorce, illness in the family), a substantial proportion of individual differences in reported negative independent life events was due to shared environmental influences (i.e., 29%–40%). These results collaborate findings from prior studies that have examined the heritability of measured life events (Bemmels et al., 2008; Billig, Hershberger, Iacono, & McGue, 1996; Kandler et al., 2012; Plomin, Lichtenstein, Pedersen, McClearn, & Nesselroade, 1990). The heritability of SOC ranged from 31%–47% across the measurement waves. Similar estimates have been reported in the few existing studies that have examined the heritability of SOC (Hansson et al., 2008; Silventoinen et al., 2014).

With respect to the relationship between life events and SOC, phenotypic correlation analyses showed moderate negative correlations between negative dependent life events and SOC, and weak negative correlations between SOC and both negative independent and positive dependent life events. Of note, a negative association between positive life events and SOC may seem counterintuitive. However, as found in previous studies (Kandler et al., 2012; Magnus, Diener, Fujita, & Pavot, 1993; Plomin et al., 1990) and in the present study, negative and positive life events were positively correlated with each other. Finding genetic variance in measured life events may partly explain this, with genetically influenced personality traits as potential third variables creating a positive covariance between negative and positive life events. For example, genetic factors that influence a person's openness to new experiences may increase the likelihood of experiencing both positive and negative life events (Kandler et al., 2012). A person's level of activity may also be associated with a

number of life events, both positive and negative. Thus, the negative association between positive life events and SOC may possibly be explained by the fact that the experience of more positive life events also implies experience of more negative life events.

As to the nature of the associations between life events and SOC, the results from the genetically informative RI-CLPMs showed that the effects of life events (whether negative dependent, negative independent or positive dependent) on levels of SOC were negligible. Rather, the phenotypic correlations were largely attributable to shared genetic influences. For example, some people may experience (or at least report) negative life events due to their genetic predisposition to perceive the world as chaotic, unmanageable, and meaningless (which characterizes a weak SOC). In conclusion, life events do not seem to influence levels of SOC in adolescence, but life events and SOC are correlated due to shared genetic influences.

In addition to provide insight into potential causal relationships between variables, the genetically informative RI-CLPM also inform us about the stability in the measured constructs. The results indicate that SOC is a relatively stable construct throughout adolescence. That is, about 40% of the of the total variance in SOC at each measurement occasion was due to time-stable between-person variance. This corroborates findings from prior studies which have found moderate rank-order stability of SOC measured with one-year intervals and up to 13 years between measurements (Eriksson & Lindström, 2005; Feldt et al., 2007; Hakanen, Feldt, & Leskinen, 2007; Honkinen et al., 2008). Thus, SOC may be considered as a relatively stable trait with similar stability as found for personality traits in general (Costa, McCrae, & Lockenhoff, 2019; Ferguson, 2010). The results also showed that negative dependent life events tend to reoccur. More specifically, about 30% of the total variance in negative dependent life events at each measurement wave was due to time-stable

between-person variance. Both the stability of SOC and the recurrence of negative dependent life events were largely determined by additive genetic influences.

Paper III: The Longitudinal Relationship between Life Events and Loneliness in Adolescence. A Twin Study

In paper III, we turn our attention to loneliness. By using the genetically informative RI-CLPM as we used in paper II, we examined whether life events predicted levels of loneliness throughout adolescence, and vice versa. As we have seen, prior studies suggest that life experiences influence feelings of loneliness in childhood and adolescence (Lasgaard et al., 2016; Rönkä et al., 2014; von Soest et al., 2020; Weeks & Asher, 2012). However, genetically informative studies on this research area are lacking. Thus, the nature of the associations between life events and loneliness are unexplored.

With respect to the etiology of loneliness, the results showed that nearly half of the variance in loneliness was due to additive genetic influences (i.e., the heritability estimates ranged from 44%-46% across the measurement waves). This corroborates findings from prior studies that have examined the heritability of self-reported loneliness (Goossens et al., 2015). In addition, the results showed that feelings of loneliness were moderately stable throughout adolescence. That is, time-stable between-person variance accounted for about 40% of the total variance in loneliness at each measurement occasion. This is in accordance with results from a recent meta-analysis, which found loneliness to be as stable as personality traits across the life span (Mund, Freuding, Möbius, Horn, & Neyer, 2020). Furthermore, results from the present study showed that the stability in feelings of loneliness was mainly attributable to additive genetic influences.

As to the relationship between life events and loneliness, there were weak positive correlations between negative life events (i.e., both dependent and independent) and

loneliness, and weak negative correlations between positive dependent life events and loneliness. When we examined the nature of these phenotypic associations, results from the genetically informative RI-CLPMs showed that neither of the life events clusters predicted levels of loneliness throughout adolescence, or vice versa. This may seem surprising, as several of the negative events in the life events scale are related to circumstances that both directly (e.g., loss of a close friend, change of schools) and indirectly (e.g., many arguments with parents, being bullied) potentially could increase feelings of loneliness. Rather, the results showed that the observed phenotypic associations between life events and loneliness were largely due to shared genetic influences. This suggests that lonely people are more likely to experience negative life events (or at least are more likely to perceive and interpret the world in a more negative way) compared to less lonely people, due to their genetic predisposition to feelings of loneliness. People who feel lonely are also shown to be more introverted and neurotic than non-lonely people (Buecker et al., 2020), suggesting that personality traits may be potential confounders that create genetic associations between life events and loneliness. For example, it may be that people with more introverted and neurotic traits also struggle with social relationships. Together, the results from this paper suggest that feelings of loneliness may be considered as a relatively stable genetically driven tendency to perceive and interpret the world, rather than reflecting a negative emotional state resulting from influences from a person's life circumstances.

Paper IV: The Role of Sense of Coherence and Loneliness in Borderline Personality Disorder Traits: A Longitudinal Twin Study

In paper IV, we examined the prediction of SOC and loneliness throughout adolescence on BPD traits in early adulthood. Results from paper II and paper III indicate that levels of SOC and feelings of loneliness are relatively stable throughout adolescence. Thus, it is meaningful to examine whether these characteristics already from early

adolescence are related to BPD traits in early adulthood. Identifying features that represent risks for later development of BPD is essential, both in terms of prevention and early intervention (Bozzatello, Bellino, Bosia, & Rocca, 2019).

In this paper, we rearranged the data from Wave-1, Wave-2, and Wave-3 into age groups. That is, instead of analyzing data from each wave (in which each wave included data from seven birth cohorts), the questionnaire data was rearranged into variables that included data from the age of 12–13 years, 14–15 years, 16–17 years, and 18 years and older (i.e., until the time of assessment of BPD traits). First, we used linear regression analyses to quantify the strength of the prediction of SOC and loneliness at the different ages on BPD traits in early adulthood. Next, a series of bivariate Cholesky decomposition models were fitted to quantify how much of the phenotypic correlations between predicted and observed BPD scores was attributable to genetic and environmental influences, respectively. The predicted BPD scores were derived from the linear regression analyses, resulting in four predicted scores. The predicted scores from a given age was included as the first variable in the Cholesky decomposition, and the observed BPD scores were included as the second variable in each model (i.e., four separate models). Finally, we included life events in the prediction of BPD traits together with SOC and loneliness, and examined whether this changed the relative contribution of genetic and environmental influences to the phenotypic correlation between predicted and observed BPD scores.

Results from linear regression analyses showed that SOC and loneliness measured already at the age of 12 years predicted BPD traits in early adulthood ($R = .25$). The strength of the prediction increased continuously with older age and shorter intervals between measurement of SOC, loneliness, and BPD traits ($R = .45$ when SOC and loneliness were measured shortly before the assessment of BPD traits). Furthermore, the strength of the prediction slightly increased when negative dependent life events were added to the

regression analyses together with SOC and loneliness. Negative independent and positive dependent life events did not have any independent effects on the levels of BPD traits.

By using Cholesky decomposition models, we were able to quantify to what extent these predictions stem from direct effects of the predictor variables and to what extent the predictors were associated with BPD for genetic reasons. The results showed that SOC and loneliness were associated with BPD traits mainly due to shared genetic influences (i.e., shared genetic influences accounted for 71%–86% of the phenotypic correlations between the predicted and the observed BPD scores). When negative dependent life events were added to the prediction of BPD traits, the proportions of the phenotypic correlations due to additive genetic and environmental influences were almost unchanged. In fact, the proportions due to additive genetic influences slightly increased. Thus, even when adding measured environments to the prediction of BPD, these did not increase the relative contribution of environmental influences. Findings in Paper II and Paper III further support these results, which showed that the stability of both SOC, loneliness and the recurrence of negative dependent life events were mainly due to genetic influences. Indeed, if a trait-like characteristic is predictive of an outcome measured at a later point in time, it is likely that it is the stable variance in the construct that drives the prediction. Together, the results from this paper suggest that the lower levels of SOC, the higher levels of loneliness and the negative life events associated with increased levels of BPD traits are mainly consequences of the genetic aspects of BPD.

Methodological Considerations

The results presented in this thesis should be considered in the context of some methodological considerations and consequently possible limitations. In this section I will discuss some selected methodological considerations.

Two Fundamental Assumptions in Twin Designs

Twin models, as any statistical model, have assumptions that may threaten the validity of results if violated. A fundamental assumption in the classical twin design is the equal environment assumption (EEA). The EEA states that MZ and DZ twins experience the same degree of similarity of environmental factors influencing the phenotype(s) studied. If MZ twins are being treated more similarly than DZ twins, and this causes similarity in the phenotype(s) studied, the EEA is violated. The higher correlation in MZ twins compared to DZ twins may then be due to environmental rather than genetic influences, leading to an overestimation of genetic influences. However, empirical evidence generally supports the validity of the EEA (Conley, Rauscher, Dawes, Magnusson, & Siegal, 2013; Felson, 2014; Kendler, Neale, Kessler, Heath, & Eaves, 1993).

Another fundamental assumption in twin designs is that DZ twins share half of their segregating genes. This is based on an assumption that parents do not share genes beyond what is expected by random chance. However, people tend to select partners and have children with people who resemble themselves phenotypically. This phenomenon is known as assortative mating. Given that all human traits are heritable (Polderman et al., 2015), assortative mating means that we also indirectly choose partners that resemble ourselves genetically. If we are studying a phenotype that has been subject to assortative mating, DZ twins will share more than 50% genes for this phenotype. This may result in an overestimation of the shared environmental influences and an underestimation of the genetic influences (i.e., assortative mating will inflate the DZ correlation relative to the MZ correlation, thus mimicking influence of C). Assortative mating is shown to be most marked for education, religion, attitudes, and socioeconomic status, whereas assortative mating is found to be low or random for physical appearance, cognitive traits, and personality domains (Ask, Idstad, Engdahl, & Tambs, 2013; Evans et al., 2002; Neale & Maes, 2004). Thus, based

on the empirical evidence for related phenotypes such as personality, we do not believe that the phenotypes studied in this thesis to a great extent are subject to assortative mating, in that people select partners based on similarities in SOC, loneliness or BPD traits.

Self-Reports

All measures used in the papers in this thesis are based on self-reports from the twins. The potential limitations of using self-reports may depend on the phenotypes studied. For example, both SOC and loneliness are subjective in nature (i.e., these constructs are intended to reflect a person's subjective feelings and perceptions of the world), which make self-reports the most appropriate approach. However, it is possible that our measure of BPD traits could have been improved if this measure had been based on a combination of reports from significant others in addition to the twins' own reports. Furthermore, one may ask to what degree the measures of childhood trauma and life events reflects the *perception* of recalled memories. For example, it might be that persons with BPD symptoms are more likely to recall an experience as abusive or negative. Whether the use of self-reports represent a limitation or not may depend on the person being asked. For example, although self-reports may be prone to the person's subjective interpretations, the responses are likely similar to the clinical situation where clinicians have to rely on the patient's descriptions.

Retrospective Reporting

The assessment of childhood trauma was based on retrospective reporting, which is the case for most studies on this research area. The accuracy of retrospective reports has been debated for a long time. For example, both normal forgetting and the presence of mental disorders have been proposed as factors that may influence the accuracy of memories of past experiences (M. L. Rogers, 1995; Williams, 1994). However, other studies indicate that people often remember unusual events and that psychiatric status does not seem to be associated with less reliable or valid reporting of early experiences (Brewin, Andrews, &

Gotlib, 1993). A review of studies published between 1980 and 2001 that have examined the validity of retrospective reports of traumatic experiences found that retrospective reports had a substantial amount of false negative reports, but that false positive reports of traumatic events were rare (Hardt & Rutter, 2004). In other words, traumatic experiences may be underreported, but people rarely invent stories of such experiences. The researchers concluded that retrospective reports of traumatic experiences in childhood are adequately valid. In further support of the validity of using retrospective reports in research, more recent prospective studies have found that memory for potential traumatic events are more accurate compared to non-traumatic events (Lalande & Bonanno, 2011), and that retrospective reporting of abuse is quite consistent over time in personality disorder samples (Spinhoven, Bamelis, Haringsma, Molendijk, & Arntz, 2011). In addition, recall bias seem to explain only a small proportion of the variance in retrospective reports of childhood trauma (Fergusson, Horwood, & Boden, 2011).

Although the amount of error variance in measures of childhood trauma probably had been lower with the use of prospective methods, such designs are often not appropriate due to both practical, economic, ethical, and time-consuming reasons (Roy & Perry, 2004). The childhood trauma interview, which is the interview that was used in the present study, has several advantages that are assumed to strengthen the validity of retrospective reports, such as a semi-structed format, a scoring manual with instructions for defining the presence of trauma, and a scoring based on concrete behavior rather than the interviewee's interpretation and judgement (Hardt & Rutter, 2004; Roy & Perry, 2004; Vrshek-Schallhorn et al., 2014).

Even so, in accordance with the review by Hardt and Rutter (2004), it is possible that discordance on trauma in some twin pairs may be due to the fact that one of the twins did not report trauma that actually has taken place. One might speculate if such underreporting by one of the twins are more common in DZ pairs compared to MZ pairs, as DZ twins are more

different in personality in general. Nevertheless, misclassifying pairs as discordant when they in reality are concordant on exposure would be a severe limitation with the discordant twin design, as the assumed non-exposed cotwin in reality has experienced the same type of trauma and therefore do not represent a valid counterfactual for the exposed twin.

Assessment and Classification of Life Events

Like the reliability and validity of retrospective reports of childhood trauma, life events checklists have also been criticized. In addition to the general limitation of possible recall bias (Monroe, 2008), the reliability and validity of life events scales has been criticized due to intra-category variability (Dohrenwend, 2006). That is, most scales include broad and general categories of life events that may reflect a variety of experiences for different persons. For example, depending on how a person interprets the question, serious illness may mean episodes of headache for some persons, while others will not make a positive response to this life event unless the person has been critically ill.

Furthermore, genetically informative studies often distinguish between life events considered dependent on a person's own behavior and/or control and life events considered independent of a person's own behavior and/or control. The rationale behind this differentiation is that while an association between independent events and some outcome is likely to be causal, the association between dependent events and the outcome may be confounded by a person's behavior, which may be genetically influenced. Thus, the association between dependent life events and the outcome may be confounded by genetic influences affecting them both (Kendler, Karkowski, & Prescott, 1999).

Results from the present thesis and previous studies (Bemmels et al., 2008; Billig et al., 1996; Plomin et al., 1990) have found higher heritability of dependent life events compared to independent life events, which supports a differentiation between them.

However, some events may not be clearly classified as dependent or independent (e.g., whether an event should be classified as dependent or independent may vary across individuals and situations). For example, some people may have many arguments with their siblings due to their own behavior, whereas for others, arguments may arise due to their siblings' quarrelsome behavior. In addition, if independent events were truly unrelated to a person's (genetically influenced) behavior, one would expect that the heritability of such life events were zero. Finding genetic variance in assumed independent life events may reflect that event-dependence may vary across individuals. It may also indicate that most life events are not purely independent of a person's genetically influenced behavior, and consequently not solely environmental in nature. Although a classification of life events into clusters based on event-dependence may be difficult, a differentiation between dependent and independent life events is still important. For example, dependent life events, potentially genetically rather than causally related to a particular outcome, may obscure potential environmental effects of independent life events on the outcome. In addition, studying the effects of life events without considering event-dependence may result in a less nuanced and less accurate picture of the relationship between life events and the outcome.

Dimensional Measure of Borderline Personality Disorder

Even in large studies of population-based samples, such as the sample used in the papers in this thesis, the number of individuals meeting the criteria for a BPD diagnosis is often too low to perform analyses with BPD as a categorically defined variable. Therefore, BPD is often studied dimensionally. This is also warranted because, as described earlier, empirical studies have shown that BPD is best conceptualized as a dimensional construct, with different symptom levels reflecting degrees of severity on a continuum (Edens et al., 2008; Torgersen et al., 2008; Trull et al., 2010).

In our dimensional measure of BPD traits, each criterion with a nonzero score were given equal weight. From a psychometric perspective, where maximizing the variation would be preferable, it may seem contradictory not to preserve the original rating scale. However, we chose this approach because it is common in clinical practice to look at the number of traits instead of weighting the strength of each trait/criterion. In addition, counting the number of criteria may be considered more robust because this approach is less dependent on the specific interviewers' tendency to use only a part of or the full width of the rating scale. This approach is also successfully used in previous studies based on a population-based twin sample (Kendler et al., 2008; Reichborn-Kjennerud et al., 2015; Torgersen et al., 2008). Despite this, much of the variance in our measure of BPD traits comes from symptoms in individuals who did not meet the criteria for a full BPD diagnosis. It may be that other results than those found in the present papers would emerge in samples that include more individuals with the full range of BPD symptoms (e.g., studies of clinical samples).

Conclusion

The aims of this thesis were to (1) examine the causes of BPD, and to (2) examine whether two central features associated with the disorder (i.e., SOC and loneliness) already from early adolescence could predict BPD traits in the beginning of adulthood.

With respect to the first aim, we approached this both by quantifying the relative contribution of genetic and environmental influences on individual differences in BPD traits, and by examining whether measured environments (i.e., childhood trauma and life events throughout adolescence) could account for some of the environmental variance in BPD traits. The findings in this thesis showed that BPD traits in early adulthood was moderately heritable (i.e., the heritability was estimated to 50%). The remaining variance was attributable to nonshared environmental influences. This nonshared environmental component contains all factors making twins from the same family dissimilar, including measurement error.

Studies that have attempted to reduce measurement error by using longitudinal designs have found higher heritability of BPD traits (around 70%) compared to cross-sectional studies (Bornovalova et al., 2009; Reichborn-Kjennerud et al., 2015). Thus, the nonshared environmental component is probably overestimated in estimates based on cross-sectional measures of BPD traits. However, even when measurement error is taken into consideration (e.g., in the longitudinal studies mentioned above), error variance alone does not explain all of the nonshared environmental variance. As described earlier, childhood trauma and adverse life events have been proposed to be important factors causing the development of this personality disorder, both theoretically and empirically in numerous studies without control for shared genetic influences. However, the findings in this thesis indicate that childhood trauma and negative life events throughout adolescence are not causally related to BPD traits. Rather, the results suggest that these measured environments are associated with BPD traits due to shared genetic influences.

