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Individual risk factors and the mother-child relationship in borderline personality disorder traits in children and adolescents

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Abstract

Borderline personality disorder (BPD) is a severe mental disorder characterized by pervasive patterns of instability of affect, self-image, and relationships. For a long time, BPD has been predominantly studied in adults, given the notion that personality in youth is still subject to changes. Research of the past decades has shown that the diagnosis of BPD is equally reliable and valid in adolescence. BPD has been started to be approached from a life span perspective that acknowledges that both adaptive and maladaptive personality traits can express and change throughout the life span. Developmental models of BPD assume an interplay of maladaptive family and parenting factors and an intrapersonal temperamental disposition to experience intense emotion. The interplay of caregiving behavior and child vulnerability is supposed to lead to insufficient co-regulation throughout the life span, impeding the development of child social communication and self-regulatory abilities. These developmental pathways have however not extensively been operationalized in youth. Lastly, research on the expression of symptoms during childhood age is rare, limiting our understanding of the early stages of the developing disorder. The aim of the current work is to extend knowledge about the interplay of intrapersonal and mother-child-relationship risk factors, explore physiological co-regulation in the context of BPD pathology, and to expand the life span perspective on BPD pathology to primary school age.

Article 1 applied a 14-year longitudinal design to study how postpartum maternal bonding impairment (MBI) as an indicator of very early mother-child relationship difficulties interacts with child temperament. MBI and low harm avoidance were risk factors for BPD traits and overall personality dysfunction. Interaction effects indicated that children low in harm avoidance and high in novelty seeking were more vulnerable to the effects of MBI, as they developed higher levels of overall personality dysfunction. Regarding BPD specifically, girls were more susceptible to the effect of MBI than boys. **Article 2** investigated the process of physiological co-regulation during mother-child interaction. The hypothalamic–pituitary–adrenal (HPA) axis shows dysregulation among BPD patients. The current work therefore studied physiological co-regulation of the HPA axis, indexed by mother-child cortisol synchrony. Patterns of cortisol synchrony varied as a function of child BPD pathology and dyadic behavior. **Article 3** aimed at gathering knowledge about differences and similarities of BPD traits during middle childhood and adolescence. BPD trait frequencies did not significantly differ between children and adolescents. BPD traits were associated with impairments such as higher comorbidity and lower quality of life in both age groups. However, age-related differences emerged indicating more strained mother-child relationships in adolescents compared to children with BPD traits.

Content

List of scientific publications for the publication-based dissertation

Article 1: Fleck, L., Fuchs, A., Moehler, E., Parzer, P., Koenig, J., Resch, F., & Kaess, M. (2021). Maternal bonding impairment predicts personality disorder features in adolescence: The moderating role of child temperament and sex. *Personality Disorders: Theory, Research, and Treatment*.

Article 2: Fuchs, A., Fleck, L., Lerch, S. Moehler, E., Koenig, J., Resch, F., & Kaess, M. (*under review*) Adolescent Borderline Personality Traits, Mental Disorder and Dyadic Relational Behavior Shape Mother-Adolescent Cortisol Synchrony. *Personality Disorders: Theory, Research and Treatment.*

Article 3: Fleck, L., Fuchs, A., Moehler, E., Williams, K., Koenig, J., Resch, F., & Kaess, M. (*under review*). Child versus adolescent borderline personality disorder traits: frequency, psychosocial correlates and observed mother-child interactions. *Personality Disorders: Theory, Research and Treatment.*

1 Introduction

In 1938, Adolf Stern first used the term “borderline” to describe patients who seemed to fall in between the diagnostic categories of “neurosis” and “psychosis” (Stern, 1938). The diagnosis of borderline personality disorder ([B]PD) was first included in the third edition of The Diagnostic and Statistical Manual of Mental Disorders (DSM-III, APA, 1980). In the current edition, the diagnosis requires patients to meet at least five of the nine following symptoms: Frantic efforts to avoid abandonment; a pattern of unstable interpersonal relationships; identity disturbance; impulsivity in at least two potentially self-damaging areas; suicidal or self-harm behavior; affective instability; chronic feelings of emptiness; intense or inappropriate anger; and stress-related paranoid ideation or dissociation (DSM-5; American Psychiatric Association, 2013). These symptoms are associated with substantial suffering, high rates of suicide attempts and treatment utilization, and BPD patients make up 15 to 25% of inpatient treatment populations (Goodman et al., 2017; Gunderson, 2009; Zanarini et al., 2008).

1.1 A life span perspective for personality disorders

For a long time, research and clinical attention regarding BPD mainly focused on adulthood. By definition, PD include maladaptive patterns of intra- and interpersonal dysfunction (American Psychiatric Association, 2013). These patterns are supposed to be of enduring nature and not limited to transient episodes. Maybe partly due to this definition, it was assumed that PD were persistent to change and improvement (Ring & Lawn, 2019). This stigma made clinicians hesitant to work with patients with PD (Sheehan et al., 2016) and created reluctance in diagnosing PD in young people, given clinicians wanted to avoid stigmatizations for their patients (Kaess et al., 2014). It was argued that child and adolescent personality – given its developing and evolving nature – would not fit the stable and pervasive characteristics required for a PD diagnosis. However, this view has led to empirical attention focusing on adult expressions of BPD, which in turn led to a limited understanding of their developmental pathways. Similarly, limiting clinical attention to adults with BPD may prevent youngsters with the same symptomatology from getting the help they need, leading to prolonged phases of suffering and maladjustment (Chanen et al., 2017). Therefore, the argument that personality in youth is insufficiently stable for a PD diagnosis had to be brought to the test before concluding about the utility of the BPD diagnosis in youth.

The stability of personality shows a relative increase with age (Briley & Tucker-Drob, 2014). On average, personality matures: Individuals in early and middle adulthood become less prone to negative emotionality, more conscientious and agentic, and achieve more agreeableness and social maturity (Bleidorn, 2015; Roberts et al., 2001). Regarding the development and stability of personality, theories have highlighted different factors (Briley & Tucker-Drob, 2014).

Five-factor theory follows the notion that the increasing stability is the product of endogenous maturational processes, with underlying biological and genetical influences (McCrae et al., 2000). Social investment theory puts greater emphasis on social and environmental influences (Roberts & Jackson, 2008). Personality may develop in response to environmental challenges and developmental transitions, and may stabilize because individuals seek niches according to their personality development. Importantly however, most changes in personality take place not only in puberty but also between ages 18 and 40 as well, and even in later adulthood personality does not completely stabilize (Roberts et al., 2006).

A couple of studies have investigated the stability of PD in youth. Chanen et al. (2004) found that in 15-18 year old patients with an initial PD diagnosis, 74% still fulfilled criteria for any PD in a 2-year follow up. Stability for BPD specifically was only at 40%, but interpretation of this finding was limited by a small case number of BPD patients. Another study reported a decline in BPD traits from age 14 to 24, but high rank-order stability (Bornovalova et al., 2009). BPD-related features in 6-12-year-old girls, i.e. impulsivity, negative affectivity and interpersonal aggression, showed high year-to-year stability (Stepp et al., 2010). Crucially, rates of remission in adult BPD have been found to be high (Gunderson et al., 2011; Zanarini et al., 2012). In sum, evidence indicates that adult BPD is not a construct of particularly high stability, nor is the stability of BPD features in youth remarkably low. Consequently, personality patterns can change - also in people with BPD. Thus, setting the age limit for diagnosing BPD to the age of 18 years is arbitrary. Since DSM-IV, diagnosing BPD no longer requires the patient to have attained adulthood.

More recent research has consequently put a focus on adolescent BPD. A major goal was to investigate whether BPD symptoms in adolescence were comparable to those in adulthood in terms of validity and reliability. Indeed, internal consistency of the nine BPD traits is comparable to that in adults, and their validity is indexed by strong associations with indicators of clinical impairment and comorbidity (Glenn & Klonsky, 2013). In 2015, a review concluded that more recent studies, especially those using measures developed especially for the use in adolescents, show that the diagnosis of adolescent BPD is valid and reliable (Fossati, 2015). Following these new insights, PD research has expanded to the age range of adolescence, but little is still known about the earlier stages of BPD. Although it is widely agreed upon that risk factors and roots of BPD lie in childhood (De Clercq & De Fruyt, 2007; Fonagy et al., 2003), there is a relative lack of studies on BPD features before adolescence. The early identification of individuals at risk could lead to earlier treatment transferal and prevent adverse life trajectories associated with adult BPD.

1.2 Category or dimension

Another recent development in BPD research concerns the categorical diagnostical approach. In spite of high rates of remission in BPD, rank order stability is high (Bornovalova et al., 2009). A categorial classification neglects this observation. Section III of DSM-5 has introduced the *alternative model for personality disorders* (AMPD), enabling a more dimensional diagnostic approach (American Psychiatric Association, 2013). In the AMPD, two criteria are used to define PD: Criterion A dimensionally indexes the degree of overall personality dysfunction. This includes deficits in intrapersonal (self-direction and identity) and interpersonal function (intimacy and empathy), which are assumed to underlie all PD. In Criterion B, pathological personality traits from five trait domains are described (negative affectivity, detachment, antagonism, disinhibition and psychoticism), which characterize the nature of the PD. Integrating the AMPD into the life span approach, experts in the field have suggested that an individual's position on Criterion B dimensions may be apparent early in life and maintain rank-order stability throughout the life span (Sharp, 2020). In contrast, Criterion A would undergo a qualitative shift during the accompanying developmental tasks of adolescence (taking on an adult role, forming an integrated sense of self), making adolescence a sensitive period for the onset of PD (Sharp, 2020). However, evidence about the developmental course of Criterion A personality function still needs to be accumulated. First studies have shown that self-reports of personality functioning provide a valid assessment in adolescents (Feenstra et al., 2011; Goth et al., 2018). There is a call to identify precursors of impaired personality functioning in children and adolescents (Birkhölzer et al., 2021).

Moreover, not only full BPD but subthreshold and even single BPD symptoms in adolescents and adults have now been shown to exert a significant impact on quality of life, social and global functioning, clinical severity and psychiatric comorbidity (Ellison et al., 2015; Kaess et al., 2017; Thompson et al., 2018; Zimmerman et al., 2011), highlighting their clinical relevance. In consequence, studies have started to use the number of BPD symptoms as a dimensional index of BPD severity instead of applying the diagnostic category (Stepp et al., 2013; ten Have et al., 2016; Yen et al., 2013). Also, the expression of BPD pathology may change during the life course (Videler et al., 2019). Youth often present with high impulsivity and a higher number of symptoms. In contrast, in later adulthood the number of symptoms decreases, but whereas the categorial cut-off may not be met anymore, general maladjustment and low social functioning remain. The use of dimensional approaches to BPD pathology has therefore also been recommended given their higher age neutrality (Videler et al., 2019) and may help us to investigate and refine our knowledge about the developmental pathways of PD.

1.3 Developmental models of borderline personality disorder

Several complementary developmental models have been proposed for BPD. Among the best known are the *emotion dysregulation* model, posing that emotional dysregulation (increased sensitivity, intense reactions to emotional stimuli and slow return to baseline) is at the core of BPD symptomatology (Linehan, 1993); the *mentalizing/reflective dysfunction* model, based on attachment theory and proposing that difficulties in making sense of their own and other's mental states make up the key feature of BPD (Fonagy & Bateman, 2008); the *interpersonal hypersensitivity* model, suggesting that psychobiological dispositions account for heightened rejection sensitivity in people with BPD (Gunderson & Lyons-Ruth, 2008); and, closely related, the *hyperbolic temperament* model, denoting a temperamental tendency to experience intense negative emotion, especially in response to interpersonal frustration (Zanarini & Frankenburg, 2007). A more recent expansion on Linehan's theory has suggested to put it into a developmental psychopathology framework (Crowell et al., 2009).

These models of BPD assume an interplay of (1) intrapersonal or biological predisposing factors such as temperament and emotional vulnerability on the one hand and (2) environmental risk factors on the other. They have in common that interpersonal factors are assigned a central role in the development of the disorder: In the etiology of BPD, insecure or disorganized attachment and traumatic interpersonal experiences play a major role. Linehan describes the environments in which later BPD patients grow up as "invalidating": individual experiences, especially those related to negative emotion, are either not acknowledged or declared inaccurate. In the expression of the disorder, interpersonal triggers are most likely to elicit the dysfunctional behavioral responses (Brown et al., 2002; Crouch & Wright, 2004; Miskewicz et al., 2015). Moreover, BPD patients show dysregulated biological responses towards social stressors (Drews et al., 2019; Weinberg et al., 2009).

Winsper (2018) has proposed a model for BPD that integrates knowledge from existing models (Figure I): Following a developmental perspective, the earliest risk concerns broader family risk factors and prenatal adversity. These may influence infancy and early childhood risk factors, contributing to difficult temperament and parenting difficulties or abuse. A particularly important role at this developing stage of the disorder may consist of the interplay between these child and parent risk factors. They likely lead to a lack of adaptive co-regulation (Hughes et al., 2012) and social communication (Fonagy et al., 2017) between parent and child. The lack of successful co-regulation and social communication contributes to emotion dysregulation and deficits in social cognition, which are supposed to constitute the core mechanisms of BPD. These deficits are likely to evoke more invalidating responses by the environment, leading to putative reciprocal effects. Social mechanisms are accompanied by biological mechanisms, such as fronto-limbic dysfunction or dysregulation of the physiological

stress response. Over time, these patterns consolidate into maladaptive emotional, cognitive, behavioral, and interpersonal traits that form the phenotype of BPD. I will base my elaborations on Winsper's model, as it integrates theories with a focus on emotional dysregulation and those with a focus on social processes into a developmental model. It therefore acknowledges a life span perspective on BPD and provides a framework explaining how individual and caregiver factors lead to insufficient co-regulation.

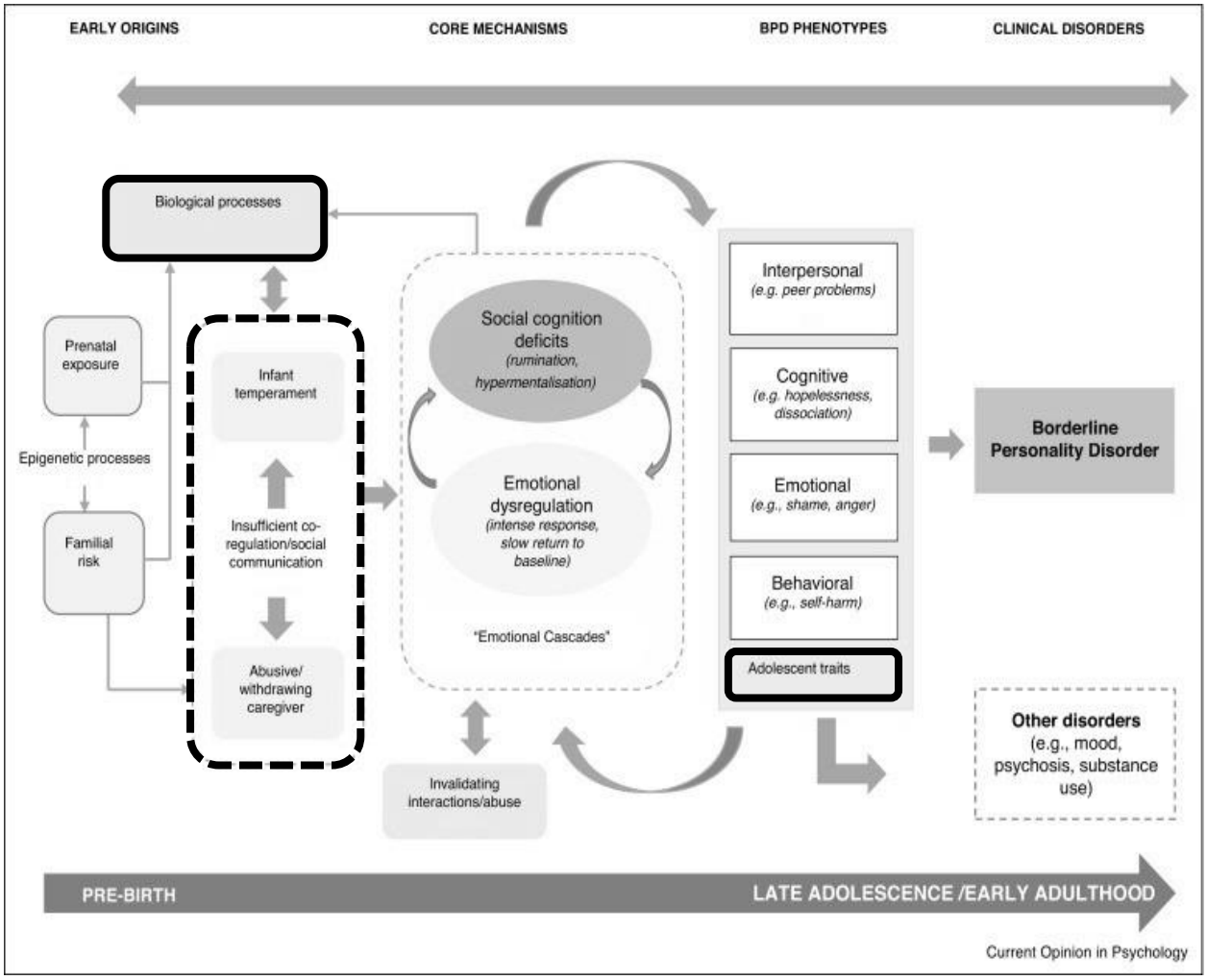

Figure I: A tentative logic model delineating the pathogenesis of Borderline Personality Disorder (BPD) from conception onwards. Adapted from "The aetiology of borderline personality disorder (BPD): contemporary theories and putative mechanisms" by C. Winsper, 2018, *Current Opinion in Psychology,* 21, p.106. Copyright 2017 by Elsevier Ltd. Adapted with permission. The current work focusses on interpersonal processes shaped by the interplay of child and caregiving factors, associated biological processes, and early BPD phenotypes/ adolescent traits (framed in bold).

I will hereunder review the theoretical and empirical background regarding central early origins and mechanisms of BPD that Winsper (2018) suggests: aspects of interpersonal risk such as maladaptive caregiving and insufficient co-regulation, and child temperament as an

intrapersonal factor. Co-regulation further involves biological processes that I will highlight. Herein, I will identify gaps in knowledge that I approached within this work.

1.3.1 Intrapersonal factors

Temperament: Temperament is viewed as a biologically based precursor of personality (Deal et al., 2005). Thus, it is a likely candidate in the study of intrapersonal predisposing factors for PD. Temperament describes individual response tendencies, in which varying brain systems are supposed to underlie organizational differences in the activation, inhibition, and maintenance of behavior (Cloninger et al., 1993). Temperament for example predicts an individual's response to novel stimuli or frustration. Individual differences in these response tendencies can already be observed in infancy (Putnam et al., 2001). In Cloninger's application of his psychobiological model of temperament to the field of PD (Cloninger & Svrakic, 2008) he proposes that extremes in temperament dimensions characterize specific PD. Regarding BPD, he suggests a temperamental profile of high novelty seeking, high harm avoidance and low reward dependence (Cloninger & Svrakic, 2008). Other temperamental dimensions that have been investigated in association with BPD are those of negative affectivity and impulsivity (Stepp et al., 2016). However, studies on Cloninger's temperamental model have investigated temperament concurrently with BPD diagnosis, whereas prospective studies on the relationship are lacking. As a methodological issue, novelty seeking, harm avoidance and reward dependence that are measured at the same time as (B)PD pathology may be highly confounded with the pathology of the disorder.

According to a diathesis-stress model of psychopathology, children with vulnerable temperamental styles may react more intensely and maladaptively to adversity. As Winsper (2018) suggests, child temperament and maladaptive parenting are likely to interact in the development of BPD. In line with this theory, adolescents with high levels of negative emotional reactivity have been found to develop more BPD symptoms when growing up in an environment of high family adversity (Stepp, Scott, et al., 2016). In the same sample, child and parental risk factors were also shown to exert reciprocal effects on each other: poor child self-control and harsh parental discipline exacerbated each other over the years, and effects of these two factors on BPD symptoms were partially mediated by this reciprocity (Hallquist et al., 2015). Therefore, the interplay between individual and environmental factors remains an important topic of research. However, up to date there are only few longitudinal studies that investigated interaction effects between temperamental dispositions and family risk factors.

Sex: Although not part of Winsper's model, another variable deserving attention as an intrapersonal factor is child sex. Early studies found a considerably higher prevalence of BPD in women compared to men (Widiger & Weissman, 1991). Later studies, drawing from community samples, found sex differences to be noticeably smaller compared to the initial

estimates (Grant et al., 2008; Sansone & Wiederman, 2014). To a relevant extent, sex ratios in patient samples can probably be attributed to sampling bias, as women might be more likely to seek treatment (Skodol & Bender, 2003). However, there are differences between male and female BPD patients regarding the type of symptoms and comorbidity (Sansone & Sansone, 2011). Moreover, sex is known as a factor moderating the response to stress and adversity throughout the development (Bale & Epperson, 2015): Whereas males might be more sensitive than females towards prenatal risk, females might be more prone to develop mental health problems, especially internalizing problems, when they experience adversity throughout childhood and adolescence. However, sex-specific vulnerability towards adversity might be specific to the type of adversity and studies assessing sex-specific pathways towards BPD are lacking.

1.3.2 Interpersonal factors

Intense, unstable relationships, marked by idealization and devaluation, and frantic efforts to avoid abandonment characterize BPD as a disorder of interpersonal functioning. Further, self-harm and suicidal gestures are often elicited by interpersonal triggers (Brown et al., 2002; Crouch & Wright, 2004; Miskewicz et al., 2015). It is therefore crucial to view BPD and its development in the context of relationships (Howard et al., 2021). Among the first relationships that an individual forms are those with the primary caregivers. Different aspects of parent-child relationships have been investigated as risk factors of BPD pathology.

Parent-child-interaction patterns. Several caregiving factors such as parental rejection and low warmth have been identified as risk factors for BPD (review: Stepp, Lazarus, et al., 2016). Most studies investigating effects of parenting behavior have used questionnaire measures. The observation of specific behaviors shown during actual parent-child interaction can, however, constitute an objective way to assess aspects of the parent-child relationship. Studies can use video-coding to evaluate interaction patterns. So far, only a handful of studies has examined mother-child interactions in connection to BPD symptoms. Measured at 18 months, in n = 56 mother-child dyads, maternal withdrawal but not intrusive behavior during a separation-reunion procedure (Strange Situation, Ainsworth et al., 1978) predicted child BPD symptoms in adolescence (Lyons-Ruth et al., 2013). Maternal hostility during a teaching task at 42 months was related to BPD symptoms in early adulthood (n = 162) (Carlson et al., 2009). During adolescence, conflict discussion tasks have been used as the context for mother-child interactions: Mother-child dyads in which adolescents engaged in self-injury (n = 17) where more likely to escalate conflict compared to healthy control dyads (n = 20) (Crowell et al., 2013). Another study showed that positive maternal and dyadic affective behavior during conflict discussion were associated with a decrease in adolescent BPD severity over time, whereas negative escalation, again, was associated with overall higher BPD severity (n = 74)

(Whalen et al., 2014). Finally, psychological control shown by mothers with BPD during a problem solving task was associated with the adolescent BPD feature of affective instability (BPD n = 28, control group n = 28) (Mahan et al., 2018). Overall, these findings indicate that stressful or challenging interaction tasks between children with BPD symptoms and their mothers are characterized by more negative behavior during the developmental phases of infancy, toddlerhood and adolescence. Apart from these few observational studies, most research regarding the parent-child relationship has still been based on adult patient (retrospective) self-reports (Boucher et al., 2017), bearing the risk of recall bias. Additionally, the extant studies using observational paradigms are predominantly limited by relatively small sample sizes. They do, however, point to the relevance of mother-child interaction patterns. Given the methodological limitation of small samples and that research is missing especially regarding observed interactions during middle childhood, more research is warranted. The exact interaction patterns that contribute to BPD symptom development might be specific to the developmental period, and existing studies do not allow for a direct age comparison.

Attachment. Attachment theory is based on the work by Ainsworth and Bowlby (1991). When caregivers serve as a secure base when the child is exploring and as a safe haven when it is in distress, they are supposed to foster secure attachment and optimal child development. This includes child self-regulatory abilities. In contrast, rejecting, inconsistent or abusive parenting can lead to insecure avoidant, preoccupied, and disorganized attachment styles. Attachment style is therefore a derivate of the quality of the parent-child relationship. Critically, caregiver-child relationships serve the organization of internal working models which are used to predict the behavior of the self and others in later attachment relationships. Based on attachment theory, Fonagy et al. (2003) prioritize the capacity for mentalization in their developmental model of BPD, which normally develops in early attachment relationships: A parent mentalizes about the offspring's feelings, needs and desires. Having one's mental state reflected by the caregiver is crucial to develop the capacity to hold one's own mental state and that of other persons in mind. This quality may be absent in abusive or neglectful parent-child relationships. A failure to mentalize may lead to misinterpretations of other's intentions and facilitate the development of BPD. In accordance, studies found that BPD patients have impaired social cognition abilities, e.g. in recognizing emotions, thoughts and intentions of others (Anupama V et al., 2018; Preißler et al., 2010).

Attachment can be assessed from later infancy onwards using the Strange Situation paradigm (Ainsworth et al., 1978). Despite this possibility, most studies investigating attachment style and BPD have been carried out cross-sectionally in adult patients, using interviews or self-reports. Findings from adult studies have consistently shown relationships between insecure, especially preoccupied and disorganized (unresolved) attachment styles and BPD (e.g.

Barone L et al., 2011; Diamond et al., 2014; Peng et al., 2021). Longitudinal studies on attachment and later BPD are rarer and have produced more mixed results. In a study observing attachment behavior at 18 months during the Strange Situation paradigm, there was no relationship with BPD symptoms in late adolescence, whereas the effect of maternal behavior was significant (Lyons-Ruth et al., 2013). In another study, the link between early attachment disorganization and BPD symptoms became nonsignificant when later childhood self-representation was entered into the model (Carlson et al., 2009). Child attachment disorganization assessed at age 8, however, significantly predicted BPD symptoms (Lyons-Ruth et al., 2013). Thus, early maternal caregiving factors and timely more proximate aspects of child interactional behavior were stronger predictors compared to early child attachment patterns. All in all, however, there are very few studies regarding the very early caregiver-infant relationship and their longitudinal relationship with BPD pathology.

Co-regulation. In their application of social baseline theory (Coan, 2008) to BPD, Hughes and colleagues (Hughes et al., 2012) stress the importance of viewing emotion regulation as both intrapersonal and interpersonal. Guided by biological principles of risk distribution, load sharing and the economy of action, humans are predisposed to use social proximity as means of emotion regulation (Coan, 2008). Co-regulatory processes are supposed to conserve individual resources. The initial source of co-regulation is the primary attachment relationship. During the first months of life, the caregiver mirrors or verbalizes the infant's internal states, aiding self-perception, and later on aids and encourages the child's self-control and regulation (Pauen, 2016). In older children, co-regulatory tasks of the caregiver include the support of the child's self-reflection and self-management. Successful co-regulation and secure attachment have a positive impact on the development of fronto-limbic circuits that underlie self-regulation (Hughes et al., 2012). In contrast, if co-regulation fails, such as in abusive or neglectful parenting, children lack the basis on which to develop sufficient self-regulatory abilities. Put into a life span perspective, growing up, children with lower self-control may act less socially competent and experience lower peer-acceptance (Crick et al., 2005; Gunnar et al., 2003; Shields et al., 1994). This might further decrease the likelihood for adaptive co-regulation within friendships or other social relationships in later life.

Biological processes in co-regulation. As suggested in Feldman's model of bio-behavioral synchrony (Feldman, 2012), co-regulatory processes take place on both behavioral and physiological levels. Although it has not been entirely established how physiological co-regulation comes about, it is hypothesized to originate from genetic predispositions, prenatal programming, and behavior in the dyad, and is supposed to constitute a biological marker of the attachment system between caregiver and child (Feldman, 2017). It is well supported that parent-child synchrony on a behavioral level, characterized by mutual adaptation an

regulation, is beneficial to child self-regulation with medium effect sizes (Davis et al., 2017). In contrast, evidence regarding the role of physiological synchrony in child development has produced more mixed results (DePasquale, 2020). Physiological synchrony is defined as "*the dynamic, within-dyad coordination of physiological activity over time between two individuals that is directly tied to an interpersonal process*" (DePasquale, 2020, p.1755). One physiological system in which parent-child synchrony could be observed is that of the hypothalamic–pituitary–adrenal (HPA) axis. The HPA axis is a neuroendocrine system that plays a central role in the body's response to stress (Smith & Vale, 2006). Its activity can be indexed by the release of the steroid hormone cortisol. The HPA axis is a stress regulatory system that has been shown to be under strong social regulation (Adam et al., 2007; Gunnar & Donzella, 2002). Some proposals about parent-child cortisol synchrony have been raised which may be relevant in the context of BPD pathology: It may reflect a matching in stress responses, which may be problematic if both interaction partners are unable to cope adaptively; it could be a mechanism of stress contagion – but was also shown to be able to buffer against psychopathology; and it may be generally stronger in instances of high emotionality, irrespective of valence (Davis et al., 2018). Whereas, in parent-infant dyads, low risk interactions and positive behavior patterns are mostly associated with positive cortisol synchrony (i.e., cortisol levels of parent and child moving into the same direction), the few studies carried out in later childhood and adolescence show inconsistent results (DePasquale, 2020). In consequence, it is currently not clear whether cortisol synchrony is generally adaptive, and which factors may shape alterations in cortisol synchrony in mother-adolescent dyads. Hence, further research is warranted. In BPD patients, HPA axis activity seems to be dysregulated (meta-analysis: Drews et al., 2019). BPD is associated with elevated continuous cortisol output and a blunted cortisol reaction in response to social stressors. This finding may be relevant in the context of co-regulatory processes during parent-child interaction. If one interactional partner is likely to show dysregulated HPA-functioning, this might affect physiological co-regulation. Moreover, cortisol synchrony could be altered as a result of more maladaptive interactional patterns between mothers and their children with BPD pathology. However, so far, no study has investigated how the presence of child BPD traits impacts the association between parental and child cortisol levels during an interaction. If we want to further dismantle the dynamics of co-regulation and how they might differ in individuals who develop BPD pathology, research on the in-the-moment processes during social interaction is strongly needed.

To sum up, the following lacks and limitations become apparent through the review of the current literature on the development of BPD pathology: a) Prospective studies are scarce, particularly regarding the very early parent-child relationship and b) the interplay of

interpersonal and child risk factors. c) The few studies that used observational paradigms to investigate mother-child interactions usually had very small sample sizes. d) Physiological co-regulation is a relatively new field of study. There are no studies so far exploring how adolescent BPD pathology and its interplay with dyadic behavior shape mother-adolescent cortisol synchrony. e) There is little evidence regarding BPD symptomatology in childhood age. Especially diagnostic interviews have rarely been applied in pre-adolescent samples.

2 Contributions of the current work

In the present work I aim to contribute to a greater understanding of the early stages and mechanisms in the development of BPD pathology by examining interpersonal correlates of early BPD expressions and their interplay with child factors. Given the salience of primary caregivers in the development of interpersonal skills and self-regulation, this dissertation focusses on different aspects of the mother-child relationship. Evidence regarding the developmental model (Figure I) is growing, particularly since BPD has been accepted as a valid diagnosis in adolescence. Still, it is not sufficient especially regarding the origins and early stages of BPD and the interaction effects of intra- and interpersonal risk factors.

Despite it being one of the core assumptions postulated in theoretical models and diathesis-stress conceptualizations of psychopathology, the interplay of intra-and interpersonal risk factors in the development of personality pathology has rarely been investigated. Longitudinal data are particularly lacking. Thus, **Article 1** focused on the effects of postpartum maternal bonding impairment (MBI) as an indicator of the very early mother-child relationship and its interactions with child temperament and sex. Outcomes were adolescent BPD traits and Criterion A personality functioning. Previous studies suggest that insufficient mother-child co-regulation plays a role in the development of child mental health, but research on physiological co-regulatory processes is still in its early phases. How do characteristics of the child and the dyad shape cortisol synchrony? **Article 2** examines mother-child cortisol synchrony in the context of child BPD traits and dyadic relational behavior. Moreover, the current study is the first to test a DSM-IV/5-based clinical interview of BPD traits in primary school age children. Can they be assessed, and do they matter? The aim of **Article 3** is to examine whether childhood BPD traits and their psychosocial correlates differ from those in adolescence, in order to achieve a better understanding about the early stages of the developing pathology. Also, Article 3 focuses on behavioral patterns during actual mother-child interaction in two different conversational contexts.