Together, these results show that the nonshared environment has a relatively large effect on individual differences in BPD traits when its total variance is partitioned into genetic and environmental variance components, but the measured environments examined in this thesis did not explain any of the environmental variance in BPD traits. One explanation may be that we have not found the potent environmental agents causing the development of BPD, even though we studied factors that frequently have been considered important in the development of this personality disorder. However, perhaps a more likely explanation may be that the nonshared environmental influences are idiosyncratic and unsystematic, but that added together they make a significant contribution to creating individual differences in BPD traits. The notion that the nonshared environmental variance is the result of unsystematic environmental influences rather than systematic effects of identifiable environmental events is referred to as ‘the gloomy prospect’ (Plomin & Daniels, 1987). Indeed, genetically

informative studies consistently fail to identify systematic effects of measured environments on behavioral outcomes (Plomin, DeFries, Knopik, & Neiderhiser, 2016; Turkheimer, 2000). Although many clinicians and researchers believe that traumatic experiences in childhood are important causes of BPD (Zanarini et al., 2019), the results in this thesis suggest that such experiences do not contribute to the development of this personality disorder. It seems likely that the gloomy prospect is true for the development of BPD traits as well. It may also be that we are looking at the wrong kind of environmental influences. For example, the nonshared environmental influences may be embedded in prenatal factors. In addition, diseases or injuries in childhood may affect brain development, resulting in emotional, cognitive, or behavioral changes that add to the nonshared environmental influences. Furthermore, measurement error blows up the estimate of the nonshared environment. Consequently, in reality the nonshared environmental influences are probably lower, and the heritability correspondingly higher, than what we estimate.

The heritability of measured environments should also be mentioned. Results from this thesis corroborate earlier work that have examined the sources of variance in measured environments. That is, we found substantial genetic variance in measures of both childhood trauma and life events throughout adolescence. These findings have important implications for the entire field of mental health and how we should think about the causal structure of life events. Although it is acknowledged in genetic research that measures of the environment are not purely environmental in nature, the research literature is full of studies that draw causal conclusions based on phenotypic correlations between environmental exposures and various outcomes. Finding genetic variance in measured environments highlights the need to use genetically informative studies also when examining the effect of assumed environmental variables. That is, an association between an environmental measure and some outcome may

not necessarily reflect a causal influence of the environmental exposure on the outcome, but can rather be due to shared genetic influences (i.e., gene-environment correlations).

The second aim of this thesis was to examine whether levels of SOC and loneliness throughout adolescence predicted BPD traits in early adulthood. Before moving on to this second aim, it is important to mention the results regarding the course of SOC and loneliness in adolescence. We found that SOC and loneliness are relatively stable, largely genetically determined, traits throughout adolescence. Furthermore, life events did not influence levels of SOC and loneliness in adolescence.

As to the second aim, identification of features related to BPD at the earliest stages of the disorder has been highlighted as an important goal in research, and not least among health care professionals (Chanen, Nicol, Betts, & Thompson, 2020). Indeed, detection of indicators that increase the risk for later development of BPD, with subsequent prevention and treatment efforts, is believed to reduce chronicity and long-term impairments in functioning associated with the disorder (Kaess et al., 2014). In this thesis, we found that lower SOC and greater feelings of loneliness already at the age of 12 years predicted BPD traits in early adulthood with some certainty. As expected, the prediction of these features on BPD traits was stronger with shorter time until assessment of BPD traits. Furthermore, we found the associations between SOC, loneliness, and BPD traits to be largely genetic in nature. In other words: the genetic predisposition to BPD manifests itself already at the age of 12 partly through low levels of SOC and feelings of loneliness.

But what is the practical implication when the associations were found to be largely genetic? It is important to have in mind that the data for this thesis is based on a naturalistic study where we observe phenomena ‘out there’, it is not an experiment where we have examined the effect of an intervention. Thus, even if SOC and loneliness are associated with

BPD traits mainly for genetic reasons, this does not necessarily mean that intervention and prevention efforts seeking to increase levels of SOC or decrease levels of loneliness do not work. Indeed, low levels of SOC and feelings of loneliness should be taken seriously already from early adolescence as they may represent a vulnerability to later development of BPD.

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Appendix

Table A1

The Life Events Scale

Negative dependent life events
I had an important change in physical appearance, which upset me (acne, glasses, physical development, etc.)[a]
I was a victim of violence (mugging, sexual abuse, robbery)[a]
I was disappointed by a friend
I was disappointed by someone in the family
I did not get into a group or activity that I wanted to get into (music group, sports team, theater, etc.)[a]
I had major problems with a teacher
I did much worse than I expected in an important exam or course[a]
I had less contact with one of my parents[a]
I had many arguments with my siblings[a]
I had many arguments with my parents[a]
I was bullied by other pupils/adolescents
I broke up with a girlfriend/boyfriend[a]
I had an abortion (girls) / my girlfriend had an abortion (boys)
I lost a close friend[a]
Negative independent life events
I lost a pet
I changed schools[a]
I became seriously ill or was injured[a]
At least one parent or another family member became seriously ill or was injured[a]
One of my parents died[a]
A brother or sister died[a]
Another family member died[a]
One of my close friends died[a]
Mom or Dad's friend moved in with us[a]
A member of my family ran away from home[a]
My parents divorced, moved apart[a]
One of my parents had problems at work[a]
One parent lost his or her job[a]
My mother began to work[a]
There has been a change in a parent's job so that my parent is away from home more often[a]
The family financial situation was difficult[a]
There was some damage or loss of family property (such as apartment, house, car or bike)[a]
There were many arguments between the adults[a]
Someone in the family had problems with the police[a]
Positive dependent life events

I received a special award (trophy, diploma etc.) for something done at school[a]
I became more popular with my friends
I joined a fun group of friends
I got a boyfriend/girlfriend[a]
I got a new friend

Note. [a] Question from the Life Event Questionnaire for Adolescents (LEQ-A; Masten et al., 1994). The wording in some of the questions were slightly changed from the LEQ-A.

Papers

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II

Journal of Research in Personality 99 (2022) 104259

Contents lists available at ScienceDirect

Journal of Research in Personality

journal homepage: www.elsevier.com/locate/jrp

The relationship between life events and sense of coherence in adolescence. A longitudinal twin study

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ARTICLE INFO

Keywords:
Sense of coherence
Life events
Adolescence
Random intercept cross-lagged panel model
Genetic

ABSTRACT

This three-wave study examined the relationship between life events and sense of coherence (SOC) throughout adolescence by using genetically informative random intercept cross-lagged panel models. We also examined the genetic and environmental contribution to variance in the measured constructs. The data come from a Norwegian population-based twin sample (N = 2,878). Life events and SOC were associated, and both showed substantial genetic variance. Negative longitudinal effects were observed from negative dependent life events to SOC, from SOC to negative dependent life events and from SOC to positive dependent life events. However, these longitudinal effects were negligible in magnitude. In summary, the associations between all three clusters of life events and SOC were almost completely accounted for by shared genetic influences.

1. Introduction

The theory of sense of coherence (SOC) was originally developed by Aaron Antonovsky, aiming to explain an individual's ability to cope with life stressors (A. Antonovsky, 1979). According to this theory, people with a strong SOC view the world as (1) *comprehensible*, (2) *manageable* and (3) *meaningful*. People with a strong SOC are therefore likely to (1) view stressors in life as clear and understandable, (2) believe that they have the necessary resources to meet the demands of the situation, and (3) find it meaningful to invest time and effort to cope with the challenges in question. People with a weak SOC, on the other hand, perceive the world as more chaotic, unmanageable and meaningless. Consequently, a strong SOC is believed to facilitate successful coping with stressful life situations (A. Antonovsky, 1993).

Conceptually, SOC may be considered as a personality characteristic characterized by a stable tendency to view the world more or less predictable, manageable and meaningful (H. Antonovsky & Sagy, 1986). Indeed, the SOC questionnaire (A. Antonovsky, 1987) includes items that are related to personality in terms of covering characteristic ways of thinking, feeling and behaving. Although SOC and the Big Five personality traits are theoretically distinct concepts, these constructs are conceptually related to each other (Feldt, Metsäpelto, Kinnunen, & Pulkkinen, 2007b). For example, individuals high on *neuroticism* are prone to feelings of hopelessness and are likely to use ineffective coping strategies, which characterizes persons with weak SOC. *Conscientious* individuals tend to plan and be organized, making it likely that they will perceive the world as structured and predictable, which are characteristic of a strong SOC. Furthermore, *extraverts* often have big social networks, and individuals who score high on *agreeableness* often get along well with other people. These characteristics may in turn increase a person's belief that he/she will receive social support when facing various stressors, a feature related to stronger SOC. In line with these considerations, empirical studies have shown that SOC is negatively related to neuroticism, and positively related to extraversion, conscientiousness, and agreeableness (Ebert, Tucker, & Roth, 2002; Feldt et al., 2007b; Hochwälder, 2012).

Antonovsky described SOC as an enduring and global way of looking at the world which develops throughout childhood and adolescence, and becomes stabilized by the end of young adulthood around the age of 30 (A. Antonovsky, 1993). Supporting this notion, a study by Feldt et al. (2007a) found that the rank-order stability of SOC was higher among persons over 30 years compared to younger adults. This is also in line with the empirical literature on the stability of personality traits, which finds that the rank-order stability increases throughout adolescence and peaks in adulthood (Costa, McCrae, & Lockenhoff, 2019). Supporting its trait-like nature, empirical studies have found moderate rank-order

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https://doi.org/10.1016/j.jrp.2022.104259
Received 22 June 2021; Received in revised form 30 March 2022; Accepted 6 June 2022
Available online 10 June 2022

stability of SOC measured with a one-year interval and up to 13 years between measurements, both in adolescence and adulthood (Eriksson & Lindström, 2005; Hakanen, Feldt, & Leskinen, 2007; Honkinen et al., 2008), similar to the rank-order stability found for personality traits (Costa et al., 2019; Ferguson, 2010; Roberts & Delvecchio, 2000).

The introduction of the theory of SOC represented a paradigm shift from a pathogenic focus on risk factors and causes of disease, to factors promoting and maintaining good health (A. Antonovsky, 1993; Eriksson & Lindström, 2005). Numerous studies have investigated the relationship between SOC and health in adult populations. Two reviews of nearly 500 papers published between 1992 and 2003 showed that SOC is strongly related to perceived health and quality of life (Eriksson & Lindström, 2006, 2007). Fewer studies have examined adolescent populations, but the existing empirical evidence supports a relationship between SOC and health in adolescence as well (Buddeberg-Fischer, Klaghofer, & Schnyder, 2001; García-Moya, Rivera, & Moreno, 2013; Honkinen, Suominen, Välimaa, Helenius, & Rautava, 2005; Moksnes, Rannestad, Byrne, & Espnes, 2011; Nielsen & Hansson, 2007; Ristkari, Sourander, Rønning, Nikolakaros, & Helenius, 2008; Torsheim, Aaroe, & Wold, 2001).

With respect to the development of SOC, Antonovsky emphasized the importance of experiences in childhood, but also experiences in adolescence and young adulthood (A. Antonovsky, 1987). He pointed to putative SOC-promoting factors like consistency in life circumstances, a balance between demands in life and available resources, social support and the importance of playing an active role in life. Negative life events, on the other hand, could potentially weaken a person's SOC (A. Antonovsky, 1987). Beyond these theoretical assumptions, little is known empirically about causal factors behind the development of SOC during childhood and adolescence (Rivera, Garcia-Moya, Moreno, & Ramos, 2013).

With respect to the association between life events and SOC, most studies have examined negative life events. Hochwälder and Forsell (2011) located 10 studies that have examined the association between negative life events and SOC in adult populations. All 10 studies concluded that SOC was lowered by negative life events. However, most of the studies suffered from some methodological shortcomings that limited the possibility to determine whether negative life events actually were related to change in SOC (e.g., absence of a measure of SOC prior to the occurrence of the negative life events). Hochwälder and Forsell (2011) addressed some of these methodological issues by measuring negative life events and SOC at two time points, one and a half year apart. They found no strong evidence supporting the hypothesis that negative life events lowered SOC.

The existing literature on adolescent populations suggests that both negative and positive life events may be related to SOC. Results from a study by Ristkari et al. (2008) showed that adolescents who had experienced parental divorce, parental illness or death of a parent had lower mean levels of SOC compared to those who had not experienced such major life events. However, the differences were small. Furthermore, studies have reported weak to moderate negative associations between SOC and stressful life events such as peer pressure, pressure of schoolwork and family conflict (Marsh, Clinkinbeard, Thomas, & Evans, 2007; Moksnes et al., 2011; Natvig, Hanestad, & Samdal, 2006). Regarding life events associated with stronger SOC, studies have reported moderate associations between SOC and positive experiences such as social support (Marsh et al., 2007; Natvig et al., 2006) and positive family relationships (Olsson, Hansson, Lundblad, & Cederblad, 2006). However, all these studies on adolescent populations are cross-sectional and thus do not allow for conclusions about the direction of effect between life events and SOC.

Although some of the methodological challenges in the existing literature that have studied the association between life events and SOC could potentially be resolved by using longitudinal study designs, *confounding* still represents a serious challenge to valid inference. If a third variable (e.g., genes) affects both life events and SOC, this may create a spurious association between them. To our knowledge, only two studies have examined the heritability of SOC in particular. In these studies, the heritability of SOC was estimated to 35% (Hansson et al., 2008) and 45% (Silventoinen et al., 2014). Furthermore, measured variables that seem environmental almost by definition, such as life events, are influenced by genes (Kendler & Baker, 2007). Genetically informative studies often distinguish between 'dependent' and 'independent' life events. Dependent life events refer to life events that may be associated with an individual's own behavior (e.g., arguments with parents) whereas independent life events refer to life events that do not seem to have anything to do with an individual's own behavior (e.g., death of a family member). As expected, prior studies have shown that the heritability of dependent life events tends to be higher compared to independent life events (Bemmels, Burt, Legrand, Iacono, & McGue, 2008; Billig, Hershberger, Iacono, & McGue, 1996; Plomin, Lichtenstein, Pedersen, McClearn, & Nesselroade, 1990).

As measured life events are partly influenced by genetic factors, genetically informative studies (e.g., twin studies) are needed to examine whether the associations between life events and SOC are truly environmental in nature. Such designs make it possible to determine the relative role of genetic and environmental contributions to phenotypic correlations between variables. However, genetically informative studies on life events and SOC are lacking. The aim of the present longitudinal twin study is to contribute to a better understanding of the relationship between life events and SOC through (1) investigating the genetic and environmental contributions to phenotypic variance in life events and SOC in adolescence and (2) examining the direction and the nature of the relationship between these constructs. Specifically, this study will investigate to what extent the phenotypic associations between life events and SOC are due to real environmental influences and to what extent they are mediated by genetic influences.

2. Method

2.1. Sample and procedure

The participants were Norwegian adolescent twins taking part in the Oslo University Adolescent and Young Adult Twin Project (Torgersen & Waaktaar, 2019; Torgersen & Waaktaar, 2020). The project involved three waves of questionnaires throughout adolescence and one face-to-face interview with the twins when they were around 18 years old. All twin pairs born in Norway between 1988 and 1994 were invited to participate. The twins were identified through the Norwegian Medical Birth Registry. In the present study, we used data on SOC and life events derived from the questionnaires. A number of other variables relevant for personality in adolescence were also assessed in the project (Torgersen & Waaktaar, 2019; Torgersen & Waaktaar, 2020), such as Big Five (Kandler, Waaktaar, Mõttus, Riemann, & Torgersen, 2019), resilience (Waaktaar & Torgersen, 2011) and loneliness (Waaktaar & Torgersen, 2012). The data collection started in 2006 when the twins were 12 to 18 years old. Questionnaires were sent to the twins three times, with two years in between. Informed consent was obtained from both the twins and their parents. The mean ages at Wave−1, Wave−2 and Wave−3 were 15.2 (SD = 1.9), 16.9 (SD = 2.0) and 19.6 (SD = 1.9), respectively. In order to maximize the number of scales in the questionnaires, the complete scales were abbreviated based on results from a pilot study (Torgersen & Waaktaar, 2019). The final sample consisted of 2,878 twins (56% females) from 1,483 families, including 1,093 monozygotic (MZ) twins and 1,785 dizygotic (DZ) twins. That is, the percentage of complete pairs was close to one hundred. The study was approved by the Norwegian Data Inspectorate and the Regional Committees for Medical and Health Research Ethics. American Psychological Association ethical standards were followed in the conduct of the study.

2.2. Zygosity determination

The zygosity of same-sex twin pairs were partially determined through a 12-item zygosity scale where questions about similarity in appearance, how often the twins have been mixed-up with each other, and whether they believe that they are monozygotic or dizygotic were asked (Torgersen, 1979). To validate the zygosity scale, cheek swabbed DNA was drawn from a subsample of twin pairs. Twin pairs with ambiguous scores on the zygosity scale were oversampled for DNA-tests. Seventeen genetic markers were tested, with an estimated probability of misclassification <0.0001. The scores on the zygosity scale were analyzed using discriminant analysis. The same-sex twins who were not gene tested were classified as MZ or DZ twins based on discriminant analysis of the zygosity scale scores.

2.3. Power analyses

Power analyses were conducted to examine how big the genetic correlations between two phenotypes in the population had to be in order to have a statistical power of 0.80 to detect it under different scenarios of heritability and shared environmental effects. The analyses were conducted in OpenMx (Neale et al., 2016), using the R functions provided by Verhulst (2017). We used the same ratio of MZ to DZ twin pairs as in the present sample (N_{MZ} = 556; N_{DZ} = 927) and assumed complete twin pairs, and continuous variables. Assuming no shared environment and a heritability of 0.30, 0.40 and 0.50, we could with a power of 0.80 detect genetic correlations of 0.57, 0.41 and 0.30, respectively. Alternatively, in the presence of a shared environmental effect of 0.10 and a heritability of 0.30, 0.40 and 0.50, we could with a power of 0.80 detect genetic correlations of 0.52, 0.36 and 0.26, respectively. With information provided by additional covariance statistics, power to detect genetic correlations will be greater in the trivariate (longitudinal) models.

2.4. Measures

2.4.1. Life events

Life events were measured by a 38-item 2-point scale in which the participants were asked about whether they had experienced any of the set of life events the past year (0 = *no*, 1 = *yes*). The scale included 29 events from the Life Event Questionnaire for Adolescents (LEQ-A; Masten, Neemann, & Andenas, 1994) translated into Norwegian. In addition, nine new life events were added (see Table A1). Life events from the LEQ-A were classified as negative dependent (e.g., "I had many arguments with my parents"), negative independent (e.g., "One of my parents died") or positive dependent (e.g., "I received a special award for something done at school") according to Masten et al. (1994). The additional life events were assigned to clusters based on evaluation of their independence (i.e., dependent or independent) and desirability (i.e., positive or negative). The sum of reported life events within each cluster was used when analyzing the data, with possible values ranging between 0 and 14 (negative dependent), 0–19 (negative independent) and 0–5 (positive dependent). The life events scale did not include positive independent life events, probably because such life events rarely appear (Kandler, Bleidorn, Riemann, Angleitner, & Spinath, 2012).

2.4.2. Sense of coherence

SOC was measured by an abbreviated 5-item version of the Sense of Coherence 13-item scale (SOC−13; A. Antonovsky, 1987) translated into Norwegian. The final scale included the following questions: "Do you have the feeling that you are being treated unfairly?", "Do you have the feeling that you are in an unfamiliar situation and don't know what to do?", "Do you have very mixed-up feeling and ideas?", "Does it happen that you have feelings inside you would rather not feel?" and "How often do you have the feeling that there's little meaning in the things you do in your daily life?". Responses were given on a 7-point Likert scale (1 = *very often*, 7 = *rarely/never*) with higher values indicating stronger SOC. For each participant an average score was calculated. A review of 127 studies using the SOC−13 showed that the Cronbach's alpha ranged from 0.70 to 0.92 (Eriksson & Lindström, 2005). In our study, the Cronbach's alpha ranged from 0.82 to 0.83 across the three study waves, supporting the reliability of the abbreviated 5-item scale.

2.5. Statistical analysis

First, phenotypic correlations were computed to examine stability across time within each of the measured constructs and to investigate the association between them. Next, cross-twin correlations were calculated to give an initial impression of the genetic and environmental contributions to variation within and covariation between the measured constructs. Twin studies make use of the knowledge that MZ twins are genetically identical while DZ twins share, on average, half of their segregating genes. These differences allow for calculations of the variance in a phenotype (and the covariance between phenotypes) caused by genetic and environmental influences. Additive genetic influences (A; i.e., genes that together operate in an additive manner, causing similarity among family members) are inferred when the MZ correlation is greater than the DZ correlation. Shared environmental influences (C; i.e., any environmental factors that contribute to similarity among family members) are inferred when the DZ correlation is more than half the magnitude of the MZ correlation. Any remaining variance in the phenotype (or covariance between the phenotypes) not accounted for by A or C is attributed non-shared environmental influences (E). The E factor thus represents any influences that contribute to phenotypic dissimilarity within both MZ and DZ twin pairs, including measurement error. Since phenotypic differences between MZ pairs can only be due to E, an initial estimate of E can be estimated through the lack of similarity between MZ pairs.

The correlation analyses were extended using biometric analyses, implemented as structural equation models. This allows us to specify and evaluate the fit of multivariate twin models, as well as calculate standard errors and confidence intervals of parameter estimates. The structural equation modeling program, OpenMx, was used for the biometric models (Neale et al., 2016). Models were fitted to raw data using full information maximum likelihood. First, we fitted Cholesky decomposition models to data from the three measurement waves for each measured construct separately to estimate the genetic and environmental contributions to variance in SOC and the three clusters of life events. The models were fitted with separate means for males and females to account for mean-level sex differences in SOC and life events. Using data from twins, a Cholesky decomposition allows us to partition the observed phenotypic variances into their latent genetic and environmental components. The Cholesky decomposition specifies as many latent factors as there are variables for each source of variance. The first latent factor loads on all of the measured variables, the second loads on all variables except the first and so on. In this way, each factor accounts for as much of the residual variance as possible, with the last factor accounting for the remaining variance in the last measured variable. For each construct, we first fitted a full ACE model, followed by reduced models (AE, CE and E). Relative model fit was determined by comparing the models' Akaike's information criterion (AIC; Akaike, 1987), with lower values indicating better model fit.

Next, genetically informative cross-lagged panel models were fitted to examine the longitudinal relationship between life events and SOC. The traditional cross-lagged panel model (CLPM) is often used to investigate causal longitudinal influences between constructs (Hamaker, Kuiper, & Grasman, 2015). In the CLPM, the autoregressive parameters reflect the rank order stability of individuals from one measurement occasion to the next. This model implicitly assumes that every person varies over time around the same mean. Thus, if there to some extent

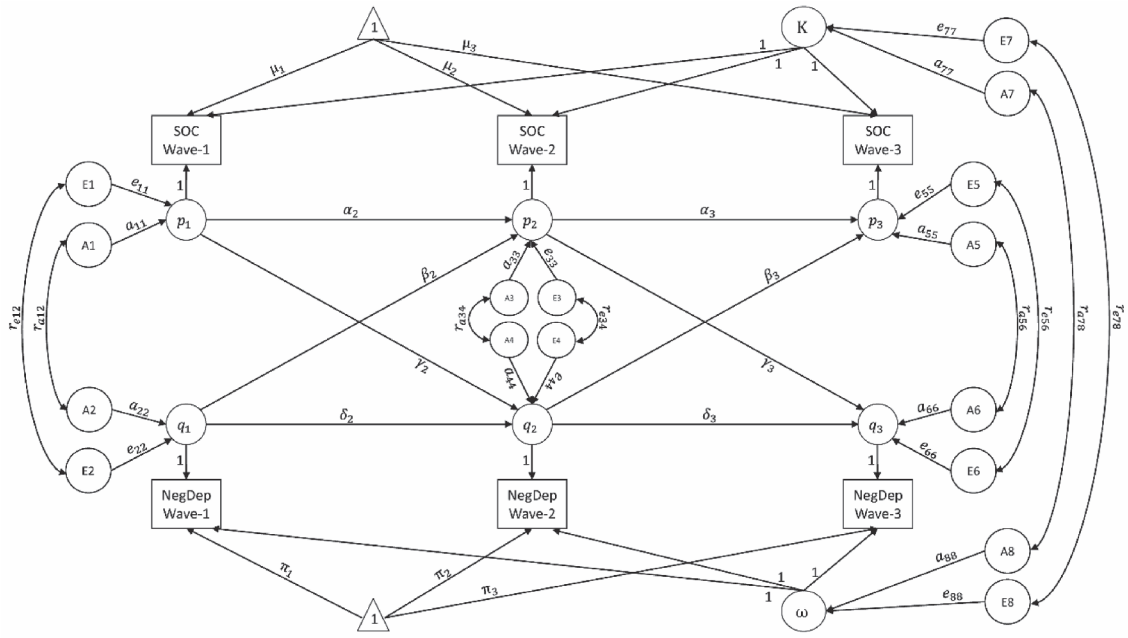

Fig. 1. Figurative Illustration of the Genetically Informative RI-CLPM for the Relationship between SOC and Negative Dependent Life Events. *Note.* For simplicity, the model is shown for one twin only and only additive genetic (A) and non-shared environmental (E) influences are shown. Triangles represent constants for the means, rectangles represent observed variables and circles represent latent variables. SOC = sense of coherence; NegDep = negative dependent life events; Wave−1, Wave−2 and Wave−3 = measurement occasions two years apart; μ_t and π_t = temporal grand means; K and ω = random intercept latent factors; p_t and q_t = within-person components; α_t and δ_t = autoregressive parameters; β_t and γ_t = cross-lagged parameters; A and a = latent additive genetic factors and paths; E and e = latent non-shared environmental factors and paths; r_a and r_e = additive genetic correlations and non-shared environmental correlations, respectively.

exist stable individual differences in mean level between persons in the phenotypes studied, the autoregressive parameters in the traditional CLPM fail to adequately account for this. This may lead to incorrect estimates of the cross-lagged parameters because the model does not separate the between-person variance from the within-person variance (Hamaker et al., 2015; Selig & Little, 2012). Hamaker et al. (2015) have proposed an alternative model, the random intercept cross-lagged panel model (RI-CLPM). This model extends the CLPM by including random intercepts that account for stable individual differences between persons (i.e., between-person variance). In this way, the cross-lagged parameters represent actual within-person processes (i.e., variance due to changes within individuals over time), which are the processes of main interest when studying reciprocal relations between variables. We first fitted three genetically informative RI-CLPMs to data, each with SOC and one cluster of life events. A graphic representation of the model with SOC and negative dependent life events is given in Fig. 1.

The RI-CLPMs were modelled following procedures as described by Hamaker et al. (2015). SOC_{it} and LE_{it} denote the measurements of SOC and life events at time point t for individual i. We modelled temporal grand means for SOC and life events (μ_t and π_t). Random intercepts (K_i and ω_i) were modelled to represent individuals' trait-like deviations from the temporal grand means. Factor loadings were constrained to one to reflect time-invariant effects. By including random intercepts, the RI-CLPM accounts for stable individual differences in mean levels of SOC and life events across the three measurement waves. The remaining variation in the data is attributed to within-person processes. Within-person components were modelled by specifying a latent variable for each observed variable (p_{it} and q_{it}), with all factor loadings constrained to one. These components (p_{it} and q_{it}), represent individuals' observed temporal deviations from their own expected score (i.e., $\mu_t + K_i$ and $\pi_t + \omega_i$). That is, individuals' time-specific deviations from their own stable level.

The autoregressive and cross-lagged paths were specified between the within-person components. The autoregressive parameters α_t and δ_t represent the amount of within-person carry-over effect. A positive α_t implies that individuals who experience stronger (weaker) SOC relative to their own stable level, are likely to experience stronger (weaker) SOC relative to their own stable level at the next measurement occasion as well. The same logic applies to δ_t. The cross-lagged parameters, β_t and γ_t, represent the degree by which within-person fluctuations in one construct predict fluctuations in another construct, after controlling for the carry-over stability effects. More specifically, a positive β_t implies that individuals who experience more (less) life events relative to their stable level of life events, are likely to experience stronger (weaker) SOC relative to their stable level of SOC at the next measurement occasion, after controlling for the carry-over stability effects in SOC. The same logic applies to γ_t.

Furthermore, we extended the RI-CLPM approach by partitioning the variance in the within-person components and the between-person components (i.e., random intercepts) into genetic and environmental sources of variance. All variances of the genetic and environmental latent factors were fixed to one and factor loadings were estimated. In addition, we modelled genetic and environmental correlations between within-time fluctuations in SOC and life events and between the random intercepts. At Wave−1, the genetic and environmental influences on p_1 and q_1 account for all within-person variance in SOC and life events. At Wave−2 and Wave−3, some of the within-person variance in SOC and life events are due to the individuals' previous state (i.e., influences from the previous age). In addition, the model allows for new sources of genetic and environmental influences at each follow-up assessment. The within-person variance in SOC and life events at Wave−2 and Wave−3 can be partitioned into four sources: (1) stability effects, (2) cross-lagged effects, (3) common effects and (4) residual effects. For example, within-person variance in SOC_2 can be partitioned into (1) genetic and environmental influences on within-person variance in SOC_1 contributing to within-person variance in SOC_2 (e.g., genetic influences: $\alpha_2{}^2 \times a_{11}{}^2$), (2) genetic and environmental influences unique to within-person variance in LE_1 contributing to within-person variance in SOC_2 (e.g., genetic influences: $\beta_2{}^2 \times a_{22}{}^2$), (3) genetic and environmental

Table 1
Descriptive statistics and phenotypic correlations.

Variable[a]	*n*	*M*	*SD*	1	2	3	4	5	6	7	8	9	10	11	12
1. Sex	2,878	0.56	0.50	–											
2. SOC_{W1}	2,567	5.12	1.28	−0.18***	–										
3. SOC_{W2}	1,911	4.98	1.27	−0.20***	0.46***	–									
4. SOC_{W3}	1,451	4.97	1.27	−0.15***	0.39***	0.47***	–								
5. $NegDep_{W1}$	2,567	2.32	2.06	0.19***	−0.53***	−0.32***	−0.23***	–							
6. $NegDep_{W2}$	1,915	2.49	2.12	0.21***	−0.37***	−0.48***	−0.33***	0.48***	–						
7. $NegDep_{W3}$	1,448	2.20	1.89	0.19***	−0.25***	−0.33***	−0.46***	0.33***	0.48***	–					
8. $NegInd_{W1}$	2,627	1.44	1.36	0.07***	−0.26***	−0.13***	−0.08**	0.36***	0.20***	0.12***	–				
9. $NegInd_{W2}$	1,918	1.46	1.43	0.08***	−0.19***	−0.27***	−0.19***	0.24***	0.40***	0.25***	0.22***	–			
10. $NegInd_{W3}$	1,453	1.39	1.37	0.10***	−0.14***	−0.14***	−0.25***	0.21***	0.26***	0.39***	0.18***	0.24***	–		
11. $PosDep_{W1}$	2,567	2.15	1.27	0.06**	−0.18***	−0.05*	0.00	0.31***	0.21***	0.15***	0.21***	0.07**	0.09**	–	
12. $PosDep_{W2}$	1,914	2.19	1.25	0.06*	−0.11***	−0.10***	−0.07*	0.18***	0.30***	0.19***	0.05*	0.22***	0.10***	0.27***	–
13. $PosDep_{W3}$	1,448	1.96	1.23	−0.02	0.01	−0.05	−0.07*	0.08**	0.18***	0.28***	−0.01	0.11***	0.18***	0.10***	0.29***

Note. SOC = sense of coherence; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events; W1, W2, W3 = Wave−1, Wave−2 and Wave−3, respectively. [a] Sex coded 0 = male, 1 = female; SOC coded from 1 to 7; NegDep coded from 0 to 14; NegInd coded from 0 to 19; PosDep coded from 0 to 5.
* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 2
Cross-twin within-trait and cross-twin cross-trait correlations.

Variable	1	2	3	4	5	6	7	8	9	10	11	12
						MZ correlations						
1. SOC_{W1}	0.48***											
2. SOC_{W2}	0.34***	0.40***										
3. SOC_{W3}	0.29***	0.31***	0.44***									
4. $NegDep_{W1}$	−0.38***	−0.29***	−0.22***	0.57***								
5. $NegDep_{W2}$	−0.29***	−0.33***	−0.23***	0.43***	0.55***							
6. $NegDep_{W3}$	−0.12*	−0.24***	−0.26***	0.31***	0.32***	0.39***						
7. $NegInd_{W1}$	−0.21***	−0.09*	−0.05	0.28***	0.12**	0.06	0.63***					
8. $NegInd_{W2}$	−0.16***	−0.17***	−0.11*	0.22***	0.28***	0.10*	0.16***	0.54***				
9. $NegInd_{W3}$	−0.03	−0.01	−0.16**	0.04	0.15**	0.22***	0.05	0.10*	0.48***			
10. $PosDep_{W1}$	−0.17***	−0.12**	−0.05	0.29***	0.22***	0.13**	0.21***	0.11**	0.06	0.53***		
11. $PosDep_{W2}$	−0.09*	−0.13***	−0.06	0.19***	0.24***	0.16**	0.04	0.16***	0.04	0.26***	0.51***	
12. $PosDep_{W3}$	0.12*	−0.02	−0.05	0.00	0.05	0.11*	−0.08	0.03	0.02	0.10*	0.25***	0.42***
						DZ correlations						
1. SOC_{W1}	0.22***											
2. SOC_{W2}	0.10**	0.07*										
3. SOC_{W3}	0.15***	0.15***	0.19***									
4. $NegDep_{W1}$	−0.18***	−0.10**	−0.14***	0.27***								
5. $NegDep_{W2}$	−0.15***	−0.08*	−0.15***	0.20***	0.21***							
6. $NegDep_{W3}$	−0.09*	−0.14***	−0.18***	0.16***	0.24***	0.34***						
7. $NegInd_{W1}$	−0.14***	−0.05	−0.07*	0.19***	0.13***	0.08*	0.49***					
8. $NegInd_{W2}$	−0.13***	−0.10**	−0.11**	0.16***	0.17***	0.19***	0.14***	0.41***				
9. $NegInd_{W3}$	−0.06	−08*	−0.16***	0.04	0.11**	0.22***	0.11**	0.17***	0.43***			
10. $PosDep_{W1}$	−0.08**	0.01	−0.02	0.11***	0.10**	0.06	0.12***	0.03	0.06	0.28***		
11. $PosDep_{W2}$	−0.05	−0.02	−0.07	0.07*	0.09**	0.11**	0.00	0.09**	0.09*	0.10**	0.27***	
12. $PosDep_{W3}$	0.00	0.03	−0.03	0.00	0.06	0.11**	−0.04	0.01	0.11**	0.01	0.08*	0.21***

Note. SOC = Sense of coherence; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events; W1, W2, W3 = Wave−1, Wave−2 and Wave−3, respectively.
* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

influences common to within-person variance in both SOC_1 and LE_1 (e.g., genetic influences: $2 \times [\alpha_2 \times a_{11} \times r_{a12} \times a_{22} \times \beta_2]$) and (4) genetic and environmental influences unique to within-person variance in SOC_2 (e.g., genetic influences: $a_{33}{}^2$).

The significance of the autoregressive and cross-lagged paths was tested by fixing all members of the parameter sets α, δ, γ and β to zero, one set at a time (i.e., four reduced models). The reduced models were compared to the full RI-CLPM by likelihood ratio χ^2 (chi square) tests. The difference in −2 log likelihood (−2ll) between two nested models is asymptotically distributed as a χ^2 with degrees of freedom equal to the difference between the number of estimated parameters in the full and in the restricted model. A non-significant χ^2 difference indicates that the restricted model should be accepted over the full model (i.e., the restricted model does not lead to a substantial loss of fit).

Finally, we fitted three genetically informative CLPMs to data, each with SOC and one cluster of life events. The CLPM can be obtained by removing the random intercepts from the RI-CLPM. Comparison of model fit between the models was examined using AIC (Akaike, 1987) and BIC (Raftery, 1995), with lower values indicating better model fit. Absolute model fit was evaluated by inspecting the comparative fit index (CFI), the Tucker-Lewis index (TLI) and the root mean square error of approximation (RMSEA). CFI and TLI values greater than 0.95 and RMSEA values < 0.06 were considered as indicating good model fit (Hu & Bentler, 1999).

3. Results

3.1. Descriptive statistics and correlations

Descriptive statistics and Pearson correlations between variables are presented in Table 1. Means, standard deviations and number of participants for the study variables by zygosity are provided in Table A2.

Table 3
Fit statistics of the Cholesky decomposition models.

Model	Model fit (AIC)[a]			
	ACE	AE	CE	E
Sense of coherence	6727.0	**6715.0**	6760.8	6927.4
Negative dependent life events	12089.5	**12082.6**	12139.3	12464.3
Negative independent life events	**7938.1**	7993.4	7947.9	8728.2
Positive dependent life events	7050.5	**7040.8**	7088.2	7427.0

Note. A = additive genes; C = shared environment; E = non-shared environment.
[a] Akaike's information criterion for the univariate Cholesky ACE, AE, CE and E models, with the best fitting model indicated in bold.

Table 4
Parameter estimates (95% CI) from the best fitting Cholesky models.

Measure	Additive genetic effects (A)	Shared environmental effects (C)	Non-shared environmental effects (E)
Sense of coherence$_{W1}$	0.47 (0.41, 0.53)	–	0.53 (0.47, 0.59)
Sense of coherence$_{W2}$	0.31 (0.23, 0.39)	–	0.69 (0.61, 0.77)
Sense of coherence$_{W3}$	0.40 (0.30, 0.48)	–	0.60 (0.52, 0.70)
Negative dependent life events$_{W1}$	0.55 (0.50, 0.60)	–	0.45 (0.40, 0.50)
Negative dependent life events$_{W2}$	0.50 (0.43, 0.56)	–	0.50 (0.44, 0.57)
Negative dependent life events$_{W3}$	0.47 (0.38, 0.54)	–	0.53 (0.46, 0.62)
Negative independent life events$_{W1}$	0.22 (0.09, 0.36)	0.40 (0.28, 0.50)	0.38 (0.34, 0.44)
Negative independent life events$_{W2}$	0.25 (0.06, 0.43)	0.29 (0.14, 0.43)	0.46 (0.40, 0.53)
Negative independent life events$_{W3}$	0.12 (0.00, 0.35)	0.39 (0.20, 0.52)	0.49 (0.41, 0.58)
Positive dependent life events$_{W1}$	0.52 (0.46, 0.57)	–	0.48 (0.43, 0.54)
Positive dependent life events$_{W2}$	0.50 (0.43, 0.57)	–	0.50 (0.43, 0.57)
Positive dependent life events$_{W3}$	0.43 (0.34, 0.51)	–	0.57 (0.49, 0.66)

Note. W1, W2, W3 = Wave−1, Wave−2 and Wave−3, respectively.

SOC and negative dependent life events were moderately stable over time, whereas negative independent life events and positive dependent life events showed less stability. As to the associations between life events and SOC, there were moderate negative within-time correlations between negative dependent life events and SOC, whereas the cross-time correlations were lower. Weak negative correlations were observed between negative independent life events and SOC, both cross-sectionally and over time. The correlations between positive dependent life events and SOC were even weaker, and surprisingly, these correlations were also negative.

When excluding the events not included in the LEQ-A (i.e., only analyzing the events from the LEQ-A), the correlations between the three clusters of life events and SOC were similar but slightly lower compared to those provided in Table 1 (see Table A3). We also calculated a sum score of all negative life events, both dependent and independent. The correlations between this total score of negative life events and SOC are provided in Table A4. Overall, these correlations were slightly lower than the correlations between negative dependent life events and SOC provided in Table 1.