The current work is based on the longitudinal follow-up of two mother-child cohorts from the community. The first cohort started out in the years 2002/2003 and originally focused on the development and consequences of vulnerable temperament. The infancy and early childhood

assessments were funded by the German Research Foundation (MO 978/1-1/2). For the purpose of the current study, an adolescent follow-up took place at age 14 (n = 76). The second cohort started in 2008/2009, including assessments during pregnancy and infancy. Earlier investigations focused on the effects of prenatal stress on child development. In the process of the current project, a childhood follow-up took place at age 9 (n = 70). The Dietmar Hopp Foundation provided funding for the respective follow-up of both cohorts (1DH1813333). The study was approved by the Ethics Committee of the Faculty of Medicine at the University of Heidelberg (S-553/2016). The current follow-up of both cohorts consisted of a three-hour appointment comprising clinical interviews regarding child mental health, the completion of questionnaires by mother and child, and two mother-child interaction paradigms accompanied by physiological assessments.

2.1 Diathesis and stress? The interplay between postpartum maternal bonding impairment and child temperament and sex

Article 1: Fleck, L., Fuchs, A., Moehler, E., Parzer, P., Koenig, J., Resch, F., & Kaess, M. (2021). Maternal bonding impairment predicts personality disorder features in adolescence: The moderating role of child temperament and sex. *Personality Disorders: Theory, Research, and Treatment*. https://doi.org/10.1037/per0000433

Article 1 was dedicated to the investigation of prospective risk factors of PD pathology. We focussed on the relationship between the early mother-child relationship and adolescent PD features, and its interplay with child variables. (Retrospective) reports of adverse childhood experiences and harsh parenting have been shown to be associated with BPD and other personality pathology in multiple studies (Johnson et al., 2006; Stepp, Lazarus, et al., 2016). A problem with the existing literature lies in the sparsity of studies investigating the longitudinal associations of parent-child relationship difficulties and later (B)PD pathology. BPD patients' reports on the relationship towards their parents may be prone to recall bias and tinted by the cognitions that are currently activated by their disorder. Moreover, most studies that have been longitudinal only started assessing the parent-child relationship when children had reached school age or adolescence (Stepp, Lazarus, et al., 2016). Therefore, little is known about the very early parent-infant bond and its long-term effect on the development of maladaptive personality features. An exception are two observational studies that found that children whose mothers acted more withdrawn during a reunion episode in infancy (Lyons-Ruth et al., 2013), and children whose mothers acted more hostile during a teaching task in toddlerhood (Carlson et al., 2009), were more likely to develop BPD symptoms in adolescence. Gathering more knowledge about the effects of the very early mother-child relationship might however be especially relevant. Maternal bonding towards her newborn is proposed to be an important

basis for further mother-child interaction and child development (Brockington et al., 2001). We speak of MBI if mothers do not experience a positive emotional response towards their infant. Instead, mothers might feel that the child does not belong to them, or even harbour feelings of rejection. Given the negative emotions and cognitions about their infant, they could be more likely to show hostile or withdrawn parenting behaviour (Brockington, 2011). Likely, these mothers will be less able to support their child in successful emotion regulation. Additionally, harsh reactions could cause even more distress in the offspring.

Models of (B)PD propose an interplay between interpersonal and intrapersonal factors. Accordingly, some children would react more vulnerable when they experience interpersonal adversity, such as MBI (diathesis-stress), or show stronger susceptibility to both negative and positive environmental influences (differential susceptibility). As intrapersonal factors, the temperamental traits of high harm avoidance and high novelty seeking have been shown to be linked to BPD in adults (Barnow et al., 2007; Fossati et al., 2001; Joyce et al., 2003) and adolescents (Kaess et al., 2013). However, research regarding Cloninger's temperament model and BPD (Cloninger & Svrakic, 2008) has been exclusively cross-sectional until now. Some evidence has already suggested an interplay between these temperamental patterns and interpersonal risk factors in BPD patients. E.g., patients high in both harm avoidance and novelty seeking, and with experiences of childhood abuse and neglect were more likely to have BPD than patients who had either the temperamental or the interpersonal risk only (Joyce et al., 2003). In line with a biosocial theory of BPD, this evidence suggests that those with greater emotional vulnerability may be more susceptible towards the impact of adverse childhood experiences. However, studies that explore this interplay between temperament and interpersonal risk longitudinally when predicting adolescent personality pathology are lacking.

Child sex may be another factor moderating the effect of childhood adversity: In a sample of adult patients who had attempted suicide, the experience of sexual abuse in childhood was associated with meeting criteria for BPD in men but not women (Spokas et al., 2009). Women have, however, been found to be more likely than men to develop PTSD after exposure to trauma (review: Tolin & Foa, 2006). Studies focussing on mother-child relationship problems rather than trauma also found that girls but not boys developed depressive symptoms (Lewis et al., 2015; Veijola et al., 1998). All in all, evidence suggests that there is a greater risk for women to develop internalizing disorders in response to adversity, but regarding BPD specifically, only the study of Spokas et al. (2009), in which men showed greater vulnerability, could be identified. Thus, research is scarce and does not yet allow for an inference as to which sex might be more vulnerable to develop BPD symptoms. Sex-specific vulnerability may

be dependent on the specific nature of the risk factor, and also to the child outcome investigated, such as the type of psychopathology.

Given the noted lacks and limitations in existing studies, this study aimed to investigate the effect of early MBI and its interplay with child temperament (harm avoidance and novelty seeking) and child sex in the prediction of personality pathology. MBI was assessed by self-report at two weeks after child birth (Postpartum Bonding Questionnaire, Brockington et al., 2001). Mothers provided a questionnaire-based report on their child's temperament at age 5 (Junior Temperament and Character Inventory 3-6R ,Goth et al., 2003). At age 14, adolescents underwent the Childhood Interview for Borderline Personality Disorder (CI-BPD, Zanarini, 2003) to assess BPD traits and completed the Levels of personality function scale 2.0 (Hutsebaut et al., 2016) to assess overall Criterion A personality dysfunction.

In the present sample, MBI predicted both BPD symptoms and overall personality dysfunction in adolescence. Low harm avoidance also was a longitudinal risk factor for BPD symptoms and overall personality dysfunction. Investigating interaction effects, the association between MBI and overall personality dysfunction was stronger for children who were higher in novelty seeking and lower in harm avoidance. Regarding BPD, the effect of MBI was found to be significant for girls but not for boys. The pattern indicated differential susceptibility: girls also seemed to benefit more when maternal bonding was good.

These data highlight the relevance of very early caregiving factors in the development of personality pathology. They also indicate that, in order to identify those children that are at the greatest risk for developing (B)PD features, child factors such as temperament and sex should be taken into account. Children with an incautious, impulsive temperamental style are more likely to develop overall personality pathology in the future, especially in the light of MBI. These children might elicit harsh parental responses more often or react more intensely to them, whereas more reserved children might elicit more supportive parenting (Bryan & Dix, 2009). Contradicting Cloninger's theory, high harm avoidance in childhood had a protective effect regarding the development of later BPD symptoms, and low harm avoidance was a risk factor. There is considerable overlap between BPD features such as anger, impulsivity and interpersonal problems, and externalizing problems such as antisocial PD, delinquent behavior and substance use, which have been formerly found to be related to low harm avoidance (Cloninger & Svrakic, 2008; Hartman et al., 2013; Hiramura et al., 2010; Masse & Tremblay, 1997). Possibly, this overlap could account for the longitudinal effect of low harm avoidance observed in this study. Child sex did not alter the effect of MBI on overall personality pathology, but girls might be more vulnerable towards the development of BPD specifically.

2.2 Physiological co-regulation: The interplay of adolescent borderline personality disorder traits and mother-child interaction predicting cortisol synchrony

Article 2: Fuchs, A., Fleck, L., Lerch, S. Moehler, E., Koenig, J., Resch, F., & Kaess, M. (*under review*) Adolescent Borderline Personality Traits and Dyadic Behavior Shape Mother-Adolescent Cortisol Synchrony.

The second publication aimed to investigate the process of mother-child physiological co-regulation in the context of BPD traits. It examined how child BPD pathology and dyadic interaction patterns predict cortisol synchrony.

It is increasingly recognized that regulatory processes are subject to both intrapersonal and interpersonal processes (Hughes et al., 2012). Evidence shows that both behavioral and physiological states are coordinated between interaction partners over time (Feldman, 2012). The HPA axis is a physiological system that responds with heightened activity towards social stressors and can be regulated by social support (Adam et al., 2007; Dickerson & Kemeny, 2004; Gunnar & Donzella, 2002). Studies are accumulating that show coordination between parent and child cortisol levels, indexing cortisol synchrony (Davis et al., 2018). Impaired co-regulatory processes assumably play a major role in BPD, its course and development (Hughes et al., 2012; Winsper, 2018). Factors contributing to impaired co-regulation presumably include deviant interactional patterns as well as biological processes. HPA activity has been shown to be dysregulated in BPD patients (Drews et al., 2019). Given the finding of HPA dysregulation, the question arises whether cortisol synchrony will also differ for dyads in which one interactional partner exhibits BPD traits.

Cortisol synchrony can either be positive (cortisol levels of both interaction partners moving into the same direction), negative (a cortisol increase in one partner is related to a decrease in the other) or absent (asynchrony) (DePasquale, 2020). Given a scarcity of studies, it is so far not clear under which circumstances cortisol synchrony might take place and when it may be adaptive. Some evidence suggests that positive cortisol synchrony is connected to sensitive maternal behavior (Atkinson et al., 2013; van Bakel & Riksen-Walraven, 2008), whereas negative synchrony might be found under conditions of risk, such as low maternal sensitivity and child disorganized attachment (Nofech-Mozes et al., 2020¸ van Bakel & Riksen-Walraven, 2008). However, other studies found positive synchrony in higher risk dyads, e.g. exhibiting lower behavioral synchrony or higher punitive parenting (Hibel et al., 2009; Pratt et al., 2017). Hence, there are still too few studies to determine the conditions under which cortisol synchrony occurs, and more research is needed regarding the factors it is shaped by.

So far, only a few studies have investigated the influence of clinical disorders on dyadic cortisol regulation, and they mostly focused on the influence of parental disorder (LeMoult et al., 2015;

Merwin et al., 2017; Pratt et al., 2017), rather than child disorder. However, child BPD pathology may however impact the nature of parent-child cortisol synchrony, given that it is characterized by interpersonal difficulties that likely show on the behavioral level during parent-child interaction (Whalen et al., 2014), and due to potentially dysregulated HPA activity in those with BPD traits (Drews et al., 2019). Currently, there are no studies that tried to disentangle the factors potentially shaping cortisol synchrony by examining both child BPD pathology and the interactional behavior shown by the dyad. Doing so might give better insight into the interplay of influential factors.

This study used state-trait modelling for dyadic analyses. *Trait* cortisol was defined as the average cortisol level across assessment points, reflecting between-person differences. S*tate* cortisol indexed the within-person deviation from their average, e.g. describing an increase or a decrease with respect to the individual's average cortisol. We investigated the effects of adolescent BPD traits and dyadic behaviour on in-the-moment mother-adolescent cortisol synchrony (state) and mother-adolescent links of average cortisol (trait). Effects of adolescent BPD traits and dyadic behavior were first probed in separate models and then in a conjoint model, investigating their interactive effects. Dyads carried out two ten-minute interactional tasks. Cortisol was sampled prior to the first interaction, ten minutes after the first interaction, and ten minutes after the second interaction. Positive dyadic relational behaviour was rated by the Coding Interactive Behavior scales of low dyadic tension and high dyadic reciprocity (CIB, Feldman, 1998).

The following results were obtained: *State cortisol*: When the effects of dyadic behavior and adolescent BPD traits were investigated in separate models, positive cortisol synchrony was observed in dyads with higher positive dyadic behavior, and those without adolescent BPD traits. Negative cortisol synchrony was observed in dyads where adolescents met at least three BPD traits. In the model combining adolescent BPD traits and dyadic behavior, dyadic positive behavior and adolescent BDP pathology interacted in shaping cortisol synchrony, yielding more nuanced results: In the absence of risk (higher positive dyadic behavior and absence of any adolescent BPD traits), there was asynchrony. When only one condition of risk was present, positive cortisol synchrony was found. When both risk factors were present (lower positive dyadic behavior and adolescent BPD traits), cortisol synchrony was negative. *Trait cortisol*: Maternal and adolescent average cortisol levels were associated only under conditions of risk (lower positive dyadic behavior in the separate model, or the combination of lower positive dyadic behavior and at least one BPD trait in the combined model).

Article 2 is the first study to systematically address in-the-moment physiological co-regulation in mother adolescent-dyads, taking adolescent BPD traits and dyadic behavior as well as their interaction into account. Our study showed that, when modelled separately, the risk factor of

adolescent BPD traits was associated with negative cortisol synchrony, whereas adolescents without BPD traits showed positive synchrony. The overall pattern in our community sample showed that mothers' and adolescents' cortisol decreased together over the course of the interaction task. Adolescents without BPD traits and their mothers may therefore adapt to the laboratory situation together. In contrast, adolescents with BPD traits increased in cortisol when their mother's cortisol decreased. Possibly, adolescent BPD pathology may impede the adaptive co-regulatory process. However, considering interaction effects in the combined model, when adolescents had BPD traits but positive dyadic behavior was higher, positive cortisol synchrony was found. These findings suggest that positive mother-child interaction patterns may work as a buffer against the effect of BPD traits on effective co-regulation and support the adolescents' regulatory systems. Whereas the direction of state cortisol synchrony was dependent on the combination of risk and protective factors, average (trait) cortisol levels were only linked under conditions of risk (negative dyadic behavior, presence of BPD traits). Hence, associations between mothers' and adolescents' average HPA activity may present an indicator of risk.

2.3 Expanding the life span perspective: Differences and similarities of borderline personality disorder traits in adolescents and primary school aged children

Article 3: Fleck, L., Fuchs, A., Moehler, E., Williams, K., Koenig, J., Resch, F., & Kaess, M. (*under review*). Child versus adolescent borderline personality disorder traits: frequency, psychosocial correlates and observed mother-child interactions.

In Article 3 we aimed to gather evidence regarding the earlier stages of BPD symptomatology. Despite the emerging life span perspective, research on the early expression of BPD traits is still scarce. The question arises whether BPD traits can be identified before adolescence, and whether they are accompanied by psychosocial correlates that also characterize adolescent and adult BPD. Adult and adolescent BPD have been shown to be associated with maternal psychopathology and stress (review of longitudinal risk factors: Stepp et al., 2016), higher parent-rated emotional and behavioral problems (Ha et al., 2014; Winsper et al., 2017), lower quality of life (Kaess et al., 2017), high comorbidity (Ha et al., 2014; Kaess et al., 2013), and more negative mother-child interactions (Carlson et al., 2009; Crowell et al., 2013; Lyons-Ruth et al., 2013; Mahan et al., 2018; Whalen et al., 2014). The few studies assessing BPD features in childhood made use of different assessment tools and combinations of BPD-related concepts such as emotional negativity and interpersonal problems (Crick et al., 2005; Rogosch & Cicchetti, 2005; Zelkowitz et al., 2001, 2007). Only the Avon Longitudinal Study applied a clinical interview (CI-BPD, Zanarini, 2003) using DSM-IV/5 BPD criteria in 11.5 year olds (Zanarini et al., 2011), thus allowing direct comparison with older samples. Therefore, little

evidence exists regarding the question whether BPD traits can be meaningfully assessed in primary school aged children, and whether they exhibit similar psychosocial correlates.

In the present study, we compared the frequencies of the nine BPD traits implementing a German translation of the CI-BPD in 9-year-old children and 14-year-old adolescents. We also analyzed the associations between BPD traits, mother-child interaction patterns and common psychosocial correlates of BPD (comorbid disorders, quality of life, emotional and behavioral problems, indicators of maternal distress, mother-child interaction patterns) in both age groups and the combined sample. We compared their patterns of significance and directions of effects. Mothers and children from both cohorts engaged in two interaction paradigms: the planning of a fun day and the discussion of a conflict. Mother, child, and dyadic interaction patterns were coded using the CIB (Feldman, 1998).

BPD traits: Results in the current study showed no significant differences in the overall number of BPD traits met by 9-year-olds vs. 14-years-olds. In our community sample, adolescents on average reported 0.57 traits (range: 0-6), and children reported 0.63 traits (range: 0-4). There were also no significant differences regarding the frequencies with which each of the nine traits were reported. In both age groups, impulsivity (adolescents: 17.11%; children: 18.57%), self-harm/suicidality (adolescents: 9.21%, children: 14.29%), and intense anger (adolescents: 9.21%; children: 10.0%) were reported most frequently.

Psychosocial correlates: In both age groups, BPD traits were related to comorbidity, lower quality of life and mother-reported emotional and behavioral problems. BPD traits were related to lower prosocial behavior in children but not in adolescents. In contrast, associations with maternal distress were significant in adolescents only. There were also more significant associations between BPD traits and more negative mother-child interaction patterns in the adolescent cohort. Still, lower maternal structuring and higher child withdrawal also characterized interactions of dyads in which younger children exhibited BPD traits. Whereas several associations appeared to be weaker in the child cohort, effects consistently pointed into the same direction. Analyses of the combined sample of children and adolescents confirmed associations with all of the expected correlates, besides maternal and dyadic behavior during fun day planning. *Effects of paradigm*: In the adolescent and the combined sample, BPD traits were associated with more negative maternal and dyadic behavior modes during the conflict discussion only. In contrast, BPD traits were associated with more negative child behavior modes in both paradigms.

This study shows that BPD traits assessed in primary school age are accompanied by impairment in multiple aspects, highlighting that their assessment is meaningful. Our study provides a first validation of the CI-BPD for use in middle childhood. Childhood BPD traits showed however weaker associations with maternal distress and negative mother-child

interaction patterns, indicating age-related differences. Mother-child relationship problems might exacerbate with prolonged persistence of the traits. This is encouraging in that early intervention might be able to avert some of the pathways otherwise leading to long-term maladjustment. Maternal and dyadic behavioral interactions patterns seem to be particularly impaired during a conflict discussion. Such conversations likely require more maternal resources, as they ask for regulation of one's own emotions as well as those of the child. Interventions could therefore train mother-child interactions, especially in the context of challenging tasks such as the conflict discussion.

3 Discussion

The aim of this work was to examine the interplay of inter- and intrapersonal risk factors and expand the life span perspective on the development of BPD pathology. Besides intrapersonal factors such as temperament, sex, and indicators of impairment, the current work had a particular focus on different aspects of the mother-child relationship: the very early mother-child bond (Article 1), physiological co-regulation, indexed by cortisol synchrony (Article 2), and mother-child interaction observed on the behavioral level during two conversational contexts (Article 3).

3.1 Implications for the developmental model of borderline personality disorder

In context of Winsper's model (2018), the presented research contributes to a better understanding of the interplay of risk factors among the "early origins". It is suggested that child temperament and abusive/withdrawn caregiving contribute to a failure in co-regulation. Article 1 provided longitudinal evidence that MBI – as a potential risk factor for hostile or withdrawn parenting – predicts (B)PD features at age 14. Although not capturing in-the-moment mother-child interactions, Article 1 may be interpreted in the context of co-regulation. Mothers experiencing MBI, i.e. feelings of indifference or rejection towards their child, experience greater parenting stress (de Cock et al., 2017). Proposedly, they have greater difficulties in mentalizing their children's needs and emotional states, and reacting sensitively to these. However, these behaviors, which help co-regulate the child's internal states, are crucial for the formation of a secure attachment relationship (Nievar & Becker, 2008; Zeegers et al., 2017), promoting later self-regulatory abilities (Senehi et al., 2018). MBI moreover interacted with children's higher novelty seeking and lower harm avoidance to predict overall adolescent personality dysfunction. Children who act impulsively don't shy away from risk and are prone to break rules. They may thus elicit intrusive parenting more often. This effect might exacerbate in mothers who experience impaired bonding. As pointed out in the application of social baseline theory to BPD, conditions such as difficult temperament and poor caregiver

co-regulation abilities (e.g. due to their own attachment histories or psychopathology) may make the management of successful co-regulation of the child's impulses and emotions particularly challenging (Hughes et al., 2012). Given its predictive quality, postpartum MBI should be considered in the developmental model as one of the earliest risk factors as an indicator of early mother-child relationship difficulties (Figure II).

Interestingly, high harm avoidance was a protective factor for both overall personality dysfunction and BPD traits specifically, whereas the opposite association with BPD was reported in the theoretical model and cross-sectional studies (Cloninger & Svrakic, 2008; Joyce et al., 2003; Kaess et al., 2013). This finding may suggest that harm avoidance measured concurrently with the disorder may reflect the pathology rather than being a risk factor per se. Negative emotionality in children is associated with less supportive parenting in samples with a low socio-economic status (SES) (Paulussen-Hoogeboom et al., 2007). In contrast, in populations with a high SES, such as the current sample, high harm avoidance might elicit more supportive parenting behaviors (Paulussen-Hoogeboom et al., 2007) which foster positive personality development in the long run. This effect could be mediated by the finding that fearful children have been found to behave more cooperatively (Bryan & Dix, 2009). The association between temperamental patterns and development of (B)PD traits may therefore be dependent on factors such as developmental timing of the temperament measurement, environmental factors such as SES, and interpersonal factors such as the mother-child bond. Given that Article 1 also indicates MBI as a risk factor especially for girls, I suggest that the model should take child sex into account when investigating developmental pathways. Girls have been shown to exhibit more interpersonal features of BPD (Silberschmidt et al., 2015; Vanwoerden et al., 2019; Zanarini et al., 2011). Hence, an interpersonal pathway towards BPD may be particularly relevant in girls. The finding also reflects other research indicating greater vulnerability of the female sex toward childhood adversity (Bale & Epperson, 2015). However, girls also seemed to benefit more from good maternal bonding.

Illuminating the influences of child BPD and dyadic behavior on mother-child cortisol synchrony, Article 2 also sheds more light on the process of in-the-moment physiological co-regulation in the context of BPD pathology. As other studies have concluded, positive cortisol synchrony may mark adaptive dyadic interactions (Atkinson et al., 2013; Bakel & Riksen-Walraven, 2008). In the current study, adolescent BPD traits were associated with negative cortisol synchrony when relational behavior was not considered in the model. This may be considered maladaptive, given that, on average, maternal cortisol levels decreased during the laboratory visit, indicating habituation. This finding may be attributable to the distinct interpersonal characteristics of BPD pathology. Negative synchrony has recently been found between mothers and children with disorganized attachment (Nofech-Mozes et al. 2020).

Given that disorganized attachment also constitutes an important risk factor for BPD, attachment deficits may contribute to BPD traits being associated with negative cortisol synchrony. However, importantly, positive dyadic behavior during the interaction was able to buffer against this effect, resulting in positive cortisol synchrony. Interactions characterized by high dyadic reciprocity and low tension may therefore be able to compensate for some of the regulatory deficits associated with BPD pathology and enable adaptive co-regulation. Overall, biological processes of co-regulation seem to differ as a function of adolescent BPD traits.

Hughes et al. (2012) suggested that adults with a history of insufficient childhood co-regulation may have learned to rely on independent self-regulation, leading to early depletion of self-regulatory resources in patients with BPD. Even if the current evidence is not sufficient to draw firm conclusions about the adaptive capacity of cortisol synchrony, our finding might indicate that individuals with BPD traits may benefit from a positive relational context. As shown in Article 3, however, adolescent BPD traits are overall associated with more negative maternal, child, and dyadic interaction patterns, and this resource of adaptive co-regulation may only be available to some. During a conflict discussion in particular, mothers of adolescents with BPD traits behaved more intrusive, showed less sensitivity towards their child's signals, and provided less structure in the solving of the conflict. Dyads with adolescents who exhibited BPD traits showed less dyadic reciprocity and greater tension. These maternal and dyadic interactional impairments were not apparent during a positive interaction task. Conflict discussions may be particularly challenging to the caregiver, as they have to regulate both their own negative emotions as well as those of the child. CIB child scales showed that children and adolescents with BPD traits interacted more negatively with their mothers during both interaction tasks, further indicating that they might be more challenging to contain. Research has indicated that adolescents may still depend on their mothers to shift from negative to positive emotion during conflict discussion (Bommel et al., 2019). When children are met with harsh maternal reactions in the context of conflict, and external regulation does not take place, negative arousal is more likely to escalate. Invalidating parental behavior during conflict, especially punishing behavior, is positively related to adolescent BPD traits (Vanwoerden et al., 2019). Mother-child interactional quality has also been found to predict child social self-concept and overall self-worth (Paulus et al., 2018). Therefore, negative maternal reactions in time of disagreement may have a detrimental impact on these aspects of child personality functioning. In contrast, children who experience validation and support, even in times of disagreement, may experience positive influences on their self-concept. Overall, Article 3 suggests that effects of mother-child interaction patterns may be context-dependent.

Article 3 also aimed to expand the life span perspective on BPD pathology by investigating similarities and differences between primary-school aged children and adolescents.

Examining BPD traits and their associated impairments, it was found that BPD traits in 9-year-old children don't differ from those in adolescents regarding their frequency, and that they are related to comorbidity, lower quality of life and more emotional and behavioral problems from a maternal perspective. I therefore suggest that the developmental model be expanded by an "early phenotypes" section, where childhood BPD traits can be identified and are associated with beginning maladjustment. Childhood temperament (Article 1) may be considered an even earlier precursor falling under this section. In both the child and adolescent cohort, behavioral and affective BPD traits were the most common, such as impulsivity, anger, and suicidality/self-harm. This corresponds with other findings indicating that these impulsive traits characterize early expressions of BPD pathology, whereas they decrease in later life, but individuals still display enduring functional impairments and interpersonal problems (Videler et al., 2019). BPD traits in the child cohort overall showed weaker associations with maternal distress and mother-child interaction patterns. Regarding a life span perspective, this may indicate that some mother-child relationship problems exacerbate or only emerge in the critical developmental phase of adolescence or only arises after prolonged BPD symptomatology.

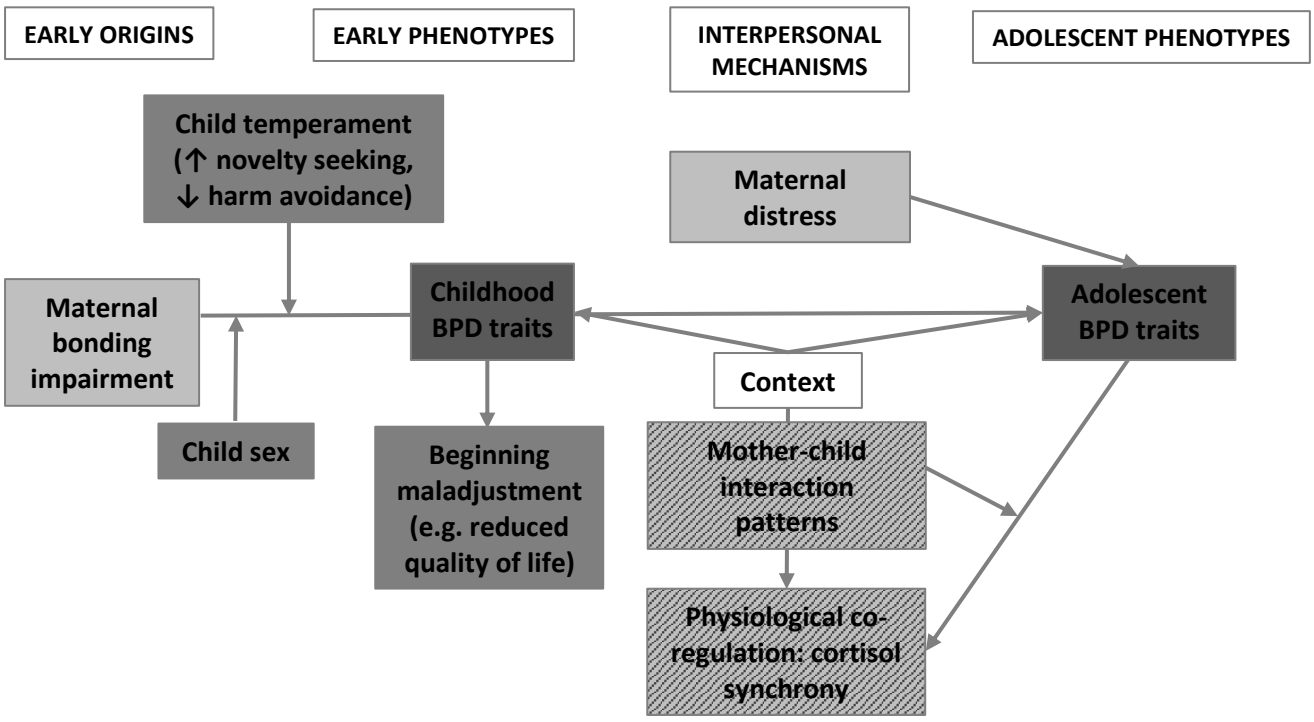

Figure II. Mother-child relationship factors in a developmental model of early BPD pathology.

3.2 Limitations and implications for future research

Future research can build on the current work to further expand the understanding of the developmental pathways to BPD pathology. Importantly, our study was a first application of the CI-BPD in primary school age. Its use to identify early symptomatology helps the

comparison and integration of study results from different age groups, refining our knowledge about BPD pathology during different phases across the life span. Based on the current study, paying attention to childhood expressions of BPD traits may be beneficial, given that they are associated with beginning impairment. Whereas there is first evidence that childhood BPD features predict later maladjustment (Zelkowitz et al., 2007), the limiting cross-sectional nature of Article 3 did not allow us to investigate whether early BPD traits specifically predict BPD in later life. Higher risk samples, e.g. with child protective service involvement, may allow us to investigate higher rates of BPD traits in future longitudinal studies.

With this work, scientific background is provided that BPD traits assessed in childhood are relevant to the well-being and adjustment. However, more research is required and it needs to be discussed at what point these traits ought to be considered as PD. Sharp (2020) has suggested that maladaptive personality traits (Criterion B in the AMPD, Section III in DSM-5) present a continuous aspect of personality function over time that can be recognized early in life. Criterion B would run along the extremes of temperamental dimensions and the internalizing-externalizing continua. Criterion A personality dysfunction – overall deficits in sense of self, self-direction, intimacy and empathy – would be marked by a discontinuous shift, starting its relevance during adolescence (Sharp, 2020). Adolescence as a developmental phase requires individuals to “bind” these aspects of personality into a coherent whole, developing an integrated sense of self and adaptive self-other relation. Therefore, only Criterion A would be able to account for an onset of BPD in adolescence. Concordance has however also been confirmed between Criterion B traits and the nine BPD criteria from DSM-IV/DSM-5 section II (Bach & Sellbom, 2016; Evans & Simms, 2018). This indicates that traits assessed with the CI-BPD also reflect Criterion B pathological personality traits. Those BPD traits observed most frequently in our cohorts (uncontrollable anger, impulsivity, self-harm-suicidality) are more descriptively in nature. They might be conceptually closer to Criterion B psychopathological trait dimensions and can be part of other developmental difficulties, such as conduct disorders. In contrast, cognitive symptoms or symptoms of disturbed relatedness (identity disturbance, feelings of emptiness, fear of abandonment), might be more closely related to Criterion A overall personality dysfunction. They may be especially relevant to the question of whether an individual would be diagnosed with a PD.