Cross-twin correlations are presented in Table 2. The correlations of main interest for the genetic analyses include the cross-twin within-trait correlations and the cross-twin cross-trait correlations between SOC and the three clusters of life events. Overall, the considerably stronger resemblance within MZ pairs than DZ pairs, with the DZ correlations about half the size of the MZ correlations, suggest additive genetic influences with negligible influence of shared environmental factors. An exception is negative independent life events, which seem to have a substantial influence of shared environmental factors.

3.2. Cholesky decomposition models

Cholesky decomposition models were fitted to data to estimate the genetic and environmental contributions to variance in the measured constructs. Table 3 presents the results of fitting these models. Consistent with the pattern of cross-twin correlations, the AE model could be accepted over the full ACE model (i.e., indicated by the lowest AIC value) for SOC, negative dependent life events and positive dependent life events. For negative independent live events, an ACE model provided the best fit of data.

Standardized parameter estimates from the best fitting models are presented in Table 4. SOC and dependent life events were moderately heritable. Negative independent life events also seem to be somewhat heritable, but a substantial proportion of individual differences in negative independent life events was due to shared environmental influences. Of note, measurement error is also included in the estimates of the non-shared environmental influences, which may lead to an underestimation of the heritability estimates (and possibly the estimates of the shared environment).

Table 5
Fit Statistics of the Genetically Informative Cross-Lagged Panel Models.

Model	*df*	*ep*	AIC	BIC	RMSEA (95% CI)	CFI	TLI
SOC and NegDep							
RI-CLPM	11,821	46	17698.0	−45405.3	0.015 (0.007, 0.021)	0.986	0.987
CLPM	11,827	38	17825.9	−45309.4	0.028 (0.023, 0.033)	0.949	0.955
SOC and NegInd							
RI-CLPM	11,885	50	14453.2	−48991.8	0.007 (0.000, 0.016)	0.995	0.995
CLPM	11,892	41	14542.5	−48939.9	0.022 (0.016, 0.027)	0.954	0.958
SOC and PosDep							
RI-CLPM	11,820	46	13776.5	−49321.4	0.008 (0.000, 0.016)	0.992	0.992
CLPM	11,826	38	13863.9	−49266.1	0.022 (0.016, 0.027)	0.938	0.945

Note. SOC = sense of coherence; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events; RI-CLPM = random intercept cross-lagged panel model; CLPM = cross-lagged panel model; *df* = degrees of freedom associated with the model; *ep* = number of parameters estimated; AIC = Akaike's information criterion; BIC = Bayesian information criterion; RMSEA = Root Mean Square Error of Approximation; CFI = Comparative Fit Index; TLI = Tucker-Lewis Index.

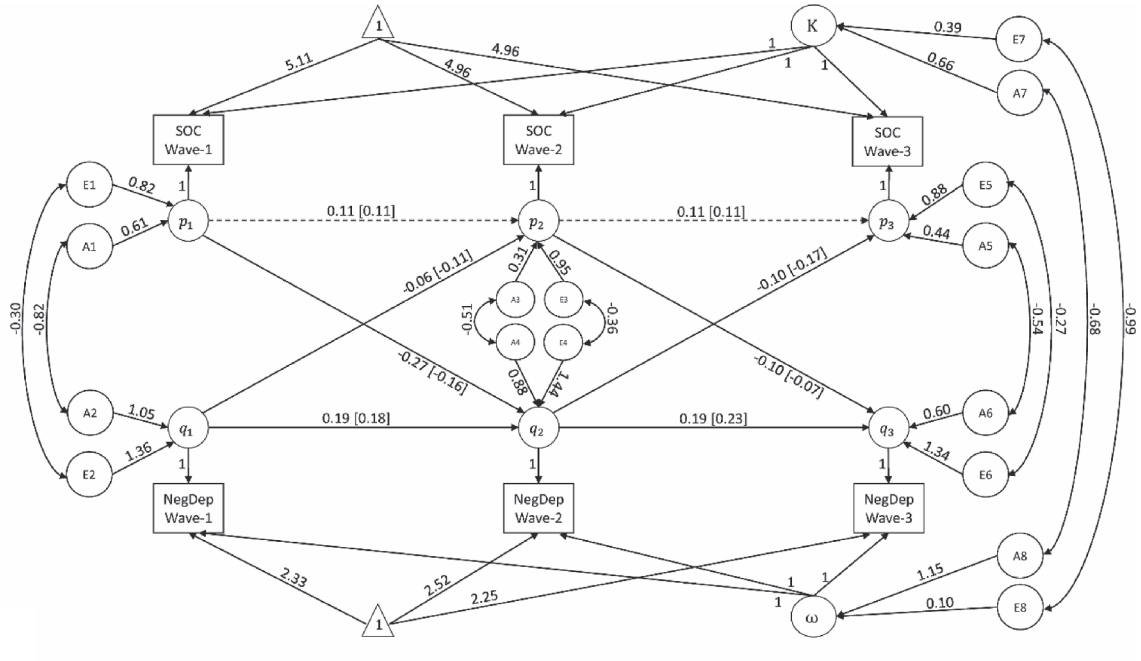

Fig. 2. Genetically Informative RI-CLPM with Unstandardized Coefficients for the Longitudinal Relationship between SOC and Negative Dependent Life Events. *Note.* Standardized coefficients are given in square brackets. Dashed lines indicate non-significant paths. SOC = sense of coherence; NegDep = negative dependent life events. See Fig. 1 for a more detailed description of model parameters.

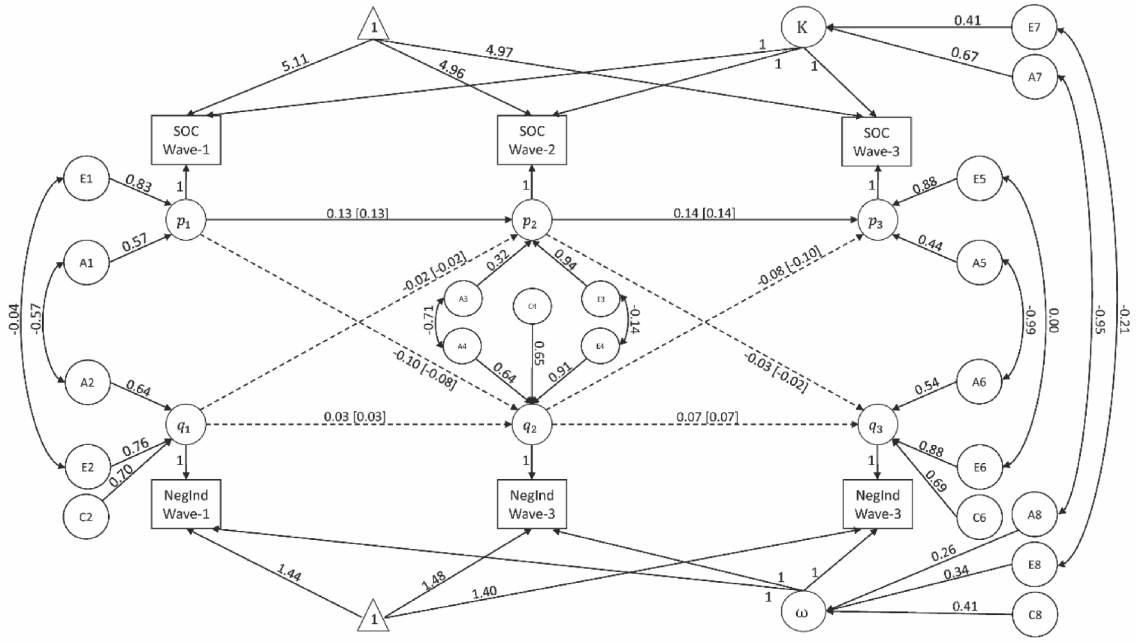

Fig. 3. Genetically Informative RI-CLPM with Unstandardized Coefficients for the Longitudinal Relationship between SOC and Negative Independent Life Events. *Note.* Standardized coefficients are given in square brackets. Dashed lines indicate non-significant paths. SOC = sense of coherence; NegInd = negative independent life events. See Fig. 1 for a more detailed description of model parameters.

3.3. Genetically informative cross-lagged panel models

Based on the best fitting Cholesky models, we included only A and E influences in the variance decomposition of SOC, negative dependent life events and positive dependent life events. In the variance decomposition of negative independent life events, we estimated all three sources of variance (i.e., A, C and E). Table 5 presents fit statistics from the genetically informative cross-lagged panel models. The RI-CLPMs provided a better fit to data compared to the traditional CLPMs (i.e., based on the lowest AIC and BIC values, the lowest RMSEA values and the highest CFI and TLI values). This indicates that there are stable individual differences between persons in SOC and/or life events, implying that it is important to account for stable between-person differences in the measured constructs before examining the reciprocal relations between them. Thus, only the results of the RI-CLPMs will be presented and discussed.

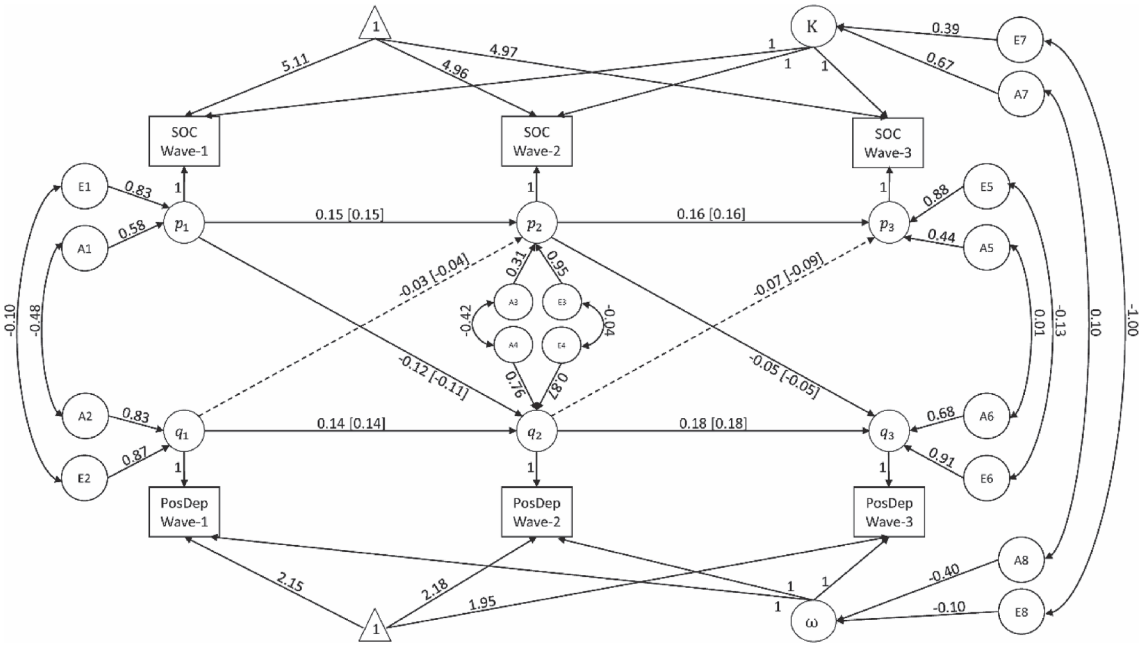

Fig. 4. Genetically Informative RI-CLPM with Unstandardized Coefficients for the Longitudinal Relationship between SOC and Positive Dependent Life Events. *Note.* Standardized coefficients are given in square brackets. Dashed lines indicate non-significant paths. SOC = sense of coherence; PosDep = positive dependent life events. See Fig. 1 for a more detailed description of model parameters.

Table 6
Proportion of variance in the measured constructs explained by the stable trait factors.

Measure	Wave−1	Wave−2	Wave−3
Sense of coherence	36.9 %[a]	37.1 %[a]	37.4 %[a]
Negative dependent life events	31.1%	29.9%	36.8%
Negative independent life events	19.2%	17.4%	18.5%
Positive dependent life events	10.5%	10.8%	11.3%

Note. [a] Mean based on the three RI-CLPMs.

Table 7
Proportion of variance in the stable trait factors due to additive genetic, shared environmental and non-shared environmental influences.

Measure	Additive genetic influences (A)	Shared environmental influences (C)	Non-shared environmental influences (E)
Sense of coherence	73.9%[a]	–	26.1%[a]
Negative dependent life events	99.2%	–	0.8%
Negative independent life events	19.2%	47.9%	32.9%
Positive dependent life events	94.1%	–	5.9%

Note. [a] Mean based on the three RI-CLPMs.

Unstandardized estimates derived from the genetically informative RI-CLPMs for the relationship between SOC and the three clusters of life events are displayed in Fig. 2, Fig. 3 and Fig. 4 (unstandardized estimated with standard errors are provided in Table A5). Standardized estimates of the autoregressive and cross-lagged paths are displayed in square brackets to enable comparison.

3.3.1. Stability in SOC and life events

Table 6 displays the proportion of variance in the measured constructs at each measurement wave explained by the stable trait factors (i.e., random intercepts). To calculate this, we divided the squared variance in the random intercept by the squared total variance in the measured construct at the given measurement wave. At Wave−1, the variance in p_1 and q_1 and the variance in the random intercepts (K and ω) contribute to the total variance (e.g., proportion of variance in SOC at Wave−1 accounted for by the random intercept = $a_{77}^2 + e_{77}^2$ / $a_{11}^2 + e_{11}^2 + a_{77}^2 + e_{77}^2$, see Fig. 1). At Wave−2 and Wave−3, stability effects, cross-lagged effects and common effects also contribute to the total variance (for further explanation, see Section 2.5. Statistical Analysis). As expected from the cross-time within-trait correlations, both SOC and negative dependent life events were moderately stable over time, whereas negative independent life events and positive dependent life events showed less stability.

Table 7 displays the proportion of variance in the stable trait factors attributable to additive genetic, shared environmental and non-shared environmental influences. To calculate this, we divided the squared genetic (and environmental) variance in the random intercept by the squared total variance in the random intercept (e.g., proportion of variance in the stable trait factor of SOC due to additive genetic influences = a_{77}^2 / $a_{77}^2 + e_{77}^2$, see Fig. 1). As expected from the cross-twin correlations, most of the stability in SOC and dependent life events were attributable to additive genetic influences whereas the stability in negative independent life events was mainly due to shared environmental influences.

3.3.2. The relationship between SOC and life events

With respect to the relationship between SOC and negative dependent life events, there was a strong negative genetic correlation between stable traits of SOC and negative dependent life events (−0.68), indicating shared genetic influences on the stability of these constructs. The non-shared environmental correlation between the two stable traits was estimated to −0.99. However, this correlation has no practical meaning because the non-shared environmental influences on the stable trait factor of negative dependent life events were negligible (0.8%, see Table 7). Furthermore, there were statistically significant negative cross-lagged effects from negative dependent life events to SOC and vice versa (i.e., significant changes in χ^2 when dropping these parameters from the

model). This indicates that individuals who experienced more (less) negative dependent life events than they typically do (i.e., quantitative deviations from the persons 'stable' amount of negative dependent life events) were likely to score lower (higher) on SOC than they typically do at the next assessment, and vice versa. Although the cross-lagged coefficients were significant, the effects were weak in magnitude. By squaring the standardized cross-lagged regression coefficients, fluctuations in negative dependent life events explained 1.2% (Wave−1 to Wave−2) and 2.9% (Wave−2 to Wave−3) of the fluctuations in SOC at the next measurement occasion whereas fluctuations in SOC explained 2.6% (Wave−1 to Wave−2) and 0.5% (Wave−2 to Wave−3) of the fluctuations in negative dependent life events at the next assessment.

As to the relationship between SOC and negative independent life events, there was a strong genetic correlation between stable traits of SOC and negative independent life events (−0.95). This may indicate shared genetic influence on the stability of these constructs, but the practical importance of this correlation is modest because most of the stability in negative independent life events was attributable to shared environmental influences (see Table 7). None of the cross-lagged coefficients was statistically significant, indicating that fluctuations in SOC were not predicted by fluctuations in negative independent life events two years earlier, or vice versa.

In addition to analyze negative dependent and negative independent life events separately, we also fitted a model to SOC and all negative life events considered together (see Fig. A1). Of course, the results from this analysis provided a less nuanced picture of the stability of negative life events and the relative proportion of genetic and environmental factors influencing this stability of reoccurrence of life events. However, results from this model showed similar results regarding the relationship between SOC and negative life events as when analyzing dependent and independent life events separately. That is, the cross lagged effects were weak in magnitude, only explaining a negligible proportion of the within-person variance in the measured constructs at each measurement occasion.

The genetic correlation between stable traits of SOC and positive dependent life events was almost zero (0.10), indicating that different genes are operating creating stability in these constructs. The non-shared environmental correlation between the two stable traits was estimated to −1.00. However, the practical importance of this correlation is negligible as the non-shared environmental influences on the stable trait factor of positive dependent life events were very weak in magnitude, explaining only 6% of the variance (see Table 7). The cross-lagged effects from SOC to positive dependent life events were statistically significant, whereas the cross-lagged effects from positive dependent life events to SOC were statistically non-significant. Although this might indicate a unidirectional effect from SOC to positive dependent life events, the effects were weak in magnitude. Fluctuations in SOC only explained 1.2% (Wave−1 to Wave−2) and 0.3% (Wave−2 to Wave−3) of the fluctuations in positive dependent life events two years later.

Finally, in all three models the concurrent genetic correlations between SOC and life events within each measurement occasion were greater in magnitude compared to the non-shared environmental correlations, indicating that the within-time correlations between life events and SOC were mainly due to shared genetic influences.

4. Discussion

The main purpose of the present study was to examine the longitudinal relationship between life events and SOC. Previous studies have shown that measured environments like life events are partly influenced by genetic factors (Kendler & Baker, 2007). Similarly, we found substantial genetic variance in measured life events, with heritability estimates across the three study waves ranging from 47% to 55% for negative dependent life events, from 43% to 52% for positive dependent life events and from 12% to 25% for negative independent life events. These results corroborate prior work, finding higher heritability of dependent life events compared to independent life events (Bemmels et al., 2008; Billig et al., 1996; Plomin et al., 1990). The heritability of SOC ranged from 31% to 47% across the three study waves, which are similar to the heritability estimates of SOC found in prior studies (Hansson et al., 2008; Silventoinen et al., 2014) and to heritability estimates reported for human traits in general (Polderman et al., 2015).

The rationale behind a classification of life events into 'dependent' and 'independent' events is that the association between dependent life events and some outcome variable is assumed to be confounded by a person's behavior, which may be genetically influenced. Therefore, although dependent life events may be causally related to a certain outcome, the association between dependent life events and the outcome may be confounded by genetic influences affecting them both. Independent life events, on the other hand, are considered outside a person's control and are therefore more likely to have direct/causal effects on the outcome in question (Kendler, Karkowski, & Prescott, 1999).

Genetic influences on environmental exposures such as life events give rise to gene-environment correlations. That is, a person's genetically influenced behaviors may play a role in the person's choice of environments and exposure to life events (i.e., active gene-environment correlation) or elicit certain reactions from the environment (i.e., evocative gene-environment correlation). For example, people with a genetic predisposition to more difficult temperament may select into risky environments where negative life events are more likely to occur or elicit negative reactions from parents resulting in many conflicts. Finding genetic influence on life events classified as independent may reflect the difficulty of finding clear criteria for categorization. It may also suggest that most life events are not exclusively independent. In addition, the same event may for some be dependent (e.g., arguments with a sibling due to the interviewee's behavior) and for others independent (e.g., arguments with a sibling due to the sibling's oppositional behavior). However, analyzing dependent and independent life events separately is still important, both conceptually for the reasons described in the paragraph above, and because dependent life events, potentially only genetically related to an outcome of interest, may obscure potential environmental effects of independent events on the outcome.