Additionally, Article 3 did not possess sufficient statistical power to investigate age group interaction effects, and similarities and differences were studied based on patterns of significance and direction of effects. It remains to be clarified whether the age-related differences indicating weaker associations between BPD traits and the mother-child relationship in middle childhood are meaningful and statistically significant. Also, BPD trait frequencies in a community sample are naturally rather low. Bigger high-risk samples may

enable us to disentangle factors that predict symptoms that reflect not only dysregulation and disruptive behavior, but also those marking a lack of a coherent sense of self. Moreover, whereas developmental research has investigated the development of identity and empathy as disparate aspects, evidence of Criterion A-defined personality functioning as a unidimensional construct has yet to be gathered. Longitudinal studies beginning in childhood should investigate the course of overall personality (dys)function from an early developmental stage to capture shifts related to age, developmental tasks, and environmental challenges.

Regarding the role of interpersonal factors in the development of BPD, the developmental model will benefit from future studies specifying how factors interact. Overall, there is a great need to view the development of BPD pathology from a transactional point of view, in which child dysregulation and the actions and reactions from caregivers and the environment influence each other over time (Fruzzetti et al., 2005). Studies investigating the developmental pathways of (B)PD pathology would create great methodological benefits by systematically evaluating both the parent-child relationship, and child BPD traits and personality functioning at multiple time points during the development. Additionally, there are some more specific targets for future research that are indicated by the current work: In Article 1, early MBI predicted the development of Criterion A personality functioning and BPD traits. Research should focus on the aspects of MBI that account for its effect: Is it mediated by either withdrawn or hostile behaviors, or may it have an effect independent of later interaction patterns? E.g., does the lack of close physical contact during the first weeks impact later self-regulatory abilities or the future mother-child relationship? We also unexpectedly found low instead of high harm avoidance to predict BPD. Future studies should investigate whether this association is specific to high SES samples and moderated by maternal behavior (Bryan & Dix, 2009; Paulussen-Hoogeboom et al., 2007).

Article 2 offers a relatively new approach to co-regulation in the context of BPD development, focusing on synchrony of physiological systems. Positive cortisol synchrony between adolescents with BPD traits and their mothers could be observed when they experienced the resource of positive dyadic relational behavior. While the findings indicate that positive synchrony may, under these circumstances, be an indicator of adaptive co-regulation, additional research might investigate whether these youth benefit in the long run. Furthermore, the study should be replicated in a clinical sample in which the resource of positive relational behavior may be available less frequently, or has to buffer against higher levels of BPD severity. Given a potential impact for interventions, studies should also investigate whether other experimental manipulations may alter cortisol synchrony. For example, the establishment of physical contact between mothers and their daughters has been shown to impact physiological arousal and arousal transmission (Lougheed & Hollenstein, 2018).

Importantly, it is not yet known whether patterns of cortisol synchrony are a precursor or a consequence of child BPD pathology. If we can improve mother-child relationship quality by means of intervention, we could examine whether this has an impact on cortisol synchrony, and whether this change is mediated by BPD pathology or vice versa, using a longitudinal design.

Lastly, whereas the current study has focused on the role of the mother in the development of BPD traits, fathers should be considered in similar ways. Due to the designation of mothers as primary caregivers, paternal factors have often been omitted in child developmental research (Cabrera et al., 2018). One study indicated that BPD may be characterized specifically by disorganized attachment towards both parents, as compared to one parent (Miljkovitch et al., 2018), indicating the absence of any secure attachment relationship. Here, the nonclinical control group was characterized by more secure attachment towards the father. In another study, attachment towards the father but not towards the mother was related to lower adolescent mentalizing abilities, and mentalizing mediated the link between attachment and emotion regulation (Gambin et al., 2021). These results suggest that there might be a unique contribution of the father-child relationship to the development of a BPD phenotype.

3.3 Implications for clinical practice

As Hughes et al. (2012, p.26) have argued, "*borderline pathology can be understood not only as a disorder of emotion dysregulation but also one of insufficient co-regulation across the life span*". The current work has put different aspects of the mother-child relationship into focus which may serve as targets for clinical intervention. It identified that both the very early mother-child bond and concurrent mother-child interaction patterns are strained in dyads in which children express BPD traits. In addition, physiological co-regulation follows a distinct pattern associated with child BPD traits. Whereas negative mother-child interaction patterns have been demonstrated to play a major role in the etiology of BPD, positive interaction patterns may promote a decrease of BPD pathology (Whalen et al., 2014). Interactions therefore provide a valuable target for intervention.

The early mother-child bond may provide a first target for indicated prevention. During the first weeks after childbirth, health care staff and midwives might screen mothers for signs of MBI. Risk factors for MBI include maternal depression and posttraumatic stress disorder, insecure attachment and childhood adversity, unplanned pregnancy and cesarean delivery (Faisal-Cury et al., 2020; Farré-Sender et al., 2018; Hairston et al., 2018; Lehnig et al., 2019; Muzik et al., 2013; Sockol et al., 2014). Hence, health care staff should pay close attention to the mental well-being of these mothers. Mothers experiencing MBI could be offered targeted treatments to improve the mother-child bond. At the same time, clinicians may pay close attention to the adjustment of children with an impulsive temperamental style and aim for

improvement of the mother-child-interaction. A transactional model of BPD in which child and maternal vulnerability impact each other is to be assumed (Fruzzetti et al., 2005; Pesonen et al., 2008). It is suggested that individuals with a temperamental disposition towards BPD more often evoke negative reactions among others, which in turn increases emotionality (Winsper, 2018). Mothers might need guidance in handling challenging reactions of emotionally vulnerable children and learn how to still acknowledge and validate their signals. Mentalization based methods for parental training have been examined in the recent years (Byrne et al., 2020). Although Byrne et al. concluded that the methodological quality of studies leaves room for improvement, it may provide a valuable approach in the context of BPD. Mentalization based treatment for parents seems especially well-suited as it may tap on the intergenerational transmission of BPD pathology. Mothers with BPD have been shown to have compromised mentalizing abilities, as they make more misattributions to the child's mental state (Marcoux et al., 2017; Schacht et al., 2013). Good parental reflective functioning can however predict secure child attachment (Slade et al., 2005) and buffer the effect of early difficult temperament on later behavioral problems (Wong et al., 2017). Thus, treatment focusing on the reflection of child mental states and needs may be particularly beneficial.

It was found that both mothers and adolescents exhibited more negative behaviors during conflict discussion in relation to the child's BPD traits. In consequence, it may be especially important to train validating communication in the context of disagreement. Next to learning positive communication patterns, it may benefit the development of a stable sense of self-worth when children experience that they are valued for their standpoints even if the interactional partner disagrees. Interpersonal skills training from dialectical behavioral therapy includes material about interpersonal validation which could be processed by both adolescent patients and their mothers. In addition, parenting interventions seem to benefit from using video feedback, as these interventions showed greater effects sizes in a meta-analysis compared to parenting interventions without video feedback (Bakermans-Kranenburg et al., 2003; Juffer et al., 2017). Using video feedback, therapists can provide parents with very concrete feedback about understanding and responding towards the child's behavior. Therefore, it aids the transmission of knowledge about interpersonal behavior into the actual interaction. Those interventions that were most successful in promoting sensitive behavior were also those most effective in promoting attachment security (Bakermans-Kranenburg et al., 2003). Turning back to the theoretical background of social baseline theory, these improvements in attachment relationships should foster child self-regulation as well as healthy expectations about co-regulation throughout the life span (Hughes et al., 2012).

4 References

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Original publications

Article 1. Maternal Bonding Impairment Predicts Personality Disorder Features in Adolescence: The Moderating Role of Child Temperament and Sex.

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Abstract

Research has shown associations between adverse parenting experiences and (borderline) personality disorder ([B]PD). A biopsychosocial model suggests that child characteristics and the environment interact in the development of symptoms. However, prospective data in this aspect are limited. This study focused on maternal bonding impairment (MBI, two weeks postpartum) and its interactions with child temperament (age 5) and child sex as predictors of BPD symptoms and general personality dysfunction in adolescence. Participants were 64 mother-child dyads from a community sample who took part in a 14-year longitudinal study. Higher MBI was a significant predictor of general personality dysfunction as defined in Criterion A of the alternative model for PD of the DSM-5. Interactions showed that the effect of MBI on general personality dysfunction was decreased for children higher in harm avoidance and increased for children higher in novelty seeking. There was also a negative main effect of harm avoidance on (B)PD features. Regarding BPD symptoms, the MBI x child sex interaction indicated differential susceptibility. Girls' but not boys' BPD symptoms were dependent on maternal bonding. Our results indicate that children at risk of developing personality pathology can be identified early in life. They stress the importance of early relationship disturbances in the development of personality pathology and refine the understanding of differential susceptibility factors in the context of MBI and PD symptom development. Our findings can be applied to target at-risk dyads for selective early prevention based on temperament and maternal bonding.

Keywords: Borderline Personality Disorder, Mother-Child Relationship, Maternal Bonding, Temperament, Differential Susceptibility

Introduction

There is increasing evidence that personality disorders (PD) should be regarded as life span developmental disorders that have their roots and antecedents in childhood (Tackett et al., 2009). All PD share general impairments in self- and interpersonal functioning such as sense of self, self-direction, intimacy and empathy, as defined in Criterion A of the alternative model for PD in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013). These general impairments underlie those pathological personality traits that are specific to each of the distinct PD. One of the best-studied PD in adolescence is borderline personality disorder (BPD). BPD is characterized by impulsive behavior and instability of affect, relationships and identity. In line with the life span hypothesis, recent evidence demonstrates that even subthreshold BPD symptoms can debilitate adolescent development (Kaess et al., 2017; Thompson et al., 2018; Winograd et al., 2008). Thus, there is a strong indication to identify at-risk children and adolescents as early as possible, before PD symptoms manifest and impede developmental processes.

Research has identified several problematic parenting behaviors such as harsh punishment and low affection which elevate the risk for the development of several PD (Johnson et al., 2006). Further, a substantial amount of research showed that childhood experiences of abuse, neglect, and inconsistent parenting play a role in the development of BPD (Stepp et al., 2016). However, many studies are limited by either using retrospective reports or by initiating assessments of parenting behavior when children had already reached school age or adolescence. In consequence, studies investigating early-life parenting as a longitudinal predictor of PD are sparse. Among the exceptions are two important longitudinal studies. Observed maternal withdrawal at 18 months (Lyons-Ruth et al., 2013) and observed maternal hostility at 42 months (Carlson et al., 2009) predicted the presence of BPD symptoms in late adolescence and early adulthood respectively. In their article, Lyons-Ruth et al. (2013) point out that, despite the literature's heavy focus on experiences of abuse, abuse may often be the tip of the iceberg only. In addition to episodic traumatic events, difficulties in everyday parent-child interactions may have an independent and maybe even equally important effect on personality development, despite often being of subtler nature. Mothers who withdraw from interactions with their child may for example miss their emotional cues, and would thus be less likely to validate them and support the child's emotional regulation. Still, little evidence exists regarding the consequences of subtler everyday-life mother-child-relationship disturbances.

Also, not much is known about the association between very early parent-child bonding and personality development. This lack of data, however, is a limitation, as the formation of an affective bond between the mother and her newborn might be critical for long term child development (Brockington et al., 2001). For example, close mother-to-infant contact as early as during the first hours after childbirth has been shown to predict the quality of later mother-

child interaction and infant self-regulation (Bystrova et al., 2009; Dumas et al., 2013). Further, assessment of maternal bonding as early as possible has the advantage of reflecting a mother's reaction to the infant's birth and her becoming a mother without too much influence of infant characteristics such as temperamental features or the emergence of social smiling. Maternal bonding impairment (MBI) is present when a mother is lacking a positive emotional response to her infant, resulting in feelings of indifference or rejection. MBI might therefore be a foundation for the development of parental withdrawal or hostile behaviors towards the offspring (Brockington, 2011). At the same time, BPD is marked by high sensitivity to rejection (Staebler et al., 2011), a factor possibly mediating the relationship between BPD symptomatology and interpersonal problems (Lazarus et al., 2016; Miano et al., 2013; Zielinski & Veilleux, 2014). This rejection sensitivity might be based on the experience of actual maternal rejection in childhood, as reflected in MBI, increasing the risk for maladaptive socio-emotional functioning throughout development (Downey et al., 1997).

Not all children are equally affected by their caregiving environment. In accordance with a biopsychosocial model of psychopathology, transactional models of BPD postulate that family behaviors and the child's vulnerabilities mutually influence each other (Fruzzetti, Shenk, & Hoffman, 2005). Likewise, personality functioning as in Criterion A is suggested to depend on a combination of biological and environmental factors (Bender et al., 2011). Therefore, the effect of familial experiences, such as MBI would be moderated by child characteristics. One of these moderators may be child temperament. Temperament is commonly viewed as a precursor of personality. In Cloninger's psychobiological model of temperament and character (Cloninger et al., 1993) he defines temperament as a biologically based emotional response tendency. He proposes that PD subtypes can be discriminated by specific combinations of the different temperament dimensions (Cloninger & Svrakic, 2008). Regarding BPD, cross-sectional studies could mostly confirm the proposed relevance of high novelty seeking (NS) and harm avoidance (HA) for the BPD diagnosis (Barnow et al., 2007; Fassino et al., 2009; Fossati et al., 2001; Joyce et al., 2003; Kaess et al., 2013; Svrakic et al., 1993). The anticipated role of low reward dependence (RD) in BPD could not be confirmed in a majority of these studies. NS describes a tendency of exploratory excitability, disorderliness and impulsiveness. HA is a tendency to react fearfully to new situations, show anticipatory worry and be easily fatigable. The combination of these two traits has been suggested to be the "temperamental substrate of affective instability" in BPD (Joyce et al., 2003, p.759). In psychiatric patients, those who reported both a "borderline" temperament (high HA and NS) and former experiences of abuse and neglect were 3-times more likely to have BPD compared to those with only one of these risk factors (Joyce et al. 2003). This finding indicates that a vulnerable temperament and adverse experiences may exacerbate each other's effects. Individuals who due to their temperamental disposition have greater difficulties regulating their emotions and

reactions could be either more vulnerable to the consequences of parenting difficulties or elicit them more often. In the study of Fossati et al. (2001), the effect of HA on BPD was not sustained after controlling for retrospective reports of parental care. Given that effects of HA and parental care were not independent from each other, they suggested that high HA, in contrast to high NS, might reflect attachment difficulties rather than a disposition. However, all of the abovementioned studies assessed Cloninger's temperament dimensions concurrently with PD diagnosis. Consequently, prospective data are lacking.

A second child characteristic that may moderate the relationship between MBI and personality pathology might be child sex. There are some studies suggesting that boys and girls would not be affected equally by childhood adversity or family disturbances. In a study among suicide attempters for example, childhood sexual abuse was associated with meeting BPD criteria in men but not in women (Spokas et al., 2009). In the context of depression, mother-child-relationship disturbances and maternal hostility have been shown to be longitudinal risk factors for girls but not for boys (Lewis et al., 2014; Veijola et al., 1998). Research reporting on possible mother-child-relationship x sex interactions is very limited in particular with regard to personality pathology. Moreover, findings are not conclusive as to which sex would be more vulnerable to the experience of family disturbances. These variations might be due to the differing nature of the investigated risk factors and might be specific to the child outcome assessed.

The aforementioned findings stress the importance of studying the parenting environment and child characteristics in interaction when investigating the developmental pathways of PD. Furthermore, research is needed to assess prospective risk factors and potential precursors as early as possible, starting in infancy at best. The current study fills this research gap by investigating the effects and interactions of MBI with childhood temperament and child sex in the prediction of general personality dysfunction and BPD symptoms.

Hypotheses

1) We hypothesized that MBI two weeks after birth predicts a) general personality dysfunction as defined in Criterion A (age 14) and b) BPD symptoms specifically (age 14).
2) We expected the effect of MBI to depend on levels of childhood NS and HA (age 5) for a) general personality pathology and b) BPD symptoms.
3) In exploratory analysis, we aimed to investigate the interaction between MBI and child sex predicting a) general personality dysfunction and b) BPD symptoms respectively.

Methods

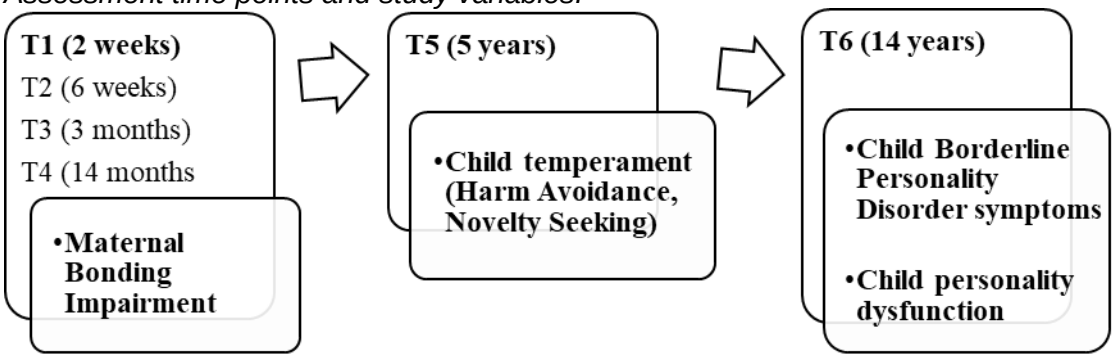
Procedure

This study was approved by the Ethics Committee of the Faculty of Medicine at the University of Heidelberg (S-553/2016). Its longitudinal design involved six assessment time points as follows: two weeks after birth (T1), six weeks after birth (T2), at three months (T3), at 14 months (T4), at 5.5 years (T5), and at 14 years (T6).

In the first part of the study (T1–T5), the aim was to examine the development and consequences of vulnerable temperament (see Möhler et al., 2006). At T6, mothers and their 14-year-old children took part in an assessment procedure that included a semi-structured clinical interview on adolescent mental health, a mother-child interaction task, and questionnaires. Figure 1 shows the assessment time points and respective study variables. To ensure participation by as many families as possible, families were given the option of an at-home visit or a laboratory visit. All mothers and their 14-year-old children signed informed consent before participation. Questionnaire and interview data from T1, T5, and T6 were included in the given study analyses. Participants received financial compensation for their participation in each of the assessment time points.

Figure 1

Assessment time points and study variables.



Note. In the current study, Maternal Bonding Impairment at T1 was used for analyses.

Participants

Mothers were recruited from local obstetric units, resulting in a community-based sample. Inclusion criteria were full-term deliveries, infant weight >2500g, Apgar scores >7, and good infant health at the first three postnatal examinations. At T1, 101 mothers and their newborns participated. At the T6 follow-up, 76 mother-child dyads attended the assessment, resulting in a 75% retention rate after 14 years. The reasons for attrition were as follows: Six families (5.9%) could not be relocated after moving, ten families (9.9%) indicated a lack of time or interest, one mother (0.9%) had deceased and eight families (7.9%) did not specify a reason for their non-participation. Of the remaining dyads, 12 had missing data for the T1 or

T5 questionnaires, resulting in a subsample of 64 mother-child dyads with complete data regarding the variables of interest.

Measures

Postpartum Bonding Questionnaire: At T1–T4, mothers completed the German version of the Postpartum Bonding Questionnaire (PBQ) by Brockington et al. (2001; Reck et al., 2006). The PBQ-score at T1 (two weeks) was used in the analyses of this study. The PBQ is a screening questionnaire developed to identify MBI. It measures impaired bonding, feelings of anger and rejection, anxiety about care and risk of abuse. Higher scores indicate greater bonding difficulties. The validity of the PBQ has been established (Brockington et al., 2006). A principal component analysis of the German version discouraged the use of the original subscales (Reck et al., 2006). As recommended, we used the total score (α = .79).

Junior Temperament and Character Inventory 3–6R: At T5 (age 5) mothers completed the Junior Temperament and Character Inventory (JTCI; Goth, Cloninger, & Schmeck, 2003). The JTCI is based on the psychobiological model of personality by Cloninger et al. (1993). The four temperament dimensions (NS: α = .89, HA: α = .92, RD: α = .75, persistence: α = .84) are assumed to have a biological basis and describe automatic emotional response tendencies based on individual differences in associative conditioning. The three character scales (self-direction: α = .84, cooperativeness: α = .87 and self-transcendence: α = .77) describe differences in intentional goals and self-concepts and are assumed to be based on propositional learning. The factor structure, internal consistency, and diagnostic validity of the JTCI have been confirmed (Goth et al., 2003).

Childhood Interview for Borderline Personality Disorder: At T6, the 14-year-old participants underwent an interview session with a trained psychologist. During this session, the Childhood Interview for Borderline Personality Disorder (CI-BPD; Zanarini, 2003) was conducted. The nine BPD symptoms can be rated as either absent (0 points), probably present (1 point), or definitely present (2 points). The validity of using the CI-BPD in adolescents has been confirmed (Sharp et al., 2012). Twenty interviews were rated by a second trained psychologist. Interrater agreement for each symptom ranged between 80% (“uncontrollable anger”) and 100% (“self-harm/suicidality” and “impulsive behaviors”), with an average of 93% agreement.

Levels of Personality Functioning Scale-Brief Form 2.0 (LPFS-BF): Following the interview appointment at T6 (age 14), adolescents completed questionnaires on their well-being, including the Levels of Personality Functioning Scale-Brief Form 2.0 (LPFS-BF) (α = .81) (Bach & Hutsebaut, 2018; Hutsebaut et al., 2016). This questionnaire assesses personality functioning in line with the alternative model for PD (American Psychiatric Association, 2013), which provides a dimensional approach to the diagnostics of personality

pathology. Higher scores indicate greater deficits in sense of self or interpersonal functioning. The LPFS-BF has been associated with other personality pathology measures and has shown good internal consistency. The questionnaire was translated into German for the purpose of this study. Translation was performed and double-checked by two translators from a professional translation agency.

Data Analysis

All analyses were conducted in Stata 16 (StataCorp. 2019. College Station, TX: StataCorp LLC.) with an $\alpha = .05$ significance level. Continuous predictor variables (MBI, HA, NS) were standardized prior to the analysis. To investigate the relationship between early MBI (two weeks), childhood temperament (age 5), child sex and PD features in adolescence (age 14), we computed separate models for the LPFS-BF and the CI-BPD. For the LPFS-BF-models, we applied (multiple) linear regression models (MLR) with the LPFS total score as the continuous dependent variable. For the CI-BPD models, a dependent variable consisting of the total number of symptoms rated as "definitely present" was created (max. score = 9). Because of the count-based nature of the CI-BPD-score, a generalized linear model (GLM) with the assumption of a binomial distribution with nine trials and maximum likelihood optimization was applied. To both the LPFS-BF and the CI-BPD models, predictor variables were added in the same consecutive manner: In model 1, MBI was included as a single predictor. In model 2, the HA and NS main effects were added together with the MBI x HA and MBI x NS interactions. Finally, child sex along with the interaction of MBI x child sex was added in model 3.

To control for the joint hypothesis that MBI would interact with childhood HA and childhood NS, model 2 was followed by a Wald test on both interaction effects. We also examined whether potential confounders (maternal age, maternal education, maternal history of psychiatric diagnosis, and child school type) changed the direction or significance of the estimates. As this was not the case, we have presented the results without these control variables.

Results

Descriptives

Ultimately, 40.8% of the T6 assessments took place at the participants' home, and 59.2% of the participants attended T6 assessments at our laboratory. The majority of mothers had a university degree (70.3%) and was still in partnership with the child's father (84.3%). A complete presentation of the demographic characteristics of the sample can be derived from Table 1. Importantly, dyads who dropped out or had missing data did not significantly differ from those with full data with respect to demographics such as maternal age ($t(99) = -1.09$, p

= .279), maternal education ($\chi 2(2) = 3.52$, $p = .172$), infant birth weight ($t(99) = -0.42$, $p = .677$) or infant sex ($\chi^2(1) = 0.05$, $p = .831$) at T1. Moreover, those with incomplete data did not significantly differ in the study outcome variables (general personality pathology: $t(74) = 1.17$, $p = .247$; BPD symptoms: $t(74) = -0.48$, $p = .633$).

Table 1
Characteristics of the study sample at T6 (N = 64)

Demographics	***M* (range)**
Mother age	48.1 years (34-60)
Child age	14.0 years (-)
	***n* (%)**
Mother partnership	
With child's father	54 (84.3%)
With different partner	4 (6.3%)
No partnership	6 (9.4%)
Mother education	
Secondary school	12 (18.8%)
University entrance diploma	7 (10.9%)
University degree	45 (70.3%)
Child sex	
Male	36 (56.0%)
Female	28 (44.0%)
Child school type	
Intermediate secondary school	10 (15.6%)
Grammar school	53 (82.8%)
Not classifiable	1 (1.6%)
Child PD features	*M* (*SD;* range)
Child LPFS-BF score	
Girls	7.21 (5.69; 0 - 21)
Boys	6.56 (5.45; 0 - 27)
Child #BPD symptoms	
Girls	.68 (1.52; 0 - 6)
Boys	.53 (.97; 0 - 4)

Preliminary Analyses

Table 2 shows unadjusted bivariate correlations between the study variables. MBI (PBQ) at two weeks (T1) was correlated with personality pathology at age 14 (T6) (LPFS-BF: $r = .27$, $p = .029$). The correlation between MBI and the CI-BPD symptom count at age 14 did not reach significance ($r = .19$, $p = .128$). The LPFS-BF showed a significant negative correlation with HA at age 5 (T5) ($r = -.28$, $p = .028$), whereas the CI-BPD did not ($r = -.17$, $p = .181$). NS at age 5 (T5) did not have a significant correlation with either the LPFS ($r = .00$, p = .969) or CI-BPD (r = .09; p = .465). There were no child sex differences regarding MBI at two weeks ($t(62) = 0.38$, $p = .701$), the temperament dimensions at age 5 (HA: $t(62) = 0.08$, $p = .936$. NS: $t(62) = -1.28$, $p = .206$) or personality pathology at age 14 (LPFS-BF: $t(62) = 0.47$, $p = .639$. CI-BPD: $t(62) = 0.48$, $p = .630$).

Table 2
Unadjusted pairwise correlation coefficients between study variables (Pearson's r)

	MBI-T1	LPFS-BF-T6	CI-BPD-T6	HA-T5
LPFS-BF-T6	.27*			
CI-BPD-T6	.19	.48***		
HA-T5	.31*	-.28*	-.17	
NS-T5	.06	.00	.09	.16

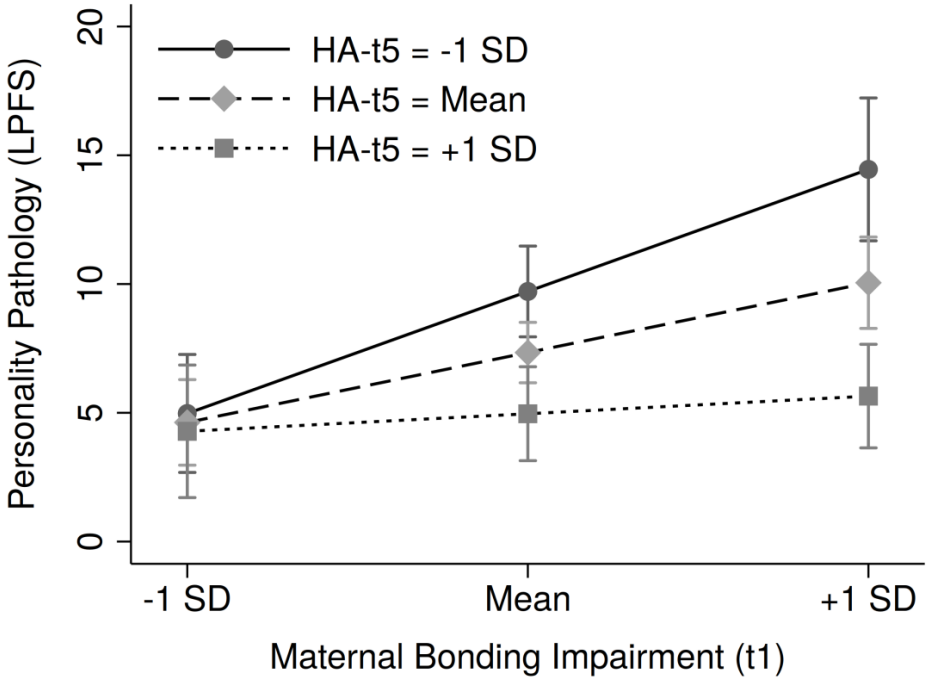
Note. LPFS-BF= Levels of Personality Functioning Scale. CI-BPD= Childhood Interview for Borderline Personality Disorder. HA= harm avoidance. NS= novelty seeking. PR= perseverance. RD= reward dependence. MBI= Maternal Bonding Impairment
*p <.05, **p <.01, ***p <.001.

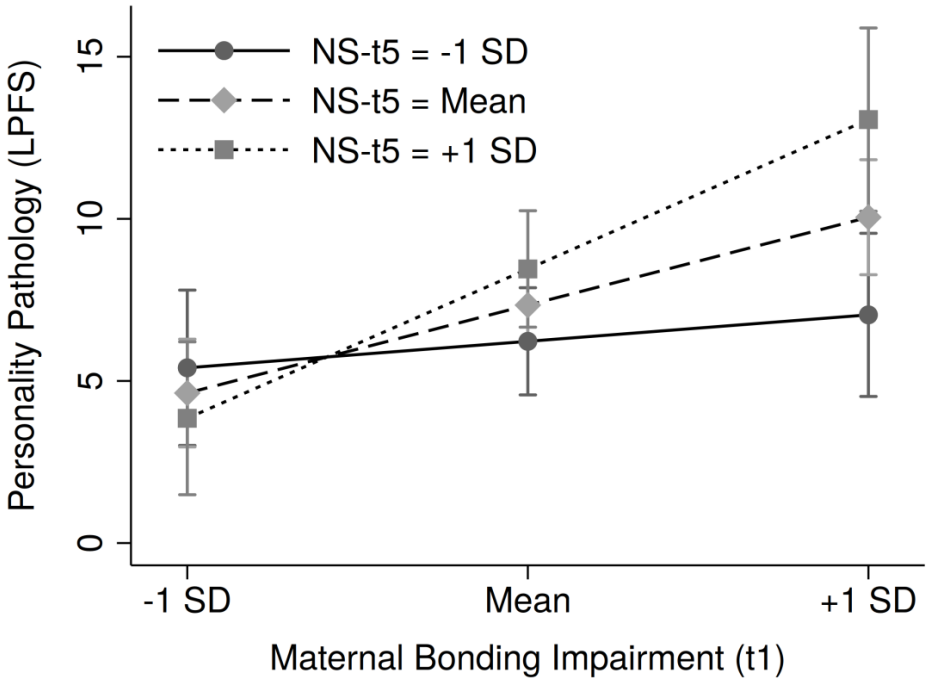
MLR: General personality pathology

MBI at two weeks was a significant single predictor of general personality dysfunction (β = .27, p = .028) and explained 7.5% of its variance (F(1, 62) = 5.03, p = .029). The second model including MBI x temperament interactions was also overall significant (R^2 = .40, F(7, 58) = 7.63, p < .001). The coefficients provided by multiple regression indicate a one unit increase in the outcome for every one unit increase in the respective predictor, while the remaining predictors in the model are kept constant at their mean (0 *SD*). Higher MBI at two weeks (β = .49, p < .001) and lower HA at age 5 (β = -.43, p = .001) were associated with greater personality pathology at age 14. In addition, there were two significant interaction effects. MBI was a stronger predictor of later personality pathology in children with higher levels of NS (β = .42, p < .003) and lower levels of HA (β = -.56, p < .001). The subsequent Wald test confirmed the combined hypothesis for the MBI x NS and MBI x HA interactions (F(2,58) = 8.54, p < .001). For a visual depiction of both moderation effects see Figure 2. The third model showed no significant interaction between MBI and child sex (β = -.00, p = .983). The effect of MBI on general personality dysfunction was similar for boys and girls. Complete statistics of the MLR models are provided in Table 3.

Figure 2

Personality pathology as functions of maternal bonding impairment and childhood harm avoidance (HA-T5) and childhood novelty seeking (NS-T5).





Note. Predictive margins at the mean and +/- 1 standard deviation with 95% confidence intervals.