Results from the genetically informative RI-CLPM analyses suggest that SOC is relatively stable in adolescence. More specifically, nearly 40% of the total variance in SOC at each measurement occasion was explained by the stable trait factor. The amount of negative life events the participants experienced also seem to be somewhat stable, with approximately 30% (negative dependent) and 20% (negative independent) of the total variance at each measurement occasion being explained by the stable trait factor. Positive dependent life events showed less stability, with only about 10% of the total variance at each measurement occasion being explained by the stable trait factor. Most of the stability in SOC and dependent life events was attributable to additive genetic influences, i.e., genetic influences are the main reason why SOC is stable and why dependent life events reoccur. In contrast, shared environmental influences explained most of the stability of negative independent life events. The proportion of stable variance in life events found in this study and the finding that the recurrence of life events is mainly due to genetic influences corroborate findings from a German twin study (Kandler et al., 2012).

If we look at the within-person fluctuations in SOC, the results indicate that both genetic and non-shared environmental influences contribute to time-specific changes. The findings that most of the stability in SOC was due to genetic influences and that both genetic and environmental factors contribute to change in SOC are in line with studies that have examined stability and change in personality in adolescence (Blonigen, Carlson, Hicks, Krueger, & Iacono, 2008; Bratko & Butkovic, 2007; Kawamoto & Endo, 2015, 2019).

As to the relationship between SOC and life events, our results indicate that life events do not seem to predict change in SOC to a substantial degree, or vice versa. Although the analyses revealed statistically significant negative reciprocal longitudinal effects between negative dependent life events and SOC and statistically significant

negative unidirectional longitudinal effects from SOC to positive dependent life events, the effects were weak in magnitude. More specifically, fluctuations in negative dependent life events explained at most 2.9% of fluctuations in SOC two years later, and fluctuations in SOC explained at most 2.6% (negative dependent) and 1.2% (positive dependent) of fluctuations in life events two years later. The negative association between SOC and positive dependent life events may seem strange at first sight. However, consistent with previous studies, the correlation analysis showed that all three clusters of life events were positively correlated with each other, indicating that individuals who reported more life events of one kind, also tended to report more life events of another kind (Kandler et al., 2012; Magnus, Diener, Fujita, & Pavot, 1993; Plomin et al., 1990). Thus, experience of more positive life events also means experience of more negative life events. For example, a person who is active, outgoing and open to new experiences may experience more life events, both positive and negative (Magnus et al., 1993). Finding genetic influences on measured life events may explain a part of this covariance between life events clusters, in which genetic factors influencing a person's level of activity and openness to new experiences increase the frequency of exposure to both positive and negative life events (Kandler et al., 2012).

Overall, the nature of the phenotypic associations between life events and SOC seems mainly to be accounted for by shared genetic influences. More specifically, the phenotypic associations between negative dependent life events and SOC seem almost exclusively to be explained by the fact that they share common genes that influence the stability of both constructs. In addition, within-time fluctuations in negative dependent life events and SOC were correlated with each other, and these concurrent associations were to a greater extent explained by shared genetic influences compared to shared non-shared environmental influences. That is, people who experienced more (less) negative dependent life events than they typically do at a specific time point, also tended to experience weaker (stronger) SOC relative to their stable level at the same time point, and these concurrent associations were largely attributable to shared genetic influences. Such within-time associations were also observed between SOC and both negative independent and positive dependent life events. Similarly, these concurrent associations were mainly due to shared genetic influences. To sum up, SOC and life events share common genetic influences. For example, some people may be more likely to experience and/or report negative life events due to their genetic predisposition to perceive the world as chaotic and unmanageable which characterizes a weak SOC.

To our knowledge, this is the first genetically informative study of the relationship between life events and SOC. Hence, it is important to validate the current findings by replicating the results in studies with data from other populations (e.g., other countries and age groups). Future studies may also expand on these results by measuring other life events to see whether the results generalize to different life events. However, genetically informative studies on the relationship between life events and related constructs like personality have reported similar results as the results in the present study. Several studies have suggested that genetically influenced personality traits may be potential candidates creating genetic variance in life events (Billig et al., 1996; Kandler et al., 2012; Saudino, Pedersen, Lichtenstein, McClearn, & Plomin, 1997). These studies and the results from the present study suggest that we need to think differently about the causal structure of life events. Measures of the environment, like life events, are influenced by genes and we need to start looking for the genetics behind the observed environment.

4.1. Limitations and strengths

The results of this study should be considered in light of some possible limitations. First, related to the discussion regarding the categorization of life events above, some of the life events may not be clearly classified as independent or dependent. For example, "moving schools" may for some individuals be classified as an independent life event whereas for others moving schools may be a dependent life event. However, the life events were classified according to classifications used in previous studies (Masten et al., 1994) and to the best of our knowledge. Second, the finding that life events do not explain much variance in SOC (i.e., the cross-lagged effects were small in magnitude) does not mean that people experiencing extreme life events do not change their SOC. For example, an extreme but infrequent life event may have a huge effect on SOC, but only a small proportion of the variance in SOC is explained by this infrequent but important life event.

Furthermore, there are several assumptions related to the classical twin design, potentially threatening the validity of twin studies. First, the equal environment assumption (EEA) assumes that MZ and DZ twins are exposed to shared environmental factors to the same degree. If MZ twins are being treated more similarly or spend more time together than DZ twins, the EEA may be violated. If the EEA has not been met, the higher correlations in MZ twins compared to DZ twins may be due to environmental influences rather than genetic influences, thus overestimating the effect of the genetic influences. However, existing empirical studies have shown that the EEA generally holds (Derks, Dolan, & Boomsma, 2006; Kendler, 1993; Kendler, Neale, Kessler, Heath, & Eaves, 1993; Plomin, DeFries, Knopik, & Neiderhiser, 2013; Tambs, Harris, & Magnus, 1995). A second assumption in twin modelling is that DZ twins share half of their segregating genes. This assumption is based on random mating (i.e., parents do not share genes beyond what is expected by random chance). However, people tend to fall in love and have children with people that resemble themselves on domains like education, religion, attitudes and socioeconomic status (Neale & Maes, 2004). If this assortative mating leads to genetic similarity in parents, DZ twins would share more than 50 % of their segregating genes, thus increasing the genetic similarity between MZ and DZ twins. Consequently, assortative mating tends to overestimate shared environmental effects and underestimate genetic effects. A third assumption is that there is no gene-environment interaction. Gene-environment interaction occurs when effects of the environment depend on an individual's genotype. Depending on the nature of the gene-environment interaction, its presence can lead to biased estimates of both additive genetic, shared environmental and non-shared environmental factors (Posthuma et al., 2003). Gene by non-shared environment interactions will overestimate the effects of the non-shared environment, whereas gene by shared environment interactions will overestimate the effects of both additive genetic and shared environmental factors.

This study also has several strengths, including the use of a longitudinal design with a genetically informative sample. Twin studies represent a powerful design to partition the phenotypic variance into genetic and environmental influences, and thereby examination of the nature (i.e., genetic and/or environmental) of the association between phenotypes of interest. The large sample size in this study provided statistical power to run separate analyses of clusters of life events. From previous studies, we know that various life events may have different effects on an outcome variable, depending on event independence. Thus, grouping of life events with regards to event independence may be crucial when studying the effect of life events on an outcome variable. Furthermore, the data come from a population-based sample. This strengthens the possibility of generalization of the results. The validity of conclusions drawn from twin studies rely on the assumption that twins are representative of the general population. Several studies have confirmed this assumption. Twins have been found not to differ from singletons with regards to personality, cognitive abilities, lifestyle characteristics and both mental and somatic health (Johnson, Krueger, Bouchard, & McGue, 2012; Kendler, Martin, Heath, & Eaves, 1995; Nilsen, Bergsjø, & Nome, 1984; Posthuma, Geus, Bleichrodt, & Boomsma, 2000). Participation bias also represents a threat to generalization of findings. For instance, persons with poorer health are shown to be less likely to participate in population-based health studies (Knudsen, Hotopf, Skogen, Øverland, & Mykletun, 2010). However,

participation bias is probably more problematic for the validity of studies of prevalence compared to studies like this which focus on associations between variables (Knudsen et al., 2010). Moreover, the analysis of recruitment and dropout of the twin material used in this study showed that attrition did not influence the heritability estimates (Torgersen & Waaktaar, 2019).

Authors' Contributions.

Eirunn Skaug conducted the data analyses and wrote the original draft. Nikolai O. Czajkowski contributed to the data analyses, interpretation of data and provided critical feedback on drafts. Trine Waaktaar collected the data and provided critical feedback on drafts. Svenn Torgersen collected the data, contributed to the interpretation of data and provided critical feedback on drafts. All authors approved the final version of the manuscript for submission.

Author note

This work was supported by the Department of Psychology, University of Oslo and by the Research Council of Norway [grant numbers 170089, 213722, 213760]. The study was approved by the Norwegian Data Inspectorate and the Regional Committees for Medical and Health Research Ethics, ref. 2015/4 (19661). RBUP, Eastern and Southern Norway contributed to data collection. The funding sources were not involved in the data analysis and interpretation of data, in writing the manuscript or in the decision to submit the manuscript for publication. None of the authors have any conflicts of interest to disclose. This study was not pre-registered.

Authors' Contributions

Eirunn Skaug conducted the data analyses and wrote the original draft. Nikolai O. Czajkowski contributed to the data analyses, interpretation of data and provided critical feedback on drafts. Trine Waaktaar collected the data and provided critical feedback on drafts. Svenn Torgersen collected the data, contributed to the interpretation of data and provided critical feedback on drafts. All authors approved the final version of the manuscript for submission.

Data Availability

Data collection was preapproved in 2005 by the Norwegian Data Protection Authority (DPA) under a clause of 20 years individual data protection and subsequent data deletion or anonymization. Thus, anonymized data may be requested after 2025.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

None.

Appendix

Table A1
The life events scale.

Negative dependent life events
I had an important change in physical appearance, which upset me (acne, glasses, physical development, etc.)[a]
I was a victim of violence (mugging, sexual abuse, robbery)[a]
I was disappointed by a friend
I was disappointed by someone in the family
I did not get into a group or activity that I wanted to get into (music group, sports team, theater, etc.)[a]
I had major problems with a teacher
I did much worse than I expected in an important exam or course[a]
I had less contact with one of my parents[a]
I had many arguments with my siblings[a]
I had many arguments with my parents[a]
I was bullied by other pupils/adolescents
I broke up with a girlfriend/boyfriend[a]
I had an abortion (girls) / my girlfriend had an abortion (boys)
I lost a close friend[a]
Negative independent life events
I lost a pet
I changed schools[a]
I became seriously ill or was injured[a]
At least one parent or another family member became seriously ill or was injured[a]
One of my parents died[a]
A brother or sister died[a]
Another family member died[a]
One of my close friends died[a]
Mom or Dad's friend moved in with us[a]
A member of my family ran away from home[a]
My parents divorced, moved apart[a]
One of my parents had problems at work[a]
One parent lost his or her job[a]
My mother began to work[a]
There has been a change in a parent's job so that my parent is away from home more often[a]
The family financial situation was difficult[a]
There was some damage or loss of family property (such as apartment, house, car or bike)[a]
There were many arguments between the adults[a]
Someone in the family had problems with the police[a]
Positive dependent life events
I received a special award (trophy, diploma etc.) for something done at school[a]
I became more popular with my friends
I joined a fun group of friends
I got a boyfriend/girlfriend[a]
I got a new friend

Note. [a] Question from the Life Event Questionnaire for Adolescents (LEQ-A; Masten et al., 1994). The wording in some of the questions were slightly changed from the LEQ-A.

Table A2
Means and number of participants for the study variables, by sex and zygosity.

Measure	MZM	DZM	MZF	DZF	DZOS
SOC_{W1}	5.45(1.10)	5.43(1.18)	5.03(1.33)	4.78(1.32)	5.08(1.27)
	$n = 391$	$n = 370$	$n = 587$	$n = 481$	$n = 738$
SOC_{W2}	5.32(1.14)	5.30(1.19)	4.75(1.34)	4.73(1.31)	5.00(1.21)
	$n = 266$	$n = 263$	$n = 452$	$n = 359$	$n = 571$
SOC_{W3}	5.16(1.22)	5.46(1.06)	4.87(1.30)	4.81(1.31)	4.88(1.26)
	$n = 192$	$n = 185$	$n = 366$	$n = 302$	$n = 406$
$NegDep_{W1}$	1.82(1.92)	1.84(1.77)	2.59(2.09)	2.73(2.07)	2.35(2.14)
	$n = 389$	$n = 370$	$n = 587$	$n = 480$	$n = 741$
$NegDep_{W2}$	1.90(1.92)	1.79(1.77)	2.92(2.25)	2.89(2.22)	2.49(2.04)
	$n = 267$	$n = 263$	$n = 453$	$n = 359$	$n = 573$
$NegDep_{W3}$	1.83(1.73)	1.55(1.54)	2.29(1.89)	2.64(2.12)	2.25(1.82)
	$n = 190$	$n = 185$	$n = 365$	$n = 302$	$n = 406$
$NegInd_{W1}$	1.31(1.34)	1.33(1.26)	1.53(1.42)	1.56(1.38)	1.40(1.34)
	$n = 403$	$n = 385$	$n = 593$	$n = 486$	$n = 760$
$NegInd_{W2}$	1.37(1.47)	1.31(1.40)	1.53(1.40)	1.73(1.52)	1.36(1.36)
	$n = 267$	$n = 262$	$n = 453$	$n = 360$	$n = 576$
$NegInd_{W3}$	1.27(1.34)	1.12(1.14)	1.39(1.40)	1.61(1.41)	1.40(1.40)
	$n = 191$	$n = 187$	$n = 365$	$n = 303$	$n = 407$
$PosDep_{W1}$	1.97(1.28)	2.03(1.31)	2.13(1.30)	2.26(1.16)	2.23(1.27)
	$n = 389$	$n = 370$	$n = 587$	$n = 480$	$n = 741$
$PosDep_{W2}$	2.12(1.26)	1.92(1.30)	2.18(1.28)	2.33(1.20)	2.26(1.23)
	$n = 267$	$n = 262$	$n = 453$	$n = 359$	$n = 573$
$PosDep_{W3}$	1.95(1.17)	1.87(1.22)	1.83(1.22)	1.96(1.27)	2.11(1.23)
	$n = 190$	$n = 185$	$n = 365$	$n = 302$	$n = 406$

Note. Standard deviations in parentheses; n = number of participants; MZM = monozygotic male; DZM = dizygotic male; MZF = monozygotic female; DZM = dizygotic female; DZOS = dizygotic opposite sex; SOC = Sense of coherence; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events; W1, W2, W3 = Wave−1, Wave−2 and Wave−3, respectively.

Table A3
Correlations between SOC and life events, including only the life events from the LEQ-A.

Variable	SOC_{W1}	SOC_{W2}	SOC_{W3}
$NegDep_{W1}$	-0.48^{***}	-0.27^{***}	-0.21^{***}
$NegDep_{W2}$	-0.32^{***}	-0.41^{***}	-0.28^{***}
$NegDep_{W3}$	-0.23^{***}	-0.28^{***}	-0.37^{***}
$NegInd_{W1}$	-0.26^{***}	-0.13^{***}	-0.09^{**}
$NegInd_{W2}$	-0.19^{***}	-0.27^{***}	-0.19^{***}
$NegInd_{W3}$	-0.14^{***}	-0.13^{**}	-0.25^{***}
$PosDep_{W1}$	-0.11^{***}	0.01	0.03
$PosDep_{W2}$	-0.07^{**}	−0.04	0.00
$PosDep_{W3}$	−0.04	−0.04	−0.01

Note. LEQ-A = Life Event Questionnaire for Adolescents (Masten et al., 1994); SOC = Sense of coherence; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events; W1, W2, W3 = Wave−1, Wave−2 and Wave−3, respectively.
$^{**}p < 0.01$. $^{***}p < 0.001$.

Table A4
Correlations between SOC and negative life events, without considering event-dependence.

Variable	SOC_{W1}	SOC_{W2}	SOC_{W3}
Negative life events$^{a}_{W1}$	-0.51^{***}	-0.30^{***}	-0.21^{***}
Negative life events$^{a}_{W2}$	-0.35^{***}	-0.47^{***}	-0.33^{***}
Negative life events$^{a}_{W3}$	-0.24^{***}	-0.30^{***}	-0.44^{***}

Note. SOC = Sense of coherence. [a] The negative life events score included all negative life events, both those considered dependent and independent.

Table A5
Unstandardized parameter estimates from the RI-CLPMs.

Parameters	Model		
	SOC and NegDep	SOC and NegInd	SOC and PosDep
Autoregressive parameters			
$SOC_{W1} \rightarrow SOC_{W2}$ (α_2)	0.11 (0.05)	0.13 (0.05)	0.15 (0.05)
$SOC_{W2} \rightarrow SOC_{W3}$ (α_3)	0.11 (0.05)	0.14 (0.05)	0.16 (0.05)
$LE_{W1} \rightarrow LE_{W2}$ (δ_2)	0.19 (0.04)	0.03 (0.04)	0.14 (0.04)
$LE_{W2} \rightarrow LE_{W3}$ (δ_3)	0.19 (0.04)	0.07 (0.04)	0.18 (0.04)
Cross-lagged parameters			
$SOC_{W1} \rightarrow LE_{W2}$ (γ_2)	−0.27 (0.07)	−0.10 (0.05)	−0.12 (0.05)
$SOC_{W2} \rightarrow LE_{W3}$ (γ_3)	−0.10 (0.07)	−0.03 (0.05)	−0.05 (0.04)
$LE_{W1} \rightarrow SOC_{W2}$ (β_2)	−0.06 (0.03)	−0.02 (0.03)	−0.03 (0.03)
$LE_{W2} \rightarrow SOC_{W3}$ (β_3)	−0.10 (0.03)	−0.08 (0.03)	−0.07 (0.04)
Random intercept variance			
A SOC (a_{77})	0.66 (0.04)	0.67 (0.04)	0.67 (0.04)
A LE (a_{88})	1.15 (0.06)	0.26 (0.17)	−0.40 (0.06)
C LE (c_{88})	–	0.41 (0.09)	–
E SOC (e_{77})	0.39 (0.06)	0.41 (0.06)	0.39 (0.06)
E LE (e_{88})	0.10 (0.10)	0.34 (0.06)	−0.10 (0.07)
Random intercept correlations			
A SOC ↔ LE (r_{a78})	−0.68 (0.05)	−0.95 (0.61)	0.10 (0.13)
E SOC ↔ LE (r_{e78})	−0.99 (1.10)	−0.21 (0.20)	−1.00 (1.06)
Within-person variance			
A SOC_{W1} (a_{11})	0.61 (0.05)	0.57 (0.05)	0.58 (0.05)
E SOC_{W1} (e_{11})	0.82 (0.04)	0.83 (0.04)	0.83 (0.04)
A LE_{W1} (a_{22})	1.05 (0.08)	0.64 (0.11)	0.83 (0.04)
C LE_{W1} (c_{22})	–	0.70 (0.08)	–
E LE_{W1} (e_{22})	1.36 (0.04)	0.76 (0.03)	0.87 (0.03)
A SOC_{W2} (a_{33})	0.31 (0.10)	0.32 (0.09)	0.31 (0.10)
E SOC_{W2} (e_{33})	0.95 (0.04)	0.94 (0.04)	0.95 (0.04)
A LE_{W2} (a_{44})	0.88 (0.09)	0.64 (0.15)	0.76 (0.05)
C LE_{W2} (c_{44})	–	0.65 (0.12)	–
E LE_{W2} (e_{44})	1.44 (0.05)	0.91 (0.04)	0.87 (0.03)
A SOC_{W3} (a_{55})	0.44 (0.08)	0.44 (0.07)	0.44 (0.08)
E SOC_{W3} (e_{55})	0.88 (0.04)	0.88 (0.04)	0.88 (0.04)
A LE_{W3} (a_{66})	0.60 (0.12)	0.54 (0.19)	0.68 (0.06)
C LE_{W3} (c_{66})	–	0.69 (0.12)	–
E LE_{W3} (e_{66})	1.34 (0.05)	0.88 (0.04)	0.91 (0.04)
Within-person correlations			
A $SOC_{W1} \leftrightarrow LE_{W1}$ (r_{a12})	−0.82 (0.07)	−0.57 (0.16)	−0.48 (0.09)
E $SOC_{W1} \leftrightarrow LE_{W1}$ (r_{e12})	−0.30 (0.05)	−0.04 (0.06)	−0.10 (0.06)
A $SOC_{W2} \leftrightarrow LE_{W2}$ (r_{a34})	−0.51 (0.21)	−0.71 (0.31)	−0.42 (0.22)
E $SOC_{W2} \leftrightarrow LE_{W2}$ (r_{e34})	−0.36 (0.05)	−0.14 (0.05)	−0.04 (0.05)
A $SOC_{W3} \leftrightarrow LE_{W3}$ (r_{a56})	−0.54 (0.22)	−0.99 (0.43)	0.01 (0.17)
E $SOC_{W3} \leftrightarrow LE_{W3}$ (r_{e56})	−0.27 (0.06)	0.00 (0.06)	−0.13 (0.06)
Means			
SOC_{W1} (μ_1)	5.11 (0.03)	5.11 (0.03)	5.11 (0.03)
SOC_{W2} (μ_2)	4.96 (0.03)	4.96 (0.03)	4.96 (0.03)
SOC_{W3} (μ_3)	4.96 (0.03)	4.97 (0.03)	4.97 (0.03)
LE_{W1} (π_1)	2.33 (0.05)	1.44 (0.03)	2.15 (0.03)
LE_{W2} (π_2)	2.52 (0.05)	1.48 (0.04)	2.18 (0.03)
LE_{W3} (π_3)	2.25 (0.05)	1.40 (0.04)	1.95 (0.04)

Note. Standard errors in parentheses. LE refers to the respective life event cluster. SOC = sense of coherence; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events; W1, W2, W3 = Wave−1, Wave−2 and Wave−3, respectively; A and a = additive genetic influences; C and c = shared environmental influences; E and e = non-shared environmental influences.