Table 3

Summary of Regression Analysis for Longitudinal Predictors of Personality Pathology (LPFS-BF) at 14 years

Model	Variable	*β*	*SE*	*p*	*F*	*df*	R^2
1				.028	5.03	1, 62	0.08
	MBI	.27	.67	.028			
2				< .001	7.63	5, 58	0.40
	MBI	.49	.62	< .001			
	HA	-.43	.65	.001			
	NS	.20	.61	.079			
	MBI x HA	-.56	.51	< .001			
	MBI x NS	.42	.62	.003			
3				< .001	4.26	9, 54	0.41
	MBI	.49	.98	.008			
	HA	-.43	.67	.001			
	NS	.20	.63	.083			
	MBI x HA	-.56	.52	< .001			
	MBI x NS	.42	.67	.007			
	Child sex	-.02	1.17	.794			
	MBI x child sex	-.00	1.33	.983			

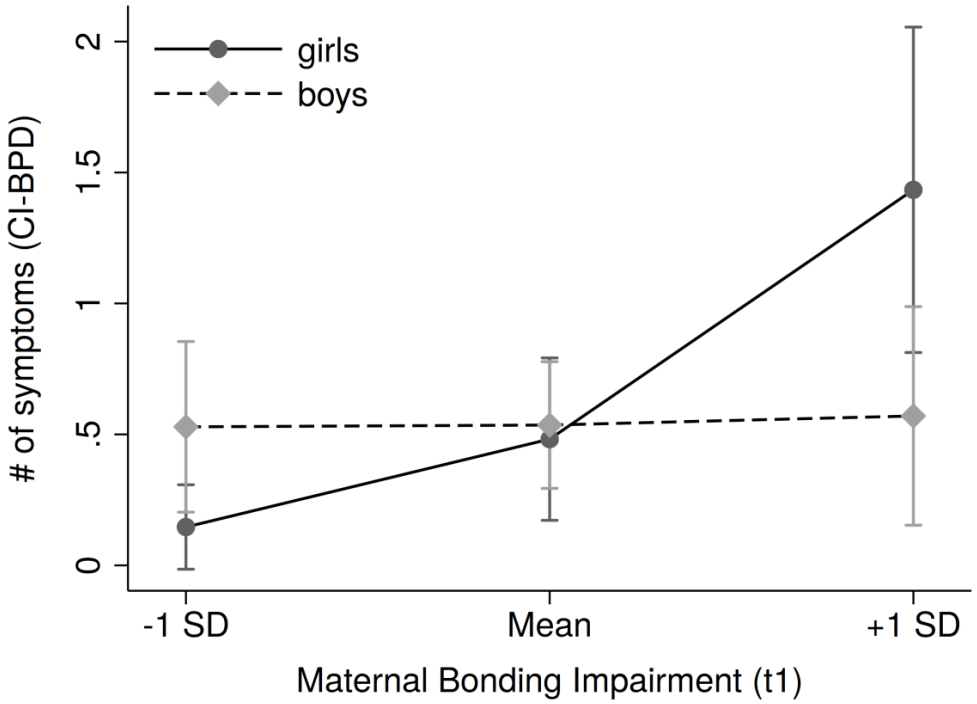
Note. LPFS-BF= Levels of Personality Functioning Scale. HA= harm avoidance. NS= novelty seeking. PR= perseverance. RD= reward dependence. MBI= Maternal Bonding Impairment.

GLM: BPD symptoms

MBI at two weeks on its own significantly increased the odds of BPD symptom development at age 14 (*OR* = 1.47, *p* = .013). The OR of 1.47 indicates that the odds of reporting a symptom increased 1.47-fold as MBI increased by one *SD*. The second model comprised the MBI x NS and MBI x HA interactions. There were no significant interaction effects between MBI at two weeks and childhood temperament at age 5 predicting BPD symptom development. However, there was a significant main effect of HA (*OR* = .51, *p* = .007). Lower HA was associated with increased odds of BPD symptom development in adolescence. The third model revealed a significant interaction between MBI and child sex (*OR* = .296, *p* = .002). With the MBI x child sex interaction in the model, the significant main effect of MBI (*OR* = 3.09, *p* <.001) applies to female sex. Therefore, the effect of MBI for boys was computed separately. MBI did not significantly predict BPD symptoms in boys (*OR* = .915, *p* = .755). The MBI x child sex interaction is shown in Figure 3. Compared to boys, girls developed more BPD symptoms under increased MBI, but also developed fewer symptoms when maternal bonding was good. A likelihood ratio-test determined that the final model was significantly superior to an intercept-only model of BPD symptoms ($\chi^2(7) = 32.06$, $p < .001$). All GLM statistics are shown in Table 4.

Figure 3

Number of BPD symptoms as a function of maternal bonding impairment and child sex.



Note. Predictive margins at the mean and +/- 1 standard deviation with 95% confidence intervals.

Table 4

Generalized Linear Model for Longitudinal Predictors of Borderline Personality Symptoms (CI-BPD) at 14 Years

Model	Variable	*OR*	*OIM SE*	95% CI	*p*
1	MBI	1.47	.27	1.08 – 1.99	.013
2	MBI	1.79	.34	1.24 – 2.60	.002
	HA	0.51	.13	0.31 – 0.83	.007
	NS	1.31	.22	0.94 – 1.82	.106
	MBI x HA	0.87	.13	0.64 – 1.18	.365
	MBI x NS	1.23	.23	0.86 – 1.77	.251
3	MBI	3.09	.87	1.78 – 5.39	<.001
	HA	0.57	.14	0.35 – 0.93	.024
	NS	1.32	.23	0.93 – 1.87	.121
	MBI x HA	0.77	.11	0.57- 1.03	.074
	MBI x NS	1.15	.22	0.79 – 1.66	.470
	Child sex	1.12	.50	0.47 – 2.68	.798
	MBI x child sex	0.30	.12	0.13 - 0.64	.002

Note. CI-BPD= Childhood Interview for Borderline Personality Disorder. HA= harm avoidance. NS= novelty seeking. MBI= Maternal Bonding Impairment.

Discussion

The present study is the first to investigate MBI and childhood temperament as predictors of PD features using a 14-year longitudinal design with assessments starting as early as two weeks following childbirth. We found that a mother's difficulties to establish an emotional bond toward her infant can have long-lasting effects on the child's personality development. Regarding general personality dysfunction, there was a stronger relationship with MBI in children who showed higher NS and lower HA at the age of 5. In the context of BPD, MBI predicted symptoms in girls but not boys.

Our finding that MBI was a prospective risk factor for personality pathology is in line with existing research reporting associations between various forms of adverse parenting experiences and PD symptoms. The newborn is fundamentally dependent on its caregiver to engage in reciprocal dyadic interactions (Feldman, 2012). These support the child's lifelong capacity for emotion regulation and mutually satisfactory social behavior, both of which are reflected in personality functioning. A mother with MBI might not be able to sensitively engage in these interactions, which require prompt and empathetic reactions to the child's signals. MBI as a form of maternal rejection might act as a developmental hardship from which children develop hypervigilance toward rejection cues (Downey et al., 1997). This hypersensitivity might, in turn, lead to interpersonal conflicts typical of BPD or to avoidance of social interaction typical of fearful forms of PD. It cannot be ruled out that the actual risk is attributable to prolonged mother-child relationship problems rather than the first two weeks after birth. Yet, it is remarkable that we were able to predict PD features from these early life data.

For both general and borderline PD features, low childhood HA was a longitudinal risk factor. According to cross-sectional findings in prior studies (e.g. Barnow et al., 2007; Kaess et al., 2013) and Cloninger's theory (Cloninger & Svrakic, 2008) , BPD would be marked by high HA. However, there has not been a longitudinal examination of this association until now. Furthermore, as for low HA, Cloninger hypothesized it to characterize anti-social PD (Cloninger & Svrakic, 2008), and it was found to be a risk factor for externalizing problems such as delinquent behavior and substance use (e.g. Hartman et al., 2013; Hiramura et al., 2010; Masse & Tremblay, 1997). It may well be that due to the overlap of externalizing symptoms and BPD symptoms such as impulsivity, uncontrollable anger and frequent interpersonal conflicts, low HA, instead of high HA, may be predictive of BPD symptoms.

The interaction effect also showed that high childhood HA was a protective factor especially in the light of MBI. A possible explanation may lie in the nature of our sample: A meta-analysis showed that while there is an overall negative association between child negative emotionality and supportive parenting, this effect is actually reversed in samples with a higher socio economic status (SES) (Paulussen-Hoogeboom et al., 2007). In this regard, child fearfulness has been shown to be associated with less maternal restrictiveness, anger

and disappointment and more synchronous maternal behavior during a mother-toddler interaction (Bryan & Dix, 2009). These effects were partially attributable to fearful children behaving more cooperative. Fearful children in our sample might therefore have elicited more supportive maternal behavior, increasing chances for a positive personality development in the long run. Mothers experiencing MBI might be more inclined to engage resources when they are met with a support-seeking, cooperative child.

At the same time, the exacerbating effects of lower HA and higher NS on the effect of MBI imply that children with an impulsive, exploratory and incautious temperament are more susceptible to the long-term consequences of MBI. These children might put themselves into potentially dangerous situations more often. This would increase the need for supportive structuring and appropriate limit-setting of a parent. In their study, Bryan and Dix (2009) found that mothers of more active children behaved less synchronous and more restrictive, while experiencing more anger and disappointment. Therefore, especially in the context of MBI, children who are less careful and misbehave more often might evoke more negative emotional reactions. Harsh maternal reactions might have an impact by further dysregulating the temperamentally predisposed offspring. The exacerbation of a cycle of negative mother-child interactional patterns could impede the development of interpersonal and self-regulatory abilities.

Contrary to our expectations, our analyses did not reveal a longitudinal moderating effect of NS on the development of BPD symptoms. We can only make tentative suggestions as to why childhood NS did not contribute to the odds of BPD symptom development specifically. This finding could be attributable to a low variance in the symptom count due to our community-based sample. The questionnaire data of the LPFS might have been better able to map the dimension of personality pathology. A replication of this study with a diverse high-risk sample might provide more insight into the circumstances under which NS and BPD might be longitudinally associated and may constitute part of a behavioral dysregulation pathway. Nevertheless, our results regarding general personality dysfunction show that it is important to investigate temperament jointly with environmental factors to specify their long-term role in personality development.

Research regarding sex differences in the response to childhood adversity and parent-child relationship problems is not conclusive yet. Our results indicated that in response to MBI, girls and boys were equally likely to develop overall personality pathology. In the context of BPD symptoms specifically, however, the established cross-over interaction indicated a differential susceptibility pattern. In our sample, girls seemed to benefit more from good maternal bonding but also showed more BPD symptoms in the context of elevated MBI. MBI did however not significantly predict BPD symptoms in boys. Our finding reflects that of Lewis et al. (2014) who showed that maternal hostility was a longitudinal predictor of depressive

symptoms only in girls. Our result could possibly indicate different pathways toward BPD symptom development for girls vs. boys. E.g., two large epidemiological studies showed that symptoms regarding behavioral dysregulation (impulsivity, physically self-destructive behavior), were more prevalent in boys than in girls (Vanwoerden et al., 2019; Zanarini et al., 2011), maybe indicating a stronger role of biological based dysregulation. In contrast, BPD symptoms marking interpersonal dysregulation (namely, fear of abandonment and unstable relationships) were more prevalent in girls. Taken together with our findings, this could indicate that an interpersonal pathway to BPD symptom development might be especially relevant for girls. However, BPD symptom counts were low in our community-based sample, and although boys and girls did not significantly differ with regards to their symptom count, variance was lower in boys. Therefore, a replication of this finding in a larger, higher-risk sample is needed.

Based on our results, indicated prevention targeting mother-child dyads who meet the criteria for the observed risk factors could be tentatively recommended as a promising avenue for future research and innovative clinical approaches. Obstetric unit employees could help to identify mothers showing signs of MBI and offer them referral to interventions focusing on the mother-child bond and improving sensitive maternal behavior. Likewise, pediatricians, educators and caregivers themselves may pay close attention to the well-being and behavior of children exhibiting signs of high NS and low HA, who seem to be particularly vulnerable to the consequences of MBI. As the psychosocial impact of PDs and especially BPD can be substantial and pervasive (Kaess et al., 2017; Skodol et al., 2002; Thompson et al., 2018; Winograd et al., 2008), they require early intervention before actual symptoms manifest and impair daily functioning, interpersonal relationships and academic success.

Limitations and future directions

The 14-year prospective design of our study offered many advantages over a cross-sectional or retrospective design. We can rule out memory bias as a mechanism of the associations between MBI and child personality pathology. It is also one of the first studies to investigate predictors of a Criterion A personality functioning measure. We were able to examine effects of early MBI longitudinally and jointly with childhood temperament, testing a diathesis-stress model of PD development. However, the following limitations should be considered. Due to the study location, our sample predominantly had a slightly elevated SES, and our sample size was relatively small. Therefore, generalizability of our results may be limited, and replication in larger study samples is needed. However, it is all the more remarkable that these associations were demonstrated in a relatively low-risk population. Larger samples would also allow for the analysis of more complex temperament profiles rather than the analysis of individual dimensions. Here, all possible combinations of high and low expressions of Cloninger's temperament traits could be compared in their longitudinal

prediction of different PD subtypes. Moreover, our study design did not allow for us to model the possible bi-directional influences between temperament and environment. Future studies could investigate the reciprocal influences between child characteristics and parental care (Fruzzetti et al., 2005). We also acknowledge that the current study could only focus on some of the possible pathways to the development of personality pathology, and does not reflect the full picture of potential etiological factors. There may also be confounding factors such as adverse events that influence both the quality of maternal bonding and child personality development and that could not be considered here. Finally, although we provide possible explanations for the protective effect of high childhood HA, this result requires replication.

Conclusion

This work contributes to the understanding of PD symptom development and highlights the importance of very early caregiving factors and child characteristics such as temperament and sex. In our study, MBI as early as two weeks after birth predicted personality pathology 14 years later, especially for children with an impulsive, incautious temperament. In the context of BPD but not that of general personality dysfunction, girls might be more susceptible to the effects of the mother-child relationship. Mother-child dyads who exhibit the established risk factors might benefit from indicated prevention targeting the mother-child bond. This should be considered an important goal for further developments and studies.

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Article 2. Adolescent Borderline Personality Traits and Dyadic Behavior Shape Mother-Adolescent Cortisol Synchrony

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Abstract

The hypothalamic-pituitary-adrenal axis, a physiological stress regulation system influenced strongly by social stimuli, produces the glucocorticoid cortisol. Associations between parent and child cortisol levels ("cortisol synchrony") are often reported and may mark dyadic regulation on a physiological level. Although dyadic behavior during interaction and adolescent borderline personality disorder (BPD) traits are linked with individual and dyadic regulation, little is known about how both factors influence parent-adolescent cortisol synchrony. We hypothesized that cortisol synchrony would differ depending on behavioral synchrony, adolescent BPD traits and their interactions. Multilevel state-trait modeling was implemented to investigate associations between concurrent mother-adolescent state cortisol and mother-adolescent average cortisol levels in a community sample of 76 German mother-adolescent dyads. Three saliva samples were collected across interaction paradigms. Behavioral synchrony was observed, and adolescent BPD traits were evaluated using clinical interviews. First, behavioral synchrony and absence of BPD traits were linked with positive associations between adolescent and maternal state cortisol (positive synchrony), BPD traits with negative associations (negative synchrony). When interaction effects were examined, results were more nuanced. In low-risk dyads (higher behavioral synchrony, no BPD traits) asynchrony was found. When risk (BPD traits) and resource (higher behavioral synchrony) were combined, synchrony was positive. Lastly, in high-risk dyads (lower behavioral synchrony, adolescent BPD traits), negative synchrony was observed. Average adolescent and maternal cortisol levels were consistently associated in dyads with higher risk. Thus, cortisol synchrony is shaped through the interplay of behavior and adolescent disorder. Further, dynamic as well as average cortisol associations may represent different processes.

Keywords: cortisol, synchrony, BPD, adolescence, mother-child interaction

Introduction

The hypothalamic-pituitary-adrenal (HPA) axis is an endogenous stress system under social regulation (Koss & Gunnar, 2018). Through a cascade of endocrine activity and the release of cortisol, the HPA axis supports the body in case of environmental demands and threats (Nicolson, 2008). Activation of the HPA axis is reliably provoked by social stressors such as negative judgement or conflict, and social support and high quality parenting reduce HPA dysregulation (Hibel et al., 2019; Koss & Gunnar, 2018; Laurent et al., 2020). Social processes have the potential to elicit arousal and dysregulation in individuals and to support individuals in managing those states (Ringwald et al., 2020). Individual physiological states and behaviors are co-constructed and synchronized in dyadic interactions, indicating that physiology and behavior in one affect physiology and behavior in the other (Timmons et al., 2015). Studies on behavioral regulatory processes in parents and children have emphasized their role in the development of children's self-regulation (Davis et al., 2017). Negative, maladaptive dyadic behavior is linked with child and adolescent mental disorder and poorer treatment outcomes (Im-Bolter et al., 2015; Miller-Slough et al., 2016). Recently, dyadic physiological regulation has gained attention, with scholars acknowledging that physiological regulation, too, needs to be conceptualized not as inherently individual but social (Feldman, 2012; Saxbe et al., 2020).

A disorder which is profoundly linked with deficits in interpersonal functioning and self-regulation (Chanen et al., 2017; Chanen & Kaess, 2012), severely impaired parent-child interactions (Franssens et al., 2021; Khoury et al., 2020) and HPA axis functioning (Drews et al., 2019) is adolescent Borderline Personality Disorder (BPD). BPD specifically has been characterized as a "disorder of impaired social regulation" across the life span (Hughes et al., 2012). Thus, while parent-child regulatory processes in physiology and dyadic behavior are of central interest in research on child and adolescent mental disorders in general, these processes may be particularly relevant and potentially altered in adolescents with BPD traits and their parents. However, studies with this very focus are lacking.

Dyadic conceptualization of regulatory processes: Parent-Child Cortisol Synchrony

A significant number of studies reports associations between parent and child cortisol levels (Hibel et al., 2019; Laurent et al., 2020; Merwin et al., 2018; Nofech-Mozes et al., 2020). However, existing studies present a range of terms and analytic methods, making it difficult to draw conclusions across multiple studies (DePasquale, 2020). To reduce variability and promote a common language in the field, we chose the frequently used term "cortisol synchrony" for dyadic cortisol associations. We define parent-child cortisol synchrony as a dynamic, within-dyad coordination of cortisol levels across time that is directly tied to an interpersonal process (DePasquale, 2020). Thus, cortisol synchrony will be used to describe

within-dyad, concurrent associations between mother and adolescent state cortisol. Significant synchrony therefore indicates that when a mother's cortisol response is higher/lower at one time point, adolescent's cortisol response is also higher/lower (at the same timepoint). At the same time, it is of interest whether these state associations are separate from overall dyadic similarity in cortisol levels (Laurent et al., 2020). In order to be able to examine both dynamic cortisol synchrony as well as associations between overall cortisol functioning, we implemented multilevel (MLM) mixed-effects modeling, also called state-trait modeling (Bolger & Laurenceau, 2013). MLM state-trait modeling allows for parsing within-dyad (WD) effects of concurrent and dynamic cortisol state associations and between-dyad (BD) effects of overall/average cortisol associations (DePasquale, 2020; Fuchs et al., 2021; Merwin et al., 2017). Thus, WD and BD effects as well as their links with risk and resource factors can be examined in more detail (Fuchs et al., 2021; Laurent et al., 2020; Lunkenheimer et al., 2021). In addition, we follow a 'statistical' approach detailing synchrony according to the direction of the observed associations, not attributing any valence to these types of synchrony per se (DePasquale, 2020). Thus, positive synchrony indicates that when a mother's cortisol is higher at one time point, adolescent's cortisol is also higher (at the same timepoint), and vice versa, without indicating whether this type of synchrony is adaptive or maladaptive. Cortisol synchrony can further be negative or not significant, suggesting asynchrony. Unfortunately, yet, little is known about different types of synchrony in different contexts or samples to be able to reliably determine which type of synchrony is adaptive or maladaptive, and more research is needed highlighting synchrony in diverse contexts and linking it with outcome measures (DePasquale, 2020; Fuchs et al., 2021). However, when synchrony is adaptive, it is suggested to serve the conservation of resources, and to support affiliative bonds and social processing, while maladaptive synchrony is thought to contribute to depletion of resources, to disrupt social affiliation, and increase the risk for psychopathology (Borelli et al., 2019; Feldman, 2012; Saxbe et al., 2020).

Parent-Adolescent Cortisol Synchrony and Dyadic Behavior

While the potential of the parent-child relationship in shaping stress and emotion regulation in early and middle childhood is widely recognized, less is known about adolescents and their interactions with parents (Byrd-Craven et al., 2020). During adolescence, a significant shift in social behavior emerges, where children strongly orient towards peers and the social group they belong to, and parent-child relationships change in content (e.g. more conflicts and negative affect (Main et al., 2016) and context (e.g. less time spent together (Collins & Laursen, 2004). Considering these changes, the lack of work examining parent-child synchrony in adolescence is a significant gap in literature (DePasquale, 2020). Whereas prior studies suggest that positive cortisol synchrony supports processes like parent-child

bonding, teaching and regulation in early and middle childhood, this may not be the case in adolescence (Atkinson et al., 2013; DePasquale, 2020; Feldman, 2012). As autonomy development is an important developmental task during adolescence, tight physiological coordination with parents may not be adaptive (Motsan et al., 2020). The few studies which have examined parent-adolescent cortisol in community (Lippold et al., 2020; Papp et al., 2009; Saxbe et al., 2014) and low-income samples (Byrd-Craven et al., 2020) reported positive associations in diurnal variations (Lippold et al., 2020; Papp et al., 2009) and over the course of laboratory visits (Byrd-Craven et al., 2020; Saxbe et al., 2014), suggesting that indeed there is cortisol synchrony beyond middle childhood.

Physiological and behavioral synchrony describe different levels of regulatory processes in a dyad. For a comprehensive understanding of dyadic regulation it is vital to examine both conjointly (Bell, 2020; DePasquale, 2020). Among the few studies integrating both physiology and behavior in their assessment of adolescents and their parents, Papp et al. (2009) found that higher negative affect in either child or mother was associated with higher positive synchrony in parent-adolescent diurnal cortisol slopes (Papp et al., 2009). Borelli et al. (2019) investigated the influence of maternal overcontrol and context on parent-child synchrony in 9–12-year-old children and reported that when at least one adverse factor (overcontrol or stress condition) was present, positive synchrony was observed. For the recovery condition, higher overcontrol was linked with negative synchrony, and the authors argued that they may have not captured a true recovery, but processing of the stress condition instead. These rather complex findings are mirrored in younger age groups, where a higher number of studies have yet produced inconsistent results. For example, positive cortisol synchrony has been found in sensitive mothers and their children (Atkinson et al., 2013; van Bakel & Riksen-Walraven, 2008), whereas negative cortisol synchrony has been observed in less sensitive mothers and their children (van Bakel & Riksen-Walraven, 2008) and in mothers and their disorganized toddlers (Nofech-Mozes et al., 2020). At the same time, positive cortisol synchrony has also been found in dyads with lower behavioral synchrony (Pratt et al., 2017) and in dyads with higher maternal punitive parenting (Hibel et al., 2009). Thus, whether or when certain forms of cortisol synchrony go hand in hand with behavioral synchrony is still unclear (DePasquale, 2020). More research drawing on clearly defined constructs, appropriate methodology and different age groups is needed to determine whether cortisol synchrony may differ depending on observed behavior.

Parent-Adolescent Cortisol Synchrony Moderated by Adolescent Borderline Personality Symptoms

Adolescence is not only a particularly interesting developmental phase for the study of parent-child regulatory processes, but also a turning point for developmental psychopathology

that is marked by heightened risk (Ullsperger & Nikolas, 2017). It thus represents an important period for the study of both social processes and mental disorder. BPD features can be reliably diagnosed in adolescence and implicate severely impaired interpersonal functioning and self-regulation (Chanen et al., 2017; Chanen & Kaess, 2012). Based on Social Baseline Theory and the assumption that regulation is an interpersonal process (Coan & Maresh, 2014), BPD has recently been described as the disorder of impaired social regulation (Hughes et al., 2012). Adolescents with BPD present with a pervasive pattern of instable relationships, self-image and affect (American Psychiatric Association, 2013), and BPD traits such as anger outbursts or impulsivity are primarily expressed in interpersonal contexts (Howard et al., 2021). BPD seems further to be associated with dysregulated HPA axis functioning in adulthood, specifically, with elevated baseline levels and blunted reactivity to social stressors (Drews et al., 2019). Thus, higher levels of adolescent BPD traits could influence parent-adolescent cortisol synchrony via a multitude of pathways such as dysregulated HPA-functioning and impaired dyadic behavior in the parent-adolescent dyad. Given the profound connection between BPD traits and regulatory impairment in interpersonal contexts (Drews et al., 2019; Howard et al., 2021; Hughes et al., 2012), BPD traits may alter parent-adolescent cortisol synchrony even above and beyond the presence of other mental disorders, preventing positive effects of adaptive dyadic regulation or enhancing negative effects of dysregulation. However, despite a potential influence of adolescent BPD traits on cortisol synchrony in adolescence studies are lacking. To the best of our knowledge, there is only one study which has highlighted a role of child mental disorder in cortisol synchrony, focusing on preschool children with autism spectrum disorder (Saxbe et al., 2017). Thus, there is much to learn about the nature of cortisol synchrony in adolescence and how it is shaped by adolescent mental disorder and borderline personality traits specifically.

Present study

Our study had several aims. First, our goal was to add to the literature by examining cortisol synchrony in mother-adolescent dyads. Following the call to ensure a fit between definition of synchrony and analytical approach (Bernard et al., 2017; Merwin et al., 2017; Suveg et al., 2019), we implemented MLM state-trait modeling (Bolger & Laurenceau, 2013) to determine presence and type of cortisol synchrony, a method matching our definition of synchrony as a WD process over the course of a dyadic interaction (DePasquale, 2020). Furthermore, MLM state-trait modeling allows for parsing WD and BD effects (DePasquale, 2020; Merwin et al., 2017), allowing us to investigate both concurrent, dynamic parent-adolescent cortisol synchrony and average cortisol associations across the interaction, which may represent different dyadic processes (Laurent et al., 2020). Based on prior findings we expected to find *positive cortisol synchrony* in our sample (Papp et al., 2009; Saxbe et al.,

2014), our analyses regarding average cortisol associations were exploratory. Our second goal was to examine whether dyadic behavior would moderate parent-adolescent synchrony. As synchrony is defined as an interpersonal process and in line with prior studies using conversational paradigms in older children, we measured cortisol synchrony during actual social interaction (Byrd-Craven et al., 2020; DePasquale, 2020; Papp et al., 2009). We observed systemic features of the adolescent-parent-relationship, i.e. behavioral synchrony, which characterizes the dyadic atmosphere rather than the behavior of one or both partners separately. As positive cortisol synchrony has been commonly found in prior work involving community samples (Papp et al., 2009; Saxbe et al., 2014), and studies examining moderating effects of behavior on cortisol synchrony remain inconsistent (DePasquale, 2020), we hypothesized to find *positive cortisol synchrony* in dyads with higher levels of behavioral synchrony, and *asynchrony* or even *negative synchrony* in those with lower levels of behavioral synchrony. Our third goal was to examine whether adolescent BPD traits would moderate parent-adolescent synchrony. Since there is first evidence of positive synchrony in adolescent community samples (Papp et al., 2009; Saxbe et al., 2014) and BPD has been described as a disorder of impaired social regulation specifically (Hughes et al., 2012), we hypothesized altered (i.e. *negative or absent) cortisol synchrony* in adolescents with a higher number of BPD traits and their mothers. Fourth, we examined whether adolescent BPD traits and behavioral synchrony would interact to jointly moderate cortisol synchrony. We assumed higher levels of behavioral synchrony to buffer effects of BPD traits such that when BPD traits were present but behavioral synchrony was higher, *positive cortisol synchrony* would be observed. Lastly, as further analyses were beyond the scope of this manuscript, we included additional exploratory research questions in the supplement a) testing whether presence of adolescent mental disorder would moderate cortisol synchrony/average cortisol associations, b) whether adolescent BPD traits remained a significant moderator of cortisol synchrony when presence of other mental disorders was controlled for and c) whether mental disorder and behavioral synchrony would interact to moderate cortisol synchrony.

Method

Participants

We examined a community sample of 76 adolescent-mother dyads who participated in a longitudinal study on temperament and the development of BPD traits (Fleck et al., 2021; Moehler et al., 2006). Adolescents (46% girls) were 14.0 (SD=0) years old, mothers on average 48.2 years old (SD=4.6). Seventy percent of mothers held a university degree, 20% had finished Intermediate Secondary School, and 10% held a University Entrance Diploma. Ninety-one percent of mothers reported to be in a relationship. Eighty-four percent of adolescents were in Grammar School, 15% in Intermediate Secondary School, and 1% visited

other school types. Out of 101 mother-child pairs who were examined at five time points starting two weeks after birth, 76 families participated in the last assessment (T6) which included physiological and behavioral data collection. The 76 mother-adolescent dyads did not differ significantly from dyads who dropped out with respect to maternal education ($\chi 2(2)=2.27$, $p=.321$) or maternal relationship status ($\chi 2(2)=3.54$, $p=.838$), infant birth weight ($t(99)=-0.74$, $p=.466$) or infant sex ($\chi 2(1)=0.09$, $p=.767$) at T1, however, mothers who continued to participate ($M=33.76$ years at T1) where older than mothers who dropped out ($M=31.96$ years at T1; $t(99)=-2.14$, $p<.05$).

Procedure

The study was approved by the Institutional Review Board at the Faculty of Medicine at the University of Heidelberg (S-553/2016). Mothers and adolescents provided informed consent. Initially, mothers were recruited via local obstetric units and offices and newspapers. Inclusion criteria were full term delivery, infant weight >2500g, APGAR scores >7 and good health of the baby during the first three postnatal doctoral exams. Exclusion criteria were inability to speak or understand the German language, acute maternal mental disorder, excessive smoking or alcohol consumption and the use of drugs or medication possibly risking fetal health. Mothers and adolescents were invited to a three-hour assessment, where clinical and socio-demographic interviews were conducted with adolescents while mothers filled in questionnaires in a separate room. Further, a parent-child interaction paradigm was administered, and cortisol samples were taken. Families were compensated 70 Euro for participation.

Measures

Cortisol sample collection

Three saliva samples were taken from mothers and adolescents each at the same time over the course of the visit. At the beginning of the visit, mother and adolescent participated in a short interview assessing sociodemographic information, which was followed by clinical interviews (adolescents) and the completion of questionnaires (mothers). After approximately two hours, mothers and adolescents completed a five-minute resting baseline where they sat quietly in separate rooms, and immediately after the first cortisol sample (baseline) was taken. Two subsequent samples were collected ten minutes after mother-adolescent dyads had engaged in a ten-minute positive (positive interaction sample) and a ten-minute negative (conflict discussion sample) interaction, respectively. On average, the positive interaction sample was taken 22.57 min (SD=0.57) and the conflict sample was taken 44.64 min (SD=0.97) after the baseline sample. The average assessment time of day was 4:13pm (SD 2:23h). Salivette (Sarstedt, Germany) sampling devices were used for saliva collection.

Families were instructed to refrain from drinking and eating for at least 60 minutes prior to the first sample. Saliva samples were stored uncentrifuged at −20 °C until assayed at Dresden University of Technology. After thawing, salivettes were centrifuged at 3,000 rpm for 5 min, which resulted in a clear supernatant of low viscosity. Salivary concentrations were measured using commercially available chemiluminescence immunoassay with high sensitivity (IBL International, Hamburg, Germany). The intra and interassay coefficients for cortisol were below 9%, respectively. Average raw cortisol levels were 2.53 for mothers (SD=1.62, min=0.48, max=11.25 nmol/l) and 2.53 for adolescents (SD=1.66, min=0.34, max=10.06 nmol/l). Screening for outliers, nine cortisol samples were identified as outliers (>=3SD from the respective time point mean). Out of these, two mothers showed outlier values on two sampling occasions, suggesting an abnormal cortisol pattern. The first mothers' cortisol levels with a maximum of 8.14 nmol/l at baseline still fell into the suggested reference range for the respective time of day and maternal age (Miller et al., 2016) and there was no indication of faulty data. The second mothers' cortisol levels with a maximum of 11.25 nmol/l at baseline were higher than the suggested reference range. However, excluding this case did not change the results and this dyad was thus kept in the analytic sample. Furthermore, to resolve skewness of raw cortisol levels, a natural log transformation was applied, and log-transformed values were used in analyses.

Behavioral Synchrony

Behavioral synchrony was observed and rated based on the Coding Interactive Behavior system (CIB, (Feldman, 1998)). Two videotaped, ten-minute dyadic interactions were coded; a positive interaction paradigm, where mothers and adolescents discussed and planned fun activities they would like to engage in together, and a conflict interaction paradigm, where mothers and adolescents discussed conflicts between them. Two main raters were trained and certified by the author of the measure and two additional raters were trained by them. Twenty-four dyads were rated by at least two raters with an inter-rater agreement 88% and Cohen's kappa=.78.

The CIB version for parent-child conversational paradigms covers 56 behavioral codes which receive ratings from 1 (low) to 5 (high). There are two dyadic behavior scales: Dyadic reciprocity (reciprocity, compatibility, and fluency, α=.88) and dyadic negativity (tension and constriction, α=.77). To determine the level of behavioral synchrony over the course of the visit, dyadic reciprocity scores during both interactions were summed up, dyadic negativity scores were subtracted, and the result averaged. Thus, behavioral synchrony was higher when dyadic reciprocity was higher and dyadic negativity was lower during both interactions.

Borderline Personality Disorder Traits

BPD traits according to DSM-IV criteria were assessed with the Childhood Interview for Borderline Personality Disorder (CI-BPD; Zanarini, 2003). For this study, the CI-BPD was translated into German language by a professional agency. The CI-BPD evaluates each of the nine DSM-criteria as either “absent” (0 points), “probably present” (1 point) or “definitely present” (2 points) during the past two years. Two psychologists were trained for reliability by the author of the measure. Further, 20 of the interviews where double coded. Inter-rater agreement per symptom ranged from 80% to 100%, with an overall agreement of 93%. A variable indicating the number of BPD criteria (traits) was calculated for each adolescent.

Analytic Plan

We implemented multilevel state-trait modeling (MLM), which accounts for the nested structure of dyadic data (Raudenbush & Bryk, 2002). Furthermore, MLM allows for simultaneous estimation of between-dyad (BD) and within-dyad (WD) effects, which offers several advantages to address current issues within the field of synchrony research. First, state-trait MLM allows to operationalize synchrony as a WD association between maternal and adolescent cortisol which is in line with our definition of cortisol synchrony as a dynamic, interpersonal process (DePasquale, 2020). Further, it allows us to focus not only on WD associations and BD associations in maternal and adolescent average cortisol levels, but also to highlight how these two processes may look differently (Khoury et al., 2020). Due to our focus on adolescent BPD traits we chose to model maternal cortisol predicting adolescent cortisol, a common approach used in prior work (e.g. Merwin et al., 2017; Suveg et al., 2019). On the BD level, maternal average (trait) cortisol was calculated by grand-mean centering (Bolger & Laurenceau, 2013). Consequently, when mothers had an average cortisol value of zero, their average cortisol over the course of the visit was equivalent to the sample average. On the WD level, in-the-moment associations were addressed by capturing whether mother and adolescent state cortisol responses coordinated at any given timepoint across the visit (Fuchs et al., 2021; Suveg et al., 2019). Maternal state cortisol was calculated by subtracting each mother’s average cortisol from her concurrent cortisol value at that specific timepoint. Maternal state cortisol levels thus represented each mother’s fluctuations around her own cortisol average and a state cortisol value of zero was equivalent to this mother’s average cortisol (Bolger & Laurenceau, 2013). A positive state cortisol value represented an increase in cortisol with respect to a mother’s average cortisol, and a negative state value cortisol indexed a decrease with respect to average cortisol. State and average cortisol predictors were set to predict ‘total’ adolescent cortisol levels. This allowed for simultaneous estimation of WD and BD effects in one model based on the assumption that outcome (adolescent

cortisol) variance is composed of both interindividual and intraindividual variance (Bolger & Laurenceau, 2013).

The unconditional means model revealed an Intraclass Correlation of 89.8% and random effects were found to be significant, confirming the appropriateness of MLM. First, we examined relationships between maternal and adolescent cortisol and sampling time. Cortisol levels significantly declined from baseline to positive interaction (Mothers: β=-.13, 95% CI[-0.172; -0.096]; Adolescents: β=-.21, 95% CI[-0.275; -0.146]), and from positive interaction to conflict discussion (Mothers: β=-.14, 95% CI[-0.175; -0.100]; Adolescents: β=-.19, 95% CI[-0.250; -0.121]). To preserve parsimony and to account for cortisol changes as a function of time we thus included a continuous variable indexing time passed since the baseline sample ("time since baseline") in all models instead of a categorical timepoint variable (Bolger & Laurenceau, 2013). We further included the planned covariate "time of day" in every analytic model to account for the influence diurnal HPA-axis activity (Bolger & Laurenceau, 2013; Nicolson, 2008). Further, to examine cortisol synchrony and associations between maternal and adolescent average cortisol levels, we included maternal average and state cortisol levels predicting adolescent cortisol. Depending on the model in question, we further added a continuous variable determining the number of BPD traits for each adolescent ("BPD traits") and/or a continuous variable depicting the level of dyadic behavioral synchrony across both interactions ("behavioral synchrony") and respective interactions (see Tables 2-4). "Number of BPD traits" and "Time of Day" were centered so that values of zero represented the average sample level. 76 dyads and 228 observations were included in analyses. Random intercept and random slope models were estimated using the lme4 package in R. Effect sizes were calculated using Cohen's f^2 (small 0.02, medium, 0.15, large 0.35) in Stata.

Results

Log transformed average cortisol levels were *M*=0.77 (*SD*=0.54) for mothers and *M*=0.72 (*SD*=0.62) for adolescents, and state cortisol levels were *M*=0.00 (*SD*=0.15) for mothers and *M*=0.00 (*SD*=0.23) for adolescents. Mean behavioral synchrony behavior was *M*=0.78 (*SD*=0.78), with a minimum value of -1.52 and a maximum value of 1.96. For 16% (n=12) of the adolescents, interviewers endorsed one BPD trait. Seven percent of adolescents (n=5) had two traits, and again seven percent had three or more traits (n=5). Thus, 71% (n=54) had no BPD traits, and 29% (n=22) had at least one BPD trait.

Correlations of study variables are presented in Supplement Table S1. There were no associations between maternal/adolescent cortisol levels for any demographic variable except maternal age, which was significantly associated with maternal baseline cortisol levels (r=.25, p<.05). No significant links were found for education, relationship status, child sex, smoking, alcohol consumption, hormonal contraception, days since first day of last period for female

participants, recent sickness, physical activity or body mass index. None of the women were pregnant. There was intraindividual stability in cortisol: Maternal baseline cortisol was significantly associated with cortisol after the positive interaction (r=.93, p<.01) and after the conflict discussion (r=.92, p<.01). Adolescent baseline cortisol was significantly associated with cortisol after the positive interaction (r=.92, p<.01) and after the conflict discussion (r=.83, p<.01). Further, maternal and adolescent cortisol levels were significantly correlated at baseline (r=.39, p<.01), positive interaction (r=.36, p<.01), and conflict discussion (r=.35, p<.01).

Multilevel State Trait Modeling

Baseline Model: Are Maternal and Adolescent Cortisol Levels Associated?

The baseline model included time since baseline, time of day, maternal state and average cortisol as predictors. Adolescent cortisol was significantly predicted by time since baseline (β=-0.01, 95% CI [-0.098, -0.006]) and time of day (β=-0.13, 95% CI [-0.190, -0.074]). We did not find cortisol synchrony in the total sample of 76 mothers and adolescents, as maternal state cortisol did not significantly predict adolescent state cortisol (β= 0.16, 95% CI [-0.149, 0.472]). Further, maternal average cortisol did not predict adolescent cortisol. There was, however, considerable intercept and slope variability between dyads (random intercept β=0.24, 95% CI [0.422, 0.587]; random slope β=0.45, 95% CI [0.410, 0.946]). Thus, there were substantial between-person differences in the association between average cortisol for mothers and adolescents, and substantial between-person differences in the within-person association between maternal and adolescent state cortisol, a variability which could potentially be explained by moderators (Laurent et al., 2020).

Are Cortisol Synchrony and Average Cortisol Associations Moderated by Behavioral Synchrony?

State cortisol. Behavioral synchrony significantly moderated cortisol synchrony (β=0.31, 95% CI [0.02, 0.60], see Supplement Table S2) such that when behavioral synchrony was *higher* (>.69 centered behavioral synchrony), maternal state cortisol positively predicted adolescent state cortisol. More precisely, when mothers showed a decrease in cortisol with respect to average at any given timepoint, adolescents showed a decrease with respect to average at the same timepoint, and vice versa.

Average cortisol. Maternal average cortisol positively predicted adolescent average cortisol (β=-0.29, 95% CI [-0.58, -0.003], see Supplement Table S2) only when behavioral synchrony was *lower* (< -.21 centered behavioral synchrony). Thus, higher maternal cortisol across the interaction was linked with higher adolescent cortisol across the interaction, and

lower maternal cortisol across the interaction was linked with lower adolescent cortisol for dyads lower in behavioral synchrony.

Are Cortisol Synchrony and Average Cortisol Associations Moderated by Adolescent BPD Traits?

State Cortisol. Adolescent BPD traits significantly moderated cortisol synchrony (β=-0.35, 95% CI [-0.53, -0.17], see Supplement Table S3). Simple slope analysis revealed that maternal state cortisol significantly predicted adolescent state cortisol when adolescents either had no BPD traits (*β*=.34; p<.05) or at least three BPD traits (Three traits: *β*=-.68; p<.05). Synchrony direction differed depending on BPD traits: When adolescents had no BPD traits, synchrony was *positive*. Thus, decreases in maternal cortisol with respect to average were linked with decreases in adolescent cortisol at any given timepoint and increases in maternal cortisol were linked with increases in adolescent cortisol. For adolescents with three or more BPD traits, synchrony was *negative.* When mothers increased/ decreased their cortisol levels at any given timepoint, adolescents showed the opposite pattern.

Average Cortisol. There were no significant effects for maternal average cortisol predicting adolescent average cortisol (β=0.21, 95% CI [-0.03, 0.45], see Supplement Table S3).

Does Behavioral Synchrony Modulate the Effects of Adolescent BPD Traits on Cortisol Synchrony and Average Cortisol Associations?

BPD Traits and State Cortisol. Behavioral synchrony modulated the way BPD traits shaped cortisol synchrony (Table 1). Specifically, dyadic behavior made a difference when BPD traits were present: When behavioral synchrony behavior was *lower* (-1SD) and adolescents had at least two BPD traits, maternal state cortisol negatively predicted adolescent state cortisol (*negative synchrony*). However, when behavioral synchrony was *higher* (+1SD) and adolescents had at least one BPD trait, maternal state cortisol positively predicted adolescent state cortisol (*positive synchrony*). When adolescents had no BPD traits, and independent from the level of behavioral synchrony, asynchrony in cortisol was observed (Figure 1). There was, however, trend-level positive synchrony in adolescents without BPD traits and their mothers when the dyad had average to higher behavioral synchrony (*p*<.10).

Table 1

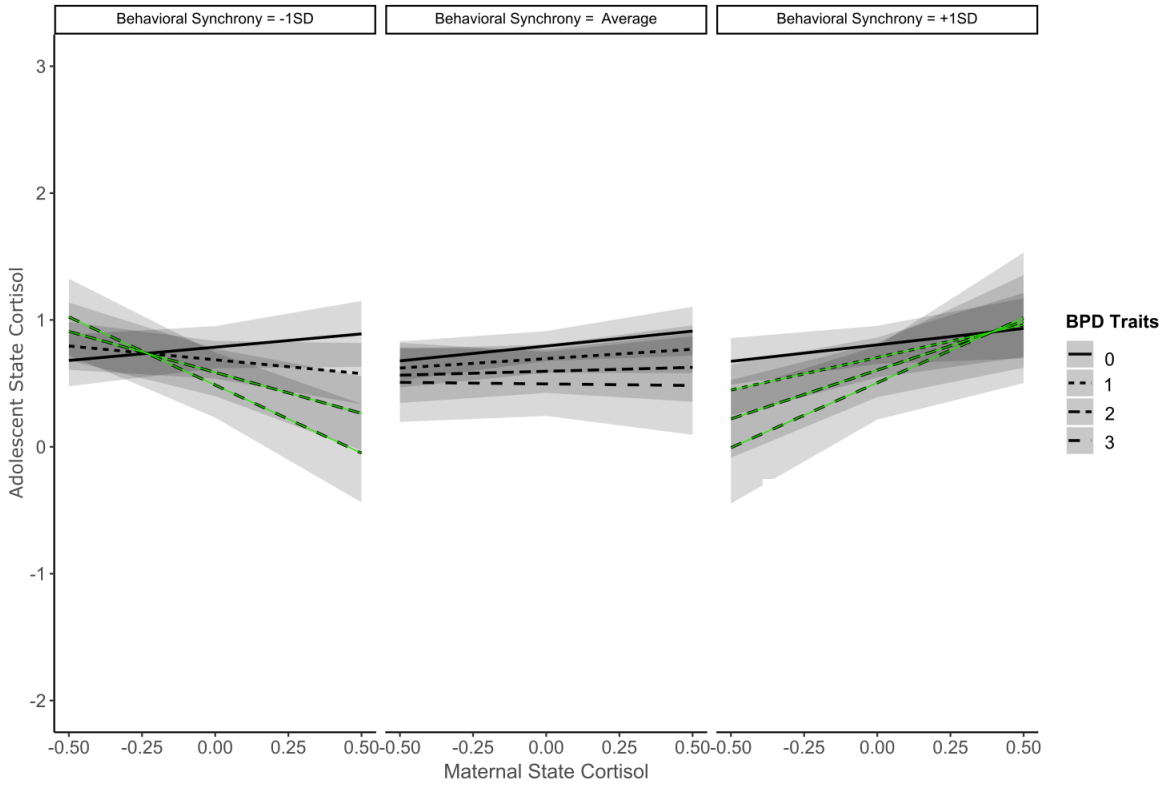
Behavioral Synchrony Modulates the Effects of Adolescent Borderline Personality Traits on Cortisol Synchrony.

Parameter	Estimate (SE)	95% CI	Cohen's f^2
Fixed effects			
(Intercept)	**0.92 (0.06)**	**[0.81, 1.03]**	
Time since Baseline [1]	**-0.01 (0.001)**	**[-0.01, -0.006]**	**0.464**
Time of Day [2]	**-0.12 (0.03)**	**[-0.18, -0.07]**	**0.001**
Behavioral Synchrony [2]	0.01 (0.07)	[-0.13, 0.15]	0.000
BPD traits [2]	**-0.10 (0.05)**	**[-0.20, -0.003]**	**0.004**
State CT [1]	0.19 (0.13)	[-0.06, 0.44]	0.015
State CT [1] * Behavioral Synchrony [2]	**0.28 (0.11)**	**[0.05, 0.51]**	**0.038**
State CT [1] * BPD traits [2]	-0.09 (0.09)	[-0.26, 0.09]	0.005
State CT [1] * BPD traits [2] * Behavioral Synchrony [2]	**0.44 (0.09)**	**[0.25, 0.63]**	**0.148**
Average CT [2]	**0.25 (0.12)**	**[0.02, 0.49]**	**0.019**
Average CT [2] * Behavioral Synchrony [2]	**-0.41 (0.14)**	**[-0.69, -0.14]**	**0.008**
Average CT [2] * BPD traits [2]	**0.35 (0.11)**	**[0.12, 0.57]**	**0.014**
Average CT [2] * BPD traits [2] * Behavioral Synchrony [2]	**-0.51 (0.21)**	**[-0.93, -0.10]**	**0.009**
Random effects			
Intercept	**0.20 (0.45)**	**[0.38, 0.53]**	
State Cortisol Slope	**0.07 (0.26)**	**[0.06, 0.56]**	

Note. Model fit: $\chi^2(12)= 294.27$. 228 Observations. Marginal R^2 =0.486. Maternal cortisol predicting adolescent cortisol. State CT=Maternal cortisol reactivity, Average CT=Maternal average cortisol. BPD traits=Number of Borderline Personality Traits. Unstandardized estimates are presented.[1]=Level 1 predictor, [2]=Level 2 predictor. Significant parameters in bold.

Figure 1.

Adolescent Borderline Personality Traits and Behavioral Synchrony Moderate Cortisol Synchrony.

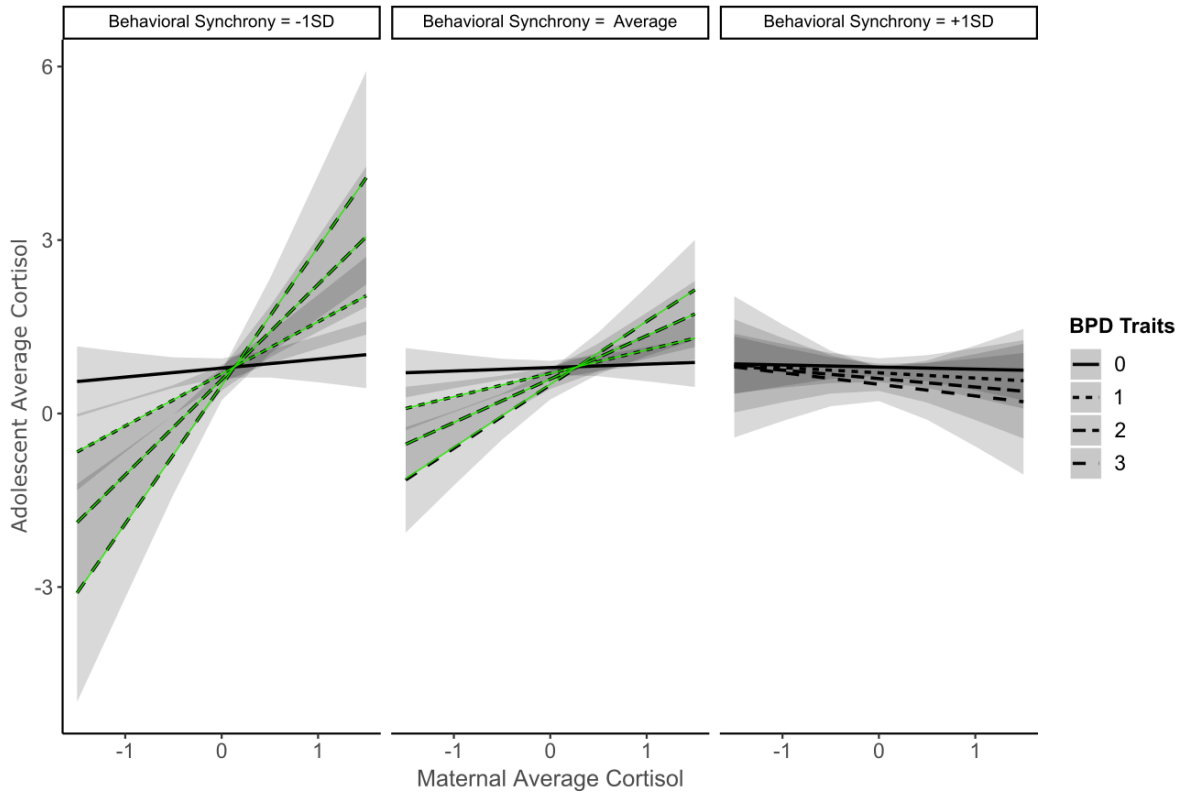


Note. Green lines: *p*<.05; black lines: *p*>.05. +/-1SD=Above/below one standard deviation. Negative cortisol synchrony when behavioral synchrony was lower and adolescents had at least two BPD traits. Positive cortisol synchrony when behavioral synchrony was higher and adolescents had at least one BPD trait.

BPD Traits and Average Cortisol. Behavioral synchrony further shaped how maternal cortisol and BPD traits predicted adolescent average cortisol (Table 1; Figure 2). When behavioral synchrony was higher (+1SD), maternal cortisol did not predict adolescent cortisol irrespective of adolescent BPD traits. When behavioral synchrony was lower (-1SD) or average level, however, results changed depending on BPD traits: For adolescents with at least one BPD trait, maternal average cortisol positively predicted adolescent average cortisol. There was no such effect when adolescents had no BPD traits. Maternal average cortisol positively predicted adolescent average cortisol when behavioral synchrony was lower. In addition, for adolescents with at least one BPD trait, maternal average cortisol significantly and positively predicted adolescent average cortisol. Lastly, main effects showed that the higher the number of BPD traits, the lower adolescent average cortisol, and the higher maternal average cortisol, the higher adolescent cortisol.

Figure 2.

Adolescent Borderline Personality Traits and Behavioral Synchrony Moderate the Link between Maternal Average CT and Adolescent Average CT.



Note. Green lines: $p<.05$; black lines: $p>.05$. +/-1SD=Above/below one standard deviation. Positive cortisol synchrony when behavioral synchrony was average or lower and adolescents had at least one BPD trait.

Results Summary

State Cortisol

When behavioral synchrony and BPD traits were examined individually, both factors were meaningful moderators of mother-to-adolescent cortisol synchrony. *Positive* cortisol synchrony was found in dyads with higher behavioral synchrony and in dyads where adolescents did not have any BPD traits. *Asynchrony* was observed in dyads lower in behavioral synchrony and in dyads where adolescents reported one or two BPD traits. Importantly, when adolescents reported at least three BPD traits, *negative* synchrony was found.

When combined in one model, behavioral synchrony and BPD traits jointly shaped cortisol synchrony and results were more nuanced. When adolescents reported more than one BPD trait, but behavioral synchrony was higher, cortisol synchrony was *positive*. *Asynchrony* in cortisol was found regardless of BPD traits when behavioral synchrony was

average. Finally, *negative* synchrony was observed when behavioral synchrony was lower and adolescents reported at least one BPD trait.

Average Cortisol

Across all models, maternal average cortisol positively predicted adolescent average cortisol under conditions of risk only, i.e., lower behavioral synchrony and lower behavioral synchrony combined with at least one adolescent BPD trait. Thus, when behavioral synchrony was higher, across all models, average cortisol levels were not significantly linked. Furthermore, even if behavioral synchrony was lower, when adolescents did not report any BPD traits, maternal and adolescent average cortisol were not significantly associated.

Discussion

Our study aimed to shed light on associations between concurrent, dynamic "state" and overall, "average" cortisol levels in adolescents and their mothers, which are theorized to indicate interpersonal physiological regulation. MLM state-trait modeling (Bolger & Laurenceau, 2013) allowed us to parse these WD and BD effects. In addition, we examined how observed behavioral synchrony as a systemic resource and adolescent BPD traits as risk factor separately and conjointly shaped mother-adolescent cortisol synchrony. Behavioral synchrony and BPD traits were relevant factors shaping cortisol synchrony, and our results mirror prior findings indicating that patterns of cortisol synchrony change depending on both risk and resources within a dyad (Atkinson et al., 2013; Hibel et al., 2019; Laurent et al., 2020; Merwin et al., 2018; Nofech-Mozes et al., 2020; Saxbe et al., 2017). However, we observed differences in cortisol synchrony patterns depending on whether behavior and BPD traits were examined separately or conjointly and whether their interactions were probed.

When behavioral synchrony and BPD traits were examined separately, in line with our hypotheses, positive cortisol synchrony was linked with higher behavioral synchrony, whereas negative cortisol synchrony was linked with BPD traits in adolescents. These results are in line with studies suggesting that positive cortisol synchrony occurs in context of adaptive dyadic interaction, could be a marker of healthy parent-child co-regulatory processes and may support them on a physiological level (Atkinson et al., 2013; van Bakel & Riksen-Walraven, 2008).

In line with our hypothesis, a higher number of BPD traits in adolescents was consistently linked with negative synchrony. BPD as a "disorder of social regulation" (Hughes et al., 2012) is characterized by severe difficulties in interpersonal relationships including the parent-child relationship, and behavioral and physiological regulatory impairments such as elevated baseline cortisol and blunted cortisol reactivity to social stressors (Chanen et al., 2017; Chanen & Kaess, 2012; Drews et al., 2019). Social impairments that are also observed in adolescent BPD are highly relevant factors for coregulatory processes in a dyad (Fuchs et al.,

2021; Lunkenheimer et al., 2021; Saxbe et al., 2017). In addition, a recent study examining infant attachment as a moderator of mother-child cortisol synchrony found negative synchrony in dyads with disorganized children, with mothers showing decreases and infants showing increases in cortisol over time (Nofech-Mozes et al., 2020). Insecure or disorganized attachment has been hypothesized to be a major predisposing factor for BPD, and a recent study reported that young adults with BPD had a greater likelihood to exhibit disorganized interactions with their mothers than adults with other or no diagnoses (Khoury et al., 2020). Deficits in attachment quality may thus be one important factor behind the moderating effect of BPD traits on cortisol synchrony.

For adolescents with BPD traits, adaptive co-regulatory processes in behavior and physiology during social interaction could function as important stabilizers supporting adolescent self-regulatory systems. In our community sample, mothers and adolescents showed a constant decrease in cortisol on average. This decrease was especially pronounced in adolescents without BPD traits but was also observed in mothers and, albeit to a lesser extent, in adolescents with BPD traits. A decrease in maternal cortisol levels, on average, thus seemed to predict decreasing cortisol levels in adolescents without BPD traits and increasing cortisol levels in adolescents with BPD traits. This may suggest that adolescents with BPD traits could not adequately benefit from adaptive maternal physiological responses. However, in order to determine whether negative synchrony in adolescents with BPD traits and their mothers is in fact maladaptive, further studies linking cortisol synchrony and adolescent outcome are indicated. In addition, it remains unclear whether these alterations in physiological synchrony are a consequence of adolescent BPD traits or may contribute to their development. Longitudinal studies on BPD pathogenesis in at-risk populations are needed to examine the role of co-regulatory processes over time.

Importantly however, while results based on separate models for behavioral synchrony and BPD indicated cortisol synchrony to be positive in context of higher behavioral synchrony, and absent or negative in context of lower behavioral synchrony and BPD traits, results were somewhat more nuanced when behavior and BPD traits were examined in one model and interaction effects were probed. In dyads with the lowest risk (higher behavioral synchrony and absence of BPD traits) we did not observe significant cortisol synchrony. In dyads where we observed higher positive synchrony as a resource, but adolescents reported at least one BPD trait, positive cortisol synchrony was found. Lastly, in dyads with two combined risk factors (lower behavioral synchrony and adolescent BPD traits), negative synchrony was observed. Interestingly, our finding of asynchrony in low-risk dyads (higher behavioral synchrony and absence of BPD traits) during potentially stressful situations could make a case for the notion that in adolescence, synchrony may not always be adaptive or developmentally normative (Motsan et al., 2020). Similarly, when the risk factor BPD traits was paired with the

resource higher behavioral synchrony, positive cortisol synchrony was found, suggesting that in these dyads, a decrease in maternal cortisol may indeed be linked with a decrease in adolescent cortisol. Hence both risk and resource factors are important to consider and jointly shape presence and/or form of cortisol synchrony.

Cortisol synchrony seems to depend on dyadic behavior especially when adolescents report BPD traits. Higher levels of behavioral synchrony are characterized by mutual adaption and low tension (Feldman, 1998). Behavioral synchrony may act as a dyadic buffer, allowing for adaptive co-regulatory processes and balancing out some of the regulatory and social impairments of BPD traits. In this case, positive cortisol synchrony may be an adaptive process. At the same time, a consistent finding throughout all models showed negative cortisol synchrony in dyads where adolescent BPD traits were combined with lower levels of behavioral synchrony. In these cases, dyadic behavior may even have exacerbated regulatory difficulties on a physiological level. Thus, negative cortisol synchrony may indicate dysfunctional dyadic regulatory processes. However, future longitudinal studies will have to examine whether negative cortisol synchrony is in fact associated with maladaptive child outcome such as emotion dysregulation, negative affect during interaction or more BPD traits.

Lastly, we were able to disentangle patterns of concurrent, dynamic parent-adolescent synchrony and average cortisol associations. While occurrence and form of state cortisol associations changed depending on which risk and resources where at play, associations between average cortisol in mothers and adolescents provided a consistent picture across all models. Maternal average cortisol was positively associated with adolescent average cortisol under conditions of higher risk (lower behavioral synchrony, adolescent BPD traits), and there was no significant association in dyads with higher positive dyadic behavior or when adolescents did not report any BPD traits. These consistent findings may suggest that mother-adolescent linkage in average cortisol represents an indicator of risk.

Limitations

Despite its strengths, our study is not without limitations. Due to only three cortisol measurements, we were limited in our analytic approach and were unable to investigate nonlinear associations between mother and adolescent cortisol reactivity. In addition, while the study was powered to find significant effects of state cortisol in three-way interactions, this was not the case for average cortisol. Futures studies would benefit from a bigger sample size. Furthermore, despite all its advantages, concurrent state-trait MLM does not allow for examination of mother-to-adolescent or adolescent-to-mother directionality. In addition, our study procedure was designed to elicit stress responses following a mother-adolescent conflict discussion. However, as reported in other studies (e.g. Byrd-Craven et al., 2020), participants on average showed a steady decline in cortisol levels, suggesting participants habituated over

time. While we were still able to examine mother-adolescent cortisol synchrony, our findings seem to suggest that conflict discussions may not be an adequate context to elicit cortisol stress responses in parents and adolescents. Further, as this was a longitudinal study focusing on mothers, we did not include fathers in our assessments. However, the one study focusing on parent-child cortisol synchrony in context of child disorder reported differences in cortisol synchrony in mother- and father-child dyads (Saxbe et al., 2017). Moving forward, it will be important to investigate how synchrony differs in mother- vs. father-adolescent dyads (Fuchs et al., 2021; Merwin et al., 2017; Saxbe et al., 2017). We further examined a highly educated, low risk community sample of German mother-adolescent dyads. As recent findings have highlighted cultural differences in cortisol reactivity (Miller & Kirschbaum, 2019), generalizability of our results may be limited to samples resembling ours in terms of socioeconomic background and culture.

Conclusion

Our findings suggest that it may not suffice to focus on one single risk factor or resource of the dyad alone, but that cortisol synchrony is shaped through risks and resources and their interplay. Future studies should integrate both mental disorder, BPD traits specifically, and dyadic behavior as moderators of synchrony and replicate how these factors shape cortisol synchrony distinctly and conjointly. Further, while we were able to show differences in cortisol synchrony depending on dyadic behavior and BPD traits, further research will have to elucidate how these differences indeed relate to child outcome. The question arises whether positive synchrony, which we observed in dyads characterized by higher behavioral synchrony and BPD traits, is adaptive in terms of successful co-regulation and adolescent healthy development. Similarly, it will be important to highlight whether negative cortisol synchrony in context of adolescent BPD is maladaptive and whether attachment quality may be of relevance in this context. The examination of both behavioral and physiological synchrony in development and persistence of BPD traits could be a fruitful future direction of research.

Supplement Material

Assessment of Other Mental Disorders

The sixth version of the Mini International Neuropsychiatric Interview for Children and Adolescents (MINI-KID; Sheehan et al., 2010) was administered to adolescents. The MINI-KID is a structured diagnostic interview assessing mental disorders according to DSM IV in children and adolescents ages six to 17. It examines the 30 most common and clinically relevant disorders in pediatric mental health (Sheehan et al., 1998) and has been found to generate reliable and valid psychiatric diagnoses (Sheehan et al., 2010). For analysis, dichotomous factor “mental disorder” was created, indicating presence or absence of any mental disorder according to the MINI-KID. In total, 27.6% (n=21) of adolescents were evaluated to have a current mental disorder. Out of these, 19.7% percent of adolescents were diagnosed with any phobic or anxiety disorder, 9.2% with any behavioral disorder and 2.6% with any mood disorder.

Additional Research Questions

Research Question 1:

Are cortisol synchrony and average cortisol associations modulated by presence or absence of adolescent mental disorders?