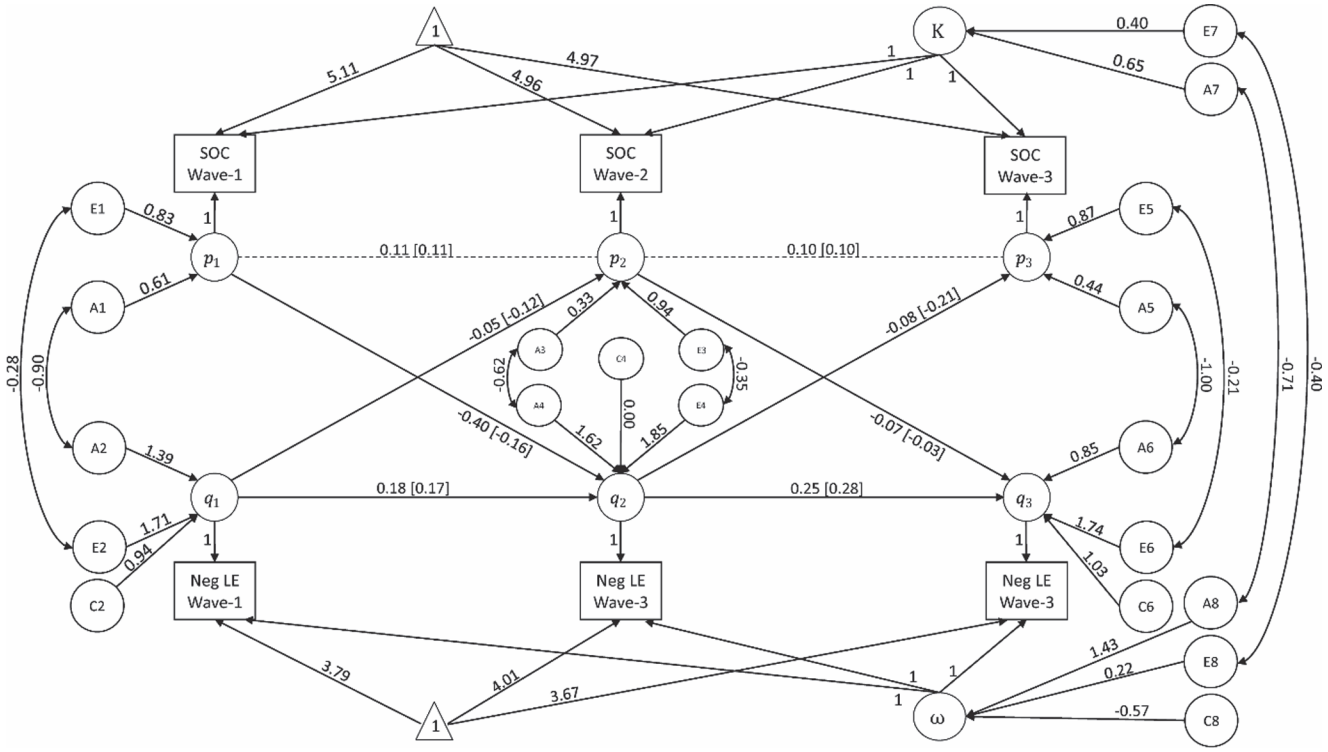

Fig. A1. Genetically Informative RI-CLPM with Unstandardized Coefficients for the Longitudinal Relationship between SOC and Negative Life Events. *Note.* Standardized coefficients are given in square brackets. Dashed lines indicate non-significant paths. SOC = sense of coherence; Neg LE = negative life events. See Fig. 1 for a more detailed description of model parameters.

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III

IV

The Role of Sense of Coherence and Loneliness in Borderline Personality Disorder Traits: A Longitudinal Twin Study

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Abstract

Background: Borderline personality disorder (BPD) implies having problems with identity and relations with other people. However, not much is known about whether these indications of BPD are present in adolescence, i.e., before personality disorders usually are diagnosed. In this study, we examined the prediction of an aspect of identity (i.e., sense of coherence [SOC]) and social relations (i.e., perceived loneliness) throughout adolescence on BPD traits in young adulthood. In addition, we examined to what degree the predictive ability could be attributed to genetic and environmental factors. We also examined whether life events in adolescence were related to BPD traits.

Methods: 3,391 twins, consisting of seven national birth cohorts from Norway, participated in the study. SOC, loneliness and life events were measured three times throughout adolescence with self-report questionnaires, with two years in between measurements. BPD traits were measured at the end of adolescence around the age of 19 with a structured interview. Regression analyses were performed to examine the prediction of SOC, loneliness and life events on BPD traits. Cholesky decomposition models were then used to determine to what degree the associations were due to genetic and environmental influences.

Results: The prediction of SOC and loneliness on BPD traits increased from R = .25 (when measured six years prior to the assessment of BPD traits) to R = .45 (when measured shortly before the assessment of BPD traits). In addition, negative life events considered dependent on a person's behavior were related to BPD traits. Negative independent and positive dependent life events did not contribute to the prediction of BPD traits. Cholesky decomposition models showed that SOC and loneliness were associated with BPD traits mainly due to shared genetic influences (i.e., the proportion due to genetic influences ranged

from 71% to 86%). Adding negative dependent life events to the prediction of BPD traits did not change these percentages.

Conclusions: These findings indicate that the weaker SOC, the stronger feelings of loneliness, and the negative life events associated with BPD traits are mainly consequences of the genetic aspects of BPD traits, rather than having direct effects on levels of BPD symptoms.

Keywords: borderline personality disorder; sense of coherence; loneliness; adolescence; longitudinal twin design

Background

Borderline personality disorder (BPD) is characterized by affective instability, intensity, anger, impulsivity, and self-destructive and unstable relations to others (American Psychiatric Association, 2013). In the alternative model for personality disorders in DSM-5, the general criteria for personality disorders includes impairment in personality functioning, defined as disturbances in self and interpersonal functioning (American Psychiatric Association, 2013). Although these impairments are common to all personality disorders, problems related to self and others seem to be more severe in patients with BPD compared to patients diagnosed with other personality disorders (Bender, Morey, & Skodol, 2011).

Numerous studies investigating the associations between the DSM personality disorders and the Big Five personality traits have concluded that normative personality traits can be used to conceptualize personality disorders (Clark, 2007). The Big Five personality traits are often found to correlate with BPD, first and foremost a profile of higher neuroticism and lower agreeableness and conscientiousness (Saulsman & Page, 2004, 2005). Also temperamental traits in childhood, such as impulsivity, aggression (Belsky et al., 2012; Cramer, 2016; Underwood, Beron, & Rosen, 2011; Vaillancourt et al., 2014), high levels of emotionality, activity, shyness, and low sociability (Stepp, Keenan, Hipwell, & Krueger, 2014) are found associated with later development of BPD symptoms (e.g., affective instability, impulsivity, unstable sense of self, and interpersonal dysregulation).

Identity disturbance is a core feature of BPD (Gunderson, Herpertz, Skodol, Torgersen, & Zanarini, 2018). However, this aspect by the disorder has received little empirical attention (Gad et al., 2019; Winsper, 2018). Although identity disturbance in adolescence has been associated with number of BPD symptoms in cross-sectional studies (Sekowski, Gambin, & Sharp, 2021; Westen, Betan, & DeFife, 2011), knowledge about the longitudinal course of identity disturbance in the development of BPD is lacking. Identity

disturbance implies problems in understanding oneself, being overwhelmed of one's affects, lacking trust in own abilities to face challenges and finding one's place in the world (Neacsiu, Herr, Fang, Rodriguez, & Rosenthal, 2015). The concept of sense of coherence (SOC) is a way of looking at identity. Having a strong SOC implies perceiving stressors we face in life as clear and understandable, having confidence that one has the resources to overcome them, and finding it worthwhile to invest time and effort to cope with the situation (Antonovsky, 1987). A weak SOC, on the other hand, means perceiving oneself and the world as more chaotic, unmanageable, and meaningless. Studies examining the relationship between SOC and BPD are lacking, but several cross-sectional studies of adolescent samples have shown that SOC are associated with perceived mental and somatic health (García-Moya, Rivera, & Moreno, 2013; Moksnes, Rannestad, Byrne, & Espnes, 2011; Ristkari, Sourander, Rønning, Nikolakaros, & Helenius, 2008).

In addition to problems related to identity, disturbance in interpersonal functioning is another core feature of BPD. The consequence of the problems with relating to other people may be alienation, aloneness, and generally a feeling of being lonely, even when surrounded by people (Hauschild et al., 2018; C. E. Miller, Townsend, & Grenyer, 2021). Regarding the relationship between BPD and loneliness, only a few cross-sectional studies on BPD patients have been published (Hauschild et al., 2018; Liebke et al., 2017). Examining the nature of the association between loneliness and BPD, results from a study of Australian and Dutch twins found that about half of the covariance between BPD features (i.e., affective instability, identity disturbance, negative relationships, and self-harm) and loneliness was due to shared genetic influences (Schermer et al., 2020). However, due to the cross-sectional nature of the studies, we do not know the direction of the associations, or whether the associations are time-limited related to an acute phase of BPD or have a more lasting association and potential

causal effect on BPD traits. Importantly, we do not know whether these aspects of BPD are present already in adolescence before personality disorders usually are diagnosed.

BPD is associated with severe impairment in psychosocial functioning, suicidality, and the presence of comorbid mental disorders (Gunderson et al., 2011; Leichsenring, Leibing, Kruse, New, & Leweke, 2011; Skodol et al., 2002). Although extensive research has shown that BPD usually has its onset in adolescence, diagnosing BPD in adolescence is a controversial issue, often leading to delayed diagnosis (Bozzatello, Garbarini, Rocca, & Bellino, 2021; Chanen, 2015; A. L. Miller, Muehlenkamp, & Jacobson, 2008). Furthermore, studies have shown that symptoms of BPD in adolescence are associated with long-term impairments in functioning (Bozzatello, Bellino, Bosia, & Rocca, 2019; Winograd, Cohen, & Chen, 2008). This emphasizes the importance of identifying features related to BPD in this age period. Identification of symptoms in adolescence that are associated with risk for developing BPD have important clinical implications, both in terms of prevention and early treatment (Bozzatello et al., 2019). Contributing to this, this paper examined whether SOC (an aspect of identity) and loneliness throughout adolescence are predictive of BPD traits in early adulthood. These constructs are closely related to how personality disorders are considered in the alternative model of personality disorders in DSM-5. In addition, impairments related to self and others are core symptoms of BPD. Both BPD (Bornovalova, Hicks, Iacono, & McGue, 2009; Reichborn-Kjennerud et al., 2015; Torgersen et al., 2012), SOC (Hansson et al., 2008; Silventoinen et al., 2014) and loneliness (Goossens et al., 2015) are influenced by genetic factors. This highlights the importance of using genetically informative designs that are able to separate the environmental effect of the predictors from the potential confounding effects of shared genetic influences.

In addition to study SOC and loneliness as predictors of BPD traits, we also examined whether life events throughout adolescence influenced levels of BPD traits in early

adulthood. Stressful life events represent important candidates that may influence levels of BPD traits. Childhood trauma in particular have been extensively studied, and such experiences have been implicated as important etiological risk factors in the development of BPD (e.g., Ball & Links, 2009). Also stressful life events in adolescence have been associated with increased BPD symptoms, such as illness in the family and maladaptive family functioning (Stepp, Olino, Klein, Seeley, & Lewinsohn, 2013). In addition, studies on adult clinical samples have shown that patients with BPD report more negative recent life events compared to patients diagnosed with other personality disorders or mood disorders, and that reporting more negative life events is associated with greater impairment in functioning (Pagano et al., 2004). However, findings from genetically informative studies have challenged the commonsense interpretation of a unidirectional effect from environment to person as they have shown that environmental measures such as life events are partly influenced by genetic factors (e.g., Kendler & Baker, 2007). Genetically informative studies on the relationship between environmental exposures and BPD in particular are scarce, but findings suggest that the association between BPD traits and both childhood trauma (Bornovalova et al., 2013; Skaug, Czajkowski, Waaktaar, & Torgersen, 2022)[1] and life events such as divorce and job loss (Distel et al., 2011) is caused by shared genetic influences. This suggests that genes influencing BPD traits also increase the likelihood of being exposed to childhood trauma and certain life events. Clearly, more genetically informative studies are needed to enhance our understanding of the relationship between assumed environmental risk factors and BPD.

The research objectives presented above can be specified into five aims. The first aim was to examine to what extent BPD traits in early adulthood can be predicted from SOC and loneliness in adolescence. The second aim was to examine whether life events in adolescence

[1] The study by Skaug et al. used data from the same dataset as the present study.

are predictive of BPD symptoms, separately and together with SOC and loneliness. The third aim was to determine to what degree associations between SOC, loneliness and BPD traits can be attributed shared genetic and environmental influences. The fourth aim, building on the third aim, was to examine whether accounting for life events in adolescence changes the estimated contribution of genetic and environmental influences. Finally, the fifth aim was to look at the development of SOC and loneliness throughout adolescence into the post-adolescence years as one kind of longtime borderline trait, and estimate the relative contribution of genetic and environmental influences of a common factor consisting of SOC, loneliness and BPD.

Method

Participants

Data for the study were drawn from the Oslo University Adolescent and Young Adult Twin Project (Torgersen & Waaktaar, 2019; Torgersen & Waaktaar, 2020). All twin pairs born in Norway between 1988 and 1994 were invited to participate. The twins completed self-report questionnaires three times throughout adolescence, with two years in between (12 to 18 years at Wave-1). In addition, the twins participated in a face-to-face interview when they were around age 19 (M = 19.1, SD = 1.2). Informed consent was obtained from both the twins and their parents. The project was approved by the Norwegian Data Inspectorate and the Regional Committees for Medical and Health Research Ethics. American Psychological Association ethical standards were followed in the conduct of the study.

In the present study, we rearranged the self-report questionnaire data from Wave-1, Wave-2, and Wave-3 data (in which each wave included data from seven birth cohorts) into data from the age of 12–13 years, 14–15 years, 16–17 years, and 18 years and older (i.e., until the time of the interview assessment of BPD traits). The whole sample consisted of 3,391

twins (56 % females) from 1,716 twin pairs. All twins, from both complete and incomplete pairs, were included in the study. Table 1 displays sample characteristics derived from the questionnaire data and the interview data. The majority of those who responded to questionnaires, also participated in the interview (i.e., 76%, 80%, 82% and 87% at age 12–13, 14–15, 16–17 and 18, respectively).

Table 1

Twin Sample Characteristics

	N single twins	N twin pairs	MZ twin pairs [a]	DZ twin pairs [a]
Questionnaire-data				
12–13 years	852	432	165	255
14–15 years	1,501	767	276	458
16–17 years	1,792	922	329	541
18 years	1,371	782	221	368
Interview-data	2,808	1,424	541	843

Note. [a] Number of complete pairs. MZ = monozygotic; DZ = dizygotic.

Zygosity Determination

The zygosity of same-sex twin pairs were partially determined through a 12-item zygosity scale where questions about similarity in appearance, how often the twins have been mixed-up with each other, and whether they believe that they are monozygotic or dizygotic were asked (Torgersen, 1979). To validate the zygosity scale, cheek swabbed DNA was drawn from 513 of the 1,006 same-sex twin pairs. Seventeen genetic markers were tested, with an estimated probability of misclassification less than $p < 0.0001$. The scores on the zygosity scale were analyzed using discriminant analysis and a cutting point for the discriminant score was established based on the results of the gene testing. Those with a discriminant score close to the cutting point were oversampled for DNA tests. It appeared

that 14 out of the 513 twin pairs were misclassified according to the discriminant analysis. Correcting for the oversampling, the questionnaire misclassified 2.13% of the same-sex twins. However, as almost all of the misclassified pairs were gene tested, only 0.64% of the same-sex twin pairs are expected to be misclassified (0.45% when including the whole twin sample).

Measures

Questionnaire Data: Sense of Coherence, Loneliness and Life Events

SOC was measured by an abbreviated 5-item version of the Sense of Coherence 13-item scale (SOC-13; Antonovsky, 1987). The abbreviation of the SOC-13 scale was performed based on results from a pilot study (Torgersen & Waaktaar, 2019). The SOC-13 scale has been shown to have good internal consistency, with Cronbach's alpha ranging from 0.70 to 0.92 across studies (Eriksson & Lindström, 2005). The Cronbach's alpha of the SOC scale used in this study ranged from 0.82 to 0.83 across the study waves, supporting the reliability of the abbreviated 5-item scale. The scale included the following questions: "Do you have the feeling that you are being treated unfairly?", "Do you have the feeling that you are in an unfamiliar situation and don't know what to do?", "Do you have very mixed-up feeling and ideas?", "Does it happen that you have feelings inside you would rather not feel?" and "How often do you have the feeling that there's little meaning in the things you do in your daily life?". Reponses were given on a 7-point Likert scale ranging from 1 (*very often*) to 7 (*rarely/never*). Average scores were computed with higher scores indicating stronger SOC.

Loneliness was measured by a 5-item scale, including a 4-item survey version of the R-UCLA Loneliness scale (Russell, Peplau, & Cutrona, 1980; "I feel in tune with the people around me", "I can find companionship when I want it", "No one really knows me well", "People are around me but not with me") and one direct measure of loneliness ("I feel

lonely"). Responses were given on a 5-point Likert scale ranging from 0 (*not typical*) to 4 (*very typical*). Positively worded items were reverse-coded, and average scores were computed with higher scores indicating higher levels of loneliness. There were strong correlations between the single direct measure of loneliness and the aggregate of the four R-UCLA items across all age groups (i.e., the correlations ranged from $r = .60$ to $r = .68$). Furthermore, the Cronbach's alpha of the full loneliness scale ranged from 0.77 to 0.84 across the study waves.