Results. State cortisol: Presence or absence of adolescent mental disorder significantly moderated cortisol synchrony (Table S4) such that there was significant positive cortisol synchrony only in mothers and adolescents who did not have a disorder (β=.33; p<.05, Figure S1a). Average cortisol: In contrast, maternal average cortisol significantly predicted adolescent average cortisol only in adolescents with a mental disorder and their mothers (β=.37; p<.05; Table S4). In this group, higher maternal cortisol across all assessments was associated with higher adolescent cortisol across all assessments, and lower maternal cortisol was linked with lower adolescent cortisol across all assessments (Figure S1b). Further, there was a main effect of adolescent mental disorder such that if a disorder was present, adolescent cortisol was lower.

Research Question 2:

Do adolescent BPD traits remain a significant moderator of the association between adolescent and maternal cortisol above and beyond the influence of other relevant mental disorders?

Results. To compare the effects of BPD traits and other mental disorders on cortisol synchrony we included both main effects and their interactions with average and state cortisol in one model. The moderating effect of BPD traits (β=-.30; p<.01) and the main effect of mental disorder remained significant (β=-.31; p<.05). Again, we found positive cortisol synchrony in adolescents with no BPD traits and their mothers and negative cortisol synchrony in

adolescents with at least three BPD traits and their mothers. Mental disorder remained negatively linked with adolescent average cortisol.

Research Question 3:

Does behavioral synchrony modulate the effects of presence or absence of adolescent mental disorders on cortisol synchrony and average cortisol associations?

Results. Mental Disorder and State Cortisol: Behavioral synchrony modulated the way presence or absence of mental disorder shaped cortisol synchrony (see Table S5, Figure S2). When behavioral synchrony was higher (+1SD) but adolescents had a disorder, *positive synchrony* was observed. When behavioral synchrony was higher and adolescents did not have a disorder, maternal and adolescent state cortisol were not significantly correlated (*asynchrony*). When behavioral synchrony was lower (-1SD) and adolescents had a disorder, *negative synchrony* was observed, whereas when behavioral synchrony was lower and adolescents did not have a disorder, *positive synchrony* was found. Mental Disorder and Average Cortisol: Mirroring findings reported in the prior mental disorder model, maternal average cortisol positively predicted adolescent average cortisol only when adolescents had a disorder (Figure S3). However, when behavioral synchrony was higher and adolescents had a disorder, there was asynchrony. Further, a positive main effect of maternal state cortisol emerged, as well as a main effect of disorder, suggesting that adolescents with mental disorder, on average, had significantly lower levels of cortisol.

Results Summary

State Cortisol

Mental disorder was a meaningful moderator of mother-to-adolescent cortisol synchrony. When mental disorder was examined without including behavior in the model, positive cortisol synchrony was observed in mothers and adolescents who did not have a disorder. However, after including behavioral synchrony as an additional moderator, positive cortisol synchrony was observed in dyads who showed higher behavioral synchrony and whose adolescents had a disorder. Asynchrony was found in the low-risk group of dyads with higher behavioral synchrony and adolescents who did not have a disorder, while negative cortisol synchrony was observed in the high-risk group of dyads with lower behavioral synchrony and adolescents who had a disorder.

Interestingly, any moderating effect of mental disorder did not remain significant when BPD traits were added to the model while the significant effect of BPD traits remained.

Negative cortisol synchrony in adolescents with at least three BPD traits and their mothers remained significant when presence of other mental disorders was controlled for.

Average Cortisol

Maternal average cortisol predicted adolescent average cortisol consistently and positively when adolescents had a mental disorder. However, when including behavioral synchrony as a moderator, this association remained significant only when dyads also showed lower behavioral synchrony. Thus, maternal and adolescent average cortisol were not significantly linked when either adolescents did not have a disorder, or adolescent had a disorder but behavioral synchrony was higher in the dyad.

Tables

Table S1

Correlations Between Study Variables.

Variable	1)	2)	3)	4)	5)	6)	7)	8)	9)	10)
1) Average CT mother[1]										
2) Average CT adolescent[1]	0.37**									
3) State CT mother[1]	-0.01	0.00								
4) State CT adolescent[1]	0.01	0.00	0.59**							
5) Number of BPD traits	-0.19	-0.43**	-0.01	-0.01						
6) Mental Disorder (present/absent)[2]	-0.13	-0.37**	-0.01	0.00	0.52**					
7) Behavioral Synchrony	-0.04	0.04	0.00	0.00	-0.10	-0.20				
8) Time of Day	-0.56**	-0.57**	0.01	0.00	0.40**	0.30**	0.07			
9) Time since Baseline	-0.02	-0.05	-0.74**	-0.68**	0.04	0.03	-0.01	0.02		
10) Age Mother	0.19	0.07	0.00	0.01	-0.08	-0.20	0.13*	-0.03	0.01	
11) Sex Child (male)	-0.06	0.17	0.00	0.00	0.01	-0.08	-0.09	-0.15	0.00	0.03

Note. CT=cortisol; [1]logarithmized cortisol values; [2]point-biseral correlation. *p<.05, **p<.01.

Table S2
Cortisol Synchrony Moderated by Behavioral Synchrony.

Parameter	Estimate (SE)	95% CI	Cohen's f^2
Fixed effects			
(Intercept)	**0.90 (0.06)**	**[0.78, 1.02]**	
Time since Baseline [1]	**-0.01 (0.001)**	**[-0.01, -0.01]**	**0.515**
Time of Day [2]	**-0.13 (0.03)**	**[-0.18, -0.07]**	**-0.002**
Behavioral Synchrony [2]	0.05 (0.07)	[-0.09, 0.20]	0.000
State CT [1]	0.13 (0.15)	[-0.17, 0.44]	0.006
State CT [1] * Behavioral Synchrony [2]	**0.31 (0.15)**	**[0.02, 0.60]**	**0.023**
Average CT [2]	0.20 (0.12)	[-0.05, 0.45]	0.004
Average CT [2] * Behavioral Synchrony [2]	**-0.29 (0.15)**	**[-0.58, -0.003]**	**-0.001**
Random effects			
Intercept $_{\mu 0j}$	**0.23 (0.48)**	**[0.41, 0.57]**	
State Cortisol Slope	**0.39 (0.63)**	**[0.36, 0.89]**	

Note. Model fit: $\chi^2(7)=183.34$, $p<0.01$. Maternal cortisol predicting adolescent cortisol. State CT=Maternal cortisol reactivity, Average CT=Maternal average cortisol. Unstandardized estimates are presented. [1]=Level 1 predictor, [2]=Level 2 predictor. Significant parameters in bold.

Table S3
Cortisol Synchrony Moderated by Adolescent Borderline Personality Traits

Parameter	Estimate (SE)	95% CI	Cohen's f^2
Fixed effects			
(Intercept)	**0.91 (0.06)**	**[0.79, 1.03]**	
Time since Baseline [1]	**-0.01 (0.00)**	**[-0.01, -0.01]**	**0.516**
Time of Day [2]	**-0.14 (0.03)**	**[-0.20, -0.08]**	**0.007**
BPD Traits [2]	-0.08 (0.05)	[-0.18, 0.02]	0.001
State CT [1]	0.14 (0.15)	[-0.15, 0.42]	0.006
State CT [1] * BPD traits [2]	**-0.35 (0.09)**	**[-0.53, -0.17]**	**0.083**
Average CT [2]	0.11 (0.13)	[-0.14, 0.37]	0.001
Average CT [2] * BPD traits [2]	0.21 (0.12)	[-0.03, 0.45]	0.001
Random effects			
Intercept	**0.23 (0.48)**	**[0.41, 0.57]**	
State Cortisol Slope	**0.28 (0.53)**	**[0.24, 0.79]**	

Note. Model fit: $\chi^2(7)=207.44$, $p<0.01$. Maternal cortisol predicting adolescent cortisol. State CT=Maternal cortisol reactivity, Average CT=Maternal average cortisol. Unstandardized estimates are presented. BPD traits=Number of Borderline Personality Traits. [1]=Level 1 predictor, [2]=Level 2 predictor. Significant parameters in bold.

Table S4
Cortisol Synchrony Moderated by Other Mental Disorder

Parameter	Estimate (SE)	95% CI	Cohen's f^2
Fixed effects			
(Intercept)	**1.00 (0.07)**	**[0.87, 1.13]**	
Time since Baseline [1]	**-0.01 (0.00)**	**[-0.01, -0.006]**	**0.517**
Time of Day [2]	**-0.13 (0.03)**	**[-0.19, -0.08]**	**0.006**
Mental Disorder (yes/no) [2]	**-0.32 (0.13)**	**[-0.56, -0.07]**	**0.003**
State CT [1]	**0.32 (0.16)**	**[0.01, 0.65]**	**0.026**
State CT [1] * Mental Disorder (yes/no) [2]	**-0.63 (0.26)**	**[-1.13, -0.11]**	**0.033**
Average CT [2]	-0.09 (0.16)	[-0.41, 0.20]	0.000
Average CT [2] * Mental Disorder (yes/no) [2]	**0.49 (0.21)**	**[0.08, 0.90]**	**0.004**
Random effects			
Intercept	**0.21 (0.45)**	**[0.39, 0.54]**	
State Cortisol Slope	**0.39 (0.62)**	**[0.34, 0.89]**	

Note. Model fit: $\chi^2(7)=200.27$, $p<0.01$. Maternal cortisol predicting adolescent cortisol. State CT=Maternal cortisol reactivity, Average CT=Maternal average cortisol. Unstandardized estimates are presented. [1]=Level 1 predictor, [2]=Level 2 predictor. Significant parameters in bold.

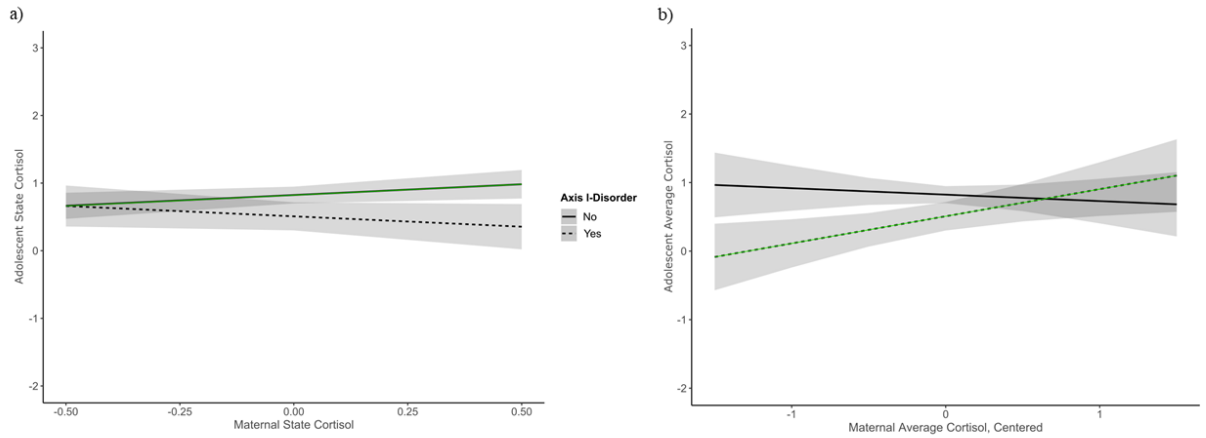
Table S5
Behavioral Synchrony Modulates the Effects of Adolescent Mental Disorder on Cortisol Synchrony.

Parameter	Estimate (SE)	95% CI	Cohen's f^2
Fixed effects			
(Intercept)	**1.01 (0.07)**	**[0.88, 1.14]**	
Time since Baseline [1]	**-0.01 (0.001)**	**[-0.01, -006]**	**0.530**
Time of Day [2]	**-0.12 (0.03)**	**[-0.18, -0.06]**	**0.000**
Behavior [2]	-0.01 (0.07)	[-0.15, 0.13]	0.000
Mental Disorder (yes/no) [2]	**-0.32 (0.13)**	**[-0.57, -0.07]**	**0.003**
State CT [1]	**0.31 (0.14)**	**[0.03, 0.59]**	**0.034**
State CT [1] * Behavior [2]	-0.19 (0.14)	[-0.49, 0.10]	0.012
State CT [1] * Mental Disorder (yes/no) [2]	-0.38 (0.21)	[-0.80, 0,05]	0.016
State CT [1] * Mental Disorder (yes/no) [2] * Behavior [2]	**1.35 (0.26)**	**[0.83, 1.87]**	**0.176**
Average CT [2]	-0.05 (0.15)	[-0.36, 0.26]	0.000
Average CT [2] * Behavior [2]	-0.05 (0.18)	[-0.40, 0.31]	0.000
Average CT [2] * Mental Disorder (yes/no) [2]	**0.46 (0.20)**	**[0.06, 0.87]**	**0.003**
Average CT [2] * Mental Disorder (yes/no) [2] * Behavior [2]	-0.41 (0.28)	[-0.97, 0.15]	0.001
Random effects			
Intercept	**0.19 (0.44)**	**[0.38, 0.52]**	
State Cortisol Slope	**0.13 (0.35)**	[0.00, 0.63]	

Note. Model fit: $\chi^2(12)= 281.36$, $p<0.01$. Maternal cortisol predicting adolescent cortisol. State CT=Maternal cortisol reactivity, Average CT=Maternal average cortisol. Behavior=Behavioral Synchrony. Unstandardized estimates are presented. [1]=Level 1 predictor, [2]=Level 2 predictor. Significant parameters in bold.

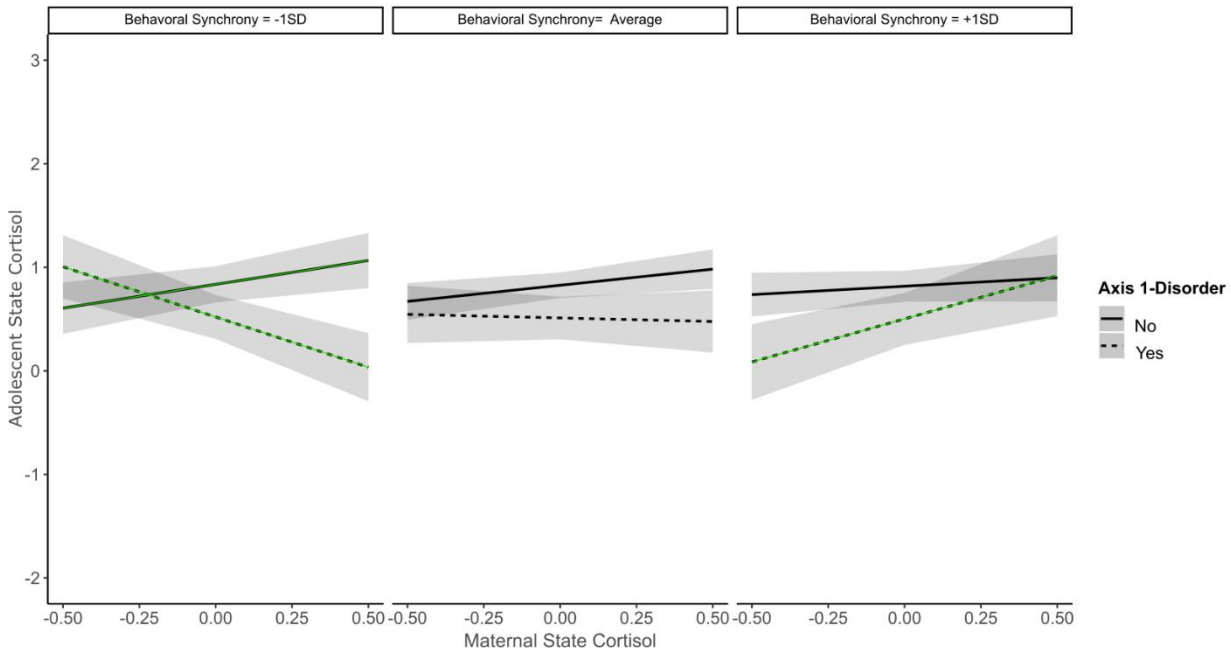
Figures

Figure S1.
Presence or Absence of Adolescent Mental Disorders Moderates Cortisol Synchrony and Average Associations.



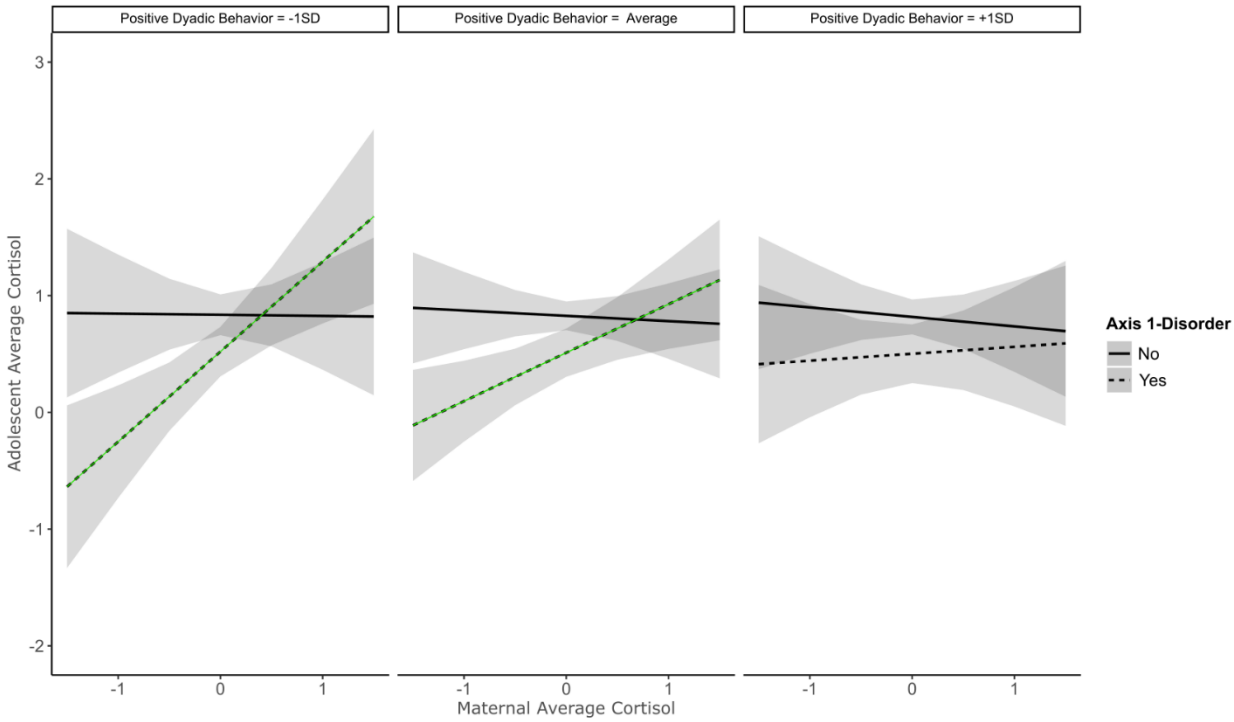
Note. Green line: *p*<.05; black line: *p*>.05. a) Significant when adolescents did not have a disorder b) Significant when adolescents had a disorder.

Figure S2.
Adolescent Mental Disorders and Behavioral Synchrony Moderate Cortisol Synchrony.



Note. Green lines: *p*<.05; black lines: *p*>.05. +/-1SD=Above/below one standard deviation. Negative cortisol synchrony when behavioral synchrony was lower, and adolescents had a disorder. Positive cortisol synchrony when a) behavioral synchrony was lower and adolescents did not have a disorder and b) when behavioral synchrony was higher, but adolescents had a disorder.

Figure S3.
Presence or Absence of Adolescent Mental Disorder and Positive Dyadic Behavior Moderate the Link between Maternal Average CT and Adolescent Average CT.



Note. Green lines: p<.05; black lines: p>.05. +/-1SD=Above/below one standard deviation. Positive cortisol synchrony when behavioral synchrony was average or lower and adolescents had a mental disorder.

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Article 3. Child versus adolescent borderline personality disorder traits: frequency, psychosocial correlates and observed mother-child interactions

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Abstract

Research has established the diagnostic validity of borderline personality disorder (BPD) in adolescence. The roots of BPD often lie in childhood, however, significantly less is known about the presence and correlates of BPD traits in school aged children and whether these are comparable to those observed in adolescents. Trained psychologists administered the Childhood Interview for Borderline Personality Disorder (CI-BPD) in a cohort of 14-year-old adolescents (n=76) and a cohort of 9-year-old children (n=70). We compared the prevalence of BPD traits in both cohorts and investigated common psychosocial correlates (comorbidity, impaired quality of life, emotional/behavioral problems, maternal distress, observed mother-child-interaction). Children and adolescents showed no significant differences regarding the type and frequency of BPD traits. In both cohorts, BPD traits were associated with comorbidity, emotional and behavioral problems and lower quality of life. In contrast to adolescents, children's BPD traits were not significantly related to maternal distress and showed less relations to interaction patterns. Negative maternal and dyadic behavior were associated with more BPD traits in adolescents during a *conflict discussion* but not during *fun day planning*. Our study suggests that BPD traits in children are similarly frequent as in adolescents and accompanied by psychosocial impairment. However, age-related differences were revealed, mostly indicating weaker associations with the mother-child relationship. Mother-child interaction patterns in youth seem to be especially relevant during conflict discussion and provide a target for intervention. Our study provides preliminary support for potential early detection of BPD pathology among children and encourages further study of its life span perspective.

Keywords: borderline personality disorder, early intervention, quality of life, mother-child-interaction, children

Introduction

Borderline personality disorder ([B]PD) is a severe mental disorder characterized by instability of affect, self-concept and relationships (American Psychiatric Association, 2013) and pervasive adverse consequences for psychosocial adjustment (Winograd et al., 2008). Historically, BPD has been considered as a stable, unmalleable disorder which was subsequently only diagnosed in adults. However, a life course perspective on personality and PD has increasingly emerged (Newton-Howes et al., 2015), acknowledging that normative as well as maladaptive expressions present and change throughout the life span. Research of the past decades has compiled increasing evidence regarding the reliability and validity of the diagnosis of BPD in adolescence (Kaess et al., 2014), including the potential of early detection and intervention of this disorder (Chanen et al., 2017). Although it is agreed upon that the roots of PD most often lie in childhood, very little research has investigated BPD features or traits before emerging adolescence. In consequence, a lot remains unknown about the early stages of the developing disorder (Crick et al., 2005). In order to expand the life span approach of PD (Newton-Howes et al., 2015) to childhood, it is warranted to investigate whether BPD traits can already be identified at an earlier age and whether patterns of the traits and their psychosocial correlates are comparable to those in adolescence and adulthood. Given that adverse psychosocial consequences of adolescent BPD persist into adulthood and pave the way for long-term maladjustment (Winograd et al., 2008), the identification of BPD traits at even earlier stages of development may bear enormous potential for indicated prevention.

Assessment and correlates of BPD features in childhood

In addition to the relative scarcity of research, the existing studies on childhood borderline pathology are characterized by different conceptualizations and assessment tools. The Childhood Interview for Borderline Personality Disorder (CI-BPD) (Zanarini, 2003) is an interview-based measure designed to assess the nine BPD criteria in children and adolescents in line with the DSM-IV/5-taxonomy. The Avon Longitudinal Study of Parents and Children (ALSPAC) used the CI-BPD in n = 6,330 11-year old children and is one of the few studies to apply an interview-based measure of BPD traits in preadolescent children. BPD traits from the community sample of 11-year-olds have been compared to those of a community sample of adults (Zanarini et al., 2011). Adults more often expressed symptoms of identity disturbance, impulsivity and fear of abandonment, whereas children were found to experience disproportionate anger more frequently. Only a few studies have investigated borderline pathology in even younger children of primary school age, and they made use of different conceptualizations (Crick et al., 2005; Rogosch & Cicchetti, 2005; Zelkowitz et al., 2001, 2007). Crick et al. (2005) developed a questionnaire-based dimensional measure (the Borderline

Personality Disorder Features Scale for Children) for children aged 9 years and older. Rogosch and Cicchetti (2005) used a “borderline precursor composite” consisting of measures of relationship difficulties, impulsive negative affect and suicidal thoughts/behaviors in children aged 6-12. Another research group developed a child-adapted retrospective chart review technique based on the Diagnostic Interview for Borderlines, rating social adaption, impulsivity, affect, psychosis, and interpersonal relations in children aged 7-12 (C-DIB, Greenman et al., 1986)). In instances, the C-DIB has also been used as a direct interview in pre-adolescents (Guzder et al., 1999). Borderline-related features as measured by parent-report have been investigated 6-12 year old children by an item selection reflecting impulsivity, negative affectivity and relational aggression (Stepp et al., 2010) respectively in 12-year old children using interview questions from the Shedler–Westen Assessment Procedure 200-item Q-Sort for Adolescents by (Westen et al., 2003) (Belsky et al., 2012).

Studies that investigated correlates of their BPD(-related) measures in children found them to be associated with experiences of childhood trauma and neglect, negative interpretation of ambiguous interpersonal situations, deficits in executive function, comorbid psychopathology (Belsky et al., 2012; Crick et al., 2005; Guzder et al., 1996, 1999; Zelkowitz et al., 2001) and to longitudinally predict mental health problems and lower functioning (Zelkowitz et al., 2007). Prospective studies found BPD traits or BPD-related features to be predicted by prenatal adversity, maladaptive parenting, lower cognitive function, childhood dysregulated behavior and bullying experiences (Belsky et al., 2012; Winsper et al., 2015, 2017). Therefore, borderline-related pathology during childhood seems to have similar correlates as adult or adolescent BPD traits (Sharp & Romero, 2007). A meta-analysis reported robust associations for BPD etiological factors like abuse, neglect, maladaptive parenting and comorbid psychopathology for both children and adolescents, although some effects (e.g. sexual abuse, comorbid anxiety disorder) seemed to be stronger in adolescent samples, whereas comorbid post-traumatic stress disorder seemed to be especially common during childhood (Winsper et al., 2016). However, the study of diagnostic interviews and BPD pathology in childhood has still not been extensive, and the variety of concepts and composites used complicates the comparison of study results. The utilization of interview tools is recommended for the assessment of BPD in youth (Sharp & Fonagy, 2015) and the combination of self-report tools and diagnostic interviews has been beneficial in identifying BPD traits (Hopwood et al., 2008). Thus, the need for applying and validating a clinical interview measure for earlier childhood age becomes evident.”

Proposed caregiver risk factors

According to the biosocial theory of BPD, emotional vulnerability may be in part due to biological predispositions, but may also develop due to an invalidating environment that does not help to label and modulate intense emotions (Linehan, 1993). Risk factors for maladaptive caregiving behavior include parental distress and psychopathology (e.g. Muzik et al., 2017). Parents who are distressed may have less resources to adequately respond to their children's signals and needs, and distress was found to increase the risk for insensitive maternal behaviour (Bödeker et al., 2019). In accordance, indicators of parental distress such as psychopathology and stress have been shown to be associated with offspring BPD symptoms and self-harm in adolescence and early adulthood (Infurna et al., 2016; Stepp et al., 2013; Tschan et al., 2015; Winsper et al., 2015). Given the association between child abuse risk and caregiver distress (Rodriguez & Tucker, 2015; M. C. Tucker et al., 2017) and the role of abuse in the development of BPD (Stepp et al., 2016), child abuse risk should also specifically be investigated in this context.

Observed parent-child interaction

Some studies investigated the relationship between BPD pathology and parent-child interaction assessed on a behavioral level. Regarding these observed interactions, to the best of our knowledge, all studies used mother-child dyads. Here, offspring BPD symptoms were associated with more negative observed mother-child interaction patterns (Carlson et al., 2009; Lyons-Ruth et al., 2013; Whalen et al., 2014). Observations can focus on dyadic, maternal, and child modes of behavior. In adolescent samples, mother-child interactions have been observed in the context of conflict discussions. Here, the dyadic escalation of conflicts was linked to self-harm and BPD severity (Crowell et al., 2013; Whalen et al., 2014), while positive maternal affective behavior predicted a decline in BPD severity. Other behaviors during conflict discussions that were associated with adolescent self-injury or affective instability were higher maternal psychological control (Mahan et al., 2018), more familial negativity and lower positive affect, higher maternal coercion and lower emotional support, and higher adolescent anger and opposition (Kaufman et al., 2020), indicating more negative interaction patterns for multiple modes of behavior for this age group. Observational paradigms in younger children have also mostly focused on stress-provoking paradigms: Maternal hostility during a teaching task in toddlerhood and maternal withdrawal during a separation-reunion situation in infancy were risk factors for developing BPD pathology later in life (Carlson et al., 2009; Lyons-Ruth et al., 2013). Concerning child modes of behavior, disorganized-controlling behaviors during a mother-child interaction at age 8 predicted later BPD pathology (Lyons-Ruth et al., 2013). Together, these findings indicate that mother-child interaction patterns play an important role in the context of

BPD traits in different developmental periods. The exact modes of behavior that are most relevant could however be specific to the developmental period and warrant further research. Additionally, existing studies mostly did not investigate all three modes of behavior (dyadic, maternal, child) simultaneously. This would be of interest in order to decompose the exact patterns relevant to the context of BPD development. Moreover, interactional patterns might be context specific (Dittrich et al., 2017), depending on the interpersonal challenge that the kind of conversation poses. Whether effects can also be detected during conversational tasks that would prompt positive conversations has yet to be investigated.

BPD-related impairments

Another factor characteristic for BPD is its association with high rates of psychiatric comorbidity and low quality of life. About 80% of adults with BPD also met criteria for a lifetime diagnosis of anxiety, mood, or substance use disorders (Tomko et al., 2014). Likewise, adolescent BPD patients show higher frequencies of psychiatric comorbidity and lower psychosocial functioning compared to clinical controls (Ha et al., 2014; Kaess et al., 2013), and higher maternal reports of their child's emotional and behavioral problems were linked to adolescent BPD pathology (Ha et al., 2014; Winsper et al., 2017), providing a second perspective on their psychopathology. They also have been found to report significantly decreased quality of life in comparison to patients without BPD traits (Kaess et al., 2017). Importantly, quality of life was also lower in patients with only subthreshold BPD symptoms. This finding supports the utility of a dimensional approach to BPD. Whether BPD traits in younger children would show similar correlates still needs to be investigated.

Present Study

Given the life span approach to personality pathology, research regarding possible age-related differences or similarities is indicated. In the present study, we compared BPD traits and their psychosocial correlates in two community cohorts of 9-year-old children and 14-year-old adolescents in order to validate their assessment and relevance in childhood. We investigated the following research questions and hypotheses: (1) Do BPD traits assessed via a clinical interview occur at similar frequencies in children vs. adolescence? Based on the lack of childhood age data on BPD traits measured according to the DSM conceptualization, this research question was investigated exploratively. (2) Do the expected psychosocial correlates predict BPD traits in a combined sample of children and adolescents? We hypothesized associations of BPD traits with the following psychosocial correlates: (a) greater presence of comorbid mental disorders, (b) lower quality of life, (c) more mother-reported behavioral and emotional problems of the child, (d) indicators of higher maternal distress (psychopathology,

perceived stress, and child abuse potential), (e) more negative maternal (e1), child (e2), as well as dyadic (e3) modes of behavior during both the planning of a fun day and, (f) during a conflict discussion task (f1-f3). (3) Do children and adolescents show different patterns regarding their psychosocial correlates of BPD traits? We explored the above associations in the adolescent cohort (hypotheses 3a-3f) and child cohort (hypotheses 4a-4f) separately and describe differences in patterns of significance or directions of effects.

Method

Procedure

The study was approved by the Ethics Committee of Faculty of Medicine at the University of Heidelberg (S-553/2016). We report how we determined our sample size, all data exclusions (if any), all manipulations, and all measures in the study (Simmons et al., 2012). Both cohorts were part of longitudinal studies. For both cohorts it was determined a priori that in order to detect medium effect sizes at $\alpha = .05$ and power = .80, a sample size of n = 100 participants would be targeted respectively. The study comprising the adolescent cohort started its assessments in the years 2002/2003 at two weeks after childbirth. Assessments of the childhood cohort started in 2008/2009 during the mothers' first trimenon of pregnancy. For both cohorts, the data presented belong to the 6^{th} assessment time point. Families were located and contacted via mail. They were given the option of an at-home visit or a visit to the laboratory. All mothers and adolescents signed their informed consent before participation in the assessments; children gave their written assent. The appointment included a semi-structured clinical interview about the child's mental health, followed by two mother-child interaction tasks, assessment of physiological measures, and the completion of questionnaires by both mother and child. Age-adapted standards were applied as to maternal presence during the interview. Children were interviewed in the presence of their mothers, following recommendations of a parental perspective for diagnostics in children (Grills & Ollendick, 2002; Merten & Schneider, 2017). Mothers were instructed that interview questions would be directed at the child, but they were invited to add to the response if they deemed it helpful. Adolescents were interviewed alone, as it has been implemented in other studies using the CI-BPD in adolescents samples (Sharp et al., 2012). Adolescents might be likely to withhold information, especially regarding risk behaviors or self-harm, in the presence of their parents. Questionnaires were completed electronically and programming prevented the omission of single questionnaire items.

Participants

Mothers from the community were recruited via local obstetric units and offices and newspapers. Inclusion criteria for the initial study phase of the adolescent cohort were full term deliveries, infant weight >2500g, APGAR scores >7 and good health of the baby during the first three postnatal doctoral exams. Exclusion criteria for mothers of the adolescent cohort were the inability to speak or understand the German language, acute mental disorder, excessive smoking or alcohol consumption and the use of drugs or medication possibly risking fetal health. Exclusion criteria for mothers of the child cohort were the inability to speak or understand the German language, pregnancy advanced beyond the 19th week, and the inability to come to the laboratory for the first postnatal assessment. The adolescent cohort started out with n = 101 mother-infant dyads. At the present follow-up, 76 dyads participated again (retention: 75%). Reasons for attrition in this cohort were: lack of time or interest (9.9%), inability to locate the family (5.9%), decease of the mother (0.9%), no reason specified (7.9%). The child cohort started out with n = 108 pregnant mothers participating. n = 70 mother-child dyads retained at the present follow-up (65%). Reasons for attrition were: lack of time or interest (14.8%), inability to locate or contact the family (13.8%), handicap of the child (0.9%), no reason specified (5.6%).

Measures

Borderline Personality Disorder Traits. The CI-BPD (Zanarini, 2003) was developed in order to assess the nine BPD traits in line with DSM-IV. It offers semi-structured questions for each of the nine traits. In an introduction, the interviewer explains that the questions refer to the period of the past two years, and that that the interviewer aims at knowing whether the behaviors, thoughts and feelings are *generally typical* of the respondent. Each trait is evaluated as being absent (0 points), probably present (1 point) or definitely present (2 points) in the course of the past two years. The CI-BPD was translated into German language by a professional translation agency for the purpose of this study. In the child cohort, some adaptations were made: Questions regarding promiscuous behavior, reckless driving and drug abuse were skipped as they were deemed irrelevant for that developmental age. Also, physical fights with siblings were relatively common in primary school aged children (Tucker et al., 2013) and therefore not rated as part of the impulsivity criterion, as they would not usually indicate pathological behavior. Remaining items for the impulsivity criterion included questions about alcohol abuse, food bingeing, spending money impulsively, losing one's temper and shouting at others, threats to physically harm someone, physical fights, deliberate damage of property, or doing something against the law. Two psychologists were trained by the measure's author and reached reliability. Thirty of the 146 interviews were double coded, and inter-rater

agreement between the reliable raters per traits ranged from 83% (uncontrollable anger) to 100% (self-harm/suicidality), with an average agreement of 94%.

Other Mental disorders. The Mini-International Neuropsychiatric Interview for children and adolescents aged 6 to 17 (M.I.N.I- KID 6.0) (Sheehan et al., 2010) was conducted. The M.I.N.I- KID is a structured diagnostic interview for mental disorders as defined in DSM-IV and ICD-10. For each of the covered disorders, the M.I.N.I- KID offers one or more screening questions, which are followed by follow-up questions if affirmed. The answers to all questions are noted in a yes/ no format.

Quality of Life. The KIDSCREEN-27 questionnaire assesses health-related quality of life and subjective well-being in children and adolescents (Ravens-Sieberer et al., 2005). The self-report version applies to ages 8-18. It covers the dimensions of physical well-being, psychological well-being, autonomy and parents, peers and social support, and school environment. Subscale T-values were averaged for a total score. Internal consistency of the total scale was α = .91.

Emotional and Behavioral Problems. Mothers completed the Strengths and Difficulties Questionnaire (SDQ)(Goodman, 2001; Klasen et al., 2003), providing a parent-report of their child's behavioral and emotional problems. The parent-rated version is applicable to children aged 4 to 16. The SDQ's subscales include: emotional symptoms (α = .80), conduct problems (α = .54), hyperactivity (α = .81), peer relationship problems (α = .64) and prosocial behavior (α = .67), and total problem score (α = .83).

Perceived Stress. The Perceived Stress Questionnaire (PSQ) (Levenstein et al., 1993) was used in order to assess mother's subjective experience of stress in the course of the last four weeks . The German 20-item version has established construct and external validity (Fliege et al., 2001). Internal consistency was α = .93.

Child Abuse Potential. The German adaption of the Child Abuse Potential Inventory (CAPI/ EBSK) (Deegener et al., 2009) assesses subjective parental stress, burdens and risk factors as an indicator for child abuse potential. The German version provides one global score with higher scores indicating higher risk (α = .89).

Maternal Psychopathology. The 18-item version of the Brief Symptom Inventory (BSI-18) (Derogatis, 2001; Spitzer et al., 2011) offers a brief assessment of overall psychological distress over the last two weeks, the "global severity index" (GSI), including symptoms of depression, anxiety and somatization (α = .82).

Observed mother-child interaction. Interactions were coded during two interaction paradigms using the Coding Interactive Behavior system (CIB)(Feldman, 1998). For the fun day interaction paradigm, mothers and children were both asked to pick a fun activity they would like to engage in with each other and to discuss these for 10 minutes. For the conflict

interaction paradigm, both picked a topic of conflict between them that they felt most irritated by in their everyday lives and discussed these, again for 10 minutes. The CIB version for parent-child conversational paradigms covers 56 behavioral codes which receive ratings from 1 (low) to 5 (high). The codes are grouped into scales which address parental, child, and dyadic behavior: *Maternal sensitivity* (positive affect, validation of the child's signals, elaboration of the child's contributions, resourcefulness, appropriate range of affect and supportive presence (α = .92)); *maternal intrusiveness* (intrusive behaviors such as interrupting the child or imposing one's own task solution, negative affect, hostility and criticizing (α = .86)); *maternal structuring* (on-task-persistence, consistency of style, appropriate structure/ limit-setting, and organization/regulation of the task (α = .76)); *child engagement* (positive affect, motivation, trust and openness towards the parent, creativity/initiation, and child confidence (α = .82)); *child withdrawal* (gaze aversion, lack of attention, negative emotionality, affective lability, withdrawal from the interaction and avoidance of the parent (α = .77)); *child compliance* (on task persistence, involvement in the conversation, compliance with parental boundaries and cooperation (α = .83)); *dyadic reciprocity* (give-and-receive interaction, compatibility and fluency (α = .87)); and *dyadic negativity* (dyadic tension and constricted interactions, e.g. long silences (α = .75)). Two main raters were trained and certified by the author of the measure and two additional raters were trained by them. 48 interactions (24 dyads) were rated by at least two raters (inter-rater agreement 88%; Cohen's kappa = .78).

As the current study was not pre-registered, we provide a list of measures that were assessed in the current study but beyond the scope of the current paper in *Online resource-Supplement List of Remaining Study Variables*.

Data Analysis

Data were analyzed using Stata 16 (College Station, TX: StataCorp LLC). Possible differences between both age groups in sociodemographic characteristics (sample description) and prevalence rates of the nine BPD traits (research question (1)) were analyzed using Fisher's exact tests, which is the preferred analytical approach for small expected cell sizes. Group differences of continuous study variables were calculated using two-tailed independent sample t-tests.

To investigate associations between BPD traits and the study variables, first all continuous predictor variables were standardized per age group and per interaction task in order to enable comparability of the estimation parameters. A score of the overall CI-BPD trait count (sum of all traits that were rated as "definitely present", max. possible score = 9) was created. The CI-BPD score is a count-based score in which every one of the nine traits is rated in a binary manner. Thus, associations with continuous or factorial predictor variables were

analyzed using generalized linear models (GLM) with maximum likelihood optimization and the assumption of a binomial distribution with nine trials. For each psychosocial correlate (independent variable), a separate GLM was performed. We established the main effects of the psychosocial predictor variables predicting the CI-BPD score in separate analyses of the combined sample (n = 146, hypotheses (2a)-(2f)), the adolescent sample (n = 76, hypotheses (3a)-(3f)) and the child sample (n = 70, hypotheses (4a)-(4f)). The strengths of associations derived from the GLM will be displayed as odds ratios (*OR*). An odds ratio of 2.00 signifies that for every one unit increase in the predictor (i.e. a one SD increase of a dimensional questionnaire score or a yes vs. no for a binary predictor), the odds of having a BPD trait increase by 2.00. In addition, we display average marginal effects (ME) in the GLM results table (Table 3). An average ME of .35 indicates that with a 1 SD increase (or yes vs. no) of the predictor, there is an increase of .35 BPD traits, on average. The significance level for all analyses was set to $p < .05$. In order to adjust for multiple testing, *p*-values from the GLM were Bonferroni-Holm corrected. Corrections were applied across those effects that were applying to the same hypothesis (e.g. (2c): *p*-values of main effects of the subscales measuring emotional and behavioral problems in the combined sample; or (3e1): *p*-values of main effects of the scales measuring maternal behavior during a fun day planning task in the adolescent sample). Note that Bonferroni-Holm corrections can lead to equal *p*-values for those comparisons that have been corrected. Data were complete apart from two children who did not complete their self-report questionnaires, resulting in n = 68 for the KIDSCREEN in the child cohort.

In order to approach discriminant validity, the effects of comorbidity, quality of life, maternal distress and mother-child interaction patterns were reanalysed controlling for the SDQ total score as a measure of non-BPD-specific emotional and behavioural problems as a covariate. Holm-corrections were performed consistent with the main analyses.

Results

Sample description

By the time of the assessment, adolescents were all 14 years old (no range). The majority went to grammar school (84%). Children in the younger cohort had a mean age of 9 years (range 8-10, *SD* = 0.55). They either attended primary school (91%) or the first grade of grammar school (9%). Mothers of both cohorts were for the majority in a partnership with the child's father (adolescent: 83%; child: 69%), had a university degree (adolescent: 70%; child: 63%), and were employed part-time (adolescent: 64%; child: 66%). Apart from maternal and child age, school type and grade attended there were no significant socio-demographic differences between the two cohorts (see Table 1).

Table 1

Socio-demographic characteristics of both study cohorts

	Adolescents	**Children**	***p* (group difference)**
Child age, M (SD)	14.0 (0.00)	9.0 (0.55)	***< .001***
Maternal age, M (SD)	48.2 (4.24)	41.2 (4.68)	***< .001***
Child sex, %			*.619*
female	46.05	41.43	
male	53.95	58.57	
Child school type, %			***< .001***
Primary school	-	91.43	
Intermediate secondary school	14.47	-	
Grammar school	84.21	8.57	
Other	1.32	-	
Maternal education, %			*.398*
Lower secondary school	-	2.86	
Intermediate secondary school	19.74	18.57	
University entrance diploma	10.53	15.71	
University degree	69.74	62.86	
Maternal employment, %			*.124*
Unemployed	-	1.43	
Housewife	3.95	5.71	
Minor	10.53	1.43	
Part-time	64.47	65.71	
Full-time	21.05	25.71	
Maternal partnership, %			*.130*
With child's father	82.89	68.57	
With different partner	7.89	15.71	
No partnership	9.21	15.71	

*Not*e. Non-corrected *p*-values. Bold values highlight significant differences.

Table 2 provides descriptive data of the interview-, questionnaire- and observation-based predictor variables investigated. It also shows group comparisons on these measures. Compared to children, adolescents reported lower physical well-being and lower well-being in the school environment. Mothers of children experienced significantly more distress than mothers of adolescents. During both interaction tasks, maternal and dyadic behavior were more negative in the adolescent cohort compared to the child cohort. For correlations between all continuous predictor variables, see *Online resource-Supplement tables Correlations per age group*.

Table 2

Descriptives and group comparisons of study variables

	Study variables	**Adolescents**	**Children**	**diff.**
	Interviews	*M* (*SD*)	*M* (*SD*)	*p*
CI-BPD	*# of traits* (definitely present)	0.57 (1.17)	0.63 (.98)	.727
		%	%	
Co-morbidity	any disorder	27.6	21.4	.385
	any phobic/ anxiety	19.7	11.4	.169
	any behavioral	9.2	8.6	.892
	any mood	2.6	7.1	.202
	Questionnaires	*M* (*SD*)	*M* (*SD*)	
Quality of life	total score	51.97 (6.82)	53.77 (7.08)	.122
Emotional and behavioral problems	total score	7.17 (4.94)	7.20 (5.85)	.974
	emotional problems	1.58 (1.96)	1.51 (2.05)	.846
	conduct problems	1.84 (1.51)	1.56 (1.43)	.246
	Hyperactivity	2.43 (2.11)	2.99 (2.57)	.157
	peer problems	1.32 (1.60)	1.14 (1.43)	.494
	prosocial behavior	7.46 (1.78)	7.96 (1.89)	.105
Maternal distress	perceived stress	33.71 (16.46)	45.96 (18.44)	**< .001**
	child abuse potential	14.69 (30.12)	17.63 (29.54)	.552
	psychopathology	.27 (.22)	.36 (.36)	**.046**
	Observed mother-child interaction	Adolescents	Children	diff.
	Fun Day	*M* (*SD*)	*M* (*SD*)	*p*
Maternal behavior	mat. Sensitivity	3.46 (.81)	3.90 (.68)	**< .001**
	mat. Intrusiveness	1.40 (.60)	1.11 (.25)	**< .001**
	mat. Structuring	4.49 (.56)	4.67 (.40)	**.028**
Child behavior	child engagement	3.69 (.77)	3.80 (.62)	.327
	child withdrawal	1.59 (.54)	1.55 (.48)	.678
	child compliance	4.47 (.59)	4.44 (.59)	.737
Dyadic behavior	dyadic reciprocity	3.80 (.83)	4.25 (.60)	**< .001**
	dyadic negativity	1.96 (.85)	1.50 (.61)	**< .001**
	Conflict			
Maternal behavior	mat. Sensitivity	3.19 (.87)	3.63 (.72)	**.001**
	mat. Intrusiveness	1.90 (.83)	1.44 (.53)	**< .001**
	mat. Structuring	4.38 (.62)	4.55 (.49)	.068
Child behavior	child engagement	3.51 (.70)	3.33 (.70)	.123
	child withdrawal	1.75 (.50)	1.90 (.54)	.091
	child compliance	4.18 (.68)	3.98 (.82)	.094
Dyadic behavior	dyadic reciprocity	3.70 (.87)	4.00 (.73)	**.022**
	dyadic negativity	2.26 (.90)	1.70 (.71)	**< .001**

Note. Non-corrected *p*-values. Bold values highlight significant differences.

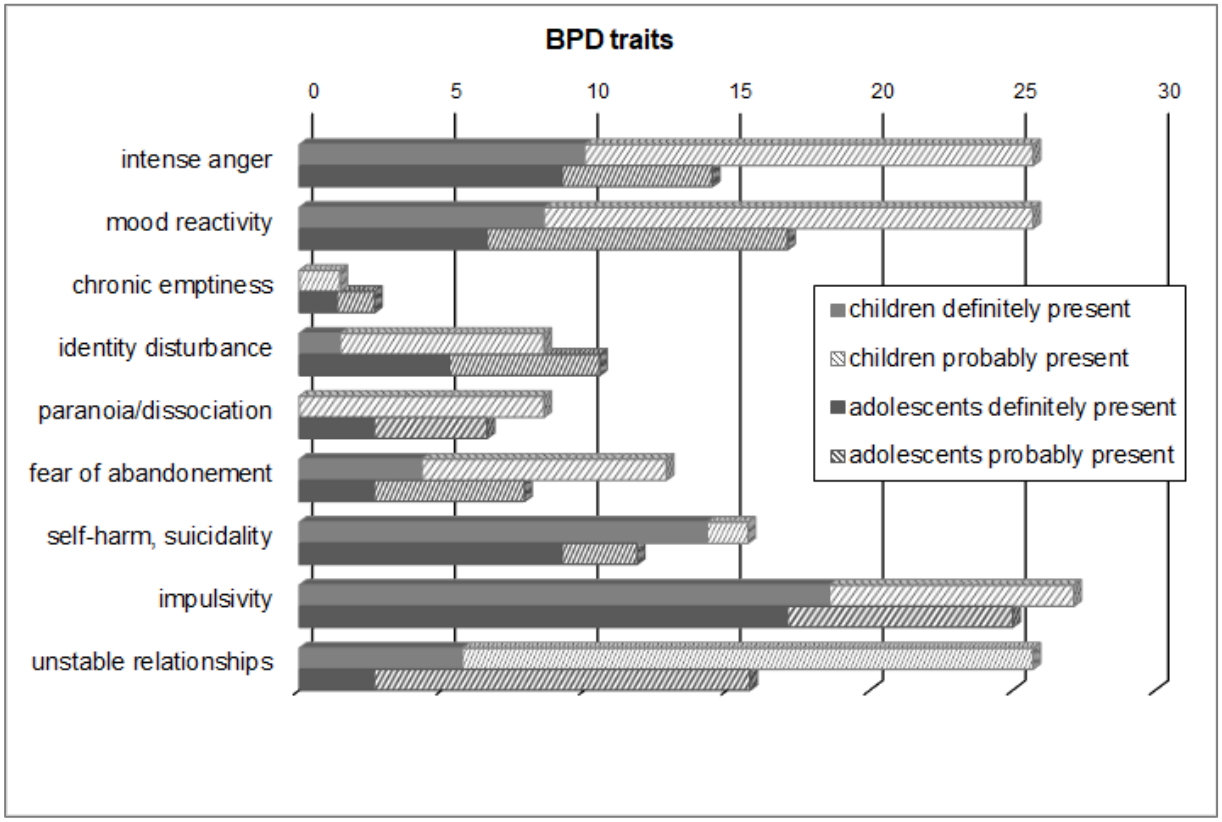
BPD traits

BPD traits that were “definitely present” most frequently, were, in both cohorts, those of impulsivity (adolescents: 17.11%; children: 18.57%), self-harm and suicidality (adolescents: 9.21%, children: 14.29%), and intense anger (adolescents: 9.21%; children: 10.0%) (see Figure 1). Exploring research question (1), there were no significant age group differences in

the absence, probable and definite presence of the nine traits (two-sided Fisher's exact: intense anger: p = .101; mood reactivity: p = .429; chronic feelings of emptiness: p = 1.000; identity disturbance: p = .472; stress-related paranoia or dissociation: p = .199; fear of abandonment: p = .633; self-harm and suicidality p = .578; impulsivity: p = .960; unstable relationships: p = .352). In the child cohort, 61.4% met BPD zero traits, 22.9% met 1 trait, 10.0% met two traits and 5.8% met more than two traits. In the adolescent cohort, 71.1% met zero BPD traits, 15.8% met one trait, 6.6% met two traits, and 6.5% met more than two traits. Children and adolescents did not differ regarding the overall number of definite BPD traits (adolescents: M = 0.57, SD = 1.17, range: 0 - 6: children: M = 0.63, SD = 0.98, range: 0 - 4; age group effect: $t(144)$ = -0.35, p = .727). Sensitivity analysis showed that the minimum detectable effect size of the t-test was medium (d = .47) at α = .05 and power = .80.

Fig. 1

Probably and definitely present BPD traits in the adolescent and child cohort (frequency in %)



Note. BPD = Borderline personality disorder.

Psychosocial Correlates

Other mental disorders: Meeting criteria for another mental disorder was associated with a higher number of definite BPD traits in the combined sample (hypothesis (2a): OR = 5.50, p <.001), the adolescent cohort (hypothesis (3a): OR = 7.90, p <.001), and the child

cohort (hypothesis (4a): *OR* = 4.19, *p* <.001). In the combined sample, of those who met at least one BPD trait, 51% met criteria for any other mental disorder (12% mood disorders; 29% phobic or anxiety disorders; 24% behavioral disorders). The association between phobic/anxiety disorders specifically and BPD traits was significant in the adolescent (*OR* = 2.90, *p* <.001) but not in the child cohort (*OR* = 1.81, *p* = .150). Of those with at least one BPD trait, 41% of adolescents and 19% of children met criteria for a phobic/anxiety disorder. Associations with mood and behavioral disorder were significant in both cohorts. For GLM results regarding all relationship between the CI-BPD and expected psychosocial correlates, see Table 3.

Quality of life: Associations between the KIDSCREEN and BPD traits were significant for the combined sample (hypothesis (2b), *OR* = .54, *p* = <.001), the adolescent cohort (hypothesis (3b), *OR* = .48, *p* <.001)*,* and the child cohort (hypothesis (4b), *OR* = .62, *p* = .008), all indicating lower quality of life for those with more BPD traits*.*

Emotional and behavioral problems: Indexed by the SDQ total score, emotional and behavioral problems were associated with BPD traits in the combined sample and both age groups (hypothesis (2c): *OR* = 2.10, *p* <.001; hypothesis (3c): *OR* = 2.00, *p* <.001; hypothesis (4c): *OR* = 2.20, *p* <.001). Regarding the SDQ subscales, BPD traits were related to lower prosocial behavior in the child (*OR* = .75, *p* = .047) but not the adolescent cohort *(OR* = .88, *p* = .384). All other subscales (emotional problems, conduct problems, hyperactivity, peer problems) were significantly associated with BPD traits in both cohorts and the combined sample.

Maternal distress: All three indicators of maternal distress were significantly related to BPD traits in the combined sample (hypothesis (2d): perceived stress, *OR* = 1.53, *p* <.001; child abuse potential, *OR* = 1.70, *p* <.001; psychopathology, *OR* = 1.31, *p* = .003) and the adolescent cohort (hypothesis (3d): perceived stress, *OR* = 1.83, *p* <.001; child abuse potential, *OR* = 1.98, *p* <.001; psychopathology, *OR* = 1.33, *p* = .026). In the child cohort, directions of effects indicated the same pattern but effects did not remain significant after Bonferroni-Holm correction (hypothesis (4d): perceived stress, *OR* = 1.28, *p* = .113; child abuse potential, *OR* = 1.41, *p* = .077; psychopathology, *OR* = 1.29, *p* = .085).

Observed mother-child interaction: During the *fun day* task, only child engagement (*OR* = .76, *p* = .022) and child withdrawal (*OR* = 1.31, *p* = .015) were significantly associated with BPD traits in the combined sample after Bonferroni-Holm correction (hypothesis 2e2). Child withdrawal was also significantly related to BPD traits in the adolescent cohort (hypothesis (3e2): *OR* = 1.37, *p* = .049) but not in the child cohort (hypothesis (4e2): *OR* = 1.25, *p* = .230). As maternal and dyadic behavior were not related to BPD traits during the *fun day* task in either of the cohorts or the combined sample, hypotheses 2e1, 3e1, 4e1, 2e3, 3e3 and 4e3 were not

confirmed. More differences between age groups occurred during the *conflict discussion* task. In the combined sample, all mother-child-interaction patterns were associated with a higher number of BPD traits in the expected directions, indicating more negative and less positive maternal, child and dyadic behaviors (hypotheses (2f1)-(2f3)). Maternal structuring significantly predicted BPD traits in both age groups. The effect of child compliance was nonsignificant in both age groups. Maternal sensitivity (*OR* = .61, *p* = .002) and intrusiveness (*OR* = 1.68, *p* <*.001*), child engagement (*OR* = .66, *p* = .021), dyadic reciprocity (*OR* = .66, *p* = .007) and dyadic negativity (*OR* = 1.57, *p* = .007) all significantly predicted BPD traits in the adolescent cohort. In contrast, these associations were pointing to the same directions but not significant in the child cohort, although the effect of dyadic negativity became nonsignificant only after Bonferroni-Holm correction (*OR* = 1.32, *p* = .094). Child withdrawal was significantly related to BPD traits in children (*OR* = 1.53, *p* = .013), but not in adolescents (*OR* = 1.31, *p* = .060).

Table 3

GLM results: psychosocial correlates of BPD traits

Study variables	**Adolescents**					**Children**					**Complete Sample**				
Comorbidity	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*
any disorder	1.26	7.90	3.96-15.75	**<.001**	**<.001**	1.07	4.19	2.23-7.82	**<.001**	**<.001**	1.16	5.50	3.50-8.64	**<.001**	**<.001**
any phobic/ anxiety	.71	2.90	1.52-5.51	**.001**	**.001**	.42	1.81	.81-4.07	.150	.150	.58	2.32	1.42-3.78	**.001**	**.001**
any behavioral	1.26	4.48	2.17-9.25	**<.001**	**.001**	1.86	6.37	3.13-12.97	**<.001**	**<.001**	1.54	5.32	3.21-8.83	**<.001**	**<.001**
any mood	4.04	18.59	6.93-49.84	**<.001**	**<.001**	1.48	4.63	2.12-10.14	**<.001**	**<.001**	2.23	7.51	4.16-13.57	**<.001**	**<.001**
Quality of life	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*
total score	-.37	.48	.36-.64	**<.001**		-.29	.62	.45-.84	**.002**		-.34	.54	.44-.67	**<.001**	
Emotional and behavioral problems	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*
total score	.35	2.00	1.57-2.56	**<.001**	**<.001**	.41	2.20	1.73-2.79	**<.001**	**<.001**	.38	2.10	1.77-2.49	**<.001**	**<.001**
emotional problems	.22	1.53	1.21-1.94	**<.001**	**.002**	.34	1.82	1.40-2.35	**<.001**	**<.001**	.27	1.66	1.39-1.97	**<.001**	**<.001**
conduct problems	.46	2.61	1.97-3.48	**<.001**	**<.001**	.47	2.49	1.90-3.26	**<.001**	**<.001**	.47	2.55	2.10-3.10	**<.001**	**<.001**
hyper-activity	.23	1.55	1.18-2.04	**.002**	**.005**	.37	1.94	1.46-2.57	**<.001**	**<.001**	.30	1.73	1.42-2.11	**<.001**	**<.001**
peer problems	.20	1.46	1.13-1.89	**.003**	**.006**	.36	1.91	1.50-2.45	**<.001**	**<.001**	.28	1.68	1.41-1.99	**<.001**	**<.001**
prosocial behavior	-.07	.88	.65-1.18	.384	.384	-.17	.75	.57-.99	**.047**	**.047**	-.12	.81	.66-.99	**.043**	**.043**

Maternal distress	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*
perceived stress	.31	1.83	1.36-2.46	**<.001**	**<.001**	.15	1.28	.94-1.73	.113	.112	.24	1.53	1.24-1.89	**<.001**	**<.001**
child abuse potential	.34	1.98	1.51-2.58	**<.001**	**<.001**	.20	1.41	1.04-1.90	**.026**	.077	.29	1.70	1.38-2.07	**<.001**	**<.001**
psycho-pathology	.15	1.33	1.04-1.70	**.026**	**.026**	.15	1.29	1.01-1.65	**.043**	.085	.15	1.31	1.10-1.56	**.003**	**.003**
Fun Day	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*
Maternal behavior															
mat. Sensitivity	-.07	.88	.64-1.20	.431	.738	-.12	.81	.60-1.09	.169	.508	-.09	.84	.68-1.05	.126	.251
mat. Intrusiveness	.08	1.17	.91-1.51	.220	.660	.09	1.18	.91-1.51	.203	.508	.09	1.17	.98-1.40	.077	.231
mat. Structuring	-.07	.88	.66-1.17	.369	.738	-.09	.85	.64-1.13	.261	.508	-.08	.86	.71-1.06	.153	.251
Child behavior															
child engagement	-.14	.76	.56-1.03	.080	.159	-.17	.75	.55-1.02	.066	.197	-.15	.76	.61-.94	**.011**	**.022**
child withdrawal	.17	1.37	1.06-1.77	**.017**	**.049**	.13	1.25	.95-1.65	.115	.230	.15	1.31	1.09-1.58	**.005**	**.015**
child compliance	-.07	.88	.66-1.17	.371	.371	-.10	.85	.64-1.12	.243	.243	-.08	.86	.70-1.05	.145	.145
Dyadic behavior															
dyadic reciprocity	-.08	.86	.63-1.16	.322	.643	-.17	.75	.56-1.00	.052	.103	-.12	.80	.65-.99	**.037**	.075
dyadic negativity	.01	1.01	.75-1.39	.901	.901	.15	1.28	.98-.1.68	.067	.103	.08	1.16	.94-1.42	.162	.162

Conflict	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*
Maternal behavior															
mat. sensitivity	-.26	.61	.45-.83	**.002**	**.004**	-.06	.90	.66-1.22	.500	.615	-.16	.74	.60-.92	**.007**	**.007**
mat. intrusiveness	.27	1.68	1.28-2.20	**<.001**	**<.001**	.08	1.15	.877-1.51	.308	.615	.18	1.38	1.15-1.65	**.001**	**.001**
mat. structuring	-.16	.73	.55-.97	**.029**	**.029**	-.22	.68	.53-.89	**.005**	**.015**	-.19	.71	.58-.86	**<.001**	**.001**
Child behavior															
child engagement	-.22	.66	.49-.89	**.007**	**.021**	-.12	.81	.59-1.11	.195	.194	-.17	.73	.59-.91	**.005**	**.009**
child withdrawal	.14	1.31	.99-1.74	.060	.121	.25	1.53	1.14-2.06	**.004**	**.013**	.19	1.41	1.15-1.73	**.001**	**.002**
child compliance	-.12	.79	.59-1.06	.114	.121	-.15	.77	.58-1.03	.082	.163	-.13	.78	.64-.96	**.019**	**.019**
Dyadic behavior															
dyadic reciprocity	-.22	.66	.49-.87	**.004**	**.007**	-.09	.86	.64-1.15	.302	.302	-.16	.75	.61-.92	**.005**	**.005**
dyadic negativity	.23	1.57	1.16-2.11	**.003**	**.007**	.16	1.32	1.00-1.74	**.047**	.094	.20	1.43	1.17-1.74	**<.001**	**<.001**

Note. GLM = Generalized linear models. BPD = Borderline personality disorder. *ME* = average marginal effects. *OR* = odd ratio. *p* adj. = Bonferroni-Holm adjusted *p*-values. Correction was performed on the multiple scales beneath their respective heading (Comorbidity, Quality of life, Emotional and behavioral problems, Maternal distress, Maternal behavior, Child behavior, Dyadic behavior), per sample (adolescents, children, combined sample) and per interaction task (fun day, conflict). Bold values highlight significant associations.

Discriminant validity: After controlling for the SDQ total score, in the adolescent cohort, the following associations with BPD traits remained significant: Comorbidity: any comorbid disorder, any mood disorder. Quality of life. Maternal distress: perceived stress, abuse potential. Fun day interaction: child withdrawal. In the child cohort, none of the psychosocial correlates remained significant after controlling for the SDQ total score. However, the effect of lower child engagement during the fun day paradigm only became significant after controlling for the SDQ. For a display of the results, see *Online resource-Supplement GLM controlled for SDQ*.

Discussion

This study systematically investigated the similarities and differences in the frequency of BPD traits as well as their psychosocial correlates between a cohort of 9-year-old children and a cohort of 14-year-old adolescents. Our results show that overall, children and adolescents exhibited comparable frequencies of the nine BPD traits. Furthermore, BPD traits were associated with comorbidity, lower quality of life, and more emotional and behavioral problems in both age groups. More differences occurred in the comparison of mother-child interaction patterns and maternal distress in relation to BPD traits, indicating stronger impairment in the adolescent cohort. Thus, whereas BPD symptomatology in childhood seems to be associated with multiple indicators of lower well-being, aspects of the mother-child relationship seem to be less strained in this age group.