Life events were measured by a 38-item scale asking whether the participants had experienced any of the set of life events the past year (0 = *no*; 1 = *yes*). Twenty-nine events came from the Life Event Questionnaire for Adolescents (LEQ-A; Masten, Neemann, & Andenas, 1994) and nine events were added to the scale after a pilot study (see Table S1). The life events were divided into three clusters; negative life events considered dependent on a person's behavior (e.g., "I had many arguments with my parents"), negative life events considered independent on a person's behavior (e.g., "One of my parents died") and positive life events considered dependent on a person's behavior (e.g., "I got a new friend"). Life events from the LEQ-A were assigned to clusters according to Masten et al. (1994), and the remaining events were classified based on the authors' evaluation. Sum scores of the respective life events clusters were used when analyzing the data, with possible values ranging from 0–14 (negative dependent), 0–19 (negative independent) and 0–5 (positive dependent).

Interview Data: Borderline Personality Disorder Traits

A Norwegian version of the Structured Interview for DSM-IV Personality (SIDP-IV; Pfohl, Blum, & Zimmerman, 1997) was used to assess BPD traits (Helgeland, Kjelsberg, & Torgersen, 2005). Each twin in a pair was interviewed by different interviewers. The SIDP-IV uses a five-year rule, which means that the ratings are based on behavior typical for the

past five years. Each criterion is scored on a 4-point scale from 0 to 3 (0 = *absent*; 1 = *subthreshold*; 2 = *present*; 3 = *strongly present*). At least five of nine criteria are required for a BPD diagnosis. The prevalence for a categorically defined BPD diagnosis in the present sample was too low to perform reliable analyses. We therefore studied BPD as a dimensional trait by calculating the number of endorsed criteria either at the clinical or subclinical level (≥ 1). Interrater reliability was assessed based on two raters' scoring of 55 audiotaped interviews, of which 53 of the recordings were of satisfactory quality to be scored. The intraclass correlation coefficient for the dimensional measure of BPD (hereafter referred to as BPD traits) was 0.77 ($p < 0.001$).

Statistical Analyses

All analyses were performed in the statistical package R (R Core Team, 2020). First, we assessed the phenotypic associations between SOC, loneliness and BPD traits using correlation and linear regression analyses. Four regression analyses were performed, each with BPD traits as the dependent variable, and SOC and loneliness at a given age as independent variables (i.e., 12–13 years, 14–15 years, 16–17 years or 18 years). We then examined whether life events contributed to the prediction of BPD by adding life events (negative dependent, negative independent and positive dependent) to the regression analyses.

Next, the classical twin design was used to partition the phenotypic correlations between the predicted scores (derived from the regression analyses) and BPD traits into genetic and environmental influences. Twin models allow the variance of an observed phenotype (and the covariance between phenotypes) to be partitioned into three sources; additive genetic (A), shared environmental (C) and non-shared environmental (E) factors. The classical twin design relays on comparing the correlation within monozygotic (MZ) pairs with the correlation within dizygotic (DZ) pairs. MZ twins are genetically identical whereas

DZ twins share, on average, half of their segregating genes. Thus, influence of A is inferred when the MZ correlation exceeds the DZ correlation. Furthermore, both MZ and DZ twins experience environments that are shared by both twins within a pair. If these experiences contribute to phenotypic similarity within pairs, they are attributed influence of C. Influence of C is inferred when the DZ correlation is more than half the magnitude of the MZ correlation. Finally, the E effects represent all experiences that contribute to phenotypic dissimilarity within pairs, including measurement error.

In the same way, bivariate twin models allow us to partition the covariance between phenotypes into genetic and environmental influences. More specifically, we fitted a series of bivariate Cholesky decomposition models to quantify how much of the phenotypic correlations between the predicted scores and BPD traits that were due to genetic and environmental factors, respectively. The predicted scores from a given age was included as the first variable, with BPD traits as the second variable in each model. Using data from twins, the bivariate Cholesky decomposition partitions the variation in the first variable into genetic and environmental sources and quantify the extent in which those genetic and environmental sources also contribute to the variance in the second variable. The remaining variance in the second variable that is not shared with the first variable is also partitioned into genetic and environmental sources (Neale & Maes, 2004). The analyses were conducted in the structural equation modeling package OpenMx (Neale et al., 2016). Models were fitted to raw data using full information maximum likelihood. We first fitted full ACE models, followed by reduced models. Model fit was evaluated based on the models Akaike's information criterion (AIC), with lower values indicating better model fit (Akaike, 1987). For each age group, we report the proportion of the phenotypic correlation between the predicted scores and BPD traits that was due to genetic and environmental factors, respectively.

Finally, we created a factor including the measures of SOC and loneliness throughout adolescence and BPD traits in young adulthood. Missing data were imputed using multiple imputation by fully conditional specification (Van Buuren, 2007). The mean factor score based on the scores from 10 iterations were computed and then used in a univariate twin model to determine the heritability of this 'longtime borderline trait'.

Results

Descriptive Statistics and Phenotypic Associations

Table 2 display descriptive statistics for each study variable from the questionnaire data. For BPD traits, measured with a diagnostic interview around the age of 19 (M = 19.1, SD = 1.2), the mean was 1.08 (SD = 1.60). Inter-scale correlations and correlations with sex are provided in Table S2. Overall, the correlations between sex and all study variables were weak, ranging from r = -.01 to r = -.22.

Table 2

Descriptive Statistics for Study Variables

	12–13 years	14–15 years	16–17 years	18 years
Variable	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*
SOC	5.42 (1.21)	5.09 (1.23)	4.88 (1.30)	4.95 (1.28)
LON	1.00 (0.68)	1.05 (0.71)	1.08 (0.75)	1.07 (0.76)
NegDep	2.01 (1.89)	2.50 (2.13)	2.48 (2.08)	2.35 (2.03)
NegInd	1.41 (1.26)	1.20 (1.31)	1.69 (1.46)	1.46 (1.43)
PosDep	2.09 (1.30)	2.04 (1.28)	2.45 (1.17)	1.99 (1.24)

Note. SOC = sense of coherence; LON = loneliness; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events.

Table 3 presents correlations and results from linear regression analyses predicting BPD traits in early adulthood from SOC and loneliness at four different ages throughout adolescence. SOC were negatively associated with BPD traits, whereas loneliness showed positive associations with BPD traits. As expected, the strength of the associations increased as the time-lag between measures of SOC, loneliness and BPD traits decreased.

Table 3

Pearson Correlations and Results from Linear Regression Analyses, Predicting BPD Traits from SOC and Loneliness

	Pearson correlation with BPD traits		Standardized Beta		
Age group	SOC	LON	SOC	LON	R
12–13 years	-.21***	.20***	-0.16***	0.14***	.25
14–15 years	-.28***	.27***	-0.21***	0.18***	.33
16–17 years	-.33***	.29***	-0.24***	0.19***	.37
18 years	-.44***	.30***	-0.38***	0.12***	.45

Note. BPD traits = borderline personality disorder traits; SOC = sense of coherence; LON = loneliness; R = coefficient of multiple correlation. $^{***}p < 0.001$.

Table 4 presents correlations between life events and BPD traits. Negative dependent and negative independent life events showed small positive associations with BPD traits, whereas the associations between positive dependent life events and number of BPD symptoms were negligible.

Table 4

Pearson Correlations between BPD Traits and Life Events Throughout Adolescence

Age group	Pearson correlation with BPD traits		
	Negative dependent life events	Negative independent life events	Positive dependent life events
12–13 years	.24***	.06	.07
14–15 years	.28***	.17***	.04
16–17 years	.34***	.14***	.07**
18 years	.35***	.17***	.01

Note. BPD traits = borderline personality disorder traits. **$p < 0.01$. ***$p < 0.001$.

When life events were included in the regression analyses (see Table 5), the coefficient of multiple correlation (R) slightly increased compared to the models predicting BPD traits from SOC and loneliness, only. Of note, it was negative dependent life events that contributed to the prediction of BPD traits. Although negative independent life events showed small bivariate correlations with BPD traits, this cluster of life events did not have any independent effect on number of BPD symptoms.

Table 5

Results from Linear Regression Analyses, Predicting BPD Traits from SOC, Loneliness and Life Events

Age group	Standardized Beta					R
	SOC	LON	NegDep	NegInd	PosDep	
12–13 years	-0.09	0.13**	0.18***	-0.02	0.00	.30
14–15 years	-0.11**	0.16***	0.16***	0.07*	-0.01	.36
16–17 years	-0.13***	0.20***	0.22***	0.00	0.03	.42
18 years	-0.29***	0.12***	0.18***	0.02	-0.04	.48

Note. BPD traits = borderline personality disorder traits; SOC = sense of coherence; LON = loneliness; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events; R = coefficient of multiple correlation.

*$p < 0.05$. **$p < 0.01$. ***$p < 0.001$.

Using data from twins allow us to examine to what degree SOC and loneliness predicts BPD traits because these phenotypes share genetic influences, and to what degree the associations are due to environmental influences contributing to variation in both the predictor variables and BPD traits. More specifically, we fitted a series of bivariate Cholesky decompositions to partition the phenotypic correlations between the predicted scores derived from the regression analyses and BPD traits into genetic and environmental influences. Table 6 presents cross-trait correlations between the predicted scores (i.e., derived from the regression analyses with SOC and loneliness as independent variables) and BPD traits. The pattern of twin correlations suggests that the associations between SOC, loneliness and BPD traits are mainly due to genetic influences, with no influence of shared environmental factors (i.e., the DZ correlations were not greater than half the size of the MZ correlations). Furthermore, the slightly lower MZ correlations compared to the phenotypic correlations suggest small non-shared environmental influences between SOC, loneliness and BPD traits. Overall, the same pattern of twin correlations was observed when negative dependent life events were added to the predicted scores (see Table S3).

Table 6

Cross-trait Correlations

	Correlation with BPD traits		
Variable [a]	Phenotypic [95% CI]	rMZ [95% CI]	rDZ [95% CI]
SOC and LON 12–13 years	.25 [.18, .32]	.21 [.09, .33]	.10 [.00, .20]
SOC and LON 14–15 years	.33 [.27, .38]	.26 [.18, .35]	.09 [.02, .16]
SOC and LON 16–17 years	.37 [.33, .41]	.33 [.26, .40]	.11 [.05, .18]
SOC and LON 18 years	.45 [.40, .50]	.38 [.29, .45]	.19 [.12, .26]

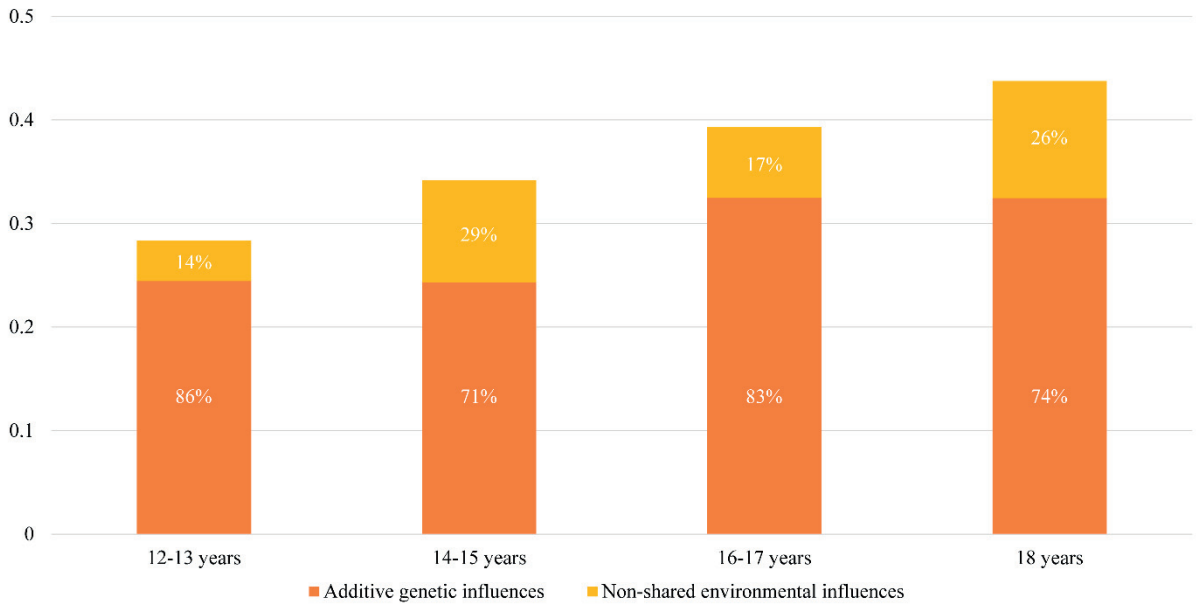
Note. BPD traits = borderline personality disorder traits; SOC = sense of coherence; LON = loneliness; Phenotypic = correlation without considering twin-pair membership; rMZ = cross-

twin correlation between monozygotic twin pairs; rDZ = cross-twin correlation between dizygotic twin pairs. [a] Predicted scores for BPD traits derived from linear regression analyses, with SOC and LON at different ages as independent variables.

According to the AIC values, an AE model (i.e., dropping the shared environmental parameters) was the best fitting model for all Cholesky decomposition models. This is also consistent with results from univariate twin analyses, where an AE model was found to have best fit for all predictor variables. All variables were moderately heritable (see Table S4). The proportions of the phenotypic correlations between the predicted scores (i.e., based on SOC and loneliness) and BPD traits due to genetic and environmental influences are displayed in Figure 1 as a set of stacked bar charts. For standardized parameter estimates derived from the Cholesky decomposition models, see Table S5. The results indicated that the phenotypic correlations between the predicted scores and BPD traits were mainly due to additive genetic influences, with additive genetic influences accounting for between 71% and 87% of the phenotypic correlations. When negative dependent life events were added to the predicted scores, the relative contribution of genetic and environmental influences were close to identical (in fact slightly higher contributions of genetic influences) to the proportions displayed in Figure 1 (i.e., the proportion of the phenotypic correlations due to additive genetic influences were 89%, 74%, 85% and 75% at age 12–13, 14–15, 16–17 and 18, respectively). See Figure S1 for a figurative illustration of the results and Table S5 for standardized parameter estimates. Genetic and environmental correlations provided support for the results provided in Figure 1. That is, the genetic correlations between the predicted scores and BPD traits were moderate to high, whereas the environmental correlations were small, around 1/4 of the genetic correlations (see Table S6).

Figure 1

Genetic and Environmental Influences on the Association between the Predicted Scores and BPD Traits



Note. BPD traits = borderline personality disorder traits; Predicted scores = predicted scores for BPD traits derived from linear regression analyses, with sense of coherence and loneliness at different ages as independent variables. The height of the bars represents the phenotypic correlation between the predicted scores and BPD traits. The percentages represent the proportions of the phenotypic correlations due to genetic and environmental influences.

Finally, we created a factor of all measures of SOC and loneliness throughout adolescence, and BPD traits (for factor loadings, see Table S7). In this way, we looked at SOC and loneliness as a kind of longtime borderline trait. Univariate twin analyses showed that the heritability of this factor was .56, 95% CI [.52, .61], which is somewhat higher

compared to the heritability of BPD traits measured at one time point (h^2 = .50, 95% CI [.44, .55]).

Discussion

The present study examined if SOC and loneliness in adolescence can predict BPD traits in early adulthood. Conceptually, these possible predictors of BPD traits are closely related to disturbances in self- and interpersonal functioning, which is how the alternative model for personality disorders in DSM-5 characterizes personality disorders (American Psychiatric Association, 2013). Furthermore, previous research has shown that BPD usually has its onset in adolescence and have highlighted the importance of early detection and intervention to prevent chronicity and reduce the risk for long-term consequences (Chanen, 2015; Kaess, Brunner, & Chanen, 2014; A. L. Miller et al., 2008). The present study demonstrated that SOC and loneliness already at the age of 12 is predictive of BPD traits in early adulthood (*M* = 19.1 years). The correlation between the predicted and the observed BPD scores increased as the time interval between the measurements decreased, and the twins got older. We cannot know if the prediction increased in strength due to decreased time interval between measurements or due to an effect of age. Previous studies have shown that both SOC (Eriksson & Lindström, 2005; Honkinen et al., 2008) and loneliness (Mund, Freuding, Möbius, Horn, & Neyer, 2020) are relatively stable constructs across the life span, showing similar rank-order stability as personality traits (Costa, McCrae, & Lockenhoff, 2019). Thus, it is reasonable to believe that SOC and loneliness measured at earlier ages are precursors of later measures of SOC and loneliness.

Regarding the association between loneliness and BPD traits, a few cross-sectional studies exist, all finding positive associations between loneliness and BPD (Hauschild et al., 2018; Liebke et al., 2017; Schermer et al., 2020). Studies examining the relationship between SOC and BPD are lacking, but previous studies have described associations between BPD

traits and features related to SOC such as poor functioning in response to stress (Belsky et al., 2012), lack of effective emotion regulation strategies and difficulties with goal-directed behavior (Salsman & Linehan, 2012). A weak SOC means, at the extreme, perceiving oneself and the world as chaotic, unmanageable, and meaningless. Hence, SOC may relate to the identity disturbance associated with BPD, with difficulties finding meaning in life, difficulties with self-direction and feelings of worthlessness (Gad et al., 2019; Neacsiu et al., 2015; Sekowski et al., 2021; Westen et al., 2011). The associations between SOC, loneliness and BPD traits may also be related to personality traits. For example, lonely people are shown to be characterized by a profile of higher neuroticism and lower extraversion compared to less lonely people (Buecker, Maes, Denissen, & Luhmann, 2020). SOC has also been associated with the Big Five traits, especially neuroticism (Feldt, Metsäpelto, Kinnunen, & Pulkkinen, 2007; Hochwälder, 2012). A research objective for future studies may therefore be to examine whether personality traits can explain some of the predictive power of SOC and loneliness on BPD traits.

The present study utilized a longitudinal twin design, allowing sequencing of predictors and outcome, and examination of the nature of the association between the predictor variables and BPD traits. The results showed that SOC and loneliness are associated with BPD traits mainly for genetic reasons, and that the relative contribution of genetic and environmental influences was more or less the same throughout adolescence. More specifically, although the associations between SOC, loneliness and BPD traits increased as the time-lag between the assessments decreased, the proportion of the correlations due to genetic and environmental influences remained quite stable (i.e., proportions due to genetic influences varied from 71% to 86%). The results showed moderate to high genetic correlations and small environmental correlations between the phenotypes, providing further support to the observation that the associations were mainly due to genetic influences. In

sum, the results indicate that the genetic factors that influence BPD symptoms also increase the likelihood of having a weaker SOC and stronger feelings of loneliness. To our knowledge, only one previous study has examined the nature of the association between loneliness and BPD. This study found that 51% of the phenotypic correlation between loneliness and BPD traits was due to genetic influences (Schermer et al., 2020). Results of the present study showed a higher proportion due to genetic influences. However, we studied the combined effect of SOC and loneliness on BPD traits, and thus our results are not directly comparable with the study by Schermer et al. (2020).

Although most of the phenotypic correlations were due to shared genetic influences, the results also indicate that common genes are not the only explanation for the association between SOC, loneliness and BPD traits. Between 14% and 29% of the correlations were due to shared non-shared environmental influences, possibly indicating that changing the environmental factors that affect a person's SOC or loneliness may also influence symptoms of BPD.