In both age groups, impulsive behavior, self-harm and suicidality, and uncontrollable anger were among the most frequent BPD traits. These difficulties in regulating destructive impulses are classified as behavioral and affective traits of BPD (Sanislow et al., 2002). In contrast, cognitive traits (identity disturbance, stress-related paranoia/ dissociation, feelings of emptiness) were not commonly reported in the present cohorts. Overall number of traits and specific trait frequencies did not differ between age groups, with the caveat that the methodological difference regarding maternal presence during the interview might tangent comparability. Compared to our study, the ALSPAC study (Zanarini et al., 2011) reported slightly higher rates for all BPD traits except self-harm and impulsivity in their 11-year-old sample. Given that a low socioeconomic status (SES) is a risk factor for BPD (Stepp et al., 2016), our lower rates of BPD traits may be explained by the relatively high SES. Also, ratings of mother-child interactions showed that, on average, maternal and dyadic behavior were in the good range. One might therefore further explore whether traits such as impulsivity and anger may reflect a reactive temperament as a component of the disorder, and if cognitive criteria may develop in children additionally experiencing external adversities such as an invalidating environment. Alternatively, it has to be noted that especially the observed traits of

impulsivity and uncontrollable anger could be part of other developmental difficulties from the externalizing spectrum, such as conduct disorder or oppositional defiant disorder.

There was some evidence that children and adolescents differ with respect to the psychosocial correlates of BPD symptomatology. Mothers of adolescents with more BPD traits reported more stress and psychopathology on their own, and their self-reports indicated higher child abuse potential, whereas these associations did not remain significant in the child cohort after a correction for multiple testing. Thus, relations between maternal distress and child BPD pathology appeared to be stronger in adolescence compared to childhood. Several studies have established maternal psychopathology as a longitudinal risk factor of BPD symptoms in adolescence or adulthood (Stepp et al., 2016). In the majority of existing studies, maternal distress was however only measured when children had reached adolescence. Given the cross-sectional study design, from our study no conclusion can be drawn regarding the direction of this effect. However, assuming a transactional model of BPD (Fruzzetti et al., 2005), it is possible that maternal distress and child dysregulation mutually exacerbate each other over time. This pattern has e.g. been shown for the link between child disruptive behavior and maternal depressive symptoms (Gross et al., 2009). A mutual exacerbation of distress and dysregulation/BPD traits over time could be resulting in stronger associations in adolescence.

We also investigated actual behavior during the mother-child interaction. During the *fun day* discussion, BPD traits were only related to more child withdrawal in the adolescent cohort but not to maternal or dyadic behavior patterns, and no significant associations were found between BPD traits and mother-child interaction patterns in the child cohort. During the *conflict discussion*, all dyadic, maternal and child modes of behavior, except child withdrawal and compliance, appeared to be more negative in dyads where adolescents met more BPD traits. These results reflect those of earlier studies in adolescent samples in which the dyadic escalation of conflict was associated with BPD severity and self-harm (Crowell et al., 2013; Whalen et al., 2014). Conflict discussions ask the parent to regulate their own as well as the child's upcoming negative emotions. Difficulties to show appropriate support and manage discussions without tension even in times of disagreement may therefore become more obvious in the context of a *conflict discussion* compared to the *fun day* paradigm. Given that more negative child behavior was associated with BPD traits during both interactions, it may directly reflect their symptomatology and therefore be less specific to the task. In the child cohort, only lower maternal structuring and - in contrast to the adolescent cohort - child withdrawal during conflict discussion were related to child BPD traits. Group comparison of the predictor variables (Table 2) indicated that maternal and dyadic behavior were overall more negative in the adolescent cohort. This is reflective of studies showing an increase of parent-child conflict from early to mid-adolescence mid-adolescence (De Goede et al., 2009;

Mastrotheodoros et al., 2020). Therefore, negative relationship patterns or conflict may play a more significant role in the context of adolescent BPD symptomatology. Adolescence presents a vulnerable developmental phase for the onset of mental disorders (Kessler et al., 2005; Paus et al., 2008). As the expression of BPD traits is often triggered by interpersonal events, more negative parent-child conflict could be exacerbating dysregulation in vulnerable adolescents. Likewise, parents may act more harshly with behaviorally dysregulated youth. Still, associations with maternal structuring and child withdrawal also indicated less optimal interaction patterns in dyads in which younger children had BPD traits.

Results regarding weaker associations with maternal distress and mother-child interaction patterns provide potential insights into the BPD life span perspective. BPD in adolescence and early adulthood is often characterized by strong impulsivity which decreases in later life, whereas interpersonal difficulties persist (Hunt, 2007; Videler et al., 2019). According to the current findings, early expressions of BPD traits show less associations with interpersonal problems in the mother-child relationship. Some mother-child relationship problems might thus only develop with prolonged persistence of BPD traits in adolescence. This might be an indication that early intervention regarding the prominent traits that mainly reflected behavioral dysregulation, some of the psychosocial consequences of BPD symptomatology may be averted.

Children and adolescents reporting more BPD traits were more likely to experience a decreased quality of life, general emotional and behavioral problems and meet criteria for another mental disorder. These results reflect findings from adolescent and adult samples (Carlson et al., 2009; Kaess et al., 2017) and show adverse consequences of BPD pathology from early onwards. Given the community-based nature of our cohorts in which only few children exhibited more than two definitely present traits, our findings once more encourage a dimensional approach to BPD pathology. As this is the first study to investigate clinical interview-based BPD traits in primary school age, a replication of our findings is warranted. Our findings may also encourage future studies to investigate whether children could benefit from intervention to prevent further psychosocial maladjustment related to early BPD traits. However, mothers of children but not those of adolescents with BPD traits experienced their children to be less pro-social with peers. This finding might be due to mothers with younger children having more opportunities to observe their children interacting with peers.

Separate analyses of adolescents and children therefore showed significant associations in one age group but not the other for some predictors. However, in analyses of the combined sample, all hypotheses except those regarding maternal and dyadic behaviour during the fun day paradigm were confirmed. Several effects appeared weaker in the child cohort. Still, effects always pointed into the same direction as in the adolescent cohort and

combination of both age groups into one sample could not cancel out the effects found in the adolescent cohort. This indicates a general pattern of higher impairment regarding the studied psychosocial correlates in youth.

We approached the issue of discriminant validity in supplementary analyses, controlling associations between BPD traits and psychosocial correlates for the SDQ total score. Several associations remained significant in analyses of the adolescent but not the child cohort. Results suggest that BPD traits assessed in adolescents might be more disorder-specific compared to those observed in children. Findings regarding negative child behaviour shown even during a positive interaction paradigm were rather robust in the adolescent and combined sample. They might reflect overarching interpersonal dysfunction or mother-child relationship problems related to BPD pathology. In contrast, effects of more negative mother-child interaction patterns during the conflict discussion were less robust and could be more generally related to child mental health problems.

Strengths and limitations

We directly compared BPD trait frequencies and their correlates between two socio-economically very similar groups. The use of an interview measure of BPD traits in preadolescent children fills an important research gap. Moreover, in using an observational assessment of mother-child interactions we provided an objective measure that sheds light on the maternal, child, and dyadic aspects of interaction. The reported associations with both self-report and observational measures of maternal and child psychosocial correlates revealed age-related similarities and differences in BPD pathology, encouraging more research about the early presentation of BPD symptomatology. However, there are some limitations that should be considered when interpreting our results: First, given that the study was nested into two ongoing and community-based longitudinal cohort studies, the respective sample sizes were rather small and did not allow for investigation of cases with “clinical” BPD. The cohorts were originally powered to detect medium main effects, but sample sizes would only have been sufficient to detect rather large age group interaction effects. Therefore, in the current study we chose for a descriptive comparison of the psychosocial correlates. Studies with larger samples will be needed in order to establish whether the observed differences are also statistically significant. Replications could also take place in higher risk populations in order to increase rates of BPD traits. Also, given the different study designs of the initial study phases, there were slightly more exclusion criteria for participation in the adolescent cohort. As a consequence of these additional exclusion criteria that could otherwise act as risk factors, our adolescent cohort might exhibit somewhat lower BPD trait frequencies as compared to the general population. BPD trait frequencies reported in our child cohort are likely to be less

affected compared to those in the general population. Moreover, mothers were present and could add to the child interview but were not present during the adolescent interview. Regarding research question (1), reports of BPD traits in the child cohort may have been over- or underestimated either as a result of the mother's second perspective or by her influence on the child's report. This difference in methodology was implemented in order to apply age-related standards for the diagnostic interview, and maternal presence was expected to comfort children and support their discourse. It was thus expected to ensure rich data in order to enable the most accurate clinical judgement on BPS trait endorsement in this age group. Therefore, we would also recommend for clinical and empirical practice the presence of a caregiver while conducting the CI-BPD in younger children to ensure assessment of the desired construct. Also, due to the cross-sectional design we could not compare important factors such as the stability of BPD traits over time. Finally, we want to note that despite the literature's and current study's focus on children with BPD and their mothers, caregiving factors contributing to the development of BPD pathology are not limited to motherhood. In fact, the few studies incorporating fathers found important associations of e.g. paternal distress and father-child attachment with adolescent BPD (Infurna et al., 2016; Miljkovitch et al., 2018), highlighting the relevance to study father-child relationships likewise.

Conclusions and future perspectives

Based on the current study, child and adolescent samples seem to show no differences regarding frequencies of the nine BPD traits. Psychosocial impairments related to childhood BPD traits greatly reflect the clinical picture of those in adolescence and adulthood regarding comorbidity, quality of life, and emotional and behavioral problems. Therefore, our results suggest that BPD traits are relevant to psychosocial adjustment early on and strengthen the life span approach to personality pathology. However, some age-related differences were revealed as well: child BPD traits were not significantly related to maternal distress, and showed fewer associations with mother-child interaction patterns during a conflict discussion. The sequelae and predictive validity of childhood BPD traits should therefore be investigated thoroughly in future studies in order to determine their relation to later dysfunction. Possibly, some of the aversive cycles related to BPD pathology can be averted with early indicated prevention. Recent research has demonstrated that BPD symptoms share a large amount of variance with the general psychopathology factor (Gluschkoff et al., 2020). Therefore, single childhood BPD traits might also reflect a broader vulnerability for general psychopathology; thus, may serve as early indicators of general risk for psychopathology. As a future perspective, it would be of great interest to determine whether these childhood traits will specifically predict the development of BPD or other mental disorders. One recent study found

that of five patients with BPD at the adolescent follow-up, four had formerly been assessed with childhood borderline pathology (Zelkowitz et al., 2007), but analyses in this study were limited by the small sample size. In order to increase case rates, longitudinal studies investigating the sequelae of childhood BPD traits could also be carried out in high risk or clinical samples.

Supplement material

Table S1
Remaining study variables not used in the current article

	Adolescent Report	Child report	Maternal Report
Questionnaires			
Impulsiveness-Venturesomeness-Empathy questionnaire (Eysenck & Eysenck, 1980; Stadler et al., 2004)	x	x	
Difficulties in Emotion Regulation Scale (Ehring et al., 2008; Gratz & Roemer, 2004)	x		
Emotion Dysregulation Scale for Children (Morrongiello et al., 2012)		x	
KIDSCREEN-27 caregiver report (Ravens-Sieberer et al., 2005)			x
Strengths and Difficulties Questionnaire self-report (Goodman, 2001; Klasen et al., 2003)	x		
Junior Temperament and Character Inventory 12-18R (Goth & Schmeck, 2009	x		
Junior Temperament and Character Inventory 7-11R (Goth & Schmeck, 2009			x
Borderline Personality Features Scale for Children (Crick et al., 2005)	x	x	
Levels of Personality functioning Scale 2.0 (Bach & Hutsebaut, 2018)	x	x	
Inventory of Peer and Parent Attachment (Armsden & Greenberg, 1987; Kullik & Petermann, 2013)	x	x	
Vulnerable Attachment Questionnaire (Bifulco et al., 2003; Reck et al., 2016)			x
Interviews			
Self-injurious thoughts and behaviors interview (SITBI-G, Fischer et al. 2014)	x		
Risk behaviors (sexual risk behaviors, school absenteeism, (pathological) internet use	x		
Data surrounding mother-child interaction paradigms			
Positive and Negative Affect Scale (Watson et al., 1988)	x	x	x
Visual analogue scale on subjective stress	x	x	x
Salivary cortisol	x	x	x
Heart rate variability	x	x	x
Steroid hormones derived from hair samples	x	x	x

Table S2

Correlations of study variables (Pearson's r): Adolescent cohort (n=76)

				1)	2)	3)	4)	5)	6)	7)	8)	9)	10)	12)	13)	14)	15)	16)	17)	18)	19)	20)	21)	22)	23)	24)	25)	26)
	Qualitiy of life	1)	total score Kidscreen																									
	Emo. + behav. problems	2)	total score SDQ	**-.42**																								
		3)	emotional problems	**-.36**	**.76**																							
		4)	conduct problems	**-.35**	**.67**	**.38**																						
		5)	hyperactivity	-.12	**.73**	**.34**	**.39**																					
		6)	peer problems	**-.35**	**.56**	**.30**	.14	.16																				
		7)	prosocial behavior	**.24**	-.23	-.11	**-.28**	-.08	-.19																			
	Maternal distress	8)	perceived stress	**-.25**	**.28**	**.36**	**.35**	.01	.08	-.02																		
		9)	child abuse potential	**-.32**	**.40**	**.24**	**.40**	.19	**.30**	.00	.19																	
		10)	psychopathology	**-.30**	.08	.10	.20	-.03	-.05	.12	**.56**	.17																
Conflict	maternal behavior	11)	mat. sensitivity	.22	**-.33**	-.10	**-.32**	**-.31**	-.18	**.34**	**-.23**	-.06	-.15															
		12)	mat. intrusiveness	-.13	**.37**	.09	**.38**	**.47**	.04	-.12	.19	.18	.19	**-.75**														
		13)	mat. structuring	.16	-.18	.01	-.22	-.13	-.18	**.26**	-.12	.08	-.20	**.75**	**-.61**													
	child behavior	14)	child engagement	.17	**-.24**	-.08	**-.30**	-.11	-.22	**.43**	-.18	-.07	-.06	**.73**	**-.53**	**.57**												
		15)	child withdrawal	-.17	.00	-.06	.14	-.04	.00	**-.40**	.07	-.03	.06	**-.59**	**.41**	**-.60**	**-.69**											
		16)	child compliance	.17	-.12	-.03	-.15	-.04	-.13	**.39**	.04	.06	-.04	**.57**	**-.40**	**.63**	**.72**	**-.78**										
	dyadic behavior	17)	dyadic reciprocity	.14	-.22	-.04	**-.32**	-.13	-.15	**.35**	**-.23**	.07	-.18	**.82**	**-.68**	**.82**	**.77**	**-.69**	**.69**									
		18)	dyadic negativity	**-.25**	**.30**	.05	**.37**	**.23**	.22	**-.41**	.16	.01	.09	**-.84**	**.63**	**-.81**	**-.70**	**.70**	**-.63**	**-.82**								
Fun day	maternal behavior	19)	mat. sensitivity	.10	-.16	-.04	-.16	-.14	-.10	**.29**	-.16	.06	.13	**.76**	**-.45**	**.55**	**.59**	**-.45**	**.43**	**.67**	**-.64**							
		20)	mat. intrusiveness	-.03	**.29**	.04	**.39**	**.37**	.00	-.14	.19	.05	-.01	**-.49**	**.58**	**-.44**	**-.29**	**.25**	**-.25**	**-.56**	**.51**	**-.52**						
		21)	mat. structuring	.09	-.22	-.11	-.21	-.17	-.11	.15	-.10	.14	-.01	**.60**	**-.39**	**.66**	**.43**	**-.40**	**.46**	**.65**	**-.59**	**.67**	**-.54**					
	child behavior	22)	child engagement	.10	-.12	-.02	**-.26**	-.05	-.05	**.45**	-.06	.14	.04	**.60**	**-.38**	**.47**	**.73**	**-.56**	**.67**	**.65**	**-.56**	**.64**	**-.29**	**.42**				
		23)	child withdrawal	-.14	.07	.03	.14	.04	.01	**-.36**	-.02	-.13	-.06	**-.45**	**.25**	**-.49**	**-.53**	**.75**	**-.71**	**-.60**	**.56**	**-.47**	**.31**	**-.57**	**-.61**			
		24)	child compliance	.14	-.06	.06	-.17	-.06	-.01	**.37**	.06	.15	-.04	**.50**	**-.28**	**.55**	**.57**	**-.68**	**.76**	**.64**	**-.55**	**.48**	**-.33**	**.61**	**.71**	**-.78**		
	dyadic behavior	25)	dyadic reciprocity	.07	-.04	.04	-.23	.03	-.01	**.33**	-.17	.18	-.02	**.70**	**-.44**	**.66**	**.64**	**-.57**	**.55**	**.84**	**-.66**	**.81**	**-.50**	**.73**	**.69**	**-.59**	**.66**	
		26)	dyadic negativity	-.11	.08	-.07	**.24**	.03	.08	**-.31**	.13	-.16	.00	**-.68**	**.37**	**-.71**	**-.55**	**.58**	**-.49**	**-.74**	**.74**	**-.76**	**.50**	**-.77**	**-.59**	**.60**	**-.63**	**-.84**
				1)	2)	3)	4)	5)	6)	7)	8)	9)	10)	12)	13)	14)	15)	16)	17)	18)	19)	20)	21)	22)	23)	24)	25)	26)

Note. Bold figures indicate significant correlations at a significance level of $p < .05$.

Table S3

Correlations of study variables (Pearson's r): Child cohort (n=70)

				1)	2)	3)	4)	5)	6)	7)	8)	9)	10)	12)	13)	14)	15)	16)	17)	18)	19)	20)	21)	22)	23)	24)	25)	26)
	Qualitiy of life	1)	total score Kidscreen																									
	Emo. + behav. problems	2)	total score SDQ	**-.34**																								
		3)	emotional problems	-.16	**.75**																							
		4)	conduct problems	-.22	**.72**	**.37**																						
		5)	hyperactivity	-.19	**.80**	**.37**	**.48**																					
		6)	peer problems	**-.53**	**.72**	**.52**	**.46**	**.37**																				
		7)	prosocial behavior	.06	-.18	-.14	-.33	-.06	-.09																			
	Maternal distress	8)	perceived stress	-.13	**.28**	**.28**	.07	**.27**	.16	-.23																		
		9)	child abuse potential	-.20	**.33**	**.42**	.03	.19	**.34**	-.07	**.43**																	
		10)	psychopathology	-.15	**.34**	**.42**	.10	.21	**.26**	-.04	**.62**	**.43**																
Conflict	maternal behavior	11)	mat. sensitivity	-.17	-.04	.01	.03	-.11	.01	-.10	.10	-.06	.09															
		12)	mat. intrusiveness	.21	.09	.09	.11	.11	-.07	.09	-.06	.08	-.02	**-.63**														
		13)	mat. structuring	-.02	-.15	-.02	-.12	**-.24**	.00	-.03	-.17	-.03	-.11	**.62**	**-.47**													
	child behavior	14)	child engagement	.09	-.09	-.06	.04	-.10	-.14	.02	.11	-.05	.05	**.61**	**-.25**	**.42**												
		15)	child withdrawal	-.02	.23	.16	.02	.27	.16	-.16	.12	.18	.12	**-.30**	**.24**	**-.47**	**-.58**											
		16)	child compliance	-.02	-.03	.03	.08	-.18	.10	.14	-.13	-.06	-.12	**.38**	**-.19**	**.61**	**.62**	**-.79**										
	dyadic behavior	17)	dyadic reciprocity	-.07	-.04	-.02	.07	-.13	.03	-.13	.02	-.03	-.03	**.68**	**-.56**	**.72**	**.68**	**-.62**	**.72**									
		18)	dyadic negativity	.12	.11	.01	.12	.15	.04	.06	.06	.01	.08	**-.63**	**.72**	**-.64**	**-.48**	**.50**	**-.44**	**-.76**								
Fun Dav	maternal behavior	19)	mat. sensitivity	-.09	-.07	-.01	.02	-.14	-.05	-.08	.06	-.04	.05	**.85**	**-.48**	**.59**	**.57**	**-.34**	**.43**	**.61**	**-.54**							
		20)	mat. intrusiveness	.09	.11	.17	-.04	.10	.03	.15	-.04	.11	-.03	**-.49**	**.63**	**-.46**	**-.28**	**.41**	**-.34**	**-.55**	**.45**	**-.60**						
		21)	mat. structuring	.02	.01	.09	.06	-.13	.08	-.03	.08	.12	-.02	**.51**	**-.23**	**.69**	**.51**	**-.44**	**.61**	**.60**	**-.38**	**.64**	**-.39**					
	child behavior	22)	child engagement	.14	.05	.02	.09	.10	-.10	-.04	.11	-.03	-.02	**.38**	.05	.18	**.72**	**-.33**	**.37**	**.36**	-.16	**.46**	-.15	**.41**				
		23)	child withdrawal	-.08	-.02	-.03	-.12	.04	-.01	-.01	.06	.08	.11	-.03	-.07	-.20	**-.33**	**.59**	**-.56**	**-.34**	.15	**-.24**	**.24**	**-.49**	**-.57**			
		24)	child compliance	.03	.11	.14	.15	-.02	.12	.01	-.01	-.02	-.05	.17	.02	**.38**	**.48**	**-.56**	**.73**	**.49**	-.21	**.35**	**-.28**	**.62**	**.64**	**-.86**		
	dyadic behavior	25)	dyadic reciprocity	.04	-.03	.02	.07	-.07	-.10	-.12	.12	-.01	.00	**.64**	**-.40**	**.63**	**.67**	**-.49**	**.57**	**.73**	**-.55**	**.77**	**-.55**	**.77**	**.65**	**-.58**	**.66**	
		26)	dyadic negativity	-.03	.11	.07	-.01	.14	.10	.17	-.10	.14	-.08	**-.48**	**.46**	**-.40**	**-.34**	**.34**	**-.33**	**-.56**	**.57**	**-.62**	**.58**	**-.50**	-.22	**.34**	**-.37**	**-.65**
				1)	2)	3)	4)	5)	6)	7)	8)	9)	10)	12)	13)	14)	15)	16)	17)	18)	19)	20)	21)	22)	23)	24)	25)	26)

Note. Bold figures indicate significant correlations at a significance level of $p < .05$.

Table S3

GLM results: psychosocial correlates of BPD traits, controlled for the effect of SDQ (emotional and behavioural problems)

Study variables	**Adolescents**					**Children**					**Complete Sample**				
Comorbidity	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*
any disorder	.87	5.10	2.41-10.78	**<.001**	**<.001**	.21	1.46	.64-3.34	.364	.728	.60	2.87	1.70-4.85	**<.001**	**<.001**
SDQ	.21	1.55	1.17-2.06	**.002**	**.002**	.37	2.01	1.49-2.73	**<.001**	**<.001**	.27	1.71	1.41-2.09	**<.001**	**<.001**
any phobic/ anxiety	.17	1.39	.66-2.92	.391	.391	.30	1.65	.72-3.76	.234	.702	.15	1.32	.78-2.23	.298	.408
SDQ	.32	1.90	1.44-2.50	**<.001**	**<.001**	.42	2.22	1.74-2.83	**<.001**	**<.001**	.37	2.05	1.72-2.45	**<.001**	**<.001**
any behavioral	.42	2.00	.86-4.66	.110	.220	.05	1.10	.32-3.76	.873	.873	.30	1.67	.85-3.28	.136	.408
SDQ	.29	1.82	1.38-2.39	**<.001**	**<.001**	.40	2.14	1.46-3.15	**<.001**	**<.001**	.33	1.90	1.53-2.36	**<.001**	**<.001**
any mood	2.05	7.61	2.50-23.18	**<.001**	**.001**	-.47	.28	.07-1.14	.075	.300	.35	1.77	.81-3.85	.152	.408
SDQ	.24	1.69	1.28-2.22	**<.001**	**<.001**	.57	2.97	1.96-4.50	**<.001**	**<.001**	.33	1.92	.81-3.85	**<.001**	**<.001**
Quality of life	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*
total score	-.20	.66	.47-.93	**.019**		-.06	.89	.65-1.22	.463		-.12	.79	.62-.99	**.040**	
SDQ	.24	1.62	1.21-2.18	**.001**		.40	2.09	1.60-2.71	**<.001**		.33	1.88	1.54-2.29	**<.001**	
Maternal distress	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*
perceived stress	.19	1.48	1.07-2.05	**.019**	**.048**	-.00	.99	.68-1.44	.966	1.000	.10	1.22	.96-1.55	.103	.309
SDQ	.28	1.77	1.36-2.30	**<.001**	**<.001**	.41	2.20	1.71-2.82	**<.001**	**<.001**	.35	2.00	1.69-2.39	**<.001**	**<.001**

child abuse potential	.19	1.47	1.07-2.01	**.016**	**.048**	-.04	.93	.69-1.27	.659	1.000	.08	1.16	.94-1.45	.168	.336
SDQ	.25	1.66	1.24-2.22	**.001**	**.001**	.43	2.26	1.72-2.95	**<.001**	**<.001**	.34	1.96	1.61-2.38	**<.001**	**<.001**
psycho-pathology	.10	1.23	.93-1.61	.141	.141	-.01	.98	.73-1.32	.902	1.000	.05	1.11	.91-1.35	.322	.336
SDQ	.33	1.94	1.52-2.48	**<.001**	**<.001**	.42	2.21	1.71-2.85	**<.001**	**<.001**	.37	2.05	1.72-2.45	**<.001**	**<.001**
Fun Day	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*
Maternal behavior															
mat. sensitivity	-.00	1.00	.72-1.39	.999	1.000	-.06	.90	.66-1.21	.478	.956	-.03	.94	.76-1.18	.616	1.000
SDQ	.35	2.00	1.57-2.56	**<.001**	**<.001**	.41	2.18	1.71-2.77	**<.001**	**<.001**	.38	2.09	1.76-2.48	**<.001**	**<.001**
mat. intrusiveness	-.01	.99	.77-1.26	.904	1.000	.02	1.05	.78-1.41	.766	.956	.00	1.00	.83-1.21	.963	1.000
SDQ	.35	2.01	1.57-2.57	**<.001**	**<.001**	.41	2.18	1.71-2.78	**<.001**	**<.001**	.38	2.10	1.76-2.49	**<.001**	**<.001**
mat. structuring	.02	1.04	.77-1.40	.808	1.000	-.11	.80	.61-1.07	.131	.393	-.04	.93	.76-1.14	.476	1.000
SDQ	.35	2.02	1.57-2.59	**<.001**	**<.001**	.42	2.24	1.75-2.85	**<.001**	**<.001**	.38	.2.09	1.76-2.48	**<.001**	**<.001**
Child behavior															
child engagement	-.10	.81	.59-1.12	.206	.412	-.23	.64	.45-.92	**.015**	**.045**	-.15	.74	.58-.93	**.012**	**.024**
SDQ	.34	1.99	1.55-2.54	**<.001**	**<.001**	.43	2.29	1.79-2.94	**<.001**	**<.001**	.38	2.11	1.78-2.51	**<.001**	**<.001**

child withdrawal	.18	1.45	1.09-1.93	**.010**	**.030**	.14	1.30	.95-1.80	.104	.104	.16	1.38	1.12-1.71	**.003**	**.009**
SDQ	.36	2.08	1.61-2.68	**<.001**	**<.001**	.41	2.19	1.72-2.79	**<.001**	**<.001**	.38	2.13	1.79-2.53	**<.001**	**<.001**
child compliance	-.09	.84	.61-1.16	.287	.412	-.17	.72	.52-.98	**.037**	.074	-.13	.78	.62-.97	**.029**	**.029**
SDQ	.35	2.03	1.58-2.61	**<.001**	**<.001**	.43	2.28	1.79-2.92	**<.001**	**<.001**	.39	2.15	1.81-2.56	**<.001**	**<.001**
Dyadic behavior															
dyadic reciprocity	-.07	.87	.64-1.19	.388	.776	-.12	.79	.58-1.07	.128	.256	-.10	.83	.66-1.03	.088	.176
SDQ	.35	2.00	1.57-2.56	**<.001**	**<.001**	.40	2.16	1.70-2.76	**<.001**	**<.001**	.37	2.09	1.76-2.47	**<.001**	**<.001**
dyadic negativity	-.00	.99	.73-1.35	.961	.961	.02	1.04	.77-1.42	.791	.791	.01	1.03	.83-1.27	.797	.797
SDQ	.35	2.00	1.57-2.56	**<.001**	**<.001**	.41	2.17	1.68-2.80	**<.001**	**<.001**	.38	2.09	1.76-2.48	**<.001**	**<.001**
Conflict	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*	*ME*	*OR*	95% CI	*p*	*p adj.*
Maternal behavior															
mat. sensitivity	-.12	.79	.57-1.11	.175	.350	-.05	.90	.66-1.24	.533	.930	-.07	.87	.70-1.09	.239	.239
SDQ	.31	1.87	1.44-2.44	**<.001**	**<.001**	.41	2.20	1.73-2.80	**<.001**	**<.001**	.37	2.06	1.73-2.45	**<.001**	**<.001**
mat. intrusiveness	.14	1.32	.98-1.76	.067	.201	.06	1.13	.82-1.55	.465	.930	.09	1.19	.97-1.46	.099	.198
SDQ	.30	1.84	1.41-2.40	**<.001**	**<.001**	.41	2.19	1.72-2.79	**<.001**	**<.001**	.36	2.04	1.71-2.43	**<.001**	**<.001**
mat. structuring	-.08	.85	.63-1.15	.299	.350	-.16	.74	.56-.96	**.024**	.072	-.12	.79	.65-.97	**.023**	.069
SDQ	.33	1.94	1.51-2.50	**<.001**	**<.001**	.41	2.19	1.71-2.79	**<.001**	**<.001**	.37	2.05	1.72-2.43	**<.001**	**<.001**

Child behavior														
child engagement	-.12	.79	.58-1.09	.153	.306	-.10	.83	.61-1.14	.259	.259	-.10	.83	.66-1.03	.096	.096
SDQ	.32	1.91	1.49-2.47	**<.001**	**<.001**	.42	2.21	1.73-2.82	**<.001**	**<.001**	.37	2.06	1.74-2.45	**<.001**	**<.001**
child withdrawal	.17	1.42	1.04-1.94	**.026**	.078	.13	1.27	.95-1.70	.100	.200	.15	1.35	1.09-1.67	**.006**	**.018**
SDQ	.36	2.07	1.61-2.66	**<.001**	**<.001**	.40	2.14	1.67-2.75	**<.001**	**<.001**	.38	2.09	1.76-2.49	**<.001**	**<.001**
child compliance	-.09	.83	.59-1.17	.283	.306	-.17	.72	.54-.98	**.034**	.102	-.13	.79	.62-.97	**.027**	.054
SDQ	.34	1.98	1.55-2.53	**<.001**	**<.001**	.43	2.28	1.78-2.92	**<.001**	**<.001**	.38	2.11	1.77-2.51	**<.001**	**<.001**
Dyadic behavior														
dyadic reciprocity	-.12	.78	.58-1.06	.110	.220	-.08	.86	.63-1.71	.345	.368	-.09	.84	.68-1.03	.096	.150
SDQ	.32	1.91	1.48-2.45	**<.001**	**<.001**	.41	2.20	1.73-2.80	**<.001**	**<.001**	.37	2.06	1.73-2.45	**<.001**	**<.001**
dyadic negativity	.11	1.26	.91-1.75	.171	.220	.11	1.23	.91-1.66	.184	.368	.10	1.22	.98-1.53	.075	.150
SDQ	.32	1.89	1.46-2.45	**<.001**	**<.001**	.40	2.17	1.70-2.76	**<.001**	**<.001**	.36	2.03	1.70-2.42	**<.001**	**<.001**

Note. GLM = Generalized linear models. BPD = Borderline personality disorder. *ME* = average marginal effects. *OR* = odd ratio. *p* adj. = Bonferroni-Holm adjusted *p*-values. Correction was performed on the multiple scales beneath their respective heading (Comorbidity, Quality of life, Emotional and behavioral problems, Maternal distress, Maternal behavior, Child behavior, Dyadic behavior), per sample (adolescents, children, combined sample) and per interaction task (fun day, conflict). Bold values highlight significant associations.

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