Negative dependent and negative independent life events were also associated with BPD traits, but positive dependent life events were not (with a small exception for those in the 16-17 age group). When a multiple regression analysis was conducted, only negative dependent life events survived together with SOC and loneliness. Adding negative dependent life events to the prediction of BPD traits did not change the proportions of the phenotypic correlations due to additive genetic and environmental influences. It is noteworthy that although 'measured environments' such as negative dependent life events predict BPD, they do not add anything to the relative contribution of environmental influences. That is, measures of the environment are not always 'environmental', as genetically informative studies have shown (e.g., Kendler & Baker, 2007). Together, the results suggest that SOC, loneliness and negative dependent life events throughout adolescence are associated with

BPD traits in early adulthood mainly due to shared genetic influences. Analyses of the genetic and environmental contribution to stability in the measured constructs further support these results, as they showed that the stability of both SOC, loneliness and the recurrence of life events were mainly due to genetic influences.

Finally, a factor of SOC and loneliness throughout adolescence, and BPD traits in early adulthood showed a heritability of 56%, which is somewhat higher than looking at BPD traits alone. The heritability of this factor is also higher than the usual 40-50% found when studying personality traits and personality disorders on a specific occasion (Livesley & Jang, 2008; Torgersen, 2009; Vukasović & Bratko, 2015), and is probably a better approximation of the real contribution of genetic influences on BPD traits. Assessing a personality trait or personality disorder at a specific occasion means a lot of time-specific influence, chance variance and measurement error. Furthermore, this time-specific measurement error cannot be separated from the estimate of non-shared environmental influences, leading to an overestimation of the non-shared environmental influences and a corresponding underestimation of genetic influences. For example, in a full-population study of individuals born in Sweden between 1973 and 1993, the heritability of clinically diagnosed BPD was estimated to 46% (Skoglund et al., 2021). Similar estimates have been reported in other cross-sectional studies that have used dimensional measures of BPD traits (Bornovalova et al., 2013; Distel et al., 2008; Kendler et al., 2008; Torgersen et al., 2008). Longitudinal studies, on the other hand, have reported heritability estimates of BPD traits up to 70% (Bornovalova et al., 2009; Reichborn-Kjennerud et al., 2015). In general, any personality trait or disorder should be assessed in a longer life perspective as they are by definition relatively long-lasting, and retrospective reports are usually highly unreliable and are missing important information.

Limitations and Strengths

The results should be considered in light of several possible limitations. First, our measure of BPD traits includes both subclinical and clinical scores and therefore may not generalize to clinical populations. Second, although the global measure of loneliness used in the present study are frequently used in the research literature, subtypes of loneliness may have differential associations with BPD traits. For example, results from a study by Lasgaard, Goossens, Bramsen, Trillingsgaard, and Elklit (2011) indicated that peer-related and family-related loneliness showed differential associations with psychopathology in adolescence. Future studies may examine the effect of multiple dimensions of loneliness on BPD traits. Third, although it is common to categorize life events based on event-dependence, some events may not be clearly classified as independent or dependent (e.g., event-dependence for some events such as 'moving schools' may vary across individuals). A clear criterion for classification of event-dependence is difficult to obtain without knowing the 'causes' behind the experiences (e.g., was the person having many arguments with his/her sibling due to own behavior or was it due to the sibling's quarrelsome behavior?). However, prior studies have reported higher heritability of dependent life events compared to independent life events, supporting a differentiation between them (Bemmels, Burt, Legrand, Iacono, & McGue, 2008; Billig, Hershberger, Iacono, & McGue, 1996; Plomin, Lichtenstein, Pedersen, McClearn, & Nesselroade, 1990). Studying classes of life events may provide a more accurate picture of the effect of life events on various outcomes. Results from the present paper supports a division of life events into different clusters as their effect on BPD traits strongly differed. Fourth, all measures are based on self-reports from the twins. Both loneliness and SOC are intended to measure a person's subjective feelings and perceptions of the world, making self-reports the most appropriate approach. However, it is possible that the measure of BPD traits could have been improved (i.e., more reliable) by reports from

significant others in addition to the twins' own reports. Whether the use of self-reports represent a limitation or not may depend on the person being asked. For example, although self-reports may be prone to subjective interpretations, the responses mimic the clinical situation where the clinician has to rely on the patient's descriptions. Fifth, some of the measures were markedly skewed. When fitting structural equation models to non-normal data, this could result in underestimated standard errors. Thus, individual parameters may be statistically significant more frequently than they should be. Sixth, there are several assumptions related to the classical twin design which may threat the validity of results if they are violated. Violations of the equal environment assumption (EEA) that MZ and DZ twin pairs experience the same degree of environmental similarity may result in overestimation of the effect of genetic influences (Evans, Gillespie, & Martin, 2002). However, empirical evidence supports the validity of the EEA (e.g., Conley, Rauscher, Dawes, Magnusson, & Siegal, 2013; Derks, Dolan, & Boomsma, 2006). Another major assumption is that DZ twin pairs share half of their segregating genes. This is based on the assumption of random mating. If mating is not random, parents may share genes beyond what is expected by chance. Consequently, DZ twin pairs will share more than 50% of their segregating genes. If the phenotypes under study have been subject to non-random mating (i.e., assortative mating), findings will overestimate the shared environmental influences and underestimate the genetic influences. Positive assortative mating would also lead to an overestimation of genetic correlations (van Rheenen et al., 2019). However, previous studies have found assortative mating to be low for related phenotypes such as personality domains (Neale & Maes, 2004).

The study also has several strengths. The present study extends previous research by using a longitudinal design which allow sequencing of predictor and outcome. Furthermore, using data from twins makes it possible to partition the covariance between phenotypes into

genetic and environmental influences. In this way, one can determine to what degree a predictor is associated with BPD traits due to a direct 'environmental' effect of the predictor and to what degree the phenotypes are associated due to shared genetic influences. Furthermore, data for the study consisted of a full cohort population-based sample, which strengthens the possibility of generalization of findings. However, future studies should seek to replicate findings in different samples from other countries.

Conclusion

SOC and loneliness already at age 12 years is associated with increased levels of BPD symptoms in young adulthood. The associations increased in strength with older age and shorter time until assessment of BPD traits. Negative dependent life events were also associated with BPD traits partly independent of the effects of SOC and loneliness. Results from the present study suggest that SOC, loneliness and life events are associated with BPD mainly because they share common genetic influences, rather than a direct/causal effect of the predictors on levels of BPD symptoms. That is, the predictors seem to be consequences of the genetic aspects related to BPD.

List of abbreviations

AIC = Akaike's information criterion; BPD traits = borderline personality disorder traits; LEQ-A = Life Event Questionnaire for Adolescents; SIDP-IV = Structured Interview for DSM-IV Personality; SOC = sense of coherence; A = additive genetic factors; C = shared environmental factors; E = non-shared environmental factors; MZ = monozygotic; DZ = dizygotic.

Declarations

Ethics approval and consent to participate

The study was approved by the Norwegian Data Inspectorate and the Regional Committees for Medical and Health Research Ethics, ref. 2015/4 (19661). Informed consent was obtained from both the twins and their parents.

Consent for publication

Not applicable.

Availability of data and materials

The dataset analyzed during the current study are not publicly available. Data collection for the study was preapproved in 2005 by the Norwegian Data Protection Authority (DPA) under a clause of 20 years individual data protection and subsequent data deletion or anonymization. Anonymized data may be requested after 2025.

Competing interests

The authors declare that they have no competing interests.

Funding

The study was supported by the Department of Psychology, University of Oslo and by the Research Council of Norway by grants 170089, 213722 and 213760. Nikolai O. Czajkowski was supported by the Research Council of Norway, grant number 288083.

Authors' contributions

ES performed the data analyses and wrote the original draft. NOC contributed to the data analyses, interpretation of data and provided critical feedback on drafts. TW collected the data and provided critical feedback on drafts. ST collected the data, contributed to writing

the manuscript, interpretation of data, and provided critical feedback on drafts. All authors read and approved the final manuscript.

Acknowledgements

The authors would like to thank RBUP, Eastern and Southern Norway for their contribution to the data collection.

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Supplementary Material

Table S1

The Life Events Scale

Negative dependent life events
I had an important change in physical appearance, which upset me (acne, glasses, physical development, etc.)[a]
I was a victim of violence (mugging, sexual abuse, robbery)[a]
I was disappointed by a friend
I was disappointed by someone in the family
I did not get into a group or activity that I wanted to get into (music group, sports team, theater, etc.)[a]
I had major problems with a teacher
I did much worse than I expected in an important exam or course[a]
I had less contact with one of my parents[a]
I had many arguments with my siblings[a]
I had many arguments with my parents[a]
I was bullied by other pupils/adolescents
I broke up with a girlfriend/boyfriend[a]
I had an abortion (girls) / my girlfriend had an abortion (boys)
I lost a close friend[a]
Negative independent life events
I lost a pet
I changed schools[a]
I became seriously ill or was injured[a]
At least one parent or another family member became seriously ill or was injured[a]
One of my parents died[a]
A brother or sister died[a]
Another family member died[a]
One of my close friends died[a]
Mom or Dad's friend moved in with us[a]
A member of my family ran away from home[a]
My parents divorced, moved apart[a]
One of my parents had problems at work[a]
One parent lost his or her job[a]
My mother began to work[a]
There has been a change in a parent's job so that my parent is away from home more often[a]
The family financial situation was difficult[a]
There was some damage or loss of family property (such as apartment, house, car or bike)[a]
There were many arguments between the adults[a]
Someone in the family had problems with the police[a]

Positive dependent life events
I received a special award (trophy, diploma etc.) for something done at school[a]
I became more popular with my friends
I joined a fun group of friends
I got a boyfriend/girlfriend[a]
I got a new friend

Note. [a] Question from the Life Event Questionnaire for Adolescents (LEQ-A; Masten et al., 1994). The wording in some of the questions were slightly changed from the LEQ-A.

Table S2

Inter-Scale Correlations

Variable	Sex [a]	SOC	LON	NegDep	NegInd
12–13 years					
SOC	-.09**	–			
LON	.06	-.37***	–		
NegDep	.13***	-.48***	.22***	–	
NegInd	-.01	-.17***	.10**	.30***	–
PosDep	.06	-.19***	-.11**	.32***	.21***
14–15 years					
SOC	-.20***	–			
LON	.04	-.42***	–		
NegDep	.22***	-.54***	.24***	–	
NegInd	.09***	-.29***	.13***	.40***	–
PosDep	.07**	-.15***	-.17***	.32***	.17***
16–17 years					
SOC	-.22***	–			
LON	.06**	-.44***	–		
NegDep	.22***	-.49***	.22***	–	
NegInd	.10***	-.28***	.10***	.39***	–
PosDep	.07**	-.13***	-.17***	.30***	.19***
18 years					
SOC	-.17***				
LON	.03	-.48***	–		
NegDep	.20***	-.49***	.22***	–	
NegInd	.10***	-.26***	.13***	.42***	–
PosDep	.02	-.05*	-.14***	.26***	.18***

Note. SOC = sense of coherence; LON = loneliness; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events. Correlations with BPD traits are provided in Table 3 and Table 4. [a] Sex coded 0 = male, 1 = female. The correlation between sex and BPD traits was $r = .11^{***}$.

$^{**}p < 0.01$. $^{***}p < 0.001$.

Table S3

Cross-trait Correlations

Variable [a]	Correlation with BPD traits		
	Phenotypic [95% CI]	rMZ [95% CI]	rDZ [95% CI]
SOC, LON and NegDep			
12–13 years	.30 [.22, .37]	.23 [.10, .34]	.18 [.08, .27]
14–15 years	.36 [.31, .41]	.27 [.18, .35]	.15 [.08, .22]
16–17 years	.42 [.38, .46]	.37 [.29, .44]	.18 [.11, .24]
18 years	.48 [.43, .52]	.38 [.30, .46]	.23 [.16, .30]

Note. BPD traits = borderline personality disorder traits; SOC = sense of coherence; LON = loneliness; NegDep = negative dependent life events; Phenotypic = correlation without considering twin-pair membership; rMZ = cross-twin correlation between monozygotic twin pairs; rDZ = cross-twin correlation between dizygotic twin pairs. [a] Predicted scores for BPD traits derived from linear regression analyses, with SOC, LON and NegDep at different ages as independent variables.

Table S4

Univariate Model Estimates From the Best Fitting Twin Models

	Additive genetic effects	Non-shared environmental effects
12–13 years		
SOC	.40 [.28, .50]	.60 [.50, .72]
LON	.45 [.34, .55]	.55 [.45, .66]
NegDep	.60 [.51, .67]	.40 [.33, .49]
14–15 years		
SOC	.46 [.37, .54]	.54 [.46, .63]
LON	.49 [.41, .56]	.51 [.44, .59]
NegDep	.54 [.47, .61]	.46 [.39, .53]
16–17 years		
SOC	.38 [.30, .46]	.62 [.54, .70]
LON	.50 [.43, .57]	.50 [.43, .57]

NegDep	.49 [.41, .56]	.51 [.44, .59]
18 years		
SOC	.42 [.32, .51]	.58 [.49, .68]
LON	.37 [.27, .47]	.63 [.53, .73]
NegDep	.54 [.45, .61]	.46 [.39, .55]

Note. 95% CI in brackets. SOC = sense of coherence; LON = loneliness; NegDep = negative dependent life events.

Table S5

Standardized Parameter Estimates from the Bivariate Cholesky Decomposition Models

Model	A_{11}	A_{12}	A_{22}	E_{11}	E_{12}	E_{22}
SOC-LON and BPD traits [a]						
12–13 years	.68 [.60, .75]	.36 [.20, .50]	.61 [.50, .69]	.73 [.66, .80]	.05 [-.05, .15]	.70 [.66, .74]
14–15 years	.74 [.68, .78]	.33 [.23, .42]	.63 [.56, .68]	.67 [.62, .73]	.15 [.07, .22]	.69 [.65, .73]
16–17 years	.70 [.64, .75]	.47 [.38, .55]	.54 [.45, .61]	.72 [.67, .77]	.10 [.02, .17]	.70 [.66, .73]
18 years	.65 [.57, .72]	.50 [.40, .59]	.51 [.39, .59]	.76 [.70, .82]	.15 [.07, .23]	.69 [.65, .73]
SOC-LON-NegDep and BPD traits [b]						
12–13 years	.78 [.71, .82]	.39 [.26, .51]	.59 [.49, .67]	.63 [.57, .70]	.06 [-.05, .16]	.70 [.66, .74]
14–15 years	.77 [.73, .81]	.36 [.26, .44]	.61 [.54, .67]	.63 [.58, .69]	.16 [.08, .24]	.69 [.65, .73]
16–17 years	.74 [.69, .78]	.51 [.43, .58]	.50 [.41, .57]	.67 [.62, .72]	.10 [.03, .17]	.70 [.66, .73]
18 years	.69 [.62, .75]	.50 [.41, .59]	.50 [.40, .58]	.73 [.66, .79]	.16 [.08, .24]	.69 [.65, .73]

Note. 95% CI in brackets. [a] Variable 1: the predicted scores for BPD traits derived from linear regression analyses with SOC and LON at different ages as independent variables. Variable 2: BPD traits. [b] Variable 1: the predicted scores for BPD traits derived from linear regression analyses with SOC, LON and NegDep at different ages as independent variables. Variable 2: BPD traits. A = additive genetic influences; E = non-shared environmental influences; $_{11}$ = Genetic and environmental influences on the predicted scores; $_{12}$ = Genetic and environmental influences on the predicted scores, contributing to variance in BPD traits; $_{22}$ = Genetic and environmental influences unique to BPD traits.

Table S6

Genetic and Environmental Correlations Derived from the Bivariate Cholesky Decomposition Models

Variable [a]	Genetic correlation with BPD traits [95% CI]	Non-shared environmental correlation with BPD traits [95% CI]
SOC and LON		
12–13 years	.50 [.29, .70]	.08 [-.07, .22]
14–15 years	.46 [.32, .60]	.21 [.10, .32]
16–17 years	.65 [.54, .77]	.14 [.03, .24]
18 years	.70 [.57, .83]	.21 [.10, .32]
SOC, LON and NegDep		
12–13 years	.55 [.37, .72]	.08 [-.07, .23]
14–15 years	.50 [.37, .62]	.23 [.12, .34]
16–17 years	.71 [.61, .81]	.14 [.04, .24]
18 years	.71 [.59, .82]	.23 [.12, .34]

Note. BPD traits = borderline personality disorder traits; SOC = sense of coherence; LON = loneliness; NegDep = negative dependent life events. [a] Predicted scores for BPD traits derived from linear regression analyses, with SOC and LON (and SOC, LON and NegDep) at different ages as independent variables.

Table S7

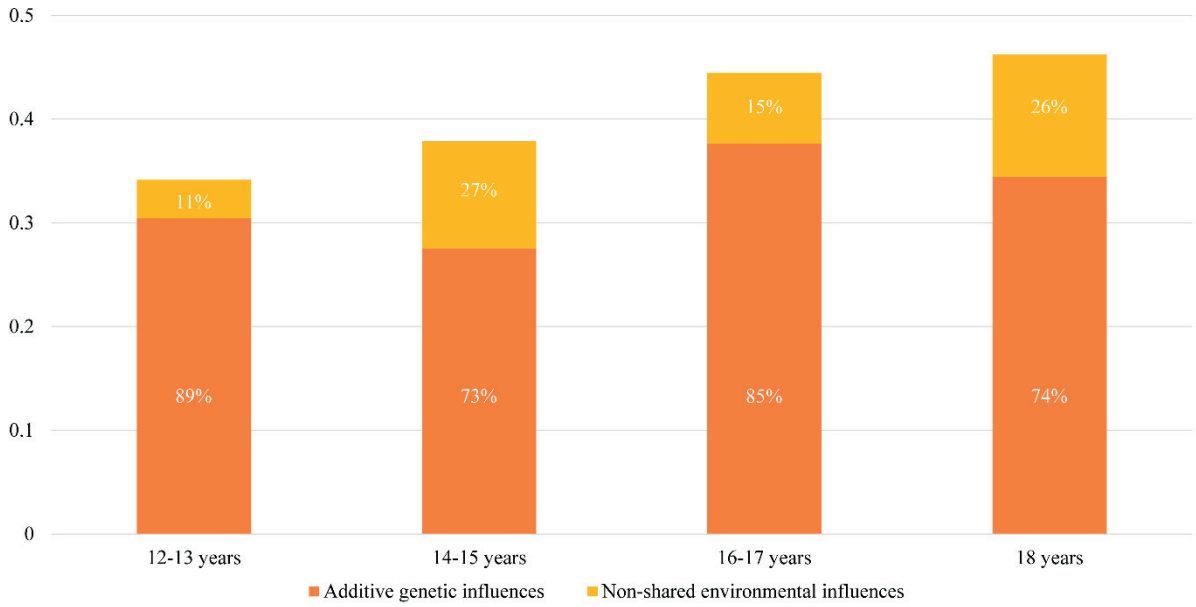
Factor Loadings

Variable	Factor
BPD traits	0.47
SOC 12–13 years	-0.45
LON 12–13 years	0.48
SOC 14–15 years	-0.58
LON 14–15 years	0.64
SOC 16–17 years	-0.47
LON 16–17 years	0.69
SOC 18 years	-0.66
LON 18 years	0.62

Note. BPD traits = borderline personality disorder traits; SOC = sense of coherence; LON = loneliness.

Figure S1

Genetic and Environmental Influences on the Association between the Predicted Scores and BPD Traits



Note. BPD traits = borderline personality disorder traits; Predicted scores = predicted scores for BPD traits derived from linear regression analyses, with sense of coherence, loneliness, and negative dependent life events at different ages as independent variables. The height of the bars represents the phenotypic correlation between the predicted scores and BPD traits. The percentages represent the proportions of the phenotypic correlations due to genetic and environmental influences